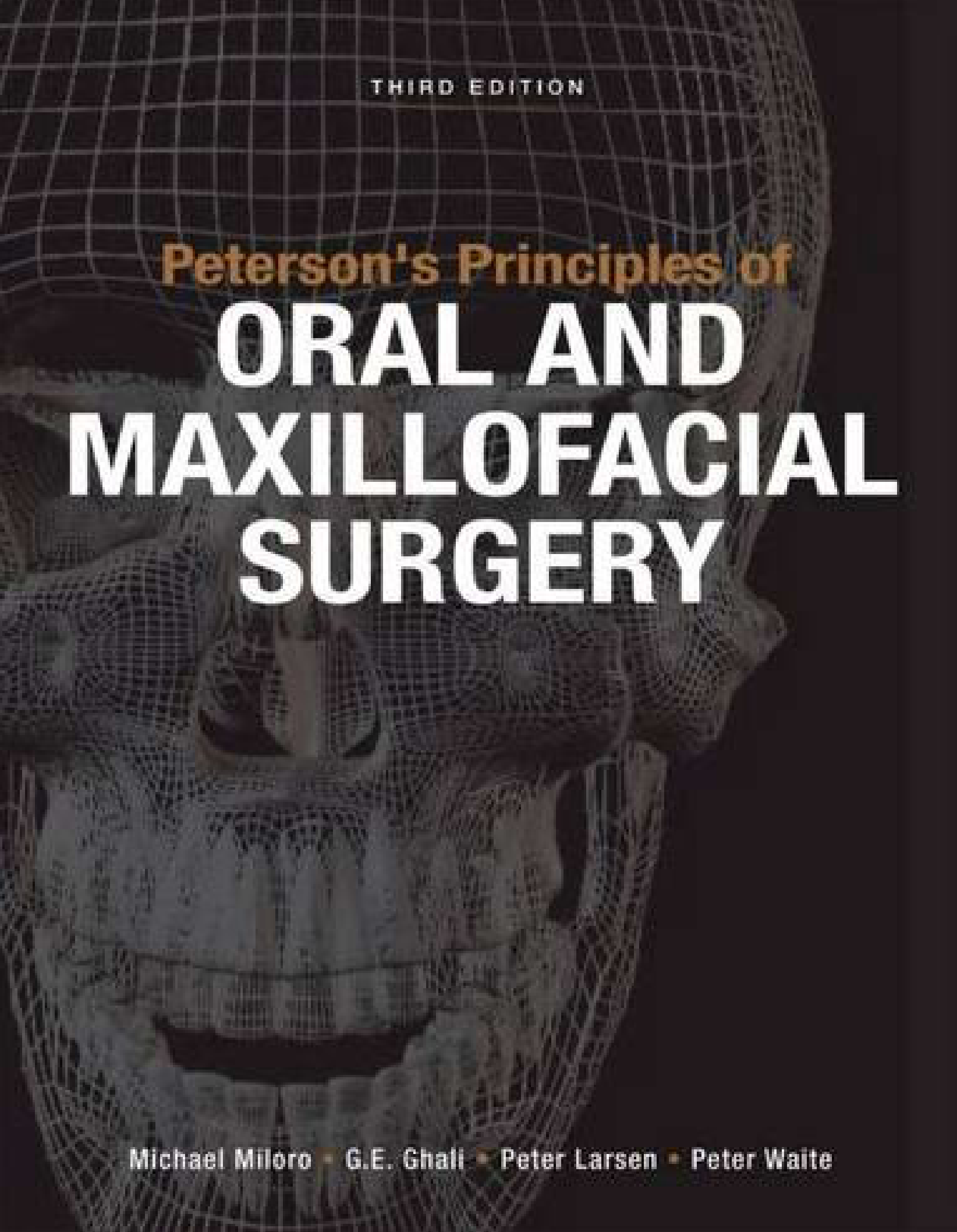

THIRD EDITION
Peterson's Principles of
ORAL AND MAXILLOFACIAL SURGERY
Michael Miloro • G.E. Ghali • Peter Larsen • Peter Waite

Peterson's Principles of Oral and Maxillofacial Surgery - Third Edition

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Preface



Dr. Gustav O. Kruger



Dr. Larry J. Peterson

Dr. Larry J. "Pete" Peterson was a teacher, and nothing would please him more than knowing that the legacy of this textbook and its teachings still continues posthumously today. Dr. Peterson began to assemble the editors and authors for the *Principles of Oral and Maxillofacial Surgery* textbook in the late 1980s, and the 1st edition was published in 1992.

Pete considered this a tribute to one of his mentors, Dr. Gustav O. Kruger, Chairman at Georgetown University School of Dentistry where Pete completed his oral and maxillofacial surgery residency training program. Dr. Kruger died in 2010 at the age of 93.

Like Dr. Peterson, whose car license plate read "OSUTCHR" (for "Ohio State University Teacher"), Dr. Kruger believed his role as an educator was most important to him. "Father really felt education was the key to a better world and instilled that in all of us," he said.

Dr. Kruger had edited his own *Textbook of Oral and Maxillofacial Surgery* in 1959, and this masterpiece underwent several editions, and enjoyed its final 6th edition in 1984. This textbook provided the basis for undergraduate and postgraduate education in the specialty of oral and maxillofacial surgery for dental students and residents throughout the country for three decades, until Dr. Peterson resurrected the idea of continuing to produce a comprehensive and complete reference in oral and maxillofacial surgery that was clinically applicable and easily readable for students and residents studying the principles of the specialty of oral and maxillofacial surgery.

This 3rd edition of the *Principles* textbook proudly continues the student and resident teachings that began by Dr. Kruger more than a half century ago.

The 3rd Edition of *Peterson's Principles of Oral and Maxillofacial Surgery,* reflects the efforts of many people, including leading oral and maxillofacial surgeons throughout the country and abroad. Although it was not an easy task to enlist the authors who have contributed to this textbook, the breadth of experience and talent of the contributing authors make this 3rd edition very special.

Oral and maxillofacial surgery encompasses an ever-expanding range of diverse topics that makes it unique among the medical and dental specialties. Dr. Kruger's *Textbook of Oral and Maxillofacial Surgery* was the first reference textbook to cover the full scope of the specialty and was available for students, residents, and surgeons to use as a reference for clinical practice.

The 3rd Edition of *Peterson's Principles of Oral and Maxillofacial Surgery* continues this tradition and provides an organized and systematic approach to the entire specialty for residents and clinicians who practice full-scope oral and maxillofacial surgery. The 3rd edition of this textbook is unique in many respects, with the inclusion of contributions from more than 100 oral and maxillofacial surgeons and other dental and medical specialists. The clear purpose of this textbook is to provide a concise, authoritative, easy-to-read, currently referenced, contemporary survey of the entire specialty of oral and maxillofacial surgery that contains the information that a

competent surgeon should possess and understand. Although some of the information may be outside of the scope of an individual practitioner, the material contained in this textbook is clearly within the scope of the specialty.

This textbook should be considered a reference for the oral and maxillofacial surgeon not only during residency but also into clinical practice, and it will serve as an excellent resource for examination preparation purposes.

As with the 1st and 2nd editions of this textbook, the authors, primarily oral and maxillofacial surgeons, were chosen because of their broad clinical experience and expertise in each specific area of the specialty. The contributions from these national and international authors clearly reflect their knowledge and expertise.

Whenever appropriate, each chapter attempts to review etiology, diagnosis, patient assessment, treatment plan development, surgical and nonsurgical treatment options, and recognition and management of complications. The information contained in this textbook is based upon a thorough evaluation of the current literature, as well as clinical expertise, and, as much as possible, is free from commercial and personal bias.

If additional information is required, suggested references have been provided so that other resources may be consulted. Considering the rapid technological advances and developments in the fields of medicine and surgery, a constant survey of the current published literature is required to maintain a working knowledge of the standards of diagnosis and treatment.

Future editions of this textbook will also reflect these changes in clinical practice. *Peterson's Principles of Oral and Maxillofacial Surgery* has established itself as *the* authoritative textbook for the specialty of oral and maxillofacial surgery.

Michael Miloro
G.E. Ghali
Peter E. Larsen
Peter D. Waite

Dedications

To my wife, Beth, and daughter, Macy, for your love, support, and understanding, and to my parents for their inspiration and encouragement. To the students and residents of oral and maxillofacial surgery for providing the impetus for this textbook, and to Pete for your mentorship.

Michael Miloro

To my wife, Hope, and my children, Gregor, Gracie, Gabrielle, and Garrisyn. To my parents, who inspired me to enter dentistry. And finally, to my residents and fellows who make it fun every day.

G. E. Ghali

To my wife, Patty, and my sons, Michael, Matthew, and Mark, for reminding me that my most important role is that of father and husband. To my father, who inspired me to enter medicine and to my mother, who convinced me that I could accomplish whatever I put my mind to. Lastly, to my colleagues and friends, who have supported me throughout my career.

Peter Larsen

I dedicate this textbook to my father, Daniel Waite, Professor and chair of OMS. His love and commitment to the specialty continue to inspire me. I thank my loving wife, Sallie, and children, for giving me the time to follow my surgical and teaching ambitions. Thanks to the residents who challenge me every day.

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SECTION 1

Medicine, Surgery, and Anesthesia

CHAPTER 1

Wound Healing

Vivek Shetty, DDS, DrMedDent, and Charles N. Bertolami, DDS, DMedSc

The healing wound is an overt expression of an intricate and tightly choreographed sequence of cellular and biochemical responses directed toward restoring tissue integrity and functional capacity after injury. Although healing culminates uneventfully in most instances, a variety of intrinsic and extrinsic factors can hamper or facilitate the process. Understanding wound healing at multiple levels—biochemical, physiologic, cellular, and molecular—provides the surgeon with a framework for basing clinical decisions aimed at optimizing the healing response. Equally important, it allows the surgeon to critically evaluate and selectively use the growing collection of biologic approaches that seek to assist healing by favorably modulating the wound microenvironment.

THE HEALING PROCESS

The restoration of tissue integrity, whether initiated by trauma or surgery, is a phylogenetically primitive but essential defense response. Injured organisms survive only if they can repair themselves quickly and effectively. The healing response depends primarily on the type of tissue involved and the nature of the tissue disruption. When restitution occurs by means of tissue that is structurally and functionally indistinguishable from native tissue, *regeneration* has taken place. However, if tissue integrity is reestablished primarily through the formation of fibrotic scar tissue, then *repair* has occurred. Repair by scarring is the body's version of a spot weld and the replacement tissue is coarse and has a lower cellular content than native tissue. With the exception of bone and liver, tissue disruption invariably results in repair rather than regeneration.

At the cellular level, the rate and quality of tissue healing depend on whether the constitutive cells are labile, stable, or permanent. Labile cells, including the keratinocytes of the epidermis and epithelial cells of the oral mucosa, divide throughout their life span. Stable cells such as fibroblasts exhibit a low rate of duplication but can undergo rapid proliferation in response to injury. For example, bone injury causes pluripotential mesenchymal cells to speedily differentiate into osteoblasts and osteoclasts. Conversely, permanent cells such as specialized nerve and cardiac muscle cells do not divide in postnatal life. The surgeon's expectation of "normal healing" should be correspondingly realistic and based on the inherent capabilities of the injured tissue. Whereas a fibrous scar is normal for skin wounds, it is suboptimal in the context of bone healing.

At a more macro level, the quality of the healing response is influenced by the nature of the tissue disruption and the circumstances surrounding wound closure. *Healing by first intention* occurs when a clean laceration or surgical incision is closed primarily with sutures or other means and healing proceeds rapidly with no dehiscence and minimal scar formation. If conditions are less favorable, wound healing is more complicated and occurs through a protracted filling of the tissue defect with granulation and connective tissue. This process is called *healing by second intention* and is commonly associated with avulsive injury, local infection, or inadequate closure of the wound. For more complex wounds, the surgeon may attempt *healing by third intention* through a staged procedure that combines secondary healing with delayed primary closure. The avulsive or contaminated wound is débrided and allowed to granulate and heal by second intention for 5 to 7 days. Once adequate granulation tissue has formed and the risk of infection appears minimal, the wound is sutured close to heal by first intention.

WOUND HEALING RESPONSE

Injury of any kind sets into motion a complex series of closely orchestrated and temporally overlapping processes directed toward restoring the integrity of the involved tissue. The reparative processes are most commonly modeled in skin[1]; however, similar patterns of biochemical and cellular events

occur in virtually every other tissue.[2] To facilitate description, the healing continuum of coagulation, inflammation, reepithelialization, granulation tissue, and matrix and tissue remodeling is typically broken down into three distinct overlapping phases: inflammatory, proliferative, and remodeling.[3,4]

Inflammatory Phase

The inflammatory phase presages the body's reparative response and usually lasts for 3 to 5 days. Vasoconstriction of the injured vasculature is the spontaneous tissue reaction to stop bleeding. Tissue trauma and local bleeding activate Factor XII (Hageman factor), which initiates the various effectors of the healing cascade including the complement, plasminogen, kinin, and clotting systems. Circulating platelets (thrombocytes) rapidly aggregate at the injury site and adhere to each other and the exposed vascular subendothelial collagen to form a primary platelet plug organized within a fibrin matrix. The clot secures hemostasis and provides a provisional matrix through which cells can migrate during the repair process. In addition, the clot serves as a reservoir of the cytokines and growth factors that are released as activated platelets degranulate (Figure 1-1). The bolus of secreted proteins, including interleukins, transforming growth factor-beta (TGF-β), platelet-derived growth factor (PDGF), and vascular endothelial growth factor (VEGF), maintain the wound milieu and regulate subsequent healing.[1]

Once hemostasis is secured, the reactive vasoconstriction is replaced by a more persistent period of vasodilation that is mediated by histamine, prostaglandins, kinins, and leukotrienes. Increasing vascular permeability allows blood plasma and other cellular mediators of healing to pass through the vessel walls by diapedesis and populate the extravascular space. Corresponding clinical manifestations include swelling, redness, heat, and pain. Cytokines released into the wound provide the chemotactic cues that sequentially recruit the neutrophils and monocytes to the site of injury. Neutrophils normally begin arriving at the wound site within minutes of injury and rapidly establish themselves as the predominant cells. Migrating through the scaffolding provided by the fibrin-enriched clot, the short-lived leukocytes flood the site with proteases and cytokines that help cleanse the wound of contaminating bacteria, devitalized tissue, and degraded matrix components. Neutrophil activity is accentuated by opsonic antibodies leaking into the wound from the altered vasculature. Unless a wound is grossly infected, neutrophil infiltration ceases after a few days. However, the proinflammatory cytokines released by perishing neutrophils, including tumor necrosis factor-alpha (TNF-α) and interleukins (IL-1a, IL-1b), continue to stimulate the inflammatory response for extended periods.[5]

Deployment of bloodborne monocytes to the site of injury starts peaking as the levels of neutrophils decline. Activated monocytes, now termed macrophages, continue with the

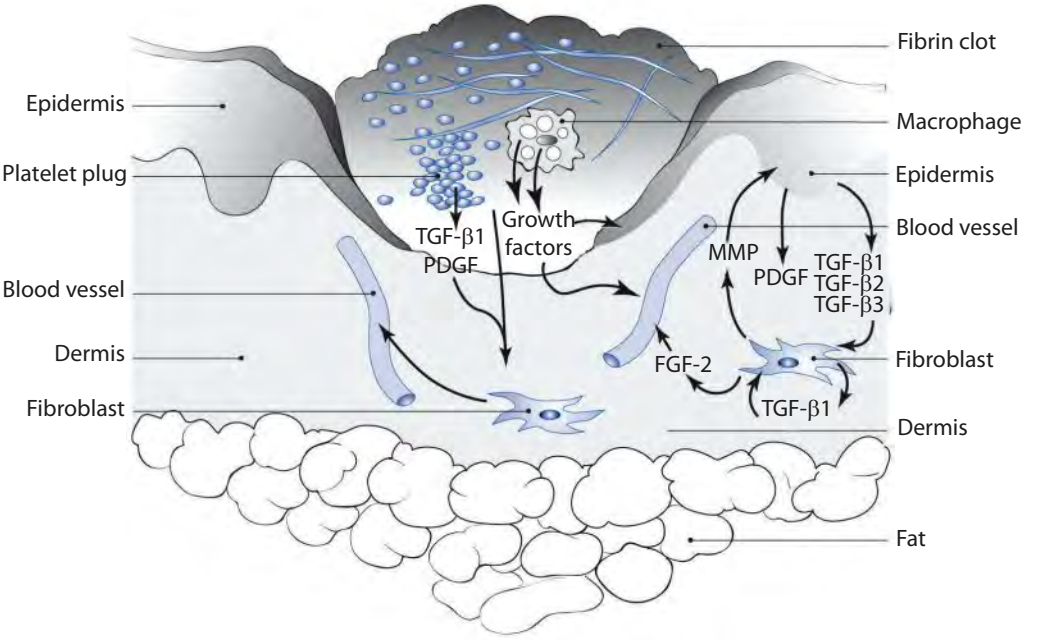


FIGURE 1-1. Immediately after wounding, platelets facilitate the formation of a blood clot that secures hemostasis and provides a temporary matrix for cell migration. Cytokines released by activated macrophages and fibroblasts initiate the formation of granulation tissue by degrading extracellular matrix and promoting development of new blood vessels. Cellular interactions are potentiated by reciprocal signaling between the epidermis and the dermal fibroblasts through growth factors, matrix metalloproteinases (MMPs), and members of the transforming growth factor-beta (TGF-β) family. FGF = fibroblast growth factor; PDGF = platelet-derived growth factor. (Adapted with permission from Bissell MJ, Radisky D. Putting tumors in context. Nat Rev Canc 2001;1:46–54.)

wound microdébridement initiated by the neutrophils. The macrophages secrete collagenases and elastases to break down injured tissue and phagocytose bacteria and cell debris. Beyond their scavenging role, the macrophages also serve as the primary source of healing mediators. Once activated, macrophages release a battery of growth factors and cytokines (TGF-α, TGF-β1, PDGF, insulin-like growth factor [IGF]-I and -II, TNF-α, and IL-1) at the wound site, further amplifying and perpetuating the action of the chemical and cellular mediators released previously by degranulating platelets and neutrophils.[6] Macrophages influence all phases of early wound healing by regulating local tissue remodeling by proteolytic enzymes (e.g., matrix metalloproteases [MMPs] and collagenases), inducing formation of new extracellular matrix (ECM), and modulating angiogenesis and fibroplasia through local production of cytokines such as thrombospondin-1 and IL-1b. The centrality of macrophage function to early wound healing is underscored by the consistent finding that macrophage-depleted animal wounds demonstrate diminished fibroplasia and defective repair. Although the numbers and activity of the macrophages taper off by the fifth postinjury day, they continue to modulate the wound healing process until repair is complete.

Proliferative Phase

The cytokines and growth factors secreted during the inflammatory phase stimulate the succeeding proliferative phase (Figure 1-2).[7] Starting as early as the third day postinjury and lasting up to 3 weeks, the proliferative phase is distinguished by the formation of pink granular tissue (granulation tissue) containing inflammatory cells, fibroblasts, and budding vasculature enclosed in a loose matrix. An essential first step is the establishment of a local microcirculation to supply the oxygen and nutrients necessary for the elevated metabolic needs of regenerating tissues. The generation of new capillary blood vessels (angiogenesis) from the interrupted vasculature is driven by wound hypoxia as well as with native growth factors, particularly VEGF, fibroblast growth factor-2 (FGF-2), and TNF-β (see Figure 1-2). Around the same time, matrix-generating fibroblasts migrate into the wound in response to the cytokines and growth factors released by inflammatory cells and wounded tissue. The fibroblasts start synthesizing new ECM and immature collagen (type III). The scaffold of collagen fibers serves to support the newly formed blood vessels supplying the wound. Stimulated fibroblasts also secrete a range of growth factors, thereby producing a feedback loop and sustaining the repair process. Collagen deposition rapidly increases the tensile strength of the wound and decreases the reliance on closure material to hold the wound edges together. Once adequate collagen and ECM have been generated, matrix synthesis dissipates, evidencing the highly precise spatial and temporal regulation of normal healing.

At the surface of the dermal wound, new epithelium forms to seal off the denuded wound surface. Epidermal cells originating from the wound margins undergo a proliferative burst and begin to resurface the wound above the basement membrane. The process of reepithelialization progresses more rapidly in oral mucosal wounds in contrast to the skin. In a mucosal wound, the epithelial cells migrate directly onto the moist exposed surface of the fibrin clot instead of under the dry exudate (scab) of the dermis. Once the epithelial edges meet, contact inhibition halts further lateral proliferation. Reepithelialization is facilitated by underlying contractile connective tissue, which shrinks in size to draw the wound margins toward one another. Wound contraction is driven by a subset of the fibroblasts that transform into myofibroblasts and generate strong contractile forces. The extent of wound contraction depends on the depth of the wound and its

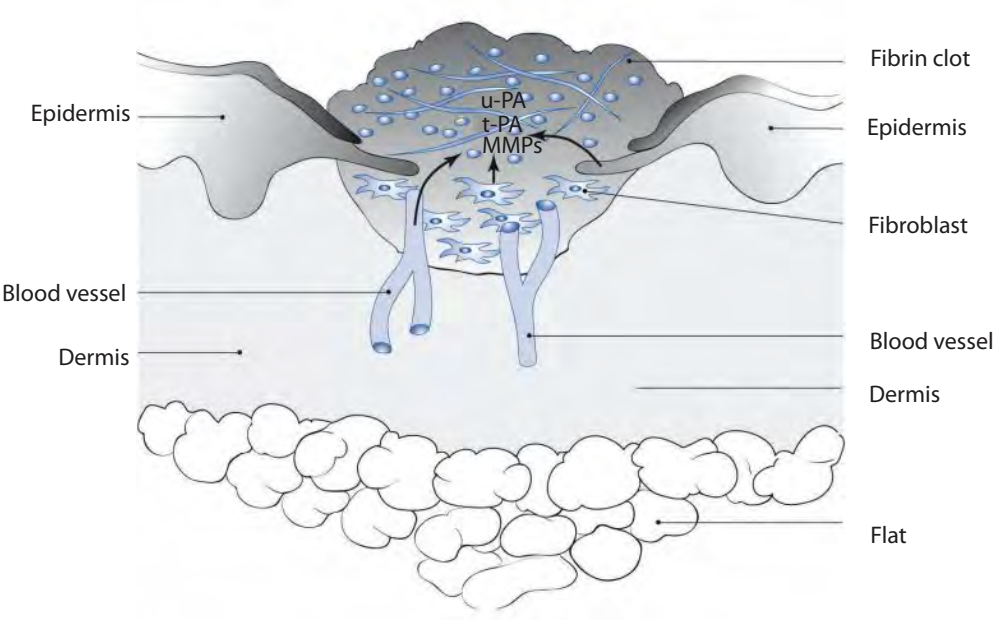


FIGURE 1-2. The cytokine cascade mediates the succedent proliferative phase. This phase is distinguished by the establishment of local microcirculation and formation of extracellular matrix and immature collagen. Epidermal cells migrate laterally below the fibrin clot, and granulation tissue begins to organize below the epithelium. MMPs = matrix metalloproteinases; t-PA = tissue plasminogen activator; u-PA = urinary plasminogen activator. (Adapted with permission from Bissell MJ, Radisky D. Putting tumors in context. Nat Rev Canc 2001;1:46–54.)

location. In some instances, the forces of wound contracture are capable of deforming osseous structures.

Remodeling Phase

The proliferative phase is progressively replaced by an extended period of progressive remodeling and strengthening of the immature scar tissue. The remodeling/maturation phase can last for several years and involves a finely choreographed balance between matrix degradation and formation. As the metabolic demands of the healing wound decrease, the rich network of capillaries begins to regress. Under the general direction of the cytokines and growth factors, the collagenous matrix is continually degraded, resynthesized, reorganized, and stabilized by molecular crosslinking into a scar. The fibroblasts start to disappear and the collagen type III deposited during the granulation phase is gradually replaced by stronger type I collagen. Correspondingly, the tensile strength of the scar tissue gradually increases and eventually approaches approximately 80% of the original strength. Homeostasis of scar collagen and ECM is regulated to a large extent by serine proteases and MMPs under the control of the regulatory cytokines. Tissue inhibitors of the MMPs afford a natural counterbalance to the MMPs and provide tight control of proteolytic activity within the scar. Any disruption of this orderly balance can lead to excess or inadequate matrix degradation and result in either an exuberant scar or wound dehiscence.

SPECIALIZED HEALING

Nerve

Injury to the nerves innervating the orofacial region may range from simple contusion to complete interruption of the nerve. The healing response depends on injury severity and extent of the injury.[8–10] Neuropraxia represents the mildest form of nerve injury and is a transient interruption of nerve conduction without loss of axonal continuity. The continuity of the epineural sheath and the axons is maintained and morphologic alterations are minor. Recovery of the functional deficit is spontaneous and usually complete within 3 to 4 weeks. If there is a physical disruption of one or more axons without injury to stromal tissue, the injury is described as *axonotmesis.* Whereas individual axons are severed, the investing Schwann cells and connective tissue elements remain intact. The nature and extent of the ensuing sensory or motor deficit relate to the number and type of injured axons. Morphologic changes are manifest as degeneration of the axoplasm and associated structures distal to the site of injury and partly proximal to the injury. Recovery of the functional deficit depends on the degree of the damage.

Complete transection of the nerve trunk is referred to as *neurotmesis* and spontaneous recovery from this type of injury is rare. Histologically, changes of degeneration are evident in all axons adjacent to the site of injury.[11] Shortly after nerve severance, the investing Schwann cells begin to undergo a series of cellular changes called *wallerian degeneration.* The degeneration is evident in all axons of the distal nerve segment and in a few nodes of the proximal segment. Within 78 hours, injured axons start breaking up and are phagocytosed by adjacent Schwann cells and by macrophages that migrate into the zone of injury. Once the axonal debris has been cleared, Schwann cell outgrowths attempt to connect the proximal stump with the distal nerve stump. Surviving Schwann cells proliferate to form a band (Büngner's band) that will accept regenerating axonal sprouts from the proximal stump. The proliferating Schwann cells also promote nerve regeneration by secreting numerous neurotrophic factors that coordinate cellular repair as well as cell adhesion molecules that direct axonal growth. In the absence of surgical realignment or approximation of the nerve stumps, proliferating Schwann cells and outgrowing axonal sprouts may align within the randomly organized fibrin clot to form a disorganized mass termed *neuroma.*

The rate and extent of nerve regeneration depend on several factors including type of injury, age, state of tissue nutrition, and the nerves involved. Although the regeneration rate for peripheral nerves varies considerably, it is generally considered to approximate 1 mm/day. The regeneration phase lasts up to 3 months and ends on contact with the end-organ by a thin myelinated axon. In the concluding maturation phase, both the diameter and the performance of the regenerating nerve fiber increase.

Bone

The process of bone healing after a fracture has many features similar to that of skin healing except that it also involves calcification of the connective tissue matrix. Bone is a biologically privileged tissue in that it heals by regeneration rather than repair. Left alone, fractured bone is capable of restoring itself spontaneously through sequential tissue formation and differentiation, a process also referred to as *indirect healing.* As in skin, the interfragmentary thrombus that forms shortly after injury staunches bleeding from ruptured vessels in the haversian canals, marrow, and periosteum. Necrotic material at the fracture site provokes an immediate and intense acute inflammatory response that attracts the polymorphonuclear leukocytes and, subsequently, macrophages to the fracture site. The organizing hematoma serves as a fibrin scaffold over which reparative cells can migrate and perform their function. Invading inflammatory cells and the succeeding pluripotential mesenchymal cells begin to rapidly produce a soft fracture callus that fills up interfragmentary gaps. Composed of fibrous tissue, cartilage, and young immature fiber bone, the soft compliant callus acts as a biologic splint by binding the severed bone segments and damping interfragmentary motion. An orderly progression of tissue differentiation and maturation eventually leads to fracture consolidation and restoration of bone continuity.

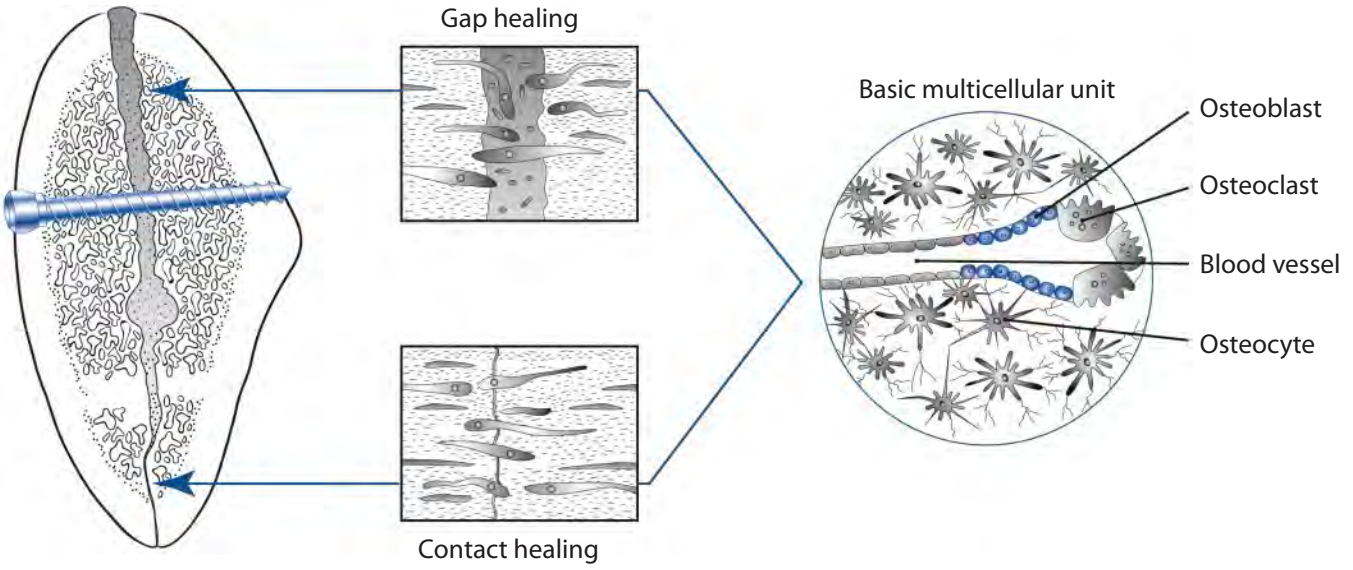


FIGURE 1-3. Direct bone healing facilitated by a lag screw. The fracture site shows both gap healing and contact healing. The internal architecture of bone is restored eventually by the action of basic multicellular units.

More commonly, the surgeon chooses to facilitate an abbreviated callus-free bone healing termed *direct healing* (Figure 1-3). The displaced bone segments are surgically manipulated into an acceptable alignment and rigidly stabilized through the use of internal fixation devices. The resulting anatomic reduction is usually a combination of small interfragmentary gaps separated by contact areas. Ingrowth of mesenchymal cells and blood vessels starts shortly thereafter, and activated osteoblasts start depositing osteoid on the surface of the fragment ends. In contact zones where the fracture ends are closely apposed, the fracture line is filled concentrically by lamellar bone. Larger gaps are filled through a succession of fibrous tissue, fibrocartilage, and woven bone. In the absence of any microinstability at the fracture site, direct healing takes place without any callus formation.

Subsequent bone remodeling eventually restores the original shape and internal architecture of the fractured bone. Functional sculpting and remodeling of the primitive bone tissue is carried out by a temporary team of juxtaposed osteoclasts and osteoblasts called the *basic multicellular unit (BMU)*. The osteoblasts develop from pluripotent mesenchymal stem cells whereas multicellular osteoclasts arise from a monocyte/macrophage lineage.[12] The development and differentiation of the BMUs are controlled by locally secreted growth factors, cytokines, and mechanical signals. As osteoclasts at the leading edge of the BMUs excavate bone through proteolytic digestion, active osteoblasts move in, secreting layers of osteoid and slowly refilling the cavity. The osteoid begins to mineralize when it is about 6 µm thick. Osteoclasts reaching the end of their lifespan of 2 weeks die and are removed by phagocytes. The majority (≤65%) of the remodeling osteoblasts also die within 3 months and the remainder are entombed inside the mineralized matrix as osteocytes.

While the primitive bone mineralizes, remodeling BMUs cut their way through the reparative tissue and replace it with mature bone. The "grain" of the new bone tissue starts paralleling local compression and tension strains. Consequently, the shape and strength of the reparative bone tissue changes to accommodate greater functional loading. Tissue-level strains produced by functional loading play an important role in the remodeling of the regenerate bone. Whereas low levels of tissue strain (~2000 microstrains) are considered physiologic and necessary for cell differentiation and callus remodeling, high strain levels (>2000 microstrains) begin to adversely affect osteoblastic differentiation and bone matrix formation.[13,14] If there is excess interfragmentary motion, bone regenerates primarily through endochondral ossification or the formation of a cartilaginous callus that is gradually replaced by new bone. In contrast, osseous healing across stabilized fracture segments occurs primarily through intramembranous ossification. Major factors determining the mechanical milieu of a healing fracture include the fracture configuration, the accuracy of fracture reduction, the stability afforded by the selected fixation device, and the degree and nature of microstrains provoked by function. If a fracture fixation device is incapable of stabilizing the fracture, the interfragmentary microinstability provokes osteoclastic resorption of the fracture surfaces and results in a widening of the fracture gap. Although bone union may be ultimately achieved through secondary healing by callus production and endochondral ossification, the healing is protracted. Fibrous healing and nonunions are clinical manifestations of excessive microstrains interfering with the cellular healing process.

Extraction Wounds

The healing of an extraction socket is a specialized example of healing by second intention.[15] Immediately after the removal of the tooth from the socket, blood fills the extraction site. Both intrinsic and extrinsic pathways of the clotting cascade are activated. The resultant fibrin meshwork, which contains entrapped red blood cells, seals off the torn blood vessels and reduces the size of the extraction wound. Organization of the clot begins within the first 24 to 48 hours, with engorgement and dilation of blood vessels within the periodontal ligament remnants, followed by leukocytic migration and formation of a fibrin layer. In the first week, the clot forms a temporary scaffold upon which inflammatory cells migrate. Epithelium at the wound periphery grows over the surface of the organizing clot. Osteoclasts accumulate along the alveolar bone crest and set the stage for active crestal resorption. Angiogenesis proceeds in the remnants of the periodontal ligaments. In the second week, the clot continues to get organized through fibroplasia and new blood vessels begin to penetrate toward the center of the clot. Trabeculae of osteoid slowly extend into the clot from the alveolus, and osteoclastic resorption of the cortical margin of the alveolar socket is more distinct. By the third week, the extraction socket is filled with granulation tissue and poorly calcified bone forms at the wound perimeter. The surface of the wound is completely reepithelialized with minimal or no scar formation. Active bone remodeling by deposition and resorption continues for several more weeks. Radiographic evidence of bone formation does not become apparent until the sixth to eighth weeks after tooth extraction. Owing to the ongoing process of bone remodeling, the final healing product of the extraction site may not be discernible on radiographs after 4 to 6 months.

Occasionally, the blood clot fails to form or may disintegrate, causing a localized alveolar osteitis. When this happens, the healing is delayed considerably and the socket fills gradually. In the absence of a healthy granulation tissue matrix, the apposition of regenerate bone to remaining alveolar bone takes place at a much slower rate. Compared with a normal socket, the infected socket remains open or partially covered with hyperplastic epithelium for extended periods.

Skin Grafts

Skin grafts may be either full thickness or split thickness.[16] A full-thickness graft is composed of epidermis and the entire dermis; a split-thickness graft is composed of the epidermis and varying amounts of dermis. Depending on the amount of underlying dermis included, split-thickness grafts are described as thin, intermediate, or thick.[17] After grafting, nutritional support for a free skin graft is initially provided by plasma that exudes from the dilated capillaries of the host bed. A fibrin clot forms at the graft-host interface, fixing the graft to the host bed. Host leukocytes infiltrate into the graft through the lower layers of the graft. Graft survival depends on the ingrowth of blood vessels from the host into the graft (neovascularization) and direct anastomoses between the graft and the host vasculature (inosculation). Endothelial capillary buds from the host site invade the graft, reaching the dermoepidermal junction by 48 hours. Concomitantly, vascular connections are established between host and graft vessels. However, only a few of the ingrowing capillaries succeed in developing a functional anastomosis. Formation of vascular connections between the recipient bed and the transplant is signaled by the pink appearance of the graft, which appears between the third and the fifth day postgrafting. Fibroblasts from the recipient bed begin to invade the layer of fibrin and leukocytes by the fourth day after transplantation. The fibrin clot is slowly resorbed and organized as fibroblastic infiltration continues. By the ninth day, the new blood vessels and fibroblasts have achieved a firm union, anchoring the deep layers of the graft to the host bed.

Reinnervation of the skin graft occurs by nerve fibers entering the graft through its base and sides. The fibers follow the vacated neurilemmal cell sheaths to reconstruct the innervation pattern of the donor skin. Recovery of sensation usually begins within 2 months after transplantation. Grafts rarely attain the sensory qualities of normal skin, because the extent of reinnervation depends on how accessible the neurilemmal sheaths are to the entering nerve fibers. The clinical performance of the grafts depends on their relative thickness. Because split-thickness grafts are thinner than full-thickness grafts, they are more susceptible to trauma and undergo considerable contraction; however, they have greater survival rates clinically. Full-thickness skin grafts do not "take" as well and are slow to revascularize. However, full-thickness grafts are less susceptible to trauma and undergo minimal shrinkage.

WOUND HEALING COMPLICATIONS

Healing in the orofacial region is often considered a natural and uneventful process and seldom intrudes into the surgeon's consciousness. However, this changes when complications arise and encumber the wound healing continuum. Most wound healing complications manifest in the early postsurgical period but some may manifest much later. The two problems most commonly encountered by the surgeon are wound infection and dehiscence; proliferative healing is less typical.

Wound Infection

Infections complicating surgical outcomes usually result from gross bacterial contamination of susceptible wounds. All wounds are intrinsically contaminated by bacteria; however, this must be distinguished from true wound infection in which the bacterial burden of replicating microorganisms actually impairs healing.[18] Experimental studies have demonstrated that regardless of the type of infecting microorganism, wound infection occurs when there are more than 1×10^5 organisms per gram of tissue.[19,20] Beyond relative numbers, the pathogenicity of the infecting microorganisms as

well as host response factors also determine whether wound healing is impaired.

The continual presence of a bacterial infection stimulates the host immune defenses, leading to the production of inflammatory mediators, such as prostaglandins and thromboxane. Neutrophils migrating into the wound release cytotoxic enzymes and free oxygen radicals. Thrombosis and vasoconstrictive metabolites cause wound hypoxia, leading to enhanced bacterial proliferation and continued tissue damage. Bacteria destroyed by host defense mechanisms provoke varying degrees of inflammation by releasing neutrophil proteases and endotoxins. Newly formed cells and their collagen matrix are vulnerable to these breakdown products of wound infection, and the resulting cell and collagen lysis contribute to impaired healing. Clinical manifestations of wound infection include the classic signs and symptoms of local infection: erythema, warmth, swelling, pain, and accompanying odor and pus.

Inadequate tissue perfusion and oxygenation of the wound further compromise healing by allowing bacteria to proliferate and establish infection. Failure to follow aseptic technique is a frequent reason for the introduction of virulent microorganisms into the wound. Transformation of contaminated wounds into infected wounds is also facilitated by excessive tissue trauma, remnant necrotic tissue, foreign bodies, or compromised host defenses. The most important factor in minimizing the risk of infection is meticulous surgical technique, including thorough débridement, adequate hemostasis, and elimination of any dead space. Careful technique must be augmented by proper postoperative care, with an emphasis on keeping the wound site clean and protecting it from trauma.

Wound Dehiscence

Partial or total separation of the wound margins may manifest within the first week after surgery. Most instances of wound dehiscence result from tissue failure rather than improper suturing techniques. The dehisced wound may be closed again or left to heal by secondary intention, depending upon the extent of the disruption and the surgeon's assessment of the clinical situation.

Proliferative Scarring

Some patients may go on to develop aberrant scar tissue at the site of their skin injury. The two common forms of hyperproliferative healing, hypertrophic scars and keloids, are characterized by hypervascularity and hypercellularity. Distinctive features include excessive scarring, persistent inflammation, and an overproduction of ECM components, including glycosaminoglycans and collagen type I.[21] Despite their overt resemblance, hypertrophic scars and keloids do have some clinical dissimilarities. In general, hypertrophic scars arise shortly after the injury, tend to be circumscribed within the boundaries of the wound, and eventually recede. Keloids, conversely, manifest months after the injury, grow beyond the wound boundaries, and rarely subside. There is a clear familial and racial predilection for keloid formation, and susceptible individuals usually develop keloids on their face, ear lobes, and anterior chest.

Although processes leading to hypertrophic scar and keloid formation are not yet clarified, altered apoptotic behavior is believed to be a significant factor. Ordinarily, apoptosis or programmed cell death is responsible for the removal of inflammatory cells as healing proceeds and for the maturation of granulation tissue into scar. Dysregulation in apoptosis results in excessive scarring, inflammation, and an overproduction of ECM components. Both keloids and hypertrophic scars demonstrate sustained elevation of growth factors including TGF-β, PDGF, IL-1, and IGF-I.[22] The growth factors, in turn, increase the numbers of local fibroblasts and prompt excessive production of collagen and ECM. In addition, proliferative scar tissue exhibits increased numbers of neoangiogenesis-promoting vasoactive mediators as well as histamine-secreting mast cells capable of stimulating fibrous tissue growth. Although there is no effective therapy for keloids, the more common methods for preventing or treating these lesions focus on inhibiting protein synthesis. These agents, primarily corticosteroids, are injected into the scar to decrease fibroblast proliferation, decrease angiogenesis, and inhibit collagen synthesis and ECM protein synthesis.

OPTIMIZING WOUND HEALING

At its very essence, the wound represents an extreme disruption of the cellular microenvironment. Restoration of constant internal conditions or homeostasis at the cellular level is a constant undertow of the healing response. A variety of local and systemic factors can impede healing, and the informed surgeon can anticipate and, where possible, proactively address these barriers to healing so that wound repair can progress normally.[23]

Tissue Trauma

Minimizing trauma to the tissues helps promote faster healing and should be a central consideration at every stage of the surgical procedure, from placement of the incision to suturing of the wound. Properly planned, the surgical incision is just long enough to allow optimum exposure and adequate operating space. The incision should be made with one clean consistent stroke of evenly applied pressure. Sharp tissue dissection and carefully placed retractors further minimize tissue injury. Sutures are useful for holding the severed tissues in apposition until the wound has healed enough. However, sutures should be used judiciously because they have the ability to add to the risk of infection and are capable of strangulating the tissues if applied too tightly.

Hemostasis and Wound Débridement

Bleeding from a transected vessel or diffuse oozing from the denuded surfaces interferes with the surgeon's view of

underlying structures. Achieving complete hemostasis before wound closure helps prevent the formation of a hematoma postoperatively. The collection of blood or serum at the wound site provides an ideal medium for the growth of microorganisms that cause infection. In addition, hematomas can result in necrosis of overlying flaps. However, hemostatic techniques must not be used too aggressively during surgery because the resulting tissue damage can prolong healing time. Postoperatively, the surgeon may insert a drain or apply a pressure dressing to help eliminate dead space in the wound.

Devitalized tissue and foreign bodies in a healing wound act as a haven for bacteria and shield them from the body's defenses.[23] The dead cells and cellular debris of necrotic tissue have been shown to reduce host immune defenses and encourage active infection. A necrotic burden allowed to persist in the wound can prolong the inflammatory response, mechanically obstruct the process of wound healing, and impede reepithelialization. Dirt and tar located in traumatic wounds not only jeopardize healing but also may result in a "tattoo" deformity. By removing dead and devitalized tissue and any foreign material from a wound, débridement helps to reduce the number of microbes, toxins, and other substances that inhibit healing. The surgeon should also keep in mind that prosthetic grafts and implants, despite refinements in biocompatibility, can stimulate varying degrees of foreign body reaction and adversely affect the healing process.

Tissue Perfusion

Poor tissue perfusion is one of the main barriers to healing inasmuch as tissue oxygen tension drives the healing response.[24,25] Oxygen is necessary for hydroxylation of proline and lysine, the polymerization and cross-linking of procollagen strands, collagen transport, fibroblast and endothelial cell replication, effective leukocyte killing, angiogenesis, and many other processes related to wound healing. Relative hypoxia in the region of injury is useful to the extent that it stimulates a fibroblastic response and helps mobilize other cellular elements of repair.[26] However, very low oxygen levels act together with the lactic acid produced by infecting bacteria to lower tissue pH and contribute to tissue breakdown. Cell lysis follows, with releases of proteases and glycosidases and subsequent digestion of ECM.[27] Impaired local circulation also hinders delivery of nutrients, oxygen, and antibodies to the wound. Neutrophils are affected because they require a minimal level of oxygen tension to exert their bactericidal effect. Delayed movement of neutrophils, opsonins, and the other mediators of inflammation to the wound site further diminishes the effectiveness of the phagocytic defense system and allows colonizing bacteria to proliferate. Collagen synthesis is dependent on oxygen delivery to the site, which in turn affects wound tensile strength. Most healing problems associated with diabetes mellitus, irradiation, small vessel atherosclerosis, chronic infection, and altered cardiopulmonary status can be attributed to local tissue ischemia.

Wound microcirculation after surgery determines the wound's ability to resist the inevitable bacterial contamination.[28] Tissue rendered ischemic by rough handling, or desiccated by cautery or prolonged air drying, tends to be poorly perfused and susceptible to infection. Similarly, tissue ischemia produced by tight or improperly placed sutures, poorly designed flaps, hypovolemia, anemia, and peripheral vascular disease all adversely affects wound healing. Smoking is a common contributor to decreased tissue oxygenation.[29] The peripheral vasoconstriction produced by smoking a cigarette can last up to an hour; thus, a pack-a-day smoker remains tissue hypoxic for most part of each day. Smoking also increases carboxyhemoglobin, increases platelet aggregation, increases blood viscosity, decreases collagen deposition, and decreases prostacyclin formation, all of which negatively affect wound healing. Patient optimization, in the case of smokers, may require that the patient abstain from smoking for a minimum of 1 week before and after surgical procedures. Another way of improving tissue oxygenation is the use of systemic hyperbaric oxygen (HBO) therapy to induce the growth of new blood vessels and facilitate increased flow of oxygenated blood to the wound.

Diabetes

Numerous studies have demonstrated that the higher incidence of wound infection associated with diabetes has less to do with the patient having diabetes and more to do with hyperglycemia. Simply put, a patient with well-controlled diabetes may not be at a greater risk for wound healing problems than a nondiabetic patient. Tissue hyperglycemia impacts every aspect of wound healing by adversely affecting the immune system including neutrophil and lymphocyte function, chemotaxis, and phagocytosis.[30] Uncontrolled blood glucose hinders red blood cell permeability and impairs blood flow through the critical small vessels at the wound surface. The hemoglobin release of oxygen is impaired, resulting in oxygen and nutrient deficiency in the healing wound. Wound ischemia and impaired recruitment of cells resulting from the small vessel occlusive disease render the wound vulnerable to bacterial and fungal infections.

Immunocompromise

The immune response directs the healing response and protects the wound from infection. In the absence of an adequate immune response, surgical outcomes are often compromised. An important assessment parameter is total lymphocyte count. A mild deficit is a lymphocytic level between 1200 and 1800, and levels below 800 are considered severe total lymphocyte deficits. Patients with debilitated immune response include human immunodeficiency virus (HIV)–infected patients in advanced disease stages, patients on immunosuppressive therapy, and those taking high-dose steroids for extended periods.[31] Studies indicate that HIV-infected patients with CD4 counts of less than 50 cells/mm^3 are at significant risk of poor wound outcome.[32] Although newer immunosuppressive drugs, such as cyclosporine, have no apparent effect on wound healing, other medications can

retard the healing process, in both rate and quality, by altering the inflammatory reaction and the cell metabolism.

The use of steroids, such as prednisone, is a typical example of how suppression of the innate inflammatory process also increases wound healing complications. Exogenous corticosteroids diminish prolyl hydroxylase and lysyl oxidase activity, depressing fibroplasias, collagen formation, and neovascularity.[33] Fibroblasts reach the site in a delayed fashion and wound strength is decreased by as much as 30%. Epithelialization and wound contraction are also impaired. The inhibitory effects of glucocorticosteriods can be attenuated to some extent by vitamin A given concurrently.

Most antineoplastic agents exert their cytotoxic effect by interfering with DNA or RNA production. The reduction in protein synthesis or cell division reveals itself as impaired proliferation of fibroblasts and collagen formation. Attendant neutropenia also predisposes to wound infection by prolonging the inflammatory phase of wound healing. Because of their deleterious effect on wound healing, administration of antineoplastic drugs should be restricted, when possible, until such time that the potential for healing complications has passed.

Radiation Injury

Therapeutic radiation for head and neck tumors inevitably produces collateral damage in adjacent tissue and reduces its capacity for regeneration and repair. The pathologic processes of radiation injury start right away; however, the clinical and histologic features may not become apparent for weeks, months, or even years after treatment.[34] The cellular and molecular responses to tissue irradiation are immediate and dose dependent and can cause both early and late consequences.[35] DNA damage from ionizing radiation leads to mitotic cell death in the first cell division after irradiation or within the first few divisions. Early acute changes are observed within a few weeks of treatment and primarily involve cells with a high turnover rate. The common symptoms of oral mucositis and dermatitis result from loss of functional cells and temporary lack of replacement from the pools of rapidly proliferating cells. The inflammatory response is largely mediated by cytokines activated by the radiation injury. Overall, the response has the features of wound healing; waves of cytokines are produced in an attempt to heal the radiation injury. The cytokines lead to an adaptive response in the surrounding tissue, cause cellular infiltration, and promote collagen deposition. Damage to local vasculature is exacerbated by leukocyte adhesion to endothelial cells and the formation of thrombi that block the vascular lumen, further depriving the cells that depend on the vessels.

The acute symptoms eventually start to subside as the constitutive cells gradually recover their proliferative abilities. However, these early symptoms may not be apparent in some tissues such as bone, where the cumulative progressive effects of radiation can precipitate acute breakdown of tissue many years after therapy. The late effects of radiation are permanent and directly related to higher doses.[36] Collagen hyalinizes and the tissues become increasingly fibrotic and hypoxic due to obliterative vasculitis, and the tissue susceptibility to infection increases correspondingly. Once these changes occur, they are irreversible and do not change with time. Hence, the surgeon must always anticipate the possibility of a complicated healing after surgery or traumatic injury in irradiated tissue. Wound dehiscence is common and the wound heals slowly or incompletely. Even minor trauma may result in ulceration and colonization by opportunistic bacteria. If the patient cannot mount an effective inflammatory response, progressive necrosis of the tissues may follow. Healing can be achieved only by excising all nonvital tissue and covering the bed with a well-vascularized graft. Owing to the relative hypoxia at the irradiated site, tissue with intact blood supply needs to be brought in to provide both oxygen as well as cells necessary for inflammation and healing. The progressive obliteration of blood vessels makes bone particularly vulnerable. After trauma or disintegration of the soft tissue cover due to inflammatory reaction, healing does not occur because irradiated marrow cannot form granulation tissue. In such instances, the avascular bone needs to be removed down to the healthy portion to allow healing to proceed.

Hyperbaric Oxygen Therapy

HBO therapy is based on the concept that low tissue oxygen tension, typically a partial pressure of oxygen (Po_2) of 5 to 20 mmHg, leads to anaerobic cellular metabolism, increase in tissue lactate, and a decrease in pH, all of which inhibit wound healing.[37] HBO therapy requires that the patient recline in a hyperbaric chamber and breathe 100% oxygen at 2.0 to 2.4 atm for 1 to 2 hours. The HBO therapy is repeated daily for 3 to 10 weeks. HBO increases the quantity of dissolved oxygen and the driving pressure for oxygen diffusion into the tissue. Correspondingly, the oxygen diffusion distance is increased threefold to fourfold, and wound Po_2 ultimately reaches 800 to 1100 mmHg. The therapy stimulates the growth of fibroblasts and vascular endothelial cells, increases tissue vascularization, enhances the killing ability of leukocytes, and is lethal for anaerobic bacteria. Clinical studies suggest that HBO therapy can be an effective adjunct in the management of diabetic wounds.[38] Animal studies indicate that HBO therapy could be beneficial in the treatment of osteomyelitis and soft tissue infections.[39,40] Adverse effects of HBO therapy are barotraumas of the ear, seizure, and pulmonary oxygen toxicity. However, in the absence of controlled scientific studies with well-defined end points, HBO therapy remains a controversial aspect of surgical practice.[41,42]

Age

In general, wound healing is faster in the young and protracted in the elderly. The decline in healing response results from the gradual reduction of tissue metabolism as one ages, which may itself be a manifestation of decreased circulatory efficiency. The major components of the healing response in aging skin or mucosa are deficient or damaged

with progressive injuries.[43] As a result, free oxidative radicals continue to accumulate and are harmful to the dermal enzymes responsible for the integrity of the dermal or mucosal composition. In addition, the regional vascular support may be subjected to extrinsic deterioration and systemic disease decompensation, resulting in poor perfusion capability.[44] However, in the absence of compromising systemic conditions, differences in healing as a function of age seem to be small.

Nutrition

Adequate nutrition is important for normal repair.[45] In malnourished patients, fibroplasia is delayed, angiogenesis decreased, and wound healing and remodeling prolonged. Dietary protein has received special emphasis with respect to healing. Amino acids are critical for wound healing with methionine, histidine, and arginine playing important roles. Nutritional deficiencies severe enough to lower serum albumin to less than 2 g/dL are associated with a prolonged inflammatory phase, decreased fibroplasia, and impaired neovascularization, collagen synthesis, and wound remodeling. As long as a state of protein catabolism exists, the wound will be very slow to heal. Methionine appears to be the key amino acid in wound healing. It is metabolized to cysteine, which plays a vital role in the inflammatory, proliferative, and remodeling phases of wound healing.

Serum prealbumin is commonly used as an assessment parameter for protein.[46,47] Contrary to serum albumin, which has a very long half-life of about 20 days, prealbumin has a shorter half-life of only 2 days. As such, it provides a more rapid assessment ability. Normal serum prealbumin is approximately 22.5 mg/dL, a level below 17 mg/dL is considered a mild deficit, and a severe deficit would be below 11 mg/dL. As part of the perioperative optimization process, malnourished patients may be provided with solutions that have been supplemented with amino acids such as glutamine to promote improved mucosal structure and function and to enhance whole body nitrogen kinetics. An absence of essential building blocks obviously thwarts normal repair, but the reverse is not necessarily true. Whereas a minimum protein intake is important for healing, a high-protein diet does not shorten the time required for healing.

Several vitamins and trace minerals play a significant role in wound healing.[48] Vitamin A stimulates fibroplasia, collagen cross-linking, and epithelialization and will restimulate these processes in the steroid-retarded wound. Vitamin C deficiency impairs collagen synthesis by fibroblasts, because it is an important cofactor, along with α-ketoglutarate and ferrous iron, in the hydroxylation process of proline and lysine. Healing wounds appear to be more sensitive to ascorbate deficiency than uninjured tissue. Increased rates of collagen turnover persist for a long time, and healed wounds may rupture when the individual becomes scorbutic. Local antibacterial defenses are also impaired because ascorbic acid is also necessary for neutrophil superoxide production. The B-complex vitamins and cobalt are essential cofactors in antibody formation, white blood cell function, and bacterial resistance. Depleted serum levels of micronutrients, including magnesium, copper, calcium, iron, and zinc, affect collagen synthesis.[49] Copper is essential for covalent cross-linking of collagen, whereas calcium is required for the normal function of granulocyte collagenase and other collagenases at the wound milieu. Zinc deficiency retards both fibroplasia and reepithelialization; cells migrate normally but do not undergo mitosis.[50] Numerous enzymes are zinc dependent, particularly DNA polymerase and reverse transcriptase. Conversely, exceeding the zinc levels can exert a distinctly harmful effect on healing by inhibiting macrophage migration and interfering with collagen cross-linking.

ADVANCES IN WOUND CARE

An increased understanding of the wound healing processes has generated heightened interest in manipulating the wound microenvironment to facilitate healing. Traditional passive ways of treating surgical wounds are rapidly giving way to approaches that actively modulate wound healing. Therapeutic interventions range from treatments that selectively jumpstart the wound into the healing cascade to methods that mechanically protect the wound or increase oxygenation and perfusion of the local tissues.[51,52]

Growth Factors

Through their central ability to orchestrate the various cellular activities that underscore inflammation and healing, cytokines have profound effects on cell proliferation, migration, and ECM synthesis.[53] Accordingly, newer interventions seek to control or modulate the wound healing process by selectively inhibiting or enhancing the tissue levels of the appropriate cytokines.

The more common clinical approach has been to apply exogenous growth factors, such as PGDF, angiogenesis factor, epidermal growth factor (EGF), TGF, basic fibroblast growth factor (bFGF), and IL-1, directly to the wound. However, the potential of these extrinsic agents has not yet been realized clinically and may relate to figuring out which growth factors to put into the wound, when to apply, and at what dose. Becaplermin (recombinant human platelet–derived growth factor-BB [rhPDGF]; Regranex, 0.01% gel; Ortho-McNeil Pharmaceutical Inc, Raritan, New Jersey) was one of the first US Food and Drug Administration approved recombinant growth factor products introduced to promote growth of soft-tissue granulation tissue in treating cutaneous wounds.[54,55] The recombinant PDGF increases fibroblast replication and induces fibroblasts to produce collagenase, which is important for connective tissue remodeling. In addition, rhPDGF increases the production of other connective tissue matrix components including glycosaminoglycans and proteoglycans. Notwithstanding its clinical efficacy, becaplermin has not found broad application due to its very high

costs. More recently, recombinant human keratinocyte growth factor-2 (KGF-2) has been shown to accelerate wound healing in experimental animal models. It enhanced both the formation of granulation tissue in rabbits and wound closure of the human meshed skin graft explanted on athymic nude rats.[56,57] Experimental studies suggest potential for the use of growth factors in facilitating peripheral nerve healing. Several growth factors belonging to the neurotrophin family have been implicated in the maintenance and repair of nerves. Nerve growth factor (NGF), synthesized by Schwann cells distal to the site of injury, aids in the survival and development of sensory nerves. This finding has led some investigators to suggest that exogenous NGF application may assist in peripheral nerve regeneration after injury.[58] Newer neurotrophins such as brain-derived neurotrophic factor and neurotrophin-3 as well as ciliary neurotrophic factor appear to support the growth of sensory, sympathetic, and motor neurons in vitro.[59–61] Insulin-like growth factors have demonstrated similar neurotrophic properties.[62] Although most of the investigations until now have been experimental, increasing sophistication in the dosing, combinations, and delivery of neurotropic growth factors will lead to greater clinical application.

Osteoinductive growth factors hold special appeal to surgeons conducting bone grafting procedures for their ability to promote the formation of new bone and avoid harvesting autogenous bone with its associated complications. Of the multiple osteoinductive cytokines, the bone morphogenetic proteins (BMPs) belonging to the TGF-β superfamily have received the greatest attention. To date over 20 members have been identified in humans with varying functions during processes such as embryogenesis, skeletal formation, hematopoesis and neurogenesis.[63] Advances in recombinant DNA techniques now allow the production of these biomolecules in quantities large enough for routine clinical applications. In particular, recombinant human bone morphogenetic protein-2 (rhBMP-2) and rhBMP-7 have been studied extensively for their ability to induce undifferentiated mesenchymal cells to differentiate into osteoblasts (osteoinduction). Yasko and colleagues[64] used a rat segmental femoral defect model to show that rhBMP-2 can produce 100% union rates when combined with bone marrow. The union rate achieved with the combination approach was three times higher than that achieved with autologous cancellous bone graft alone. Similarly, Toriumi and coworkers[65] showed that rhBMP-2 could heal mandibular defects with bone formed by the intramembranous pathway. The widespread application of osteoinductive cytokines depends in large part on a better understanding of the complex interaction of growth factors and the concentrations necessary to achieve specific effects.

Most recently, INFUSE® Bone Graft (Medtronic Spinal and Biologics, Memphis, TN) was approved as an alternative to autogenous bone grafts in interbody spine fusion, fresh tibial fractures, and oral and maxillofacial bone grafting procedures.[66] Containing rhBMP-2 at a concentration of 1.5 mg/cc delivered on an absorbable collagen sponge (ACS), the INFUSE® Bone Graft is increasingly applied in maxillary sinus augmentations, and for localized alveolar ridge augmentations. Despite the reported successes, the universal adoption of BMP is tempered by high costs and lingering safety concerns and complications specific to BMP use. Ongoing research is focused on clarifying guidelines for BMP use and on developing more affordable BMP formulations with dosing that predictably results in the desired clinical outcome while minimizing possible side effects.[67]

Gene Therapy

The application of gene therapy to wound healing has been driven by the desire to selectively express a growth factor for controlled periods of time at the site of tissue injury.[68] Unlike the diffuse effects of a bolus of exogenously applied growth factor, gene transfer permits targeted, consistent, local delivery of peptides in high concentrations to the wound environment. Genes encoding for select growth factors are delivered to the site of injury using a variety of viral, chemical, electrical, or mechanical methods.[69] Cellular expression of the proteins encoded by the nucleic acids help modulate healing by regulating local events such as cell proliferation, cell migration, and the formation of ECM. The more popular methods for transfecting wounds involve the in vivo use of adenoviral vectors. Existing gene therapy technology is capable of expressing a number of modulatory proteins at the physiologic or supraphysiologic range for up to 2 weeks.

Numerous experimental studies have demonstrated the efficacy of gene therapy in stimulating bone formation and regeneration. Mesenchymal cells transfected with adenovirus-hBMP-2 cDNA have been shown to be capable of forming bone when injected intramuscularly in the thighs of rodents.[70,71] Similarly, bone marrow cells transfected ex vivo with hBMP-2 cDNA have been shown to heal femoral defects.[72] Using osteoprogenitor cells for the expression of bone-promoting osteogenic factors enables the cells not only to produce bone growth–promoting factors but also to respond, differentiate, and participate in the bone formation process. These early studies suggest that advances in gene therapy technology can be used to facilitate healing of bone and other tissues and may lead to better and less invasive reconstructive procedures in the near future.

Dermal and Mucosal Substitutes

Immediate wound coverage is critical for accelerated wound healing. The coverage protects the wound from water loss, drying, and mechanical injury. Although autologous grafts remain the standard for replacing dermal mucosal surfaces, a number of bioengineered substitutes are finding their way into mainstream surgical practice. Available human skin substitutes are grouped into three major types and serve as excellent alternatives to autografts. The first type consists of grafts of cultured epidermal cells with no dermal components.

The second type has only dermal components. The third type consists of a bilayer of both dermal and epidermal elements. An example of bioengineered skin substitutes is Apligraf®, an allogeneic living epidermal and dermal skin derived from cultured neonatal foreskin (Organogenesis, Canton, Massachusetts).[73] This bioengineered, full-thickness skin product consists of a living permanent bilayer skin graft with active cellular and growth factor components. It does not elicit an immune response because its Langerhans cells have been extracted. Currently, Apligraf® is FDA approved for covering venous and diabetic foot ulcers. The chief effect of most skin replacements is to promote wound healing by stimulating the recipient host to produce a variety of wound healing cytokines. The use of cultured skin to cover wounds is particularly attractive inasmuch as the living cells already know how to produce growth factors at the right time and in the right amounts. Thus, the ultimate goal of bioengineers is to develop engineered skin that contains all of the components necessary to modulate healing and allow for wound healing with a surrogate that replicates native tissue and limits scar formation.

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CHAPTER 2

Medical Management and Preoperative Patient Assessment

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Patients who undergo oral and maxillofacial surgery are often young and relatively healthy. Medical complications are uncommon in this patient population. However, medical conditions may be present in any patient and may lead to increased morbidity and mortality unless these conditions are sought in those with a history of disease and recognized in those without such a history. A thorough medical and social history will elicit medical co-morbidities when known and provide clear guidelines regarding management in most patients. A thorough physical examination will often confirm the presence of systemic disease or identify it in those without a prior history. A thorough knowledge of medical co-morbidities before any surgical procedure will allow patients to be stratified according to surgical risk and managed in an optimal fashion. Preoperative patient assessment is best completed by the surgeon who has a vested interest in the patient's well-being. When medical conditions and co-morbidities are recognized preoperatively, appropriate workup or referral is easily organized. The involvement of other medical and surgical subspecialties should be readily sought when indicated. A systematic approach to preoperative patient assessment is required. This requires a didactic approach to a patient interview. This mandates obtaining a chief complaint; history of the chief complaint; other complaints; a medical history with review of all systems, allergies, medications, social history, family history, surgical history; and functional status. The sensitivity of a thorough history to identify a previously unrecognized medical condition should not be underestimated.

The physical examination should also be standardized to include all systems. The examination is, however, often focused based on the chief complaint and a thorough history. In addition to identifying the need for oral and maxillofacial surgery, the primary purpose of the physical examination is to identify pertinent positive and negative findings. The history and physical examination will then enable the surgeon to request additional investigations and medical referrals that allow risk stratification.

The ultimate goal of the preoperative evaluation is to identify medical concerns and provide for the most efficacious perioperative treatment algorithm that minimizes patient morbidity. The risk assessment for each patient undergoing surgery requires an understanding of the surgical stress and the patient's medical condition. Head and neck surgery is considered to be intermediate in perioperative risk. Oral and maxillofacial procedures performed in an ambulatory setting would be considered low risk. The patient's medical conditions impart additional risk factors that are best evaluated using a combination of the American Society of Anesthesiologists (ASA) classification (Table 2-1) and an assessment of functional status.

Additional classification systems may be appropriate for risk stratification for specific organ systems depending on

TABLE 2-1. **American Society of Anesthesiology Physical Status Classification**

Classification	Description
I	Healthy patient
II	Mild systemic disease with no functional limitation
III	Severe systemic disease with functional limitation
IV	Severe systemic disease that is a threat to life
V	Moribund patient
VI	Brain-dead patient awaiting organ transplantation

Adapted from American Society of Anesthesiologists. Relative Value Guide, 2003. Park Ridge, IL, American Society of Anesthesiologists, 2003.

TABLE 2-2. **Duke Activity Status Index**

Activity	METs	Functional Capacity
Walk around house	1.75	Poor
Walk two blocks on level ground	2.75	Poor
Activities of daily living	2.75	Poor
Light house work (dishwashing)	2.7	Poor
Moderate housework (vacuuming)	3.5	Poor
Yard work	4.5	Moderate
Sexual relations	5.25	Moderate
Climb stairs	5.5	Moderate
Golf	6	Moderate
Swimming, basketball	7.5	Excellent
Running	8.0	Excellent

MET = metabolic equivalent (1 MET = 3.5 mL/kg/min O_2 use).
Adapted from Hlatky MA, Boineau RE, Higginbothom MB, et al. A brief self-administered questionnaire to determine the functional capacity (the Duke Activity Status Index). *Am J Cardiol* 1989;64:651–654.

TABLE 2-3. **Cardiac Predictors of Perioperative Morbidity**

Condition	Goldman et al[2]	Detsky et al[3]
Myocardial infarction		
<6 mo	10	10
>6 mo		5
Angina		
Unstable		10
With one flight of stairs		10
With ADLs		20
Congestive heart failure	11	
<1 wk prior		10
>1 wk prior		5
Dysrhythmia		
PVC > 5/min	7	7
Nonsinus rhythm	7	5
Critical aortic stenosis	3	20
Age > 70 yr	5	5
Emergency operation	4	10
Poor general health (COPD, ESRD, ESLD, electrolytes)	3	5

ADLs = activities of daily living; COPD = chronic obstructive pulmonary disease; ESLD = end-stage liver disease; ESRD = end-stage renal disease; PVC = premature ventricular contraction

the medical disease present. The Duke activity status index is particularly useful to classify patients according to functional status[1] (Table 2-2). Age itself is a poor surrogate risk indicator but serves to further assess patients for surgical risk.

CARDIOVASCULAR ASSESSMENT AND DISEASE

Cardiovascular disease is a heterogeneous group of diseases that correlate with perioperative risk. It is crucial that the presence of cardiovascular disease be sought in the history and physical examination. Risk factors for cardiovascular disease may also provide insight into a patient's likelihood of cardiac complications. A multitude of cardiac indices have been put forward to help classify patients. Some are more complicated than others. The Goldman index was first published in 1977 and was later modified in 1986 by Detsky and coworkers (Table 2-3).[2,3]

Patients were stratified according to their total scores. Class I patients with scores of 0 to 5 had a 0.7% and a 0.2% incidence of serious complications and cardiac death, respectively. In contrast, class IV patients with scores of 26 or higher had a 22% and a 56% incidence, respectively.

More recently, the American College of Cardiology and the American Heart Association (ACC/AHA) task force have put forward additional recommendations for the evaluation of cardiac patients undergoing noncardiac surgery.[4] Patients are initially stratified as having major, intermediate, or minor predictors. Major clinical predictors of risk include acute coronary syndromes, decompensated congestive heart failure (CHF), significant arrhythmias, and severe valvular disease. Elective surgery is not recommended in this group of patients. When emergent noncardiac surgery is required, consideration should be given to cardiac catheterization. Intermediate clinical predictors include mild angina, prior myocardial infarction (MI), compensated CHF, and diabetes mellitus (DM). Minor predictors include advanced age, abnormal electrocardiogram (ECG), nonsinus rhythm, low functional capacity, history of cerebrovascular accident (CVA), and uncontrolled hypertension (HTN). In both the intermediate and the minor clinical predictor groups, a careful assessment of their functional status is required. Those with moderate or excellent functional status according to the Duke criteria may proceed to surgery. Those patients with poor functional status as defined by less than 4 metabolic equivalents (METs) should receive noninvasive cardiac testing.

Noninvasive cardiac testing typically involves a stress test using specific exercise protocol with age-related heart rate requirement and real-time ECG monitoring. The sensitivity of the stress test is low depending in part on how many vessels are stenosed. More sensitive noncardiac testing is typically more appropriate in some patients. The tests include exercise echocardiography, dobutamine stress echocardiography, and dipyridamole thallium stress testing. Any patient identified as high risk through this testing should proceed to coronary angiography before any noncardiac surgery.

Myocardial work is primarily determined by four factors related to myocardial oxygen demand: heart rate, preload, afterload, and contractility. *Heart rate* is influenced by a number of factors including oxygen demand, physical activity, hormonal and neurogenic mechanisms. *Preload* represents all factors that contribute to passive ventricular wall stress at the end of diastole. It approximates the end-diastolic ventricular pressure. Volume status significantly influences preload. An accurate determination of preload requires a central line to measure central venous pressure or pulmonary capillary wedge pressure. *Afterload* represents all factors that contribute to the ventricular wall stress during systole. The total peripheral resistance has the most influence on afterload, although intrathoracic pressure may also influence afterload as is the case with mechanical ventilation. *Contractility* refers

to the ability of the myocardium to contract. The opportunity to influence myocardial work and oxygen demand exists through modulation of any of these four factors.

Coronary Artery Disease

Patients with coronary artery disease (CAD) are often identified during the history and physical examination. When identified preoperatively, CAD should prompt risk stratification following the ACC/AHA algorithm. Patients with acute coronary syndromes should not undergo noncardiac surgery. Tests to consider include:

- Chest radiograph to evaluate for cardiomegaly, pulmonary edema, or pleural effusion.
- ECG to evaluate for left ventricular hypertrophy, ST segment changes, inverted T waves, Q waves, and arrhythmias.
- Transthoracic Doppler echocardiography for wall motion abnormalities, ejection fraction, and chamber pressures.
- Stress test to assess for functional cardiac ischemia. This can be combined with echocardiography.
- Perfusion nuclear imaging to assess cardiac perfusion at rest and with function.
- Cardiac angiography.

Asymptomatic patients with CAD may, however, develop symptoms in the perioperative period. Risk factors for CAD are DM, HTN, smoking, hypercholesterolemia, and a family history. CAD may result in stable angina or one of the acute coronary syndromes (ACSs). Stable angina often presents with precordial pain radiating to the left arm, neck, and jaw upon exertion. It is relieved by rest or the use of sublingual nitroglycerin. ACSs include unstable angina, non–ST-elevated MI, and ST-elevated MI. Symptoms are similar to stable angina but occur with less exertion than is usual, or at rest, and do not abate with further rest. The history surrounding the onset of chest pain has a diagnostic sensitivity of 90% when the symptoms are classic. An ECG may show ST segment depression or inverted T waves indicating ischemia. ST segment elevation indicates frank MI. The treatment of any patient suspected of having ACS begins with the correct diagnosis. The diagnosis can be confirmed with:

- A 12-lead ECG (ST elevation, inverted T waves, Q waves).
- Cardiac enzymes (CK-MB [MB isoenzyme of creatine kinase], troponins).

The initial treatment should include a standardized approach to reduce morbidity and mortality. All of the following should be instituted concurrently:

- Oxygen via facemask.
- Nitrates administered sublingually (or intravenously).
- Morphine for pain and to decrease sympathetic output.
- Aspirin 325 mg administered sublingually.

Additional treatment may include intravenous heparin and/or platelet glycoprotein IIa/IIIb antagonists to impair clotting and beta blockers to decrease myocardial oxygen demand. Definitive treatment may include the administration of thrombolytic agents, percutaneous angioplasty with stenting, or coronary artery bypass grafting.

The use of perioperative beta blockers has been shown to reduce the likelihood of cardiac events including MI. Patients with a recent MI may be at risk for re-infarction following the initial infarct. This has led to the historical suggestion of avoiding elective surgery for a period of 6 months. The current management of MI has radically changed since the early 1990s and, given the number of possible cardiac interventions after an MI, the risk of re-infarction may be much less than was originally thought. This may allow elective surgery to be performed within a much shorter period of time after an infarction, perhaps after only 6 weeks.[5]

Congestive Heart Failure

CHF is a result of inadequate cardiac output. Compensated CHF is considered an intermediate clinical predictor whereas decompensated CHF is considered a major clinical predictor within the ACC/AHA algorithm and a contraindication to elective surgery. The New York Heart Association (NYHA) classification may also be used to help stratify surgical patients (Table 2-4).

The risk of surgical patients with CHF decompensating depends on their class, with NYHA class I patients having a 3% risk and class IV patients having a 25% risk.[6] Causes of CHF include MI, valvular heart disease, HTN, anemia, pulmonary embolism (PE), cardiomyopathy, thyrotoxicosis, and endocarditis. Heart failure can be left-sided, right-sided, or both. Left-sided failure presents with exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnea, cardiomegaly, rales, and a third heart sound (S_3) gallop. Right-sided heart failure results in elevated jugular venous pressure, peripheral edema (especially lower extremities), and atrial fibrillation (AF). The diagnosis is best made with a transthoracic echocardiogram, although the measurement of brain natriuretic peptide (BNP) can be used to help diagnose and monitor CHF progression. Posteroanterior and lateral chest radiography will often reveal cardiomegaly and may show pulmonary edema and plural effusions if decompensated. Treatment is primarily aimed at reducing afterload and increasing the cardiac contractility. Treatment may include:

- Angiotensin-converting enzyme inhibitor (ACEI; e.g., captopril, lisinopril, ramipril).
- Diuretic (e.g., furosemide or hydrochlorothiazide).

TABLE 2-4. **New York Heart Association Classification**

Class	Symptoms
I	Asymptomatic
II	Symptomatic with moderate activity, comfortable at rest
III	Symptomatic with minimal activity, comfortable at rest
IV	Symptomatic at rest

- Beta blocker (metoprolol or carvedilol).
- Digoxin.

Patients with CHF should have their regular medications maintained in the perioperative setting. Particular attention to fluid and electrolyte balance is crucial. Potassium should be monitored closely because hypokalemia may be present secondary to the use of non–potassium-sparing diuretics or renal hypoperfusion. This may require correction with 10 to 40 mEq of oral or parenteral potassium. Parenteral replacement should be done carefully with no more than a 10 mEq/hr increase. Digitalis is often used to increase contractility in patients with CHF. Patients taking digitalis are at risk for fluctuating digitalis blood levels secondary to interactions with other drugs. This can lead to digitalis toxicity. In addition, reducing the preload can reduce cardiac output just as much as excessive preload can precipitate decompensation. Fluid replacement must be judicious and is often assisted by a central line so that central venous pressure can be monitored.

Valvular Heart Disease

Valvular heart disease is recognized through a thorough history and physical examination. Although the requirements for antibiotic prophylaxis have changed, the potential for valvular abnormalities to affect cardiovascular parameters must be recognized. As with most systemic diseases, functional status serves as a wonderful barometer with which to evaluate the severity of the valvular abnormality. Perioperative changes in cardiac rate, rhythm, blood pressure, preload, afterload, and contractility secondary to anesthesia and surgery may have profound influences on cardiovascular stability and morbidity.

Aortic stenosis (AS) may be congenital or secondary to rheumatic fever or dystrophic calcification. It may also be a result of obstructive hypertrophic subaortic stenosis. The classic presentation is syncope, angina, and exertional dyspnea. Patients are intolerant to changes in heart rate and peripheral vascular resistance. Auscultation of a high pitched midsystolic crescendo-decrescendo murmur at the right upper sternal border is consistent with AS. Critical AS is an independent risk factor for perioperative morbidity and mortality.[6] *Critical stenosis* is defined as an aortic valve area of less than 0.75 cm^2 and/or a valvular pressure gradient of 50 mmHg. A transthoracic echocardiogram is required to quantitate disease severity. As a result of the increased ventricular systolic pressures, left ventricular hypertrophy (LVH) develops with increased myocardial oxygen demand and a propensity for ischemia even without preexisting CAD. LVH may be identified on the ECG as well as with auscultation of the cardiac apex where a fourth heart sound (S_4) gallop may be appreciated. Patients with AS cannot tolerate heart rate increases or a reduction in peripheral resistance that further decreases coronary perfusion.

Aortic regurgitation (AR) may be secondary to rheumatic heart disease, bicuspid aortic valve, endocarditis, or aortic root disease. It may present with pulmonary edema, hypotension, and CHF. Auscultation of a diastolic decrescendo murmur, a high-pitched crescendo decrescendo murmur, and diastolic rumble are consistent with AR. The ECG will often reveal LVH whereas a chest x-ray will reveal left ventricular and aortic root enlargement. Treatment of patients often aims to reduce total peripheral resistance with calcium channel blockers or ACEIs. Beta blockers are best avoided because they increase the regurgitation by prolonging diastole.

Mitral stenosis (MS) is often due to preexisting rheumatic heart disease. It may present with dyspnea, orthopnea, pulmonary edema, AF, and a right ventricular heave. Auscultation of an opening snap and a mid-diastolic low-pitched rumble at the apex are consistent with MS. An ECG may reveal notched or enlarged P waves when atrial enlargement has developed. AF may be the final outcome. As with AS, increases in heart rate are not well tolerated owing to the potential for acute increases in pulmonary pressures and reduced cardiac output. Mitral regurgitation (MR) may be secondary to MS, mitral valve prolapse (MVP), rheumatic heart disease, endocarditis, or MI. It may present with pulmonary edema, hypotension, dyspnea on exertion, and a holosystolic high-pitched murmur at the cardiac apex.

Prosthetic Valve Replacement

Prosthetic valves can be alloplastic or biologic. A biologic valve can be a heterograft or a xenograft. Valve function is best evaluated with an assessment of the functional capacity of the patient and a transthoracic echocardiograph. Mechanical valves always require anticoagulation whereas biologic valves may not require anticoagulation after 3 months, although this depends on other risk factors. Patients with prosthetic valves are susceptible to endocarditis, valve failure, red cell destruction, and thromboemboli formation. Patients should have their warfarin discontinued before surgery if bleeding is likely to be a problem. The risk for thromboembolism and CVA is high if anticoagulation is discontinued and, accordingly, patients may need to be bridged with low-molecular-weight heparin (LMWH) as an outpatient or unfractionated heparin (UFH) as an inpatient. The adequacy of the warfarin anticoagulation should be measured with the prothrombin time or International Normalized Ratio (INR). The therapeutic value of the INR is typically between 2.5 and 3.5. Adequacy of anticoagulation with UFH is measured with the partial thromboplastin time (PTT), which should be between 50 and 90 seconds. When LMWH is used to bridge the patient, the adequacy of anticoagulation is typically not measured, although an assay for Factor Xa can be used to measure anticoagulation for both UFH and LMWH. The half-life of UFH is 1 to 2 hours, which necessitates cessation 6 hours before a surgical procedure. LMWH has a longer half-life, which requires cessation the night before the procedure.

Antibiotic prophylaxis is required in patients with prosthetic heart valves or those with a history of endocarditis,

unrepaired cyanotic congenital heart disease, or repaired congenital heart disease during the first 6 postoperative months. The antibiotic of choice is amoxicillin 2 g or clindamycin 600 mg (if penicillin allergic) orally 1 hour before the procedure. When antibiotics are to be given parenterally, ampicillin 2 g or clindamycin 600 mg (if penicillin allergic) should be administered within 1 hour of the procedure start time.

Arrhythmias

Patients with diagnosed or occult arrhythmia present a management challenge to the surgeon and anesthesiologist. Arrhythmias typically compromise cardiac output to some degree. The distinction between supraventricular and ventricular arrhythmias must be made early. The latter requires prompt attention and due concern. Anesthesia and surgery are capable of unmasking occult arrhythmias through stress-mediated physiologic change, proarrhythmic drugs, and hypoxia. The evaluation of patients with arrhythmias includes an ECG. When the arrhythmia is intermittent in nature, the ECG may fail to identify the rhythm. In this situation, it may be prudent to obtain a 24-hour continuous monitoring record that can be analyzed to identify the abnormal rhythm.

Common supraventricular arrhythmias include supraventricular tachycardia (SVT) (also known as paroxysmal atrial tachycardia), atrial flutter, and AF. SVT is benign but results in heart rates of 150 beats per minute (bpm) or greater in an adult. The net result may be a reduction in cardiac output and blood pressure. It is typically due to a reentrant conduction abnormality and may precipitate myocardial ischemia. SVT may be initiated by premature atrial contractions (PACs), which are otherwise considered very common. Beta blockers and calcium channel blockers are often used to reduce the frequency of SVT. Recalcitrant SVT may require electrophysiologic ablation of the reentrant pathway.[7] Cardiac stimulants such as alcohol and coffee should be limited. Exogenous and endogenous epinephrine may also precipitate this arrhythmia. When SVT develops in the perioperative setting, it demands treatment. Carotid massage may be used in patients without carotid bruits or stenosis. This results in stimulation of the carotid sinus with an increase in vagal tone. In addition, a Valsalva maneuver can be performed, which also acts through vagal stimulation. Failure to correct the SVT in a timely fashion or the development of angina or unstable vital signs demands more aggressive treatment with intravenous adenosine or electrical cardioversion in a monitored setting in which acute cardiac life support measures can be instigated.

Atrial flutter is characterized by an atrial rate approaching 300 bpm. There is often a rapid ventricular response (RVR) depending on how many of the atrial contractions are conducted through to the atrioventricular (AV) node. This often presents as a 2:1 or 3:1 block and a ventricular rate of 100 to 150 bpm. Mortality in patients with atrial flutter who undergo surgery is higher. Treatment often requires calcium channel blockers or amiodorone to control the RVR.

AF is an exceedingly common arrhythmia. The ventricular rate varies and is irregular giving rise to the term "irregularly irregular" to describe the pulse. Cardiac output is reduced owing to the lack of an atrial "kick," which often produces fatigue in older patients. Atrial enlargement, thyrotoxicosis, CAD, and stimulants such as alcohol, caffeine, cocaine, and nicotine can precipitate AF. AF predisposes the myocardium to mural thrombi and embolic events. Paroxysmal or chronic AF usually requires long-term anticoagulation with warfarin to reduce this risk. The treatment of AF usually requires chemical cardioversion with digitalis or amiodorone. The latter may also be used to prevent the recurrence of AF.[8] Calcium channel blockers or beta blockers may be used to help manage the RVR. Electrical cardioversion remains an effective treatment modality for acute-onset AF when chemical cardioversion is unsuccessful or vital signs dictate a more urgent restoration of sinus rhythm.

Ventricular arrhythmias are generally more concerning that atrial arrhythmias, given the potential for some to deteriorate into a nonperfusing rhythm. Premature ventricular contractions (PVCs) are common in healthy patients but may also be associated with fever, hypoxia, inhalational anesthetics, Swan-Ganz catheters, electrolyte disturbances, or myocardial ischemia. Isolated PVCs are often not pathologic, but three or more consecutive PVCs are considered ventricular tachycardia. The discovery of excessive PVC activity on a preoperative ECG mandates referral to a cardiologist for workup. The onset of PVCs in the perioperative period is more worrisome and may reflect myocardial ischemia, MI, or electrolyte abnormalities.[9,10]

Ventricular tachycardia may be perfusing or nonperfusing. When nonperfusing, the advanced cardiac life support (ACLS) protocol is adopted. This requires basic life support and early electrical cardioversion. Additional drugs that may be of benefit are amiodorone and magnesium.[11] Elective surgery should be deferred in all patients with ventricular dysrhythmias. Long-term therapy may involve the use of antiarrhythmic drugs, radiofrequency ablation, or implantable cardiac defibrillators.

Heart blocks arise from an abnormality in conduction from the sinoatrial node to the AV node. This slows cardiac conduction leading to PR intervals that are longer than 0.2 second. Heart blocks can be divided into four categories. *First-degree blocks* are associated with a prolonged PR interval but all atrial impulses are conducted through to the AV node with a corresponding ventricular contraction. First-degree blocks are usually not symptomatic and require no treatment. When the heart rate is excessively slow, they may require atropine. *Second-degree blocks* are divided into Mobitz I and Mobitz II variants. The Mobitz I variant involves a progressive increase in the PR interval over several beats until one atrial impulse fails to be conducted through the AV node, resulting in the lack of a ventricular contraction. This type of heart block is generally asymptomatic and requires no treatment. An excessively slow heart rate may again require atropine. The Mobitz II variant involves a

constant but prolonged PR interval with the loss of conduction through the AV node after several beats. This results in the loss of a ventricular contraction at a regular time period, usually every third or fourth beat. *Third-degree heart block* has no conduction through the AV node with atria and ventricles contracting independently. The heart rate is often in the 30s and this produces symptoms due to a reduction in cardiac output and blood pressure. Treatment of a second-degree heart block Mobitz II or third-degree heart block often requires external or internal pacing to maintain a satisfactory heart rate. Long-term management involves implantation of a pacemaker. The occurrence of any heart block in the perioperative period requires further investigation to determine causes, particularly electrolyte abnormalities, drug toxicity, and myocardial ischemia. The development of second-degree Mobitz II and third-degree heart block will most often require perioperative pacing.[12]

Hypertension

HTN is one of the most common diseases, and a large number of surgical patients will have HTN. HTN is defined as a systolic blood pressure in excess of 140 mmHg or a diastolic blood pressure in excess of 90 mmHg measured on two separate occasions (Table 2-5). It is one of the most common reasons for postponing elective surgery. Most HTN is of unknown etiology and is referred to as "essential hypertension." Secondary HTN is a result of known pathology including renal disease, Cushing's syndrome, Conn's syndrome, pheochromocytoma, hyperthyroidism, aortic regurgitation, and medication induced. Blood pressure can be classified based on the systolic and the diastolic blood pressures.

Hypertensive urgency requires referral to a primary care physician or internist as soon as possible and preferably within 24 hours to avoid the potential for hypertensive emergency and end-organ damage including CVA, acute renal failure, and myocardial ischemia. *Hypertensive emergency* is defined by any elevated blood pressure associated with end-organ damage including encephalopathy, heart failure, pulmonary edema, and renal failure. Hypertensive emergency requires immediate transport to an emergency room for blood pressure control. Elective surgery should be postponed for hypertensive urgency and emergency.[4,13]

Major risk factors for HTN include smoking, hyperlipidemia, DM, age older than 60 years, gender, and family history. If untreated, it commonly causes CAD, cardiomegaly, left ventricular dysfunction, CHF, renal disease, retinopathy, and CVA.

TABLE 2-5. **Hypertension**

Class	Systolic (mmHg)	Diastolic (mmHg)
Normal	<120	<80
Pre-HTN	120–139	80–89
Stage I HTN	140–159	90–99
Stage II HTN	160–179	100–109
Hypertensive urgency	>180	>110

HTN = hypertension.
Adapted from the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension* 2003;42:1206.

The diagnosis of HTN should not be made for the first time in the postoperative setting because many perioperative factors, including pain, may elevate blood pressure in an otherwise nonhypertensive patient. All patients with a history of HTN should be maintained on their usual blood pressure medication in the perioperative period. However, several antihypertensive medications reduce the patient's ability to compensate for anesthesia-induced hypotension. It may be necessary in some patients to reduce the antihypertensive medication dose on the morning of surgery. Beta blockers, calcium channel blockers, and clonidine should be continued in the perioperative period. Diuretics, ACEIs, and angiotensin-receptor blockers may be held the morning of surgery if anesthesia-induced hypotension is likely to be a problem. The choice of anesthetic agents for a general anesthesia may also be modified if there are concerns for blood pressure lability. Most of the inhalational anesthetics will reduce blood pressure to some degree. The intravenous agents such as propofol may also produce a similar blood pressure reduction. Induction with etomidate should be considered if there is a need to maintain blood pressure.

The signs and symptoms of HTN are subtle unless hypertensive emergency develops. An S_4 gallop is often present owing to the LVH that may also be evident on the ECG. The treatment of hypertensive emergency may include selective beta blockers such as labetolol which is an alpha 1, beta 1, and beta 2 receptor antagonist. It will reduce both the heart rate and peripheral resistance to decrease blood pressure. Esmolol is a selective beta 1 blocker that can also be used, but this requires a continuous intravenous infusion because the half-life of the drug is less than 5 minutes. ACEIs can also be used to reduce peripheral resistance, although only enalaprilat is available for intravenous administration. Hydralazine is a selective alpha 1 antagonist that can reduce peripheral resistance and lower blood pressure very quickly, but caution should be exercised in the elderly because it produces a reflex tachycardia that can exacerbate myocardial ischemia.

The treatment of patients with stage I and stage II HTN includes numerous classes of medications. Medication is often added over time and titrated to effect. Many patients will require multiple medications to control blood pressure. Weight loss, exercise, and reduced dietary sodium consumption are also important to help manage blood pressure. Patients who develop HTN in the perioperative setting or those with a history of controlled HTN who are difficult to maintain in a normotensive range may benefit from treatment. Unless significantly elevated, it is generally not ideal to diagnose HTN in the immediate perioperative setting because many physical and emotional factors may influence the blood pressure. Inadequate pain control is often responsible for an elevated blood pressure and is best addressed by increasing analgesia. Simply asking patients to rate their pain on a visual analogue scale will often identify whether pain is a significant issue and a likely etiology of the HTN. When

treatment is thought to be necessary, a stepwise approach is required and, although most patients will have essential HTN, consideration of secondary causes is warranted. A diuretic such as hydrochlorothiazide is an excellent first-choice medication. It results in potassium wasting and elevated serum calcium so electrolytes should be monitored in the long term. A selective beta 1 blocker such as metoprolol is also a good initial or subsequent choice. This is particularly useful in patients with a history of MI. An ACEI such as captopril is a reasonable third choice. Once an appropriate dose is found, it can be changed to another longer-acting ACEI such as lisinopril or ramipril, which require only once-a-day dosing. ACEIs are ideal for patients with DM owing to the renal protection that these drugs provide. Angiotensin-receptor blockers and calcium channel blockers are additional choices for medications.

Automatic Implantable Cardioverter Defibrillators and Pacemakers

A growing number of patients who present for surgery will have an automatic implantable cardioverter-defibrillator (AICD) or cardiac pacemaker. These devices are able to monitor cardiac rhythm, pace, and defibrillate. Some devices have the ability to sense oxygen saturation, right ventricular pressures, temperature, and body movement. Patients with an AICD or pacemaker who require surgery will have a cardiac history that should be determined. An ECG will indicate whether the AICD or pacemaker is pacing the myocardium as evident by the pacing spike and subsequent QRS complex. The decision to reset the AICD or pacemaker immediately before the surgical procedure should be made only in consultation with the cardiologist and anesthesiologist. This requires a magnetic key and knowledge of exactly which device (brand and model number) has been implanted. Defibrillators should be turned off to prevent accidental discharge or damage to the device. The AICD can have the defibrillator turned off and the pacer left on for surgery. Monopolar electrocautery should be avoided in all patients with an AICD or pacemaker. The current generated can damage an AICD or pacemaker even when turned off and it will be sensed by the device if the device is left on, which could result in inappropriate activity. Bipolar electrocautery can also affect the AICD, although this is less likely. If electrocautery was used during surgery, the device should be assessed immediately upon completion of the procedure to ensure that the device is functioning normally. All patients with an AICD should have an external means of pacing and defibrillating available throughout the surgery in case a cardiac dysrhythmia develops.

RESPIRATORY DISEASE

All patients require a thorough history and physical examination to identify respiratory disease. Furthermore, the very nature of oral and maxillofacial surgery dictates that the potential for upper airway obstruction is a potential concern with many procedures. The functional status of the patient, as with most systemic diseases, provides significant insight into the disease severity and the potential for perioperative complications. Much can also be learned by simply watching the patient in terms of the respiratory rate and work of breathing. Smoking tobacco is relatively common and this should always be considered in all patients. Postoperative respiratory complications are more common in those who smoke.[14,15] The risk declines by 50% after only 8 weeks of smoking cessation, although patients who stop smoking for less than 8 weeks before surgery have a higher risk for complications than those who continue to smoke.[16] Cigarette smoke also results in elevated levels of carboxyhemoglobin (CoHb), which predisposes the patient to perioperative hypoxia. Pulse oximetry cannot detect CoHb and the displayed saturation level will, therefore, be incorrectly elevated. An arterial blood gas (ABG) is required to measure the CoHb. CoHb and nicotine both increase the potential for cardiac complications including ventricular fibrillation.[17] This additional risk can be eliminated with smoking cessation for 24 hours.

The need for additional preoperative pulmonary testing is dictated by the history, physical examination, functional status, smoking history, and age. The most common screening tool continues to be the chest x-ray, although the yield is relatively low unless diffuse parenchymal disease, pulmonary edema, pneumonia, or advanced chronic obstructive pulmonary disease (COPD) is present. Pulmonary peak flow can be easily measured with an office peak flowmeter. This can help quantitate obstructive airway disease and identify those patients who are having an exacerbation of preexisting disease. Pulmonary function testing (PFT) is the gold standard in evaluating pulmonary function. PFT measures lung volumes and lung dynamics including flow rates throughout the respiratory cycle. The forced expiratory volume in 1 second (FEV_1) and its relationship to the forced vital capacity (FVC) are particularly useful to quantitate disease severity and stratify patients. The measurement of the ABG is typically done in conjunction with the PFTs. The measurement of the partial pressures of oxygen and carbon dioxide, pH, and base excess allows the net effect of pulmonary disease on oxygenation to be seen.

Respiratory disease is a common diagnosis among surgical patients. The net result of most respiratory diseases is hypoxemia that, in broad terms, may develop from one of four mechanisms: hypoventilation, diffusion impairment, ventilation-perfusion mismatching, and shunting. *Hypoventilation* as defined by a decrease in respiratory rate or vital capacity that can be due to asthma, chronic COPD, obstructive sleep apnea, pneumonia, chest trauma, narcotics, neurologic disease, and idiopathic pulmonary fibrosis. *Diffusion impairment* may be due to idiopathic pulmonary fibrosis or acute respiratory distress syndrome. *Ventilation-perfusion mismatching* can be the result of many processes including PE, whereas *shunting* is often seen with pulmonary edema, pneumonia, atelectasis, cardiac septal defects, and chronic liver disease.

The perioperative management of a patient in respiratory distress with hypoxemia and increased work of breathing

requires intervention. The management of these patients entails a general initial approach while the cause for the hypoxemia is sought. Treatment involves the administration of oxygen using one of several methods, all of which increase the fractional concentration of inspired oxygen (FiO_2). The effect of varying the method of oxygen delivery on the FiO_2 should not be underestimated.

- Nasal cannula (1 L/min increases the FiO_2 by 4% to a maximum of 40% at 6 L/min).
- Rebreathing facemask (increases the FiO_2 to 60% at 10 L/min).
- Non-rebreathing facemask (increases the FiO_2 to 90% at 10 L/min).
- CPAP/BiPAP (continuous positive airway pressure/bilevel positive airway pressure) with positive pressure (increases the FiO_2 to 80%).
- Intubate/tracheostomy (increases the FiO_2 to 100%).

Several diseases should be considered in any patient with signs or symptoms of disordered respiration. The diagnosis, and ultimately the treatment, depends on a good history, physical examination, and appropriate studies.

Asthma

Asthma is classically defined by bronchial hyperresponsiveness and reversible bronchoconstriction due to smooth muscle contraction leading to reduced minute ventilation and hypoxemia. Signs and symptoms of asthma include episodic dyspnea, shortness of breath, nocturnal awakenings, limitation in activity, wheezing that is predominantly expiratory, cough, chest tightness, chest pain, and status asthmaticus. The likelihood of an asthma exacerbation in the perioperative period is dependent on many factors including the occurrence of pain, stress, aspiration during induction, and the presence of an unrecognized preoperative upper respiratory tract infection. The frequency, severity, duration, and response to therapy of recent asthma attacks are helpful predictors. Furthermore, a history of emergency room visits and the need for intubation should be reasons for concern. Requirements for multiple asthma medications including inhaled steroids should also raise the suspicion of severe disease. Patients with significant asthma as evident by these indicators are probably ASA class III patients and are probably not good candidates for office-based sedation or general anesthesia. Whereas well-controlled asthma poses minimal risk to patients, an exacerbation can occur in any asthmatic. Bronchospasm that heralds an asthma attack can occur rapidly and be difficult to manage even with positive-pressure ventilation and intubation. This is in part due to the combined effect of smooth muscle contraction, edema, and mucous plugging.

The diagnosis of asthma can be made with an appropriate history of asthma-like symptoms and PFT that indicates a reduction in FEV_1, normal vital capacity, reduced FEV_1/FVC (proportional to severity), and an increase in FEV_1 by 10% with bronchodilator treatment. Peak expiratory flow rate is best used to monitor asthma over time and identify potential exacerbations. A decline in the peak flow rate to less than 80% of baseline would suggest exacerbation and elective surgery should be postponed.

The goal of asthma management is to maintain all preoperative medications. The choice of medication is usually determined by classifying asthma as intermittent, mild persistent, moderate persistent, or severe persistent. The frequency and severity of symptoms, FEV_1, and peak flow are all used to categorize patients into one of these groups. The choice of medication is individualized but often progresses through an algorithm that includes:

- Short-acting beta agonist (SABA) such as albuterol.
- Inhaled corticosteroids such as fluticasone.
- Anticholinergic medications such as ipratropium or tiotropium.
- Long-acting beta agonists (LABAs) such as salmeterol or formoterol.
- Leukotriene receptor antagonists such as montelukast and zafirlukast.
- Cromolyn.
- Theophylline.

Perioperative exacerbations may occur despite adequate control with medication. The treatment of exacerbations often requires that a SABA be administered by metered-dose inhaler (MDI) or nebulizer every 2 hours as needed. Continuous nebulized treatment may be beneficial in children. Anticholinergic medication can also be administered by MDI or nebulizer every 6 hours as needed. A short course of oral or parenteral corticosteroid over a week may also be needed.[18]

Chronic Obstructive Pulmonary Disease

Emphysema and chronic bronchitis are the two major respiratory diseases within COPD. Emphysema is characterized by dilated and collapsed airways with alveolar destruction secondary to alpha 1 antitrypsin deficiency as a result of smoking or congenital deficiency. Chronic bronchitis is characterized by increased airway secretions and mucous production, often secondary to smoking. A patient with emphysema is often described as a "pink puffer" whereas a patient with chronic bronchitis is often described as a "blue bloater." The net result of both diseases is retention of carbon dioxide and eventually hypoxemia.

The symptoms and signs of COPD include chronic cough, sputum production, shortness of breath, decreased functional status, wheezing, and a barrel-shaped chest. As disease advances, patients may begin to expire with pursed lips, which increases intrathoracic pressure to help stent their airways open. The diagnosis of COPD can be made with an appropriate history supplemented with additional tests. PFTs reveal a reduction in FEV_1, no change in vital capacity, and a reduction in the FEV_1/FVC. ABGs are particularly helpful and reveal an increase in the partial pressure of carbon dioxide, a decrease in the partial pressure of oxygen, and respiratory alkalosis. The chest x-ray reveals a loss of lung markings, hyperinflation, and a flattened diaphragm.

COPD should be optimized before surgery. Smoking cessation should have occurred concurrently with the initial diagnosis of COPD, but if patients continue to smoke, complications will be higher. Smoking cessation should occur longer than 8 weeks before the planned surgical procedure. Elective surgery in the face of a COPD exacerbation is contraindicated. The perioperative management of patients with COPD aims to maintain the preoperative medication regime that is typically determined by classifying COPD as mild, moderate, severe, and very severe. All patients should be well hydrated and use humidified oxygen when needed to reduce the inspissation of secretions. Patients with mucopurulent sputum are best treated with a course of antibiotics such as azythromycin, trimethoprim/sulfamethoxyzole, or amoxicillin.[19] This is best completed before the planned surgery.

Complications in patients with COPD undergoing surgery can be perioperative or frequently postoperative. The perioperative concerns are typically anesthesia related. Nitrous oxide is best avoided due to its blood/gas coefficient and the potential to accumulate within the multiple bullae associated with COPD, which can rupture. Ventilator plateau pressures should be kept to a moderate level to further reduce this risk. Peak flow pressures will typically be increased to allow for a short inspiratory and prolonged expiratory time. The latter is crucial to reduce the likelihood of auto-PEEP (auto positive end-expiratory pressure). Despite maintaining normal preoperative medications, patients with COPD may experience postoperative exacerbations that require further treatment. This usually includes the administration of the following:

- Oxygen delivery with nasal cannula or facemask.
- SABA such as an albuterol MDI or nebulizer as needed.
- Anticholinergic medication such as an ipratropium MDI or nebulizer as needed.
- Corticosteroid burst given orally or intravenously over 5 days (prednisolone 1 mg/kg/day).
- The need to intubate patients who are doing poorly should be considered early.

The potential concern of administering oxygen to COPD patients who rely on a hypoxic respiratory drive is more theoretical than real. Any patient with COPD who is hypoxemic as a result of the disease should be treated aggressively with oxygen.

Pneumonia

Nosocomial pneumonia may occur in the postoperative period. It may be associated with the use of mechanical ventilation, at which time it is referred to as "ventilator-associated pneumonia." The development of pneumonia may be influenced by the use of perioperative antibiotics, the placement of nasogastric feeding tubes, and the propensity for aspiration. The most common causative organisms associated with nosocomial pneumonia are *Streptococcus pneumoniae, Staphylococcus aureus, Pseudomonas aeruginosa, Acinetobacter* species, and occasionally, anaerobic organisms. The net result is hypoxemia and sepsis. Signs and symptoms include dyspnea, shortness of breath, fever, chest pain, decreased breath sounds, and fremitus. The diagnosis should be considered in any patient on a ventilator who develops fever and the need for increased ventilatory support including inspired oxygen pressure and PEEP. The peak pressures are often also elevated secondary to reduced lung compliance. Additional findings are a chest x-ray or computed tomography (CT) scan indicating infiltration or consolidation of lung parenchyma and parapneumonic pleural effusion, a bronchoalveolar lavage with cultured organisms, positive blood culture, and an increased white blood cell count. The treatment of pneumonia includes the use of parenteral antibiotics, which may involve either monotherapy or combination therapy depending on the organisms identified. The need to convert patients from an endotracheal tube to a tracheotomy and the timing of this continue to be controversial.[20]

Pulmonary Embolus

More than 95% of PEs are from the deep veins of the legs. These travel to the lungs, leading to respiratory and cardiovascular compromise. Most emboli are clinically silent owing to their small size. Risk factors include smoking, contraceptives, pregnancy, advance age, malignancy, abdominal and orthopedic surgery, and hereditary coagulation disorders such as antithrombin III deficiency, Factor V Leiden, and protein C and S deficiency. Prevention with sequential calf compressors, thromboembolic deterrent stockings, and perioperative UFH or LMWH is important. All patients who undergo surgery should be considered at risk for PE. In addition to the risk factors listed previously, general anesthesia and surgery produce a prothrombotic state that is compounded by bed rest. Early ambulation and appropriate preventive measures afford the best opportunity to reduce the risk of PE.

Signs and symptoms include chest pain, shortness of breath, dyspnea, sinus tachycardia, hemoptysis, a swollen and painful leg, and pain on dorsiflexion of the foot (Homan's sign). The workup of possible PE may involve several investigations. An initial ABG with a large alveolar-arterial (A-a) gradient would be suspicious for PE. A CT scan of the chest with contrast (PE protocol) can identify subsegmental pulmonary arteries and emboli within them. This has largely replaced the ventilation-perfusion scanning. A duplex ultrasound of the leg veins should also be obtained. The sensitivity of this scan is highest for deep venous thrombosis (DVT) between the knee and the groin. The D-dimer assay is a relatively simple blood test that measures the level of fibrin degradation products. A positive D-dimer may be due to a multitude of conditions whereas a negative D-dimer virtually excludes DVT/PE, reflecting the high negative predictive value. An ECG most often reveals a nonspecific sinus tachycardia, although the classic pattern of an S wave in lead I and a Q wave and inverted T wave in lead III may be seen, reflecting right ventricular strain. Pulmonary angiography remains the gold standard but is infrequently used because it is invasive.

The treatment of PE may include an initial heparin bolus followed by a heparin drip. Alternatively, enoxaparin or

fondaparinaux can be administered subcutaneously. Long-term anticoagulation should also be simultaneously initiated with warfarin with a goal INR of 2.5 to 3. Massive PE leading to right ventricular strain may require chemical thrombolysis or surgical thrombectomy. Patients unable to be anticoagulated and patients with recurrent PE despite anticoagulation are candidates for inferior vena cava filters to prevent the development of PEs. The filters may be permanent or temporary and are themselves at risk for obstruction.

Atelectasis

Atelectasis is a common postoperative outcome characterized by the segmental collapse of lung alveoli. It leads to a progressive decline in lung compliance, impaired segmental ventilation, retained secretions, and a decrease in functional residual capacity. The signs and symptoms of atelectasis include decreased breath sounds, inspiratory crackles at the bases, increased work of breathing, and a low-grade fever. The diagnosis is based on the clinical picture and temporal relationship to the immediate postoperative period supported when indicated by chest x-ray. Treatment includes incentive spirometry and early ambulation.

Pulmonary Edema

Pulmonary edema can develop from cardiac and noncardiac causes. In the setting of normal cardiac function, it may be the result of fluid overload or it can follow extubation as a result of upper airway obstruction leading to negative-pressure pulmonary edema (NPPE). The latter is more common in young males. The net result is significant alveolar transudation and hypoxemia. Risk factors include obesity, obstructive sleep apnea, and maxillofacial surgical procedures. Signs and symptoms include increased work of breathing, dyspnea, shortness of breath, decreased breath sounds, and bilateral crackles. The diagnosis is based on the clinical suspicion, the development in the immediate postoperative period, and chest x-ray with diffuse infiltrates.

Treatment usually requires supplemental oxygen, support of the airway, and possible re-intubation and a short period of mechanical ventilation with PEEP. Diuretics may be used to facilitate fluid removal but are typically not required.

Airway

A preoperative assessment of the airway is mandatory, and the oral and maxillofacial surgeon is in a unique position to provide this evaluation. Complications related to establishing an airway and maintaining ventilation continue to be a source of significant patient morbidity and mortality. The oral and maxillofacial surgeon must carefully assess the potential for airway difficulty and must be in a position to appropriately prevent it when possible and manage it when it develops during sedation or general anesthesia.

A thorough evaluation of the patient's airway begins with the patient's history. A history of prior sedation or general anesthesia that presented with a difficult airway or complication should be sought. This may require obtaining anesthesia records to verify the nature of the difficulty and the management rendered. The physical examination should address several airway-related issues. The patient's weight and body mass index should be obtained. Obese individuals present potentially difficult airways and, due to their weight, have a reduced functional residual capacity, making early oxygen desaturation likely. Pregnancy will produce an identical picture. A decrease in cervical range of motion, a decrease in the maximum incisal opening, or the presence of a retrognathic mandible all portend to a potentially difficult airway. Additional patients that may present with a difficult airway include those with congenital conditions such as Pierre Robin, Treacher Collins, Goldenhar, Klippel-Feil, and Down syndromes; oropharyngeal infections; oropharyngeal tumors; radiotherapy patients; and patients with temporomandibular joint ankylosis. In all patients, the oral cavity and oropharynx should be examined and classified using the Mallampati scheme (Table 2-6).[21]

The difficulty of direct laryngoscopy and intubation correlates with advancing Mallampati class. In all patients undergoing sedation or general anesthesia, the Mallampati classification should be determined and used to predict the likelihood of a difficult airway. Supplemental measures to obtain and secure an airway should always be immediately available. For office-based procedures, the ability to support the airway with jaw lift, oral airway, nasal airway, and positive-pressure bag-valve-mask ventilation is mandatory. There will always be a small percentage of difficult airway patients who cannot be ventilated with these techniques. One or more additional techniques are needed. The surgeon should be proficient in the insertion of a laryngeal mask or endotracheal tube and the performance of a cricothyroidotomy. Patients who present with potentially difficult airways might be better candidates for hospital-based procedures. This has the advantage of a more controlled environment where the possibility of using indirect laryngoscopes, fiberoptic intubation, or emergent tracheostomy can be readily performed.[22,23] Furthermore, additional well-trained personnel are typically available and able to assist in an emergency. Ultimately, it is the responsibility of the surgeon and anesthesiologist to provide a secure airway and one or more back-up plans should the unexpected difficult airway be encountered.

TABLE 2-6. **Mallampati Classification**

Class	Anatomy
I	Visualized entire tonsillar pillars, uvula, soft palate, hard palate
II	Visualized upper tonsillar pillars, base of uvula, soft palate, hard palate
III	Visualized base of uvula, soft palate, hard palate
IV	Visualized hard palate

RENAL DISEASE

Renal disease and renal failure correlate directly with surgical morbidity and mortality.[24,25] HTN, DM, polycystic kidney disease, pyelonephritis, and autoimmune kidney disease can all result in diminished renal function. The normal glomerular filtration rate (GFR) is approximately 120 mL/min. Generally, the GFR needs to decrease to about 30 mL/min before a rise in serum creatinine or urea is seen. This represents a 75% loss in renal function before a significant increase in serum creatinine occurs. As renal disease progresses, the normal urine production declines from 0.5 to 1 mL/kg/hr until the patient eventually becomes oliguric or anuric. As the renal function decline continues, the patient will develop anemia, hypoalbuminemia, and electrolyte abnormalities of sodium, potassium, calcium, magnesium, and phosphorus. Furthermore, the ability to regulate acid-base homeostasis is impaired because the kidney can no longer effectively excrete hydrogen ions or conserve bicarbonate. The effective removal of metabolic waste products and urea is also impaired, resulting in azotemia and uremia, respectively. Renal disease also results in extrarenal disease including bone marrow suppression, thrombasthenia, pericardial effusion, and immunosuppression. The basic assessment of the patient with renal disease should include urine output, serum creatinine, serum electrolytes, bicarbonate, and hematocrit.

Urine output depends on the level of hydration and patient age. Typically, urine output approximates 1 mL/kg/hr in a young adult. Serum creatinine and urea provide indirect evidence of renal function by correlating only roughly with the GFR owing to active renal secretion of the former and active renal resorption of the latter. The GFR can be calculated by the Cockcroft-Gault formula, which requires the patient's age, sex, weight, and serum creatinine. More accurate methods to calculate GFR are available but these require 24-hour urine collection and the administration of a compound like inulin that is filtered but neither secreted nor reabsorbed. Urinalysis with microscopy is only useful in the initial diagnosis of renal disease when the presence of protein, red blood cells (RBCs), white blood cells, casts, and crystals can be identified. Urinalysis can be used to monitor renal disease progression in certain diseases such as DM.

Perioperative renal impairment can occur in a patient with preexisting renal disease or in otherwise healthy patients. Abnormalities in renal function should be classified as prerenal, renal, or postrenal. Prerenal causes of renal failure include hypovolemia, shock, hypotension, and heart failure. Renal causes of renal failure include glomerulonephritis (GN), acute tubular necrosis (ATN), interstitial nephritis (IN), and pyelonephritis. GN may be secondary to autoimmune disease, DM, human immunodeficiency virus (HIV), and amyloidosis. ATN may be secondary to renal ischemia, hypotension, rhabdomyolysis, drugs, and intravenous contrast media. IN is most often drug related. GN is readily identified by RBC casts in the urine, ATN by muddy brown casts, and IN by eosinophiluria. Postrenal causes of renal failure are usually obstructive in nature secondary to prostate hypertrophy, malignancy, and renal stones.

The initial diagnosis of acute renal failure may be made when urine output begins to decline, serum creatinine increases, and serum electrolyte abnormalities develop. The assessment for a patient with new-onset renal failure should include serum creatinine and urea, serum electrolytes, urinalysis with microscopy, and the fractional excretion of sodium (FENa). The latter helps to distinguish prerenal, renal, and postrenal causes of renal failure. Urine osmolality, urine sodium, and the blood urea nitrogen (BUN)–to–creatinine ratio can also be used to help distinguish among the three broad causes of renal failure. The FENa is calculated as follows:

$$\text{FENa} = \frac{[\text{Urine Na}]/[\text{Plasma Na}]}{[\text{Urine Cr}]/[\text{Plasma Cr}]}$$

A FENa less than 1% is consistent with prerenal causes, a FENa above 2% is consistent with renal causes, and a FENa above 4% is consistent with postrenal causes.

Perioperative management of the patient with renal disease should focus on volume status because patients may become fluid overloaded quickly. The use of intravenous fluid replacement should be done conservatively and without the addition of potassium. Clinically significant anemia should be managed acutely with packed cells, although the potential for significant fluid shifts exists. Chronic management usually mandates erythropoietin subcutaneously or intravenously. Long-term iron supplementation is also appropriate. Electrolyte abnormalities especially hyperkalemia can develop quickly and can result in severe cardiac arrhythmias. Hyperkalemia above 5.5 mEq/L mandates an ECG. A wide QRS complex, loss of P waves, and peaked T waves require urgent treatment with calcium gluconate to stabilize the cardiac membrane, kayexalate to bind gastrointestinal potassium, and dextrose/insulin to drive the intracellular movement of potassium. Dialysis may be needed when the hyperkalemia is excessively high or ventricular cardiac arrhythmias develop.

Many medications such as nonsteroidal anti-inflammatory drugs (NSAIDs) and some antibiotics require discontinuation if they are nephrotoxic. Many other medications need to be renally dosed if they are renally excreted. This typically means a reduced frequency of drug administration rather than a reduced dose and early consultation with a pharmacist or nephrologist is indicated.

Patients with end-stage renal disease require dialysis, which is typically performed three times a week as an outpatient. Patients with acute renal failure or those with end-stage renal disease who are inpatients may also be managed with continuous renal dialysis, which is typical with patients in the intensive care unit. Less frequently, patients with renal disease receive peritoneal dialysis. Surgery should be performed the day after dialysis so that electrolytes are optimized and fluid shifts minimized. All medications should be renally

dosed and the diet modified to ensure that it is consistent with the needs of the patient. This typically means a low-salt and low-protein diet.

LIVER DISEASE

Liver disease can have a significant impact in the perioperative period. A careful history will often reveal patients who are at risk for liver disease including those with alcoholism, substance abuse, prior transfusions, hepatitis, tattoos, and sexual promiscuity. Signs and symptoms may be conspicuous by their absence, although fatigue, pruritus, jaundice, palmar erythema, spider telangiectasia, splenomegaly, gynecomastia, testicular atrophy, and increased abdominal girth are all suggestive. Screening for liver disease in an otherwise healthy patient is of little benefit. The end stage of liver disease is cirrhosis, which results in portal hypertension with esophageal varices and hemorrhoids with the inherent potential for severe gastroesophageal bleeding. Perioperative morbidity and mortality are significantly higher in this patient population. Surgery is generally contraindicated in patients with acute or fulminant hepatitis, alcoholic hepatitis, or severe chronic hepatitis.

The liver is an important organ for the metabolism of many drugs, and liver disease can adversely affect drug metabolism. Liver disease also influences mechanical ventilation owing to the respiratory compromise it produces through hepatopulmonary syndrome, pleural effusions, and pulmonary HTN. Liver disease also predisposes patients to excessive bleeding owing to the impaired synthesis of the vitamin K–dependent Factors II, VII, IX, and X.

Patients with cirrhosis have typically been risk stratified according to the Child's classification, but the Model for End-Stage Liver Disease (MELD) system may be superior.[26] The MELD score is determined from an equation that includes the serum creatinine, serum bilirubin, and INR. Several modifications to this system have been suggested with the goal of more accurately predicting survival in patients with end-stage liver disease. An online calculator for determining a patient's MELD score can be found on the United Network for Organ Sharing website (www.unos.org/resources/meldpeldcalculator.asp). Patients with a MELD score less than 10 can undergo elective surgery, those with a MELD score of 10 to 15 may undergo elective surgery with caution, and those with a MELD score greater than 15 should not undergo elective surgery.

Liver function is easily assessed with simple blood tests including:

- Liver enzymes alanine aminotransferase, aspartate aminotransferase, and gamma-glutamyltranspeptidase.
- Bilirubin.
- Prothrombin time, INR, PTT.
- Serologic markers for hepatitis (A, B, or C) are of limited value unless in the setting of acute hepatitis or when establishing exposure or carrier status.

The perioperative goals of the patient with liver disease are multiple and depend to some degree on what the surgical procedure is and whether it is being performed under local anesthesia, sedation, or a general anesthesia. The elevated prothrombin time and INR can be corrected with vitamin K if time allows, although it may take 12 hours for the vitamin K–dependent factors to be synthesized in sufficient quantities to significantly decrease the INR. Alternatively, fresh frozen plasma (FFP) or activated Factor VII can be administered with an immediate improvement in the INR. Desmopressin (DDAVP) can also be administered to increase von Willebrand's factor (vWF), which is normally produced in endothelium, megakaryocytes, and the liver. This results in increased platelet function through enhanced platelet binding. Ascites should be managed with diuretics, beta blockers, and the removal of excessive peritoneal fluid. Intravenous fluid should be limited, as should total sodium intake. Electrolyte abnormalities such as hypokalemia must also be addressed with cautious supplementation. When present, elevated plasma ammonia levels should be managed with lactulose and dietary modification. Antibiotic prophylaxis to reduce the risk of peritoneal infection secondary to bacterial translocation should be considered. Fluid balance can remain a challenge, and careful management of blood volume, cardiac output, and urine production is required. The potential for severe and life-threatening gastroesophageal bleeding exists in all patients with liver disease and consideration to reducing this risk with histamine (H_2) antagonists is warranted.

BLOOD DISORDERS

Anemia

Patients may present with a history of anemia or it may be suspected based on nonspecific symptoms of fatigue and tiredness. Occasionally, patients may report blood loss in the stool or emesis or from menorrhagia. Anemia is an absolute or relative reduction in the hemoglobin concentration or hematocrit. The net result is hypoxemia due to a lack of oxygen-carrying capacity. Anemia may be due to a decrease in RBC production or an increase in RBC destruction. Patients with anemia require a diagnosis, and elective surgical procedures should be delayed if the anemia is significant. No absolute value can be used to decide what is significant, although when the anemia is symptomatic, this should be considered important. Females with a history of heavy menstrual bleeding and breakthrough bleeding have a reason to be anemic, and referral to a gynecologist is needed. Female patients with gastrointestinal losses and all males with anemia require appropriate workup to identify the source of bleeding.

Anemia is diagnosed with a complete blood count (CBC), which will reveal a reduced red cell number, reduced hemoglobin, and a reduced hematocrit. RBC indices will also be available as part of the CBC and include the mean corpuscular volume (MCV) and mean corpuscular hemoglobin concentration (MCHC). The initial approach to a patient with

anemia begins with determining whether it is a result of decreased production or increased destruction. The reticulocyte count can distinguish the two, with hypoproliferative disorders associated with a reduction in the reticulocyte count and anemia due to an increased red cell destruction would be associated with an increased reticulocyte count. Decreased RBC production can be divided into microcytic, normocytic, and macrocytic anemia. All have the common feature of decreased RBC production with a decreased reticulocyte count below 2%. Microcytic anemia is defined by an MCV less than 80 fL and may be the result of iron deficiency, thalassemia, sideroblastic anemia, or sickle cell disease. Additional tests are required to distinguish among the four entities. *Iron deficiency* is the most common and easily recognized by a decrease in serum iron and serum ferritin and an increase in total iron-binding capacity (TIBC). *Thalassemia* typically has a positive family history and diagnosis requires gel electrophoresis. *Sideroblastic anemia* is uncommon and associated with an increase in serum iron and ferritin, although diagnosis requires bone marrow biopsy. *Sickle cell trait/disease* also typically provides a positive family history and can be confirmed with a blood smear or genetic testing.

Normocytic anemia is defined by an MCV of 80 to 100 fL and may be the result of renal failure, chronic disease, or aplastic anemia. Macrocytic anemia is defined by an MCV greater than 100 fL and is most often due to vitamin B_{12} or folic acid deficiency. The serum vitamin B_{12}, folic acid, methylmalonic acid, and homocysteine can be used to distinguish the two.

Anemia due to increased RBC destruction is associated with an increased reticulocyte count above 2% and may be due to hereditary spherocytosis, autoimmune hemolysis, cold agglutinin disease, or mechanical destruction. A blood smear, direct and indirect Coomb's test can be used to identify the cause. The presence of schistocytes in the blood smear can be the result of disseminated intravascular coagulation (DIC), prosthetic heart valve, thrombotic thrombocytopenic purpura (TTP), or hemolytic-uremic syndrome (HUS).

The general workup of the anemic patient should include a CBC, peripheral smear, MCV, and reticulocyte count. Additional tests should be requested based on the initial results and may include serum iron, ferritin, TIBC, vitamin B_{12}, folate, Coomb's test (direct and indirect), methylmalonic acid, homocysteine, and a bone marrow biopsy.

Iron-deficiency anemia may be treated with iron supplements whereas megaloblastic anemias should be treated with vitamin B_{12} or folate depending on the deficiency. Dietary change should also be instigated in all cases except pernicious anemia because a lack of intrinsic factor renders dietary vitamin B_{12} ineffective. Megaloblastic anemia can be seen in vegetarians and those who avoid leafy green vegetables. Dietary inadequacy may also be seen in chronic alcoholics.

Sickle cell anemia deserves special mention because it is seen frequently in African Americans. Sickle cell trait is heterozygous and of minor clinical significance. Sickle cell disease is homozygous and clinically important. Deoxygenated RBCs polymerize and sickle. This leads to vaso-occlusive crises causing pain, cardiomyopathy, and infarcts of bone, lungs, and kidneys. Most patients undergo autosplenectomy owing to the progressive splenic infarction. Intravascular hemolysis also leads to gallstones in young patients. The treatment of sickle cell patients requires

- Hydration.
- Supplemental oxygen.
- Hydroxurea to decrease the incidence and severity of pain crises.
- Narcotic pain medication during crises.
- Aggressive treatment of infection.
- Vaccination as a result of the autosplenectomy.

Myeloproliferative Disease

This is a group of disorders characterized by a clonal proliferation of myeloid cells. All of these entities have the potential to transform into acute leukemias, although this occurs slowly. Myeloproliferative diseases may be suspected based on symptoms but may be identified from routine blood screening in otherwise asymptomatic patients. Myeloproliferative diseases can be classified according to the cell line responsible. Although bone marrow biopsy is the most sensitive diagnostic test, a CBC and smear is often suggestive. The four categories of myeloproliferative diseases include polycythemia vera, essential thrombocytosis, myelofibrosis, and chronic myelogenous leukemia. *Polycythemia vera* is an elevated RBC count and may present with headache, CVA, angina, pruritus, amaurosis, claudication, and splenomegaly as a result of the hyperviscosity syndrome. *Essential thrombocytosis* is an elevated platelet count and presents with symptoms similar to those of polycythemia vera. *Myelofibrosis* is defined by an increase in bone marrow fibrous tissue and is associated with fatigue, weight loss, extramedullary hemopoiesis, and massive splenomegaly. *Chronic myelogenous leukemia* is discussed in the next section.

Leukemia

The leukemias are a group of malignant diseases of lymphocytes or myeloid cells. They are mentioned for completeness. The leukemias are classified according to cell type: acute lymphoblastic leukemia, acute myelogenous leukemia, chronic myelogenous leukemia, and chronic lymphocytic leukemia. All leukemias tend to present with similar perioperative concerns. Anemia, thrombocytopenia, and ineffective leukocytosis present multiple surgical challenges. Infections are more common and should be treated aggressively. The medical management of leukemia causes further immunocompromise, increasing patient morbidity.

Lymphoma and Multiple Myeloma

Lymphoma is a malignancy of lymphoid tissue that resides predominantly within lymphoid tissue. It can be classified as

Hodgkin's lymphoma and non-Hodgkin's lymphoma (NHL) depending on the presence of Reed Sternberg cells, which are present in the former. Waldenström's macroglobulinemia is a low-grade form of NHL that secretes immunoglobulin leading to a hyperviscosity syndrome, peripheral neuropathy, and Raynaud's phenomenon.

Multiple myeloma (MM) is a malignant proliferation of plasma cells that produce a monoclonal antibody (M component). The disease progresses slowly but can lead to anemia, bone pain, hypercalcemia, renal disease, nephrotic syndrome, and hyperviscosity syndrome

The treatment of lymphoma and MM includes chemotherapy and occasionally radiotherapy. The additional use of bisphosphonates in MM to reduce lytic lesions and bone pain has greatly improved the quality of life but has resulted in the development of bisphosphonate-related osteonecrosis of the jaws (BRONJ) in a proportion of patients.

Thrombocytopenia

Thrombocytopenia is due to decreased platelet production, increased destruction, or splenic sequestration. The net result is bleeding episodes that occur spontaneously or with minimal trauma when the platelet count falls below 50,000 cells/mm^3. The causes of thrombocytopenia are multiple but the surgeon should be aware of idiopathic thrombocytopenic purpura (ITP), TTP, HUS, and DIC. ITP is an autoimmune disorder that can follow an upper respiratory infection and is treated with steroids or splenectomy. Although often self-limiting, persistent mild perioperative thrombocytopenia may require steroids to increase the platelet count depending on the number and the nature of the planned procedure. TTP and HUS are characterized by hemolytic anemia, thrombocytopenia, and renal failure, although the former is also associated with seizures. DIC may be seen in the perioperative period, particularly the multitrauma and septic patient. Patients are at risk for significant perioperative bleeding. All patients with thrombocytopenia should be optimized before any surgical procedure. This entails managing the underlying condition and increasing the platelet count, which may necessitate platelet transfusion. Patients with DIC should not be considered surgical candidates.

Coagulopathy

Abnormalities of platelets, the intrinsic clotting pathway, and the extrinsic clotting pathway are all causes of coagulopathy. A personal or family history of a bleeding diasthesis or a history of excessive bleeding with prior surgical procedures or extractions is a sensitive means of assessing potential bleeding problems. The propensity for bleeding is best assessed with a CBC to evaluate the platelet count; platelet aggregation tests to assess platelet function; and the prothrombin time (PT), INR, and PTT to evaluate the coagulation cascade. Routine screening in an asymptomatic patient without a history of bleeding is not recommended, however.[27–32]

Thrombasthenia may be due to inherited defects as seen with Bernard-Soulier thrombasthenia, Glanzmann's thrombasthenia, or von Willebrand's disease (vWD). The latter is a common autosomal disease associated with a decrease in vWF and factor VIII. There are several types of vWD that are classified depending on whether the vWF defect is quantitative or qualitative. When the defect is quantitative, treatment with the vasopressin analogue (DDAVP) will increase the vWF and Factor VIII level. The preoperative platelet count and the nature of the surgery will determine whether this increase is sufficient for the planned procedure. When the platelet count is particularly low or the defect qualitative, Factor VIII concentrate, recombinant vWF, or cryoprecipitate will be necessary. A hematologist should be involved early in the patient's management to optimize the treatment. Platelet defects may also be acquired. This is very common and the potential impact should not be underestimated. A recent history of NSAIDs, clopidogrel, ticlopidine, or platelet IIb/IIIa inhibitors should be sought. Many patients will fail to report the use of aspirin unless specifically asked. Lastly, patients with end-stage renal disease may present with uremia, which may reduce platelet function significantly.

The general aim with surgical patients is to achieve a platelet count above 100,000 cells/mm^3. In addition, platelet function may be abnormal and should be assessed and corrected if possible. The use of clopidigrel and ticlopidine is not usually problematic for minor surgery, but the potential for bleeding exists and this needs to be considered before any major surgery. In the case of expected major blood loss at surgery, consultation with the patient's internist and subsequent discontinuation of clopidigrel and ticlopidine is recommended 1 week before the procedure. Platelet replacement may be required for patients with either thrombocytopenia or thrombasthenia. It should be remembered that the development of autoantibodies to prior platelet transfusions may result in a less than predictable response to subsequent platelet transfusions.

Hemophilia A and B

Hemophilia A is an X-linked recessive inherited disorder characterized by a deficiency in Factor VIII. Males are affected, although females may be carriers and can have hemophilia if they are homozygous. Factor VIII is a key coagulation factor in the extrinsic cascade and any deficiency can be measured with the PTT. The Factor VIII level can also be measured to quantitate disease severity. Hemophilia A can be classified based on the Factor VIII level:

- Mild hemophilia with Factor VIII level of 5% to 25%.
- Moderate hemophilia with Factor VIII level 1% to 5%.
- Severe hemophilia with Factor VIII level lower than 1%.

The treatment of hemophilia A depends on the Factor VIII level and the nature of the proposed surgical procedure. Minor surgical procedures may require only modest

elevations in Factor VIII level to 50% of the normal value. Major surgical procedures usually dictate that the level be restored to a normal level. The most common treatment involves replacement with recombinant Factor VIII. DDAVP can be used in mild cases and results in a mild increase in Factor VIII level through endothelial cell release. Cryoprecipitate can also be used but carries the risk of disease transmission because it is obtained from pooled human donors. If antibodies have developed to prior Factor VIII replacement therapy, then activated Factor VII may be required. With a half-life of 12 hours, Factor VIII replacement needs to be given twice a day. Despite an improvement in the PTT or factor, local measures at the time of surgery are also of paramount importance in helping to reduce the risk of postoperative bleeding. Antifibrinolytics should also be considered in this patient population. Epsilon-aminocaproic acid (EACA) and tranexamic acid can be administered systemically or applied topically as a mouthwash/gargle and have been shown to reduce the number of postoperative bleeding episodes. A growing number of patients are maintained on home therapy and learn to self-administer Factor VIII when necessary or have a family member do so. These patients can have minor surgery performed on an ambulatory basis. They should be instructed in conjunction with their hematologist/hemophilia nurse to administer the appropriate number of units at home preoperatively and postoperatively.

Hemophilia B (Christmas disease) is less common that hemophilia A and is autosomal recessive in the mode of inheritance. It is due to a deficiency in Factor IX. The half-life of Factor IX is 18 hours and, accordingly, replacement is required every 18 hours. Distinguishing between hemophilia A and B can initially be difficult because they both present with an elevated PTT. A Factor IX assay is required to make the diagnosis of hemophilia B. Treatment is with Factor IX replacement, which should increase the Factor IX level and normalize the PTT.

Warfarin Therapy

Many patients present who are anticoagulated with warfarin. They may have a history of AF, prosthetic heart valve, peripheral vascular disease, DVT, or PE. The adequacy of treatment is measured by evaluating the PT or the INR. The actual therapeutic level required is determined more by the medical condition necessitating the anticoagulation. Warfarin is a potent inhibitor of the vitamin K–dependent proteins II, VII, IX, X, C, and S. Proteins C and S are involved in the fibrinolytic pathway and, as such, a deficiency of these proteins tends to promote coagulation. The type of surgery planned, the INR, and the underlying medical need for warfarin will dictate whether to discontinue it before surgery, although considerable controversy exists. For minor surgery, such as extractions and the surgical removal of teeth, maintaining warfarin with an INR of less than 3.0 or 3.5 seems appropriate, given the potential for significant embolic complications in those who discontinue warfarin. Local hemostatic measures during surgery are paramount and should be combined with postoperative tranexamic acid or EACA.[33]

Major surgery dictates a different approach. Several options exist and should be tailored to the patient. Early discussion and treatment planning with the patient's treating physician is needed before any decision making.

- Stop warfarin 4 days before the procedure and resume after surgery (if medically appropriate to discontinue).
- Stop warfarin 4 days before the procedure. Bridge with enoxaparin 1 mg/kg subcutaneously twice a day and resume warfarin after surgery. Hold enoxaparin the night before and the morning of the procedure and resume the evening of the procedure.
- Stop warfarin 4 days before the procedure. Bridge with UFH infusion and resume warfarin dose after surgery. Hold heparin 6 hours before the procedure and resume 6 hours after surgery.
- Use local hemostatic measures during surgery.
- Administer tranexamic acid or EACA orally or topically.

Active bleeding or the emergent need for surgery dictates a different approach. Warfarin can be reversed with the administration of vitamin K, although it typically takes 10 to 12 hours for the reversal to occur. It also has a short half-life and may need to be redosed every 6 hours. Acute bleeding necessitates immediate treatment and replacement of clotting factors with FFP.

Hypercoagulable Diseases

There are multiple inherited and acquired conditions that predispose patients to thrombosis. The acquired states include antithrombin III deficiency, protein C and S deficiency, Factor V Leiden deficiency, and Factor II mutation. Acquired states include prolonged immobilization, pregnancy, oral contraceptives, malignancy, smoking, nephritic syndrome, and systemic lupus erythematosus. Acquired causes should be eliminated whenever possible. Patients may be receiving warfarin, and this may influence the perioperative management. Additional perioperative steps that can reduce the risk for DVT and PE should be instituted.

- Encourage ambulation as soon as possible.
- Use sequential calf compressors.
- Use thromboembolic deterrent stockings (TEDS).
- Use UFH at 5000 units subcutaneously every 8 hours or enoxaparin 30 mg subcutaneously every 12 hours

DVT should be suspected if leg pain, leg swelling, lower extremity pitting edema, pain on dorsiflexion (Homan's sign), chest pain, shortness of breath, unexplained sinus tachycardia, or an elevated A-a oxygen gradient is noted. Appropriate workup should include a duplex ultrasound of the lower extremity, D-dimer assay, and spiral CT of the chest if PE is suspected. Appropriate management requires heparin anticoagulation and long-term warfarin treatment. Patients who cannot tolerate warfarin or who develop additional

episodes of DVT on warfarin sodium (Coumadin) may require an inferior vena cava filter.

ENDOCRINOLOGY

Diabetes Mellitus

DM affects a significant proportion of the population and is characterized by autoimmune destruction of the pancreatic islet cells or the development of insulin resistance. Elevated blood glucose level and the by-products of glucose metabolism lead to cardiovascular disease, cerebrovascular disease, nephropathy, neuropathy, and retinopathy. Elevated or decreased blood glucose levels may also lead to altered mental status and metabolic encephalopathy. Signs and symptoms of DM include polyphagia, polydipsia, polyuria, acanthosis nigricans, peripheral skin pigmentation/ulcers, peripheral neuropathy, decreased visual acuity, nonketotic hyperosmolar coma, diabetic ketoacidosis (DKA), altered mental status, and electrolyte abnormalities.

The diagnosis of DM can be established using a number of methods. A random blood glucose above 200 mg/dL is suggestive of DM. A fasting blood glucose between 100 and 125 mg/dL is considered to represent a prediabetic state. A fasting blood glucose above 126mg/dL is diagnostic of DM. If the random or fasting blood glucose levels are inconclusive, then the oral glucose tolerance test can be used. This requires the ingestion of 75 mg of glucose after an 8-hour fast. A post blood glucose is then measured at 2 hours with a blood glucose level of 140 to 199 mg/dL considered to represent impaired glucose tolerance and a blood glucose of greater than 200 mg/dL diagnostic of DM.

A significant number of patients with DM will require surgical procedures. For diabetic patients undergoing procedures under local anesthesia, no change in their medication regime is warranted, provided that they will resume a normal diet postoperatively. Diabetic patients undergoing a general anesthesia or intravenous sedation will be fasting and require a change to their normal regime. Management of the diabetic patient can be divided into the preoperative and postoperative periods. Furthermore, management depends on whether the patient is treated with insulin or oral hypoglycemic agents.

Preoperative

The general goal is to avoid excessive hyperglycemia and hypoglycemia. This generally represents maintaining the blood glucose between 80 and 140 mg/dL. The following guidelines for intravenous sedation or general anesthesia are considered ideal but may not be appropriate for all patients and all situations.

Surgery in Diabetic Patients Not Treated with Insulin

For all surgery:

- Hold any oral agents on the day of surgery.
- For patients with "fair" metabolic control (fasting blood glucose < 180 mg/dL), cover with regular or rapid-acting insulin such as lispro, aspart, glulisine as needed using the sliding scale.
- For patients with "poor" metabolic control (fasting blood glucose > 180 mg/dL), start continuous insulin infusion (CII) because the use of sliding-scale insulin (SSI) is unlikely to obtain adequate blood glucose control.

Surgery in Diabetic Patients Treated with Insulin

For minor surgery:

- Hold any oral agents (if treated with combination therapy) on the day of surgery.
- For patients with "fair" metabolic control, hold the short-acting insulin and give half the dose of intermediate-acting insulin (neutral protamine Hagedorn [NPH]) the morning of surgery.
- While NPO, infuse 5% dextrose in normal saline (D_5NS) plus KCl (10–20 mEq/L) at 100 mL/hr.
- Check blood glucose every 4 to 6 hours while NPO and supplement with short-acting insulin using the sliding scale (see later).
- Patients treated with basal (glargine) insulin should receive their usual basal insulin dose. Similarly, patients treated with CII therapy (insulin pump) should receive their usual basal infusion rate.
- Restart preadmission insulin therapy once food intake is tolerated.
- For patients with "poor" metabolic control (fasting blood glucose > 180 mg/dL), start CII because the use of SSI is unlikely to obtain adequate blood glucose control.

For major surgery:

- Hold any oral agents on the day of surgery.
- Start insulin infusion before surgery and continue during the perioperative period because the use of SSI is unlikely to obtain adequate blood glucose control.

The sliding scale for insulin administration is derived from individuals with relatively normal insulin sensitivity. Patients who respond poorly to the calculated insulin dose may have reduced insulin sensitivity and adjustment to the formula is needed or consideration of changing to a CII. SSI requires that blood glucose be measured every 6 hours. Regular insulin is then administered subcutaneously according to the formula:

$$\text{Units regular insulin} = \frac{\text{BSL} - 140}{40}$$

where BSL = blood sugar level.

Perioperative complications in patients with DM depend on the perioperative glucose level, extent of end-organ damage, and nature of the surgical procedure. Potential complications include wound infection, hypoglycemia, hyperglycemia, and DKA. Potential complications related to the extent of the end-organ damage also exist. The cardiovascular system poses the greatest risk in the perioperative period in terms of CAD and peripheral vascular disease. Diabetic sensory neuropathy may permit myocardial ischemia and silent MI to go

unrecognized.[34] Furthermore, autonomic dysfunction may delay gastric emptying and increase the risk of aspiration. Ensuring that patients with DM fast for a longer period before anesthesia can reduce this risk but also compounds the challenge of preventing immediate preoperative hypoglycemia.

The glucose control in the preoperative period can be estimated by measuring the hemoglobin A_{1c}. The latter measures the amount of glycosylated hemoglobin over the prior 3 months, which reflects the average glucose level over that time.

DKA can occur in insulin-dependent diabetics, usually as a result of inadequate insulin, infection, or drug use. It is associated with severe hyperglycemia and ketosis. There is a 1% mortality rate even when treated appropriately. The clinical manifestations include polyuria, polydipsia, dehydration, hypotension, nausea, emesis, abdominal pain, altered mental status, and Kussmaul's respiration. The diagnosis is supported by the presence of an anion gap metabolic acidosis, hyperglycemia, pseudohyponatremia, and ketoacidosis (acetoacetate and β-hydroxybutyrate) in serum and urine. The treatment of DKA requires aggressive intravenous hydration with normal saline, intravenous regular insulin as an initial bolus and infusion, potassium replacement, and bicarbonate when severe acidosis or cardiac instability is present. The latter is typically defined by a pH of less than 7. The fluid used for hydration should be changed to a dextrose-containing fluid such as $D_5{}^1/_2$NS when the glucose level reaches 250 mg/dL. DKA may take many hours to treat because the hourly reduction in the glucose level with this regime should not exceed approximately 100 mg/dL/hr.

Nonketotic hyperosmolar hyperglycemic coma can occur in older non–insulin-dependent diabetics. Precipitating factors are the same as for DKA. The mortality rate is higher than DKA owing in part to the older patients. The key clinical features are severe dehydration and altered mental status. Significant hyperglycemia and an increase in serum osmolality (>350 mOsm/L) are typical. The treatment includes aggressive rehydration with normal saline. This should be accompanied by the administration of regular insulin as a bolus and infusion.

Thyroid

Surgical patients may present with a history of hypothyroidism or hyperthyroidism. Hypothyroidism is typically characterized by the progressive destruction of thyroid tissue. It occurs in Hashimoto's thyroiditis in which an autoimmune lymphocytic infiltrate develops with antithyroid peroxidase antibodies. Subacute thyroiditis is also autoimmune and often follows a flulike illness and presents with jaw pain but is usually self-limiting and resolves in a few months. Hypothyroidism may also be iatrogenic, resulting from the medical treatment of hyperthyroidism. The signs and symptoms of hypothyroidism include fatigue, weight gain, cold intolerance, constipation, facial edema, delayed deep tendon reflexes, and altered mental status. The diagnosis is usually established by observing a decreased free thyroxin (T_4) and elevated thyroid-stimulating hormone (TSH) level. Mild hypothyroidism is not a contraindication to surgery.[35] The treatment of hypothyroidism requires levothyroxine. The adequacy of treatment can be measured through TSH levels, which should approach normal. Myxedema is the only emergent hypothyroid condition that can develop as a result of infection, surgery, medications, or any other stressful event. The diagnosis of acute myxedema is based on a history of hypothyroidism and the development of altered mental status, seizures, coma, or hypotension. Treatment requires immediate intravenous levothyroxine and corticosteroids. Any patient who presents with acute myxedema preoperatively requires postponement of the surgery until appropriate treatment has been rendered and the patient made euthyroid.[36]

Hyperthyroidism is most often due to Graves' disease, an autoimmune disease characterized by the presence of thyroid-stimulating antibodies. There is the potential for thyroid storm with fever, cardiac arrhythmias, high-output cardiac failure, coma, and death. The signs and symptoms of hyperthyroidism include tachycardia, anxiety, tremors, heat intolerance, weight loss, AF, diarrhea, elevated systolic blood pressure, exophthalmos, and pretibial myxedema. The diagnosis can be established by the presence of an increased free T_4, decreased TSH, and the presence of thyroid-stimulating antibodies. The treatment of hyperthyroidism may require several approaches, although the development of thyroid storm mandates a rapid and aggressive approach. Many patients with hyperthyroidism will have been treated with radioactive iodine or thyroidectomy, which should render them euthyroid or even hypothyroid.[37,38] These patients provide no additional perioperative risk. Others may have mild hyperthyroidism that requires beta blockers and either propylthiouracil (PTU) or methamizole to reduce thyroxin secretion in the perioperative period. A significant concern for any patient with a history of hyperthyroidism is the potential for thyroid storm. It can develop quickly with fever, tachycardia, hypertension, cardiac failure, altered mental status, and death. Treatment requires a prompt diagnosis and the sequential administration of beta blockers, PTU or methamizole, and sodium iodide. One problem is that the signs and symptoms that present may be confused with malignant hyperthermia (MH), neuroleptic malignant syndrome, or pheochromocytoma. Additional treatment includes hyperventilation to help manage the hypercarbia secondary to the hypermetabolic state and rapid cooling to manage the increasing body temperature. Decreasing ambient temperature, cool intravenous fluids, and cooling blankets may also be needed. For either hypothyroidism or hyperthyroidism, elective surgery should be delayed until the treatment makes the patient euthyroid as determined by thyroid hormone levels. Emergent or urgent procedures should be undertaken without delay and the patients should be monitored closely. The potential need to supplement patients with thyroid hormone, corticosteroids, and cardiovascular support or the need to block the production and effects of excess thyroid hormone should be recognized early.

Adrenal

Patients with adrenal disease may present with excessive adrenal function or adrenal insufficiency. Cushing's syndrome is characterized by excessive plasma cortisol levels. It can be secondary to adrenal hyperplasia, pituitary adenoma, ectopic adrenocorticotropic hormone (ACTH) production, or exogenous corticosteroid administration. Patients present with truncal obesity, moon facies, abdominal striae, hirsutism, hyperglycemia, HTN, and purpura. The diagnosis is confirmed with a 24-hour urine free cortisol level, high dexamethasone suppression test, and abnormal ACTH level. Conn's syndrome is characterized by excessive aldosterone levels. This is usually secondary to adrenal hyperplasia or adenoma of the zona glomerulosa but can occur as a result of renal disease and increased serum renin. Patients present with HTN, hypernatremia, hypokalemia, hyperchloremia, and alkalosis. The diagnosis is confirmed with elevated serum aldosterone levels. The perioperative concerns relate primarily to fluid balance, electrolytes, and glucose level, all of which require appropriate correction as needed.

Adrenal insufficiency may be primary or secondary. Primary adrenal insufficiency is due to destruction of the adrenal glands from autoimmune disease, infection, or infarction and is referred to as *Addison's disease.* Secondary adrenal insufficiency is due to a lack of ACTH due to pituitary failure. Primary and secondary adrenal insufficiency can be distinguished based on the presence of hyperpigmentation, ACTH level, and the cosyntropin stimulation test. The inadequate levels of adrenal cortisol place the patient at risk for an acute adrenal crisis when stressed with surgery or any infection. Acute adrenal crisis is characterized by nausea, emesis, abdominal pain, fever, lethargy, severe hypotension, altered mental status, and electrolyte abnormalities. Patients with adrenal insufficiency are usually managed with prednisolone replacement therapy. The steroid administration, while providing adequate replacement for daily physiologic needs, is insufficient to manage the patient's requirements in the perioperative period. This requires a significant increase in the perioperative steroid dose. Occasionally, patients may also be receiving mineralocorticoid replacement (fludrocortisone) if the adrenal insufficiency has resulted in decreased aldosterone production. For minor procedures such as extractions, the patient should double the usual corticosteroid dose on the morning of the procedure. Blood pressure should be monitored during and after the procedure. Hypotension should be treated with intravenous hydrocortisone (100 mg) or an equipotent dose of another steroid. For major procedures, the patient should receive intravenous corticosteroids in the perioperative period. Doses should be equivalent to or greater than 300 mg of cortisol per day, which represents the normal physiologic endogenous steroid production in the face of major surgical stress. Hydrocortisone can be administered every 6 hours and the dose or frequency increased to maintain blood pressure. Alternatively, a more potent steroid with a longer half-life such as dexamethasone can be administered. Patients with primary adrenal insufficiency may also have fluid and electrolyte abnormalities from the relative lack of aldosterone. Judicious fluid replacement and management of hyponatremia and hyperkalemia are required.

Pituitary Disease

Diabetes insipidus (DI) can occur as a result of a decrease in pituitary antidiuretic hormone (ADH) production or as a result of renal insensitivity to ADH. These two disorders are referred to as *central* and *nephrogenic* DI, respectively. They present with severe polyuria, polydipsia, increased serum osmolality (>300 mmol), and mild hypernatremia. The diagnosis of DI can be made with the water deprivation test in which inappropriate urine production occurs in the face of restricted water intake. The continued urinary loss of free water results in the increased serum osmolality and hypernatremia. The distinction between central and nephrogenic DI is made by administering DDAVP. Central DI responds to DDAVP with an appropriate decrease in free water urinary loss and a corresponding correction of the serum osmolality and hypernatremia. The long-term management of central DI is nasally administered DDAVP, whereas nephrogenic DI is treated with sodium restriction and liberal water intake. The perioperative management of the surgical patient with DI revolves around fluid and electrolyte balance.

NEUROLOGIC DISEASE

Trauma

The spectrum of neurologic disease is wide with some patients presenting significant perioperative challenges. Acute neurologic deterioration in the setting of trauma is a special circumstance that also demands particular attention. The initial management of the trauma patient requires a systematic approach. The primary survey is designed to identify and correct life-threatening conditions and entails an assessment of the airway, breathing, circulation, and disability. The latter is a neurologic assessment that can be influenced by many factors including the airway, breathing, and circulation. The assessment of the neurologic status continues to be the use of the Glasgow Coma Scale (GCS), which helps determine severity, treatment, and prognosis of head-injured patients (Table 2-7).[39]

Injury may be classified as mild, moderate, or severe based on the score. Severe injury as determined by a GCS of 8 or less typically mandates intubation, although less severe injuries may also require a secure airway. The nature of the neurologic injury will depend in part on the mechanism of injury. Gunshot wounds are typically associated with penetrating and avulsive injuries, whereas motor vehicle crashes, sporting injuries, and domestic violence are more likely to result extradural, subdural, subarachnoid, and parenchymal hemorrhage or diffuse axonal damage. Early involvement of the neurosurgical team to manage and treat the injury is paramount. Generalized goals of treatment are to limit further damage by

TABLE 2-7. **Glasgow Coma Scale**

Action	Score
Eye Opening	
Spontaneously	4
To speech	3
To pain	2
No response	1
Verbal Response	
Orientated	5
Confused	4
Inappropriate words	3
Incomprehensible	2
No response	1
Motor Response	
Obeys commands	6
Localizes pain	5
Withdraws to pain	4
Flexes to pain	3
Extends to pain	2
No response	1

Adapted from Teasdale G, Jennett B. Assessment of coma and impaired consciousness: a practical scale. *Lancet* 1974;2:81–84.

controlling hemorrhage, minimizing cerebral edema, and maintaining cerebral perfusion. The role of the maxillofacial surgeon is limited, but the importance of securing the airway is critical and may necessitate intubation or an emergent airway.

Seizures

Seizures can occur in the perioperative period in healthy patients and those with a known seizure disorder such as epilepsy. Seizures can be classified as partial and generalized. Unremitting generalized seizure activity is termed *status epilepticus*. The potential causes of seizure are many. Epilepsy is a frequent cause of seizures, and a careful history regarding the frequency and severity of seizure activity should be sought. This will enable the surgeon to determine whether local anesthesia, sedation, or a hospital-based procedure is appropriate. Patients with well-controlled seizures can generally undergo anesthesia and surgery in the office or hospital. Irrespective of the procedure location, seizure medication should always be maintained. A multitude of medications are currently available to control seizures. Traditional medications such as phenytoin, valproic acid, carbamazepine, ethosuximide, and phenobarbital are used less frequently owing to the side effects including sedation. Bone marrow suppression with carbamazepine and liver enzyme inhibition with valproic acid may also occur. Newer drugs such as gabapentin, pregabalin, lamotrigine, topiramate, and tiagibine are becoming more popular owing to their efficacy and side effect profile.

Additional potential causes of seizure activity include anesthesia-related drugs, drug and alcohol withdrawal, degenerative central nervous system disease, head trauma, CVA, and metabolic derangements. Two particular anesthetic drugs that can reduce the seizure threshold are local anesthetics and meperidine. Local anesthetic overdose can suppress inhibitory neurons and increase seizure activity. Meperidine has an active metabolite, normeperidine, that can promote seizure activity particularly in the face of renal insufficiency. Benzodiazipine, barbiturate, and alcohol withdrawal can also promote seizures owing to loss in neuronal inhibition from these drugs. Head injury can also promote seizures from raised intracranial pressure, extravascular blood, and diffuse axonal injury. Metabolic derangements may be encountered in relatively healthy surgical patients, and this may also lead to seizures. Sodium, calcium, magnesium, glucose, urea, and ammonia are particularly important in this regard. Fluid and electrolyte balance is paramount, and all abnormalities should be corrected promptly.

New-onset seizures require treatment as well as a search for the cause. Signs and symptoms of seizure include altered mental status, confusion, loss of consciousness, and abnormal movement. The diagnosis of seizure may require an electroencephalogram (EEG). The initial treatment of perioperative seizures may require intravenous loading and maintenance with phenytoin. Fosphenytoin may be preferred owing to the more predictable bioavailability. Benzodiazepines may also be used to terminate a seizure but play no role in the long-term management of recurrent seizure activity. The transition to oral seizure medications is best managed in conjunction with neurology.

Cerebrovascular Accident

Atherosclerosis of the carotid and cerebral blood vessels places patients at risk for CVA and transient ischemic attacks (TIAs) in the perioperative period. Carotid atherosclerosis may be suspected based on the presence of a carotid bruit. A carotid duplex ultrasound is typically used to better define the severity of the stenosis. Occasionally, angiography, CT angiography, or magnetic resonance angiography may be used. Stenosis greater than 70% may benefit from endarterectomy before any elective surgical procedure.[40] CVA may be the result of thromboembolic events or hemorrhage whereas TIA is typically a result of the former. AF also increases the risk for CVA owing to atrial thrombosis and subsequent embolic events. Thrombosis of the lower extremity veins can also lead to CVA or TIA in the presence of atrial or ventricular septal defects. Patients with AF who are typically on warfarin should continue the medication in the perioperative period whenever possible. Patients with a history of TIA or thromboembolic CVA are often treated with aspirin or ticlopidine, which should be continued in the perioperative period if the nature of the planned surgical procedure allows. Surgical patients with a history of hemorrhagic CVA need to have their blood pressure well controlled. They may also have a history of HTN. Excessive elevations or reductions in blood pressure may also predispose to either CVA or TIA.

Myasthenia Gravis

Myasthenia gravis (MG) is an autoimmune disease characterized by the development of antibodies against the acetylcholine (Ach) receptor. It is often the result of thymus hyperplasia or a thymoma. Stressors including surgery, infections, and certain medications such as aminoglycosides, phenytoin, and meperidine may predispose patients to a myasthenic crisis. The signs and symptoms of a myasthenia crisis may include weakness, fatigue, ptosis, diplopia, dysarthria, dysphonia, dysphagia, respiratory distress, or failure to wean from the ventilator. Patients who present with a history of MG present challenges mostly related to their ventilatory status. This has an impact on their ability to undergo sedation, general anesthesia, and ventilator separation. Close collaboration with the neurologist managing the MG is strongly recommended. The diagnosis of an MG requires repetitive muscle activity with demonstrative progressive weakness, an electromyogram with decreased amplitude on repetitive nerve stimulation, and the edrophonium test, during which there is a rapid but transient improvement in symptoms due to increased Ach at the motor endplate. The diagnosis can also be supported by the presence of anti-Ach receptor antibody and a thymoma on CT or magnetic resonance imaging of the chest. The perioperative treatment of MG may involve anticholinesterase medication such as pyridostigmine, intravenous immunoglobulin (IVIG), and plasmapharesis.

OTHER CONDITIONS

Malignant Hyperthermia

MH is a serious, life-threatening condition characterized by a state of hypermetabolism leading to hyperthermia and massive rhabdomyolysis. It is an autosomal dominant genetic defect of one of several receptors within the sarcoplasmic reticulum. A mutation in the ryanodine receptor accounts for most cases, although several other receptors such as the CACNA 1S calcium channel receptor are involved in some patients. Certain anesthetic drugs including all volatile agents and succinylcholine can cause the rapid accumulation of calcium within the sarcoplasmic reticulum within skeletal muscle. The patient with a history suspicious of MH would benefit from a definitive diagnosis before any future anesthesia. This typically requires a skeletal muscle biopsy and the caffeine halothane contraction test, which has a sensitivity and specificity of 97% and 75%, respectively.[41] Genetic susceptibility testing is currently available for the ryanodine and CACNA 1S receptor mutations.[42] More often than not, the diagnosis is made only after the rapid development of MH.

One of the earliest features of developing MH is hypercarbia as seen through an increase in end-tidal carbon dioxide. This is followed by tachycardia, muscle rigidity, metabolic acidosis, and hyperthermia. Massive rhabdomyolysis with renal failure, electrolyte abnormalities including hyperkalemia, cardiovascular collapse, and death ensues unless treated aggressively. The treatment of MH mandates immediate cessation of the volatile anesthetic agent or succinylcholine, administration of dantrolene, cooling, and fluid resuscitation. Dantrolene is a powerful muscle relaxant that requires reconstitution with water and rapid administration. The dose of dantrolene needed is typically large and, accordingly, as many as 20 or 30 vials should be available.

Autoimmune Disease

There are a multitude of autoimmune diseases that are likely to affect surgical patients. These include rheumatoid arthritis, systemic lupus erythematosus, progressive systemic sclerosis, scleroderma, ankylosing spondylitis, mixed connective tissue disease, Sjögren's syndrome, polyarteritis nodosa, and polymyositis. The autoimmune disease as well as the medical treatment of the disease results in suppression of the immune system and increases the likelihood of perioperative complications. In addition, organ systems may be the target of the disease resulting in airway, pulmonary, cardiovascular, musculoskeletal, renal, hematologic, and neurologic difficulties. Wound healing may also be impaired in many of these patients and surgical infections are more common.

Three autoimmune diseases worth elaborating on are rheumatoid arthritis, systemic lupus erythematosus, and ankylosing spondylitis. Surgical patients with one of these diseases are likely to be encountered. Rheumatoid arthritis, despite the name, is a multisystem disease. In addition to the classic symmetrical involvement of the joints, nonarticular involvement can include the pericardium, pleura, lung, blood vessels, muscle, bone marrow, and skin. This can result in pericarditis, pleuritis, pneumonitis, vasculitis, myopathy, bone marrow suppression, and ulcers. Anemia of chronic disease is also often present. Rheumatoid factor may or may not be present in patients. Temporomandibular joint involvement may occur and result in limited opening, arthralgia, degenerative joint disease, and apertognathia. Cervical involvement may limit neck extension, further complicating anesthesia. The patient with limited opening may present anesthetic challenges, and planning for a difficult airway is essential. Many of the medications used to treat rheumatoid arthritis result in complications themselves. NSAIDs reduce platelet function; glucocorticoids result in adrenal insufficiency and Cushing's syndrome; and the slow-acting antirheumatic drugs (SAARDs), disease-modifying anti-rheumatic drugs (DMARDs), and anticytokine drugs are all immunosuppressive. SAARDS and DMARDS include hydroxychloroquine, sulfasalazine, methotrexate, and leflunomide. Anticytokine drugs continue to grow in number. Several categories of drugs exist including the interleukin 1 receptor antagonist anakinra, B cell–depleting agent rituximab, T cell costimulator blocking agent abatacept, and the tumor necrosis factor inhibitors etanercept, adalimumab, and inflixamab. Ideally, patients with rheumatoid arthritis should have their disease control maximized before an elective surgical procedure. However, cessation of anticytokine medication before

an elective procedure may be appropriate, given the potential for infection in this group, and this should be discussed with the rheumatologist. Cessation of medication typically requires 1 week before and after the surgical procedure.

Systemic lupus erythematosus is another autoimmune disease with multisystem involvement. Features may include anemia, thrombocytopenia, rash, polyarthritis, photosensivity, uveitis, coagulopathy, neurologic manifestations, psychosis, and renal disease. Perioperative concerns relate primarily to the anemia, renal disease, adrenal suppression, and risk for DVT. Care should be exercised in the use of radiographic contrast media and drugs may need to be renally dosed. Appropriate risk reduction for DVT mandates sequential calf compressors in the perioperative period, early mobilization, and heparin or enoxeparin prophylaxis. Additional steroids to compensate for adrenal suppression is likely, but this will depend on the nature of the surgical procedure and the usual daily steroid dose.

Ankylosing spondylitis is an autoimmune disease characterized by progressing spondylosis and immobility. Kyphosis develops insidiously and results in a rigid cervical spine that provides significant challenge for anesthesia. This is often combined with temporomandibular joint involvement and limited opening. Sedation is generally contraindicated in this group owing to the inability to secure an airway should it become necessary. General anesthesia is also problematic and often requires fiberoptic intubation or an awake tracheostomy. The kyphosis also reduces pulmonary compliance and functional residual capacity.

Immunodeficiencies

Deficiencies in cell-mediated immunity predispose patients to infection from bacteria, mycobacteria, viruses, fungi, and parasites. Cell-mediated immunity can be assessed by observation of the prototypical cell-mediated response of the delayed-type hypersensitivity reaction. This is classically seen with the Mantoux test, in which an intradermal injection of tuberculin is given. A more predictable method of evaluating cell-mediated immunity is by measuring the ratio of CD4 helper T cells to the CD8 suppressor cells. This provides quantitative information but might not necessarily correlate with normal function. One of the most common causes of reduced cell-mediated immunity is HIV. However, the advent of highly active antiretroviral treatment (HAART) has significantly reduced morbidity and mortality by reducing the viral load and increasing the CD4 count in most patients.

Deficiencies in humoral immunity also predispose patients to infection. Humoral immunity is best assessed by measuring the serum level of the five classes of immunoglobulin. Prior vaccination may also lead to the development of circulating antibody, which can be readily measured to determine the adequacy of the humoral system.

Deficiencies in the nonspecific immune pathway often present with abnormalities of neutrophil function. This predisposes patients to bacterial and fungal infections. Although the absolute neutrophil count can be measured, neutrophil function should also be evaluated. An absolute neutrophil count below 500 cells/mL requires patient isolation and prophylactic antibiotic and antifungal treatment. Deficiencies in complement also predispose patients to bacterial infections. Individual complement factors can be measured as can the entire complement cascade function through the CH50 assay.

Immunocompromised patients should have a thorough history to ascertain whether there is a history of recurrent infections, opportunistic infections, generalized lymphadenpathy, or weight loss. A thorough understanding of any identified autoimmune disease or immunodeficiency is necessary to further appreciate the effect on organ systems. The clinical examination should identify signs of immunodeficiency, which manifest as opportunistic infections. Of particular importance are oral and esophageal candidiasis, gingivitis and periodontitis, oral ulceration, diarrhea, and persistent cough and shortness of breath. Laboratory investigations may include a CBC with differential, CD4/CD8 ratio, serum immunoglobulins (IgG, IgM, IgE, IgD, and IgA), functional assays for neutrophil and complement function, and a chest x-ray

Immunocompromised patients need to be aggressively treated when infections occur. This includes early surgical intervention and broad-spectrum antibiotics. The use of perioperative and postoperative antibiotics should be considered in all but the simplest elective surgical procedures. Optimizing the immune status is also critical, although difficult to do in the short term. Treatment of the underlying cause of the immunodeficiency is paramount. This is particularly true for HIV. HAART has no role in the acute setting but should be implemented as soon as possible. Optimizing blood glucose control reduces patient morbidity and should be the goal in all patients. Consideration should also be given to reducing or eliminating perioperative steroids, which will further suppress the inflammatory response and immune system.

Patients who have lost their spleen through surgery or sickle cell disease are a special group. They are at risk for bacterial infections, particularly from bacteria with polysaccharide capsules such as *Pneumococcus*, *Meningococcus*, and *Hemophilus*. These patients should be vaccinated against these organisms. Early and aggressive treatment of infections is appropriate.

A significant number of patients take immunosuppressive agents to modulate autoimmune disease, treat malignancy, or suppress organ transplant rejection. A multitude of drugs are used, and these can result in neutropenia and lymphopenia. This places patients at risk for poor wound healing, infection, and overwhelming sepsis. Treatment of the underlying autoimmune disease should be optimized before any elective surgery. The judicious discontinuation of certain medications in the perioperative period should be discussed with the patient's rheumatologist or internal medicine specialist. Perioperative antibiotics should be considered in most patients, and any infections that do develop should be treated aggressively.

HIX affects more than 1 million Americans and results in the progressive destruction of CD4 helper T cells. This may lead to acquired immunodeficiency syndrome (AIDS) with opportunistic infections. Infection with HIV should be considered in any patient with signs and symptoms of opportunistic infections. The diagnosis of HIV infection requires a screening enzyme-linked immunosorbent assay (ELISA) test and a confirmatory Western blot. The CD4 count is used to assess disease progression and is often combined with polymerase chain reaction (PCR) to evaluate viral load and response to HAART. There are four broad drug categories in HAART. These include nucleoside/tide reverse transcriptase inhibitors, non-nucleoside reverse transcriptase inhibitors, protease inhibitors, and fusion inhibitors. The CD4 count should be known before all surgical procedures. This allows for risk stratification and appropriate prophylaxis against opportunistic infections. Every organ system can be affected by HIV/AIDS, and management of these patients can be challenging.

For a CD4 count below 200 cells/mL, *Pneumocystis jiroveci* pneumonia (formerly known as *Pneumocystis carinii* pneumonia; PCP) prophylaxis with trimethoprin/sulfamethoxazole, dapsone, atovaquone, or pentamadine is needed. As the CD4 count declines below 100 cells/mL, prophylaxis against toxoplasmosis becomes important with trimethoprin/sulfamethoxazole the first choice. When the CD4 count falls bellow 50 cells/mL, the potential for *Mycobacterium avium* complex (MAC) necessitates prophylaxis with a macrolide antibiotic. Patients with HIV who will require surgery may benefit from receiving prophylactic treatment in the perioperative period even if currently not receiving such medication.

Substance Abuse

The oral and maxillofacial surgery patient may present with a problem related to substance abuse. This may include alcohol, opioids, or elicit drugs. A thorough understanding of the substance abuse will allow appropriate social and surgical management of the patient. In general terms, a distinction should be made between patients who abuse drugs and those who are dependent on them. *Abuse* is best defined as an individual who continues to use a drug despite social, interpersonal, and legal consequences. *Drug dependence* has the same features of abuse as well as ongoing physical harm. Patients with dependence are at risk of withdrawal when drugs are discontinued. Withdrawal is invariably emotionally and physically painful but, in addition, can be life threatening depending on the drug involved.

Alcohol withdrawal is much more concerning than opioid withdrawal. The "CAGE" questionnaire is a simple means of screening for alcohol abuse and potential dependency.[43] The four questions that are asked are Have you ever felt the need to **c**ut down?, Have you ever felt **a**ngry at someone's criticism of your drinking?, Have you ever felt **g**uilty about your drinking?, and Have you ever had an **e**ye opener? Two or more positive answers correlates with a high likelihood of alcohol abuse. Alcohol withdrawal may be characterized by a state of neuronal excitability. This may lead to the severe syndrome of delirium tremens (DTs) with associated hallucinations, autonomic instability, hypertension, seizures, and coma.[44] Alcohol withdrawal can be avoided by continuing to provide alcohol in the perioperative period or by providing an alternative sedative medication. Benzodiazepines are the most appropriate medications to consider. Chlordiazepoxide and lorazepam are two common agents. Chlordiazepoxide can be given preemptively as a 4-day tapered oral regime beginning with 50 mg every 4 hours on day 1, 50 mg every 6 hours on day 2, 25 mg every 4 hours on day 3, and 25 mg every 6 hours on day 4. Most surgeons prefer to carefully evaluate potential patients and treat when the first signs of central nervous system excitation are seen. Early signs include restlessness, agitation, and tremulousness. Lorazepam can then be administered orally, intramuscularly, or intravenously every 6 to 8 hours. Additional areas of concern in patients with alcohol dependence include liver disease, anemia, and nutritional deficiency. Patients are often deficient in folate and thiamine, and these should be repleted.

Opioid abusers present different challenges, and the withdrawal syndrome is not life threatening. Opioid abusers are likely to require much larger doses of analgesic medications in the perioperative period. This is due to their induced liver enzymes and cross-tolerance. The potential for patients to divert medication is also problematic. Opioid abusers may exaggerate pain in the outpatient postoperative setting in an attempt to obtain more medication. However, patients who require chronic pain medication should receive medication. The drug, dose, frequency, duration, and side effects should be recorded. Patients should be seen monthly to evaluate their progress and identify potential side effects and abuse problems.

Obesity

Patients with obesity present many challenges in addition to the actual surgical procedure. Obesity is best measured by calculating the body mass index (BMI; Table 2-8). This is calculated by dividing the mass in kilograms by the square of the height in meters. The BMI allows patients to be risk stratified.[45]

TABLE 2-8. **Body Mass Index**

Category	BMI Range
Severely underweight	<16.5
Underweight	16.5–18.4
Normal	18.4–24.9
Overweight	25.0–30.0
Obese class I	30.1–35.0
Obese class II	35.1–40.0
Obese class III	>40.0

BMI = body mass index.
Adapted from World Health Organization. *BMI Classification.* Available at http://apps.who.int/bmi/index.jsp?introPage=intro_3.html

Surgical risks increase exponentially with the BMI once obesity is reached. The most common perioperative complications tend to be pulmonary. Obesity results in a restrictive pattern of lung disease due to decreased chest compliance and a superiorly displaced diaphragm, especially in the supine patient. Additional complications include a reduced functional residual capacity and the propensity to desaturate rapidly, airway obstruction, bronchospasm, atelectasis, PE, and pneumonia. Obese patients are also likely to suffer obstructive sleep apnea (OSA), which potentially increases the likelihood of co-morbid conditions such as HTN, coronary artery disease, and CVA. Office procedures under local anesthesia eliminate potential airway concerns. Intravenous sedation is more problematic and may not be appropriate for the obese and morbidly obese patient because the ability to secure an airway may be very challenging or not possible in the emergent setting. When sedation is chosen, the level should be light enough for the patient to maintain his or her own airway and respirations. Patients undergoing hospital-based procedures will still provide significant perioperative challenges, but these are more readily managed in this setting. The potential immediate postoperative need for CPAP in this group, particularly with a history of OSA, should not be underestimated. ASA recommends that any patient with OSA be carefully screened to determine whether they are candidates for ambulatory surgery. Factors that need to be considered include the severity of the OSA, coexisting disease, type of surgery, type of anesthesia, postoperative opioid needs, and the post-discharge observation.[46] A growing number of hospitals and anesthesia services are requiring patients with a proven diagnosis of OSA to be planned for overnight hospital admission with continuous pulse oximetry to monitor oxygen saturation.

Drug pharmacokinetics are also different in the obese patient, particularly for lipid-soluble drugs. The redistribution of lipid-soluble drugs to large body stores increases the volume of distribution, which significantly delays emergence from anesthesia.

Postoperatively, patients should be encouraged to rest with an elevated head of bed, ambulate as soon as possible, use incentive spirometry, and receive subcutaneous heparin for DVT prophylaxis.

Geriatric Patients

Elderly patients can provide significant challenges in the perioperative period. Those challenges depend on their general health status, co-morbidities, and age. Advancing age is itself not considered a predictor of complications, but medical conditions such as HTN and coronary artery disease are more common in this age group. Furthermore, physiologic changes ensure that the compensatory mechanisms are less able to accommodate anesthetic and surgical stresses. The medical history, current medications, and an assessment of the functional capacity are still reasonably sensitive tools to detect potential problems. Common complications that can occur in the elderly include atelectasis, pneumonia, MI, DVT, PE, CHF, and altered mental status.

Pulmonary function typically declines and is seen as a reduced lung compliance, vital capacity, and functional residual capacity. The A-a oxygen gradient also increases with age. There is roughly a 5-mmHg reduction in the arterial partial pressure of oxygen for each decade of life. A healthy 20-year-old would, therefore, have a partial pressure of oxygen of 100 mmHg on room air whereas a 70-year-old would have a partial pressure of 75 mmHg. Furthermore the elderly patient's response to hypoxia and hypercarbia is blunted. The potential to desturate rapidly or become apneic is high. Pulmonary vascular changes also ensure that ventilation-perfusion mismatching increases, further reducing pulmonary function.

Cardiac function also declines with a reduction in the maximal heart rate. This reduces a significant compensatory mechanism that would otherwise allow cardiac output to increase. The development of CAD is ubiquitous, and even without a prior history, MI, acute coronary syndrome, or angina, the presence of CAD should be suspected. HTN is also often present to some degree and is likely to have reduced ventricular compliance through LVH. This may be recognized as an additional S_4 on auscultation or with an ECG. This further reduces the heart's ability to increase cardiac output. Autonomic dysfunction, arteriosclerosis, and dystrophic calcification may also lead to postural hypotension, carotid stenosis, and aortic valve stenosis, respectively.

Renal function declines insidiously and, although there may be no clinical symptoms or signs of renal disease, the GFR is reduced. This is best assessed with the creatinine clearance. Serum creatinine is an indirect measure of creatinine clearance but may remain relatively normal owing to the reduced muscle mass. The most clinically important renal concerns in the elderly patient are related to fluid and electrolyte balance. Judicious fluid administration and appropriate monitoring of fluid balance are important. Urinary retention, particularly in males, may also be problematic secondary to benign prostatic hypertrophy or prostate cancer.

Elderly patients often fail to exhibit the classic signs of infection. Pyrexia and an elevated white cell count may be conspicuous by their absence. The signs may be subtle and deterioration rapid. Fatigue, malaise, and altered mental status may be early indicators of infection, electrolyte abnormality, MI, or PE. The ability to make the appropriate diagnosis relies on a strong index of suspicion.

The preoperative evaluation of the elderly patient requires a thorough history, physical examination, and ancillary studies as needed. The latter depends on the history, physical examination, nature of the planned procedure, and the type of anesthesia proposed. Additional studies may include a CBC, chemistries, ECG, chest x-ray, carotid or cardiac ultrasound, and PFTs. Other health care providers who are managing the patient's medical conditions may need to be contacted to further clarify the severity of systemic diseases and the patient's suitability for the planned anesthesia and surgical procedure.

Perioperative and postoperative medications should be chosen carefully and used judiciously to avoid unwanted responses. Dose reduction should be the rule for most medications.[47] Generally, the fewer the medications introduced, the less likely the patient is to develop side effects.

Pediatric Patients

The pediatric patient provides significant challenges just as does the geriatric patient. The physiology and anatomy of pediatric patients dictate a unique approach to their management. The airway presents challenges in that the mouth is small, the tongue large, the epiglottis large and floppy, and the vocal cords more anterior. Intubation can, therefore, be more difficult and the use of a Miller blade for direct laryngoscopy may be more appropriate. The functional residual capacity is reduced, and the predisposition to rapid and early desaturation is always present. Cardiac output is proportional to heart rate because the pediatric patient has little capacity to increase stroke volume. Blood volume is higher than in adults at approximately 80 mL/kg. Fluid administration and resuscitation need to be carefully administered and are weight based. Urine output is higher than adults and may approximate 2 to 3 mL/kg/hr in infants and toddlers. This declines with increasing age. Children also have large surface areas and can rapidly develop hypothermia. This mandates appropriate techniques to keep them warm including warming blankets and fluid warmers. Drugs should be administered based on weight and should be checked to ensure that they are appropriate for the pediatric patient. Certain NSAIDs such as aspirin should be avoided owing to the potential for developing Reye's syndrome, whereas certain antibiotics such as the tetracyclines and fluoroquinolones should also be avoided owing to their adsorption into mineralizing tissue such as teeth, bones, and cartilage.

Pregnancy

Pregnancy results in many maternal physiologic changes. Cardiac output, minute ventilation, and renal perfusion increase. There is a relative anemia due to dilution. Pulmonary function declines largely due to the reduction in functional residual capacity with the gravid uterus. As the pregnancy progresses, compression of the inferior vena cava while supine reduces cardiac filling with a reduction in cardiac output and blood pressure. Second- and third-trimester pregnancies, therefore, require a lateral decubitus position when supine to avoid this problem. The gravid uterus also increases intra-abdominal pressure, resulting in delayed gastric emptying and increased gastroesophageal reflux disease. The potential for aspiration is, therefore, increased. When general anesthesia is needed, this necessitates a rapid induction with cricoid pressure to help reduce the risk. Nausea and emesis, if present, typically improve as the pregnancy progresses but can further compound the risk of aspiration. The frequency of urination also increases overtime as the bladder is compressed by the enlarging uterus. Additional complications that may develop include preeclampsia, eclampsia, and the HELLP syndrome. *Preeclampsia* is characterized by HTN, proteinuria, headache, and edema. The onset of tonic-clonic seizures heralds *eclampsia*. The HELLP syndrome can accompany either preeclampsia or eclampsia and is characterized by hemolysis, elevated liver enzymes, and low platelets. These are serious and potentially life-threatening conditions that mandate early and aggressive intervention by the obstetrician and gynecologist.

Any drug administered to the mother should be considered capable of crossing the placenta and entering the fetal circulation. Different plasma protein concentrations in the fetus may further compound maternal drug administration by leading to fetal trapping of those drugs. Teratogenic drugs are most problematic in the first trimester during fetal organogenesis. The U.S. Food and Drug Administration (FDA) introduced a classification system for drugs and pregnancy that guides the choice of drugs (Table 2-9).

It would be ideal to use only category A drugs during pregnancy, but most drugs fall into category B or C as defined by the FDA. The decision should be individualized for the patient. Category D and X drugs are contraindicated in pregnancy. Even after childbirth, maternal drugs may result in potential harm to the infant through breast feeding. Many drugs are present in breast milk and precautions should be taken that may include choice of another drug that is not

TABLE 2-9. **Drug Classification in Pregnancy**

Class A	Adequate and well-controlled studies have failed to demonstrate a risk to the fetus in the first trimester of pregnancy.
Class B	Animal reproduction studies have failed to demonstrate a risk to the fetus and there are no adequate and well-controlled studies in pregnant women OR Animal studies have shown an adverse effect, but adequate and well-controlled studies in pregnant women have failed to demonstrate a risk to the fetus in any trimester.
Class C	Animal reproduction studies have shown an adverse effect on the fetus and there are no adequate and well-controlled studies in humans, but potential benefits may warrant use of the drug in pregnant women despite potential risks.
Class D	There is positive evidence of human fetal risk based on adverse reaction data from investigational or marketing experience or studies in humans, but potential benefits may warrant use of the drug in pregnant women despite potential risks.
Class X	Studies in animals or humans have demonstrated fetal abnormalities and/or there is positive evidence of human fetal risk based on adverse reaction data from investigational or marketing experience, and the risks involved in use of the drug in pregnant women clearly outweigh potential benefits.

present in breast milk, a safer drug, or avoidance of breast feeding for a period of time.

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CHAPTER 3

Pharmacology of Outpatient Anesthesia Medications

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Intravenous (IV) sedation has a long history of use in oral surgery practice. Oral surgeons have been the historical leaders in the development of office-based ambulatory anesthesia practice. The development of newer IV agents and techniques have led to the increased acceptance of these practices as being safe and cost-effective. Currently, the vast majority (>70%) of surgical procedures are performed on an ambulatory basis, and up to 25% of surgical procedures are performed with office-based sedation or general anesthesia.

Although it is neither possible nor the intention of the author to present the full scope of anesthetic medications including emergency medications in this chapter, we review the pharmacology of many agents used in office-based sedation and general anesthesia practice. Where applicable, the use of these agents in oral surgical practice is highlighted.

PHARMACODYNAMICS AND PHARMACOKINETICS

Pharmacodynamics

Pharmacodynamics is the study of the pharmacologic actions and clinical effects of a drug in the body.[1] The clinical response of most anesthetic and sedative medications derives from their actions in the central nervous system (CNS).

At a cellular level, the most frequent mechanism by which drugs exert their pharmacologic effects is through interactions with specific protein receptors embedded in cell membranes, which then initiate a specific set of intracellular actions. These protein receptors can be characterized as ion channels or transmembrane receptors. Ion channels allow the passage of specific ions, including chloride, potassium, sodium, and calcium, into or out of the cell. Alterations in the intracellular concentration of these ions initiate characteristic cellular effects such as depolarization of a cell membrane or movement of storage vesicles. Opening of ion channels may be triggered by either changes in membrane voltage or binding by a specific ligand. Voltage-sensitive ion channels open and close depending on cell membrane voltage, whereas a ligand-gated ion channel undergoes conformational changes when a drug or natural ligand binds to it, altering ion channel opening and closing. The γ-aminobutyric acid (GABA) receptor is an example of a ligand-gated chloride ion receptor.

Transmembrane receptors are also ligand-regulated and typically rely on second-messenger systems to carry out the pharmacodynamic effect. When a specific ligand binds to the extracellular portion of these transmembrane receptors, a conformational change in the domain of the receptor exposed toward the cytoplasm activates either a specific enzyme or a second-messenger system. Second-messenger systems, such as G proteins and cyclic adenosine monophosphate, are complex cascades of signaling proteins that, once triggered, will produce the intended effect. An example of an enzyme-activated system is insulin, which binds to its specific receptor, activating an intracellular enzyme called *tyrosine kinase*, resulting in increased glucose uptake. Muscarinic acetylcholine (ACh) receptors also use a second-messenger cascade involving intracellular calcium.

Some lipid-soluble drugs do not engage membrane receptors, but instead exert their pharmacodynamic effect intracellularly via receptors found in the cytoplasm. Hormones and steroid medications cross the cell membrane and bind to cytoplasmic receptors, which then alter cellular functions such as gene transcription. A small number of medications may also alter enzyme activity outside of cells, such as

anticholinesterase drugs that block the activity of acetylcholinesterase.

Drugs are commonly classified as either agonists or antagonists for a specific receptor. *Agonist* drugs function to exert the normal property associated with receptor activation. $GABA_A$ agonists, like benzodiazepines, bind at specialized sites on the GABA complex activating the receptor, allowing an influx of chloride, hyperpolarizing the cell, and reducing neuronal activity, thus promoting the normal activity associated with GABA activation. *Antagonist* drugs exert the opposite effect of the natural ligand or agonist drug activity. *Competitive antagonists* bind at the normal ligand-binding site but exert no pharmacologic effect. Instead, the antagonist "takes up space" at the binding site, thus blocking agonist drug activity. The higher the concentration of antagonist, the greater the blocking effect. Agonist activity returns once the antagonist concentration decreases or if additional agonist is administered to overcome the antagonist concentration. Flumazenil is an example of a competitive antagonist at the benzodiazepine receptor. The shorter duration of flumazenil than that of some benzodiazepines means that resedation may occur, especially after a small initial dose. Nondepolarizing neuromuscular blockers are another example of competitive antagonists used in anesthesia, in this case, for the ACh receptor. *Noncompetitive antagonists* do not bind at the ligand site but instead attach to a different location on the receptor, altering the configuration of the binding site and preventing normal ligand binding. Administration of an additional agonist does not affect noncompetitive antagonist activity, because they do not compete for the same binding site. Many pesticides are examples of noncompetitive antagonist agents.

Pharmacokinetics

Pharmacokinetics is the study of the factors that affect the plasma concentration of a drug in the body, encompassing the processes of absorption, distribution, metabolism, and elimination.[1] Commonly identified by the route of administration, such as per oral (PO), IV, intramuscular (IM), or inhalation, *absorption* describes the point of entry of the drug into the body. Orally administered agents undergo *first-pass metabolism;* PO medications are absorbed by the intestinal mucosa and carried via the portal circulation to the liver where they undergo partial metabolism before entrance into the central circulation. This process potentially reduces the plasma concentration of drug that reaches the effector site, such as the CNS. Because the degree of gastrointestinal absorption and first-pass metabolism is unpredictable, PO sedative drugs can have less reliable clinical effects. Most anesthetic agents used in oral surgical practice are delivered intravenously, intramuscularly, or by inhalation. In contrast to oral agents, these routes of administration do not undergo first-pass metabolism. Both IV and inhalation administration provide direct entry into the central circulation, reaching peak plasma concentration very quickly after drug administration. Inhalation pharmacokinetics are discussed in "Inhalation Anesthetics," later in this chapter.

Distribution describes the movement of the drug between body compartments. The main factors influencing distribution include the allocation of blood flow to a specific compartment, the concentration gradient of the drug between compartments, the chemical structure of the drug, and plasma protein binding of the drug. After administration, the majority of the drug initially redistributes to the vessel-rich compartments. This vessel-rich group includes the brain, heart, kidney, and liver, representing 10% of total body mass but 75% of cardiac output. Because the major site of anesthetic agent activity is the brain, early distribution to the CNS results in early anesthetic effects.

The transfer of the drug from the central circulation to the brain is also determined by the concentration gradient between the two compartments. A lower concentration in one compartment favors the transfer from a region of higher concentration. After initial IV administration, the initial drug concentration in the brain is low relative to the plasma concentration; thus, the drug will rapidly transfer into the brain based on this differential concentration gradient. As the plasma concentration falls by continued redistribution to other vessel-rich organs, and later to less vessel-rich organs such as skeletal muscle (~20% of cardiac output), anesthetic drug not bound to receptors in the brain will transfer back into the central circulation for further redistribution to other tissue sites. As the brain concentration of sedative agent falls, the clinical effects of sedation also decrease.

Characteristics of the drug itself affect its distribution throughout the body. Lipophilic drugs readily cross the blood-brain barrier and cellular membranes and generally exert their effects rapidly. Likewise, lipophilic drugs can quickly exit the CNS, shortening the duration of their effects. Hydrophilic medications either cross very slowly or must be transported by specific mechanisms. The size or molecular weight of the drug molecules influences movement across capillary walls; smaller molecules will cross more readily. A number of transport systems have been identified that cause some variability in drug onset and offset however.

The degree to which the drug binds to plasma proteins such as albumin and alpha 1 acid glycoprotein will affect the amount of free drug available to cross into the brain. Most sedative agents are highly plasma protein–bound. For example, initial doses of diazepam are 98% bound to plasma protein and unavailable to cross into the CNS. As the free drug plasma concentration decreases through further redistribution, and later *metabolism* and *elimination*, plasma protein–bound drug is released back into the plasma as free drug and is able to cross the blood-brain barrier. In this way, drug bound to plasma protein may be thought of as a reservoir of drug that may contribute to prolonged sedative effects. Once plasma protein–binding sites have been filled, an additional consequence is that further administration of small quantities of drug can have profound effects because the majority of the

additionally administered agent will be free drug that is able to cross the blood-brain barrier. Careful titration of IV agents, especially after initial administration and filling of protein binding sites, is important to avoid oversedation due to this mechanism. Hypoproteinemia secondary to advanced age, malnutrition, or severe liver failure can also dramatically increase the concentration of free drug, and dose reduction may be required.

As redistribution continues, a fraction of drug plasma concentration is delivered to the liver, the primary organ of drug metabolism, undergoing transformation from a lipid-soluble entity to a water-soluble form. There are four main pathways of hepatic metabolism: oxidation, reduction, hydrolysis, and conjugation. Phase I reactions include the first three pathways, converting the drug into a water-soluble metabolite or intermediate form. Phase II reactions involve most forms of conjugation, in which an additional group is added onto the metabolite in order to increase its polarity. Subsequent elimination via the kidney, the main excretory organ, requires hydrophilicity to avoid reabsorption of the excreted drug. Water-soluble drugs and metabolites are eliminated chiefly by the kidney, but also via the bile, lungs, skin, and other organs.

Phase I hepatic reactions, including the cytochrome P-450 (CYP-450) group of enzymes, which carry out the oxidation and reduction reactions, occur in the hepatic smooth endoplasmic reticulum (hepatic microsomal enzymes). The CYP-450 group of enzymes has been characterized into several isoforms (e.g., CYP-3A4, CYP-2D6, and CYP-1A2). The conjugation reaction of glucuronidation is also conducted by the hepatic microsomal enzymes. The hepatic microsomal enzymes are unique in that certain chemicals and drugs, including those used in anesthesia, can stimulate their activity. This is termed *enzyme induction* and generally requires chronic exposure of the drug to the enzyme system for at least several days or weeks. An isolated exposure to anesthetic agents is unlikely to induce hepatic enzyme activity. However, if the patient's daily medications induce hepatic enzymes, then increased metabolism of newly administered medications is possible. Induction is isoform-specific—a coadministered drug will be affected by enzyme induction only if both drugs are metabolized by the same enzyme system. Hepatic microsomal enzymes can also be inhibited by certain drugs, thus reducing metabolism of drugs by a specific enzyme system. For example, patients taking cimetidine for treatment of gastric ulcers may experience prolonged residual CNS effects from diazepam, because cimetidine inhibits the hepatic enzymes that normally metabolize diazepam. Various tables have been published that list drugs that are substrates, inducers, and inhibitors of the various cytochrome enzyme systems.

Nonhepatic forms of metabolism are important for certain anesthetic medications, and are useful in patients with significant liver or kidney disease. Drugs susceptible to Hofmann elimination spontaneously degrade at body pH and temperature. Ester hydrolysis by nonspecific and specific (e.g., pseudocholinesterase) esterases is also less dependent on renal and hepatic functions.

Redistribution, metabolism, and elimination reduce the plasma concentration of the drug, increasing the transfer of drug from tissue sites (e.g., brain) back into the central circulation for further redistribution, metabolism, and elimination. Different mathematical models involving these processes have been developed that describe the offset of activity of anesthetic agents. The fall of 50% of the plasma concentration of the drug secondary to redistribution is termed the *alpha half-life*. The removal of 50% of the drug from the body due to metabolism and/or elimination is termed the *beta half-life*, or elimination half-life. Offset of clinical effects and awakening from a bolus of an IV anesthetic agent is more dependent on redistribution of the drug away from the brain and is, therefore, better approximated by the alpha half-life than the beta half-life. In some cases, residual CNS effects can be predicted by a long elimination half-life. The beta half-life has more use for orally administered agents and particularly describes central compartment concentration in a one-compartment model.

The pharmacokinetics of a continuous infusion of IV anesthetic agents may be better described by the *context-sensitive half-time*. This value represents the time necessary for the plasma drug concentration to decrease by 50% after discontinuing a continuous infusion, depending on how long the anesthetic agent has been administered.[2] Figure 3-1 describes the context-sensitive half-time for a number of common anesthetic agents. Currently, computer-controlled pumps administer continuous infusions based on a specific amount of drug per time, but the newest infusion pumps can be programed to calculate and provide target plasma concentrations of an agent to a specified anesthetic or analgesic level. In the future, these pumps will likely be integrated with concurrent electroencephalogram consciousness monitoring to individualize anesthetic drug delivery.

SPECIFIC ANESTHETIC AGENTS

Benzodiazepines

Benzodiazepines are the most commonly used sedative and anxiolytic medications in oral surgery. Their relatively high margin of safety, in addition to the availability of an effective reversal agent (flumazenil), makes their use attractive during operator-anesthetist procedures in an outpatient setting.

Benzodiazepines are composed of a benzene and diazepine ring fused together.[3] Agonist agents contain a 5-aryl substitution that is not present on the antagonist reversal agent (Figure 3-2). This structure binds to inhibitory GABA receptors found throughout the brain, particularly in the cerebral cortex. Binding to the benzodiazepine receptor increases the frequency of pore opening in the chloride-gated channel, thus increasing inward chloride flow, hyperpolarizing cell membranes, and reducing neuronal transmission.

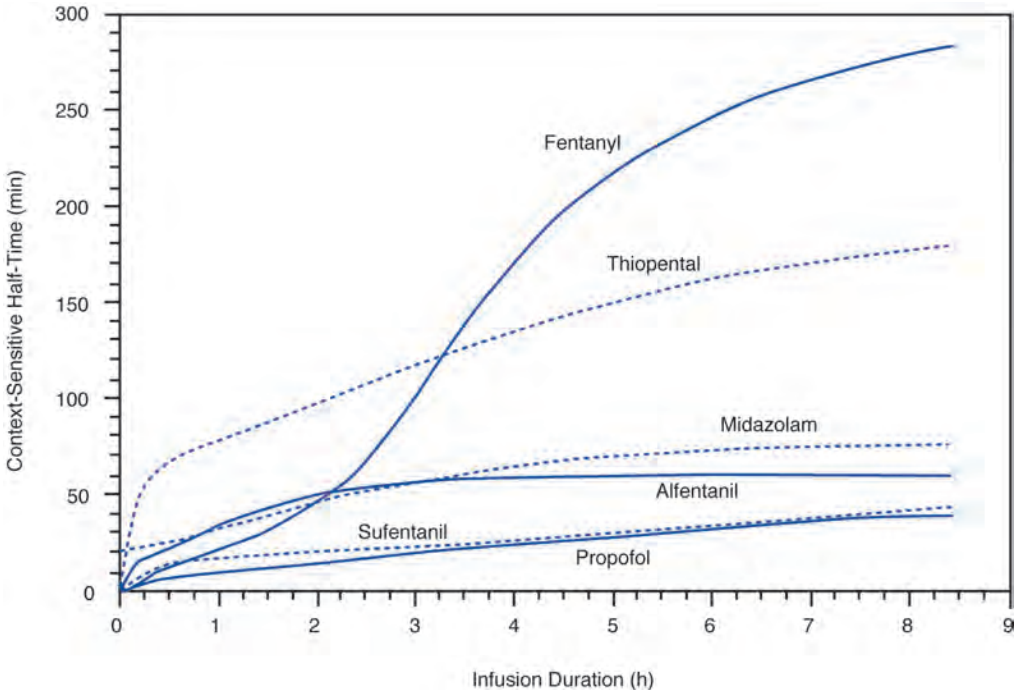


FIGURE 3-1. Context-sensitive half-time of a number of anesthetic agents. (Adapted from Hughes MA, Glass PSA, Jacobs JR. Context-sensitive half-time in multicompartment pharmacokinetic models for intravenous anesthetic drugs. Anesthesiology 1992;76:334–341.)

Characteristics shared by benzodiazepines include sedation, anxiolysis, anterograde amnesia, muscle-relaxing properties, and anticonvulsant activity. Indeed, any IV benzodiazepine agonist may be used to suppress acute seizure activity. These drugs do not produce analgesia.

Benzodiazepines are commonly used for preoperative sedation both immediately before the procedure and as a sleep adjunct the night before surgery. In clinical practice, they are also used for moderate sedation, and at higher doses can produce deep sedation and even general anesthesia.

In a nervous patient, anxiolysis from benzodiazepines can produce noticeable reduction in blood pressure and heart rate, but these medications have little direct effect on cardiovascular parameters. Given alone in slowly titrated doses, benzodiazepines also have minimal effects on ventilation. Large-bolus doses will, however, induce unconsciousness and apnea. In addition, even small doses, when given in combination with an opioid, can synergistically enhance opioid-induced respiratory depression.

Benzodiazepines are metabolized by hepatic enzymes into hydrophilic forms. These metabolites, some of which may be clinically active, are then excreted by the kidney in urine.

Serious adverse effects of properly titrated benzodiazepines are few, but paradoxical excitement, in which patients may become overly disinhibited and disoriented, is a possible complication. Flumazenil is useful in the reversal of paradoxical excitement and benzodiazepine-related respiratory depression.

Diazepam

Diazepam is lipid soluble and is carried in an organic solvent such as propylene glycol or a soybean oil emulsion.

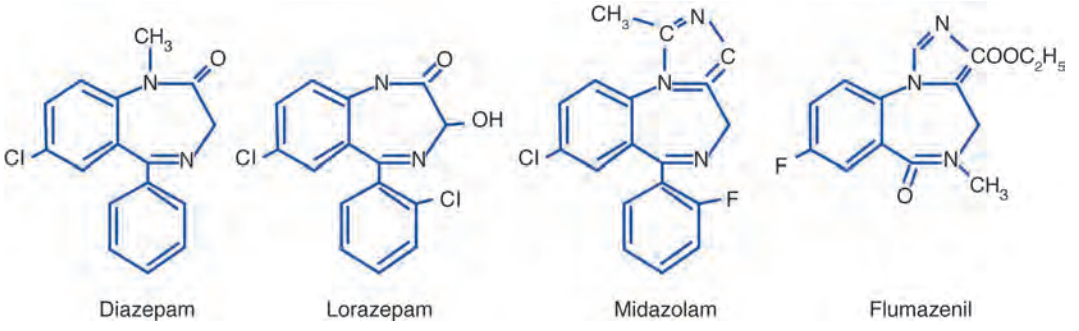


FIGURE 3-2. Chemical structure of benzodiazepine agonists and antagonists.

IV injection can be painful, although injecting into a larger vein or preadministration of lidocaine or an opioid can reduce discomfort. IM injection is painful and absorption can be unpredictable.

Diazepam is still used for IV moderate sedation, given in 2.5- to 5-mg increments every few minutes. Onset of sedation occurs in several minutes and recovery from clinical sedation by diazepam is similar to that of midazolam. However, the much longer elimination time of diazepam, and numerous active metabolites, may contribute to lingering sedative effects. Diazepam can also be given orally (5–10 mg) for preoperative anxiolysis and minimal sedation.

This highly lipid-soluble drug accumulates in fat tissues with slow reentry of very small quantities into the central circulation, leading to an elimination half-life of 24 to 96 hours. Diazepam is metabolized into two pharmacologically active metabolites, desmethyldiazepam and oxazepam, each with long elimination half-lives as well. The active metabolites and parent drug are partially eliminated in bile and can result in reemergence of sedation several hours after completion of the procedure owing to enterohepatic metabolism. Upon ingestion of a fat-rich meal, bile is released into the gut, and active drug components in the bile are reabsorbed by the intestinal mucosa and undergo first-pass metabolism. These still active drugs are then re-introduced into the central circulation and into the CNS, resulting in possible resedation.

Midazolam

Midazolam has an imidazole ring attached to its diazepine ring. The imidazole ring is open, rendering the compound water-soluble at pH less than 4, but the ring closes at physiologic pH producing the lipid-soluble benzodiazepine. Midazolam can, therefore, be delivered in an aqueous acidic solution, rather than propylene glycol, resulting in less pain on IV and IM injection.[4] It is two to three times as potent as diazepam, with a faster onset, much faster elimination, and shorter duration of lingering effects. Its active metabolites are not thought to produce significant sedative effects. Respiratory depression is more of a concern with midazolam than with diazepam after bolus IV administration.

Midazolam is currently more popular than diazepam for IV sedation for short oral surgical procedures. For conscious sedation, 0.05 to 0.15 mg/kg intravenously in divided doses is titrated to effect, typically given in 1- or 2-mg boluses every few minutes. Peak effect is seen in approximately 5 minutes. Dosage should be adjusted downward when given concurrently with other medications such as opioids or propofol. An IM injection of 0.15 to 0.2 mg/kg to a maximum of 10 to 15 mg depending on patient age is also possible. As an alternative, midazolam may be given orally at 0.5 mg/kg (maximum 15 mg) for preoperative sedation, such as before mask induction or IV start, or up to 1 mg/kg (maximum 15 mg) for procedural sedation, usually mixed into a flavored syrup or in a commercially available premixed product; this route may be better accepted by pediatric patients.[5] Clinical effect from PO administration will be seen after 15 to 20 minutes in the pediatric patient.

Lorazepam

Lorazepam is a long-acting benzodiazepine with a slow onset. Its use for PO and IV sedation is, therefore, limited but is an option for oral preoperative anxiolysis, particularly the night before surgery or for long operative appointments. Dosage for an adult is 0.05 mg/kg, not to exceed 4 mg total.

Triazolam

Triazolam is available only in an oral formulation as 0.125- and 0.25-mg tablets. This sleep adjunct can be used off-label for anxiolysis and moderate sedation at a dose of 0.25 to 0.5 mg for an adult. It is a very short-acting benzodiazepine and its effects are observed in 30 to 45 minutes, peaking at a little over an hour, with clinically effective sedation lasting from 60 to 90 minutes or longer. This drug has recently been highly promoted by dentists using multiple repetitive oral dosing. Although benzodiazepines are inherently safe drugs, patient selection, timing of additional doses, as well as knowledge of medical compromise and drug interactions along with good airway management skills are critical to safe clinical practice with oral sedation techniques that utlize multiple dosing regimens.

Flumazenil

Flumazenil is a highly specific competitive antagonist for the benzodiazepine receptor and is used as a reversal agent for benzodiazepine agonists.[6] It will reverse benzodiazepine sedation, excessive disinhibition, and the additive ventilatory depression related to benzodiazepines when combined with opioids. Flumazenil is electively adminstered at 0.2 mg intravenously initially, followed by 0.1 mg at 1-minute intervals as necessary, to a total of 1 mg. In emergency situations, 0.5 to 1 mg or more may be administered in a bolus dose. Reversal effects may take several minutes to manifest. The effect of flumazenil will last 30 to 60 minutes and may require redosing because agonist drug activity may outlast the reversal effects. Flumazenil should not be administered to epileptic patients using benzodiazepines for seizure control and should be used cautiously with other epileptic patients.

OPIOIDS

Opioid medications are used in oral surgery primarily for analgesia and to augment sedation or euphoria. It is important to note that opioid medications do not produce amnesia or classic sedation, nor do they induce loss of consciousness or sensation of touch at clinically relevant doses. Patients given opioid medications alone will retain awareness and memory. Instead, opioids are often used in combination with sedative-hypnotic medications such as benzodiazepines and

propofol to provide analgesia and augment the desired level of sedation/general anesthesia.

Whereas the term *opiate* refers to any drug derived from opium, *opioid* medications include all substances, natural and synthetic, that bind to the opioid receptors.[7] Common opioid medications are shown in Figure 3-3. Endogenous opioids such as endorphins and enkephalins, and administered opioid medications like morphine, bind to opioid receptors located in presynaptic and postsynaptic neurons throughout the CNS as well as in peripheral afferent nerves.

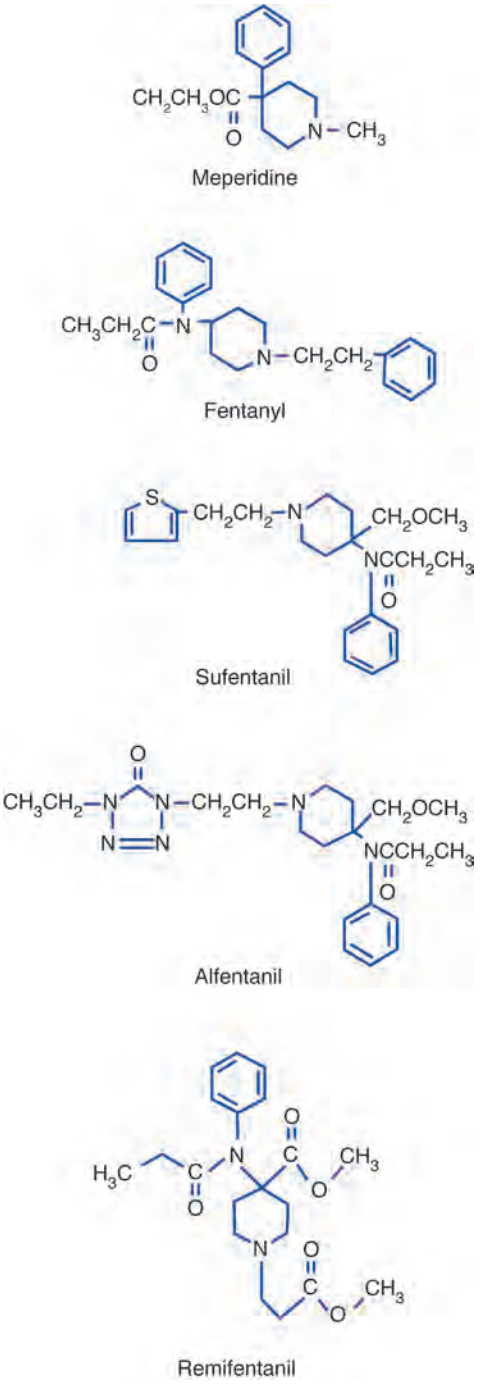


FIGURE 3-3. Chemical structure of synthetic opioid agonists.

Agonist activity at these receptors either modifies or decreases neuronal transmission of pain signals. Several subtypes of opioid receptors (e.g., μ, κ, δ) with differential effects have been identified. The μ and κ receptors are predominantly responsible for analgesia, and most clinically used opioids are agonists for the μ receptor. A subset of opioids, termed *agonist-antagonist* opioids, exhibit more κ receptor activity and can be antagonists at μ receptors. Thus, agonist-antagonist opioids are contraindicated for patients on long-term opioids, such as those using these agents for chronic pain or those on methadone maintenance for treatment of opioid substance abuse.

Respiratory depression is the most common and pronounced side effect of opioid agonists as used in anesthetic practice. This effect can be significantly exacerbated with concurrent administration of other medications such as benzodiazepines, barbiturates, propofol, and other opioids. Respiratory depression is dose dependent, resulting from a decrease in the respiratory response to arterial carbon dioxide (CO_2) levels in the brainstem respiratory centers. Decreased respiratory rate and arterial hypoxemia may result without supplemental oxygen (O_2) and appropriate monitoring (e.g., pulse oximetry). Opioids are often titrated incrementally to balance the analgesic effect against respiratory depression.

Bradycardia as a direct effect is more apparent with high doses of opioids and is due to centrally mediated vagal response. This effect is common with opioids such as morphine, fentanyl, and the synthetic derivatives, but less common with meperidine. A mild decrease or stabilization of the heart rate may be desirable in patients with cardiovascular disease.

Most opioids are metabolized by hepatic enzymes and excreted into the urine and bile. The exception is remifentanil, which is metabolized by plasma esterases.

Opioids suppress the cough reflex and are a common ingredient in cough medicines. These antitussive effects can be beneficial during sedation, especially when used in patients with hyperreactive airways (e.g., smokers). However, several opioids can cause the release of histamine and caution should be used when histamine-triggering opioids are administered to an asthmatic patient. Other manifestations of histamine release include a decrease in blood pressure secondary to vasodilation, and pruritus and erythema, especially at the site of injection.

Other adverse effects such as nausea and vomiting, constipation, urinary retention, and biliary tract spasm may increase patient discomfort postoperatively, particularly with repeated oral or neuraxial administration. These reactions are frequently misinterpreted by the patient and other health care providers as an "allergic" reaction.

Morphine

Morphine is the standard agent by which other opioids are compared. It has poor lipid solubility and, therefore, a slow onset. Majority peak effect after IV administration occurs in

15 to 20 minutes and the analgesic effect lasts approximately 3 to 4 hours. Because of its slow onset and longer duration of activity, it is commonly used in anesthesia practice for postoperative pain management rather than IV sedation. Morphine is normally given in 1- to 2-mg IV increments for postoperative analgesia.

Morphine has several notable characteristics. Histamine release from morphine can result in skin flushing and a decrease in blood pressure and may be of concern in an asthmatic patient. Morphine is metabolized by hepatic enzymes into two metabolites that are subsequently eliminated by the kidney. One of these metabolites, morphine-6-glucuronide, is more potent than morphine itself, and prolonged opioid effects in patients with renal failure can be significant.

Hydromorphone

Hydromorphone has a more rapid onset than morphine, 2 to 5 minutes, with a duration of action similar to that of morphine. It is eight times more potent than morphine, so 1.3 mg equals approximately 10 mg of morphine. It produces no histamine release and has no active metabolites. For these reasons, it is preferred by some anesthesiologists for postoperative pain control during emergence from anesthesia.

Meperidine

Meperidine is a synthetic opioid with a relatively rapid onset time and duration of action between 2 and 3 hours. It is used for both IV sedation and postoperative pain control. Meperidine is usually given in 12.5- to 25-mg IV increments titrated to effect.

The drug has several identifying characteristics. Like morphine, it also has an active metabolite, normeperidine, that has half the potency of meperidine but significant CNS toxicity. Postsedation delerium may occur, especially in the elderly. With repeated dosing, particularly in renally compromised patients, accumulation of normeperidine may lead to seizures. When mixed with monoamine oxidase inhibitors, meperidine administration may produce a dangerous excitatory hyperthermic reaction. Meperidine is also associated with the release of histamine; thus, appropriate precautions should be taken. Unlike the other opioids, it is not associated with bradycardia. Its structure resembles atropine and it possesses mild anticholinergic effects such as a mild increase in heart rate (offset by direct vagal stimulation) and xerostomia. Meperidine is commonly used to reduce shivering postoperatively, an action likely associated with partial agonist activity at the κ receptor.

Fentanyl

Fentanyl is a synthetic opioid, and its high lipid solubility leads to its high potency, rapid onset (1 min), and shorter duration of action (10–20 min). With such characteristics, fentanyl is a frequent choice for IV conscious sedation for short office-based procedures. It is typically given in 25- to 50-μg increments toward a total dose of approximately 1 to 2 μg/kg. It is also given during induction of general anesthesia, for both analgesia and attenuation of airway reflexes during intubation.

Fentanyl does not induce histamine release and is, therefore, not associated with vasodilatory or bronchospastic effects. However, at higher doses, it can cause more pronounced bradycardia than morphine. Fentanyl is a potent respiratory depressant. At high doses and with rapid bolus administration, fentanyl and other synthetic derivatives have been associated with chest wall and glottic rigidity, making ventilation impossible; there are reports that even lower doses (e.g., 100 μg) can trigger this centrally mediated effect. Fentanyl-associated chest wall rigidity is treated with either naloxone or succinylcholine (SCh), and positive-pressure O_2 and other resuscitation equipment should be immediately available. The incidence of fentanyl rigidity is reduced by a preceding dose of a benzodiazepine or other hypnotic drug.

Remifentanil, Sufentanil, and Alfentanil

Remifentanil, sufentanil, and alfentanil are synthetic fentanyl derivatives used primarily for analgesia during general anesthesia. Remifentanil, in particular, is associated with a rapid onset and extremely short duration of action, resulting in a significantly shorter recovery time. Metabolized by nonspecific plasma esterases, its clearance is very rapid and independent of both hepatic and renal functions. It has a very short context-sensitive half-time of 4 minutes with virtually no cumulative effect, even after hours of continuous infusion. These features make remifentanil ideal for use in a titratable continuous infusion. Of note is the fact that because the actions of this medication are so short-lived, postoperative pain will not be addressed by intraoperative remifentanil, and alternative pain control with another narcotic such as a nonsteroidal anti-inflammatory drug (NSAID) or local anesthesia should be considered toward the end of the procedure.

Remifentanil is used in a total IV infusion anesthetic technique to maintain anesthesia during dental/oral surgery, often in combination with propofol. For analgesia during general anesthesia, it is used at 0.25 to 1 μg/kg or 0.5 to 2 μg/kg/min. During sedation, the dose ranges from 0.05 to 0.10 μg/kg/min.

Remifentanil, like fentanyl, can cause chest wall rigidity and caution should be used during bolus administration. It is also a highly potent respiratory depressant; even at lower doses, apnea may be pronounced. If spontaneous ventilation is desired, the remifentanil infusion is usually titrated to maintain an adequate respiratory rate. None of these synthetic derivatives causes the release of histamine.

Sufentanil and alfentanil are shorter-acting agents than fentanyl but not as rapid in offset as remifentanil. These agents are commonly used as a continuous infusion adjunct for intubated general anesthesia during cardiac or prolonged surgery, particularly when residual opioid effects are desirable postoperatively. They are not as commonly used for office-based oral surgical anesthesia.

Nalbuphine

Nalbuphine is the most frequently used IV agonist-antagonist opioid. It has a relatively short onset and duration of action of 2 to 4 hours at sedation doses of 5 to 10 mg for the adult patient. Although nalbuphine and other agonist-antagonist opioids do possess a ceiling effect for respiratory depression at higher doses, at equianalgesic and clinically relevant sedation doses, the respiratory depressant effects are similar to μ agonist opioids. Nalbuphine does not release histamine.

Unlike all the other agents noted above which are U.S. Drug Enforcement Administration (DEA) schedule II controlled substances, nalbuphine is not currently a scheduled controlled substance and does not require state and federal documentation of use.

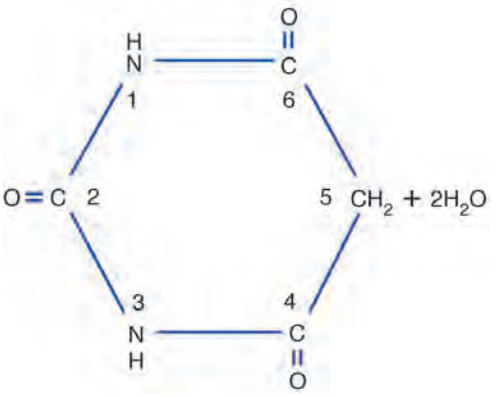


FIGURE 3-4. Chemical structure of propofol.

Naloxone

Naloxone is a pure opioid antagonist that is active at all opioid receptor subtypes. It will reverse both the ventilatory depressive and the analgesic effects of opioids. It can also be used to reverse chest wall or glottic rigidity from fentanyl and its derivatives. In patients taking opioids chronically (e.g., chronic pain management, illicit opioid users, methadone therapy for opioid abuse), naloxone must be used with caution, if at all, because the antagonist effect may precipitate acute opioid withdrawal and acute congestive heart failure may result.

The initial dose is 0.4 to 2 mg intravenously for acute reversal. Naloxone can also be titrated in 0.04-mg increments when gradual adjustment of respiratory depression is required. Because the duration of naloxone activity is 30 to 45 minutes, reemergence of respiratory depression may occur and additional dosing may be needed.

INDUCTION AGENTS

The general anesthesia induction agents are a category of drugs used to induce general anesthesia but they are also used for various sedative techniques in oral surgery practice. In the past, the barbiturates were the mainstays of this category, but over the last decade, propofol has become the major induction agent in anesthetic practice. Besides the barbiturates, which are rarely used today, other medications are available for sedation and induction of general anesthesia including etomidate and ketamine.

Propofol

Propofol has become one of the most popular sedative-hypnotic drugs used for ambulatory surgery. Propofol, 2,6-diisopropylphenol (Figure 3-4), is highly lipid soluble and available as a milky white 1% suspension in soybean oil, glycerol, and egg phosphatide. Like benzodiazepines and barbiturates, propofol is thought to interact with the GABA receptor, causing increased chloride conductance and hyperpolarization of neurons. At higher doses, propofol can produce amnesia and loss of consciousness. It is also an anticonvulsant, although spontaneous excitatory movements may be noted after administration.[8]

Depending on the dose and technique, propofol is used for all levels of sedation and general anesthesia. For induction of general anesthesia, a bolus of 1.5 to 2.5 mg/kg intravenously produces unconsciousness within 30 seconds. In the intermittent bolus technique frequently used for deep sedation in oral surgery, small increments of propofol (10–30 mg) are periodically administered, after a baseline moderate sedation with a benzodiazepine and opioid is obtained, in order to produce a state of deep sedation for local anesthetic administration and other stimulating portions of dentoalveolar surgery. Propofol can also be used as a continuous IV infusion.[9] The dosages for moderate sedation range from 25 to 100 μg/kg/min, deep sedation from 75 to 150 μg/kg/min, and general anesthesia from 100 to 300 μg/kg/min depending on the use of intubation. The overlap of dose ranges, from moderate sedation to general anesthesia, highlights the lower margin of safety of this drug, especially if the intended level of sedation is moderate sedation. Use of additional benzodiazepines and opioids further blurs the dosing guidelines for various forms of sedation. FDA labeling prohibits use of propofol by those involved in the conduct of the surgical or diagnostic procedure.

Propofol is extensively metabolized by hepatic enzymes. In addition, extensive redistribution and other mechanisms of metabolism and elimination most likely occur, because the rate of propofol clearance from the plasma exceeds hepatic blood flow. This rapid plasma clearance may account for the decreased cumulative effect of this drug in the body, contributing to rapid awakening. The context-sensitive half-time for this drug is short, reaching a maximum of 40 minutes even after 2 to 6 hours of continuous infusion. Context-sensitive half-times are even shorter with brief infusions.

Propofol decreases systemic blood pressure by as much as 20% to 40% from baseline through both central and peripheral mechanisms. Propofol also blocks sympathetic tone and allows parasympathetic vagal responses to predominate, thereby blunting the reflex tachycardia that would normally be associated with such a drop in blood pressure. Hypotension may, therefore, be very significant after bolus administration

or continuous infusion of propofol, particularly in elderly, medically compromised, and hypovolemic patients.

Propofol also leads to dose-dependent respiratory depression and can produce apnea at higher doses. It is not associated with histamine release and has bronchodilatory properties.

Recovery from anesthesia with propofol has several unique characteristics. Compared with other induction agents, propofol is associated with a more rapid awakening and recovery, with less residual CNS effects. Many patients also experience mild euphoria on awakening, which enhances reported satisfaction with the anesthesia postoperatively. Even at subhypnotic doses, propofol is associated with decreased postoperative nausea and vomiting (PONV).[9] All these features make propofol an attractive choice for outpatient procedures in which decreased time to discharge is desirable.

Several considerations should be taken when using propofol. The solution can cause significant pain on injection, especially in smaller vessels. This may be attenuated with preadministration of opioids or 1% cardiac lidocaine. Unlike barbiturates, however, it does not cause vasospasm when inadvertently injected into an artery.

Anaphylaxis is rare but has been reported in patients with a history of allergic reactions to other medications, especially neuromuscular blocking drugs. A history of egg allergy does not necessarily preclude the use of propofol, because the egg protein contained in the suspension is lecithin, whereas most egg allergies consist of a reaction to egg albumin. True soy allergy would, however, be a contraindication to propofol use. All propofol formulations are pH neutral and can support bacterial growth; therefore, the observation of sterile technique and discarding of an opened vial or filled syringe after 6 hours are recommended. Cracked glass containers or discolored contents should be discarded, because sepsis is a possibility. The original proprietary agent, Diprivan, uses EDTA (ethylenediaminetetraacetic acid) as an antibacterial agent, whereas one generic version contains metabisulfite. Although this generic agent should not be used in patients with known sulfite sensitivity, it appears that allergic reactions and bronchospasm are very unlikely, although not completely unheard of, in other patients including asthmatics. Another generic version uses benzyl alcohol to retard bacteria growth.

Etomidate

Like midazolam, etomidate contains an imidazole structure (Figure 3-5). It is water soluble but available in a 0.2% solution in propylene glycol. In the same way as the other induction medications, etomidate interacts at the GABA receptor.

Etomidate is used primarily as an induction agent for general anesthesia at 0.2 to 0.4 mg/kg intravneously. Its main advantage over barbiturates and propofol is cardiovascular stability. Although systemic blood pressure can decrease by up to 15% with etomidate, changes in heart rate are minimal. It also does not depress myocardial contractility. Etomidate is usually reserved for patients with unstable cardiac disease.

Spontaneous respiration may be maintained. Respiratory depression is less pronounced with etomidate than with barbiturates, although apnea is still possible with higher doses.

Etomidate is metabolized by both hepatic enzymes and plasma esterases. This rapid clearance leads to awakening

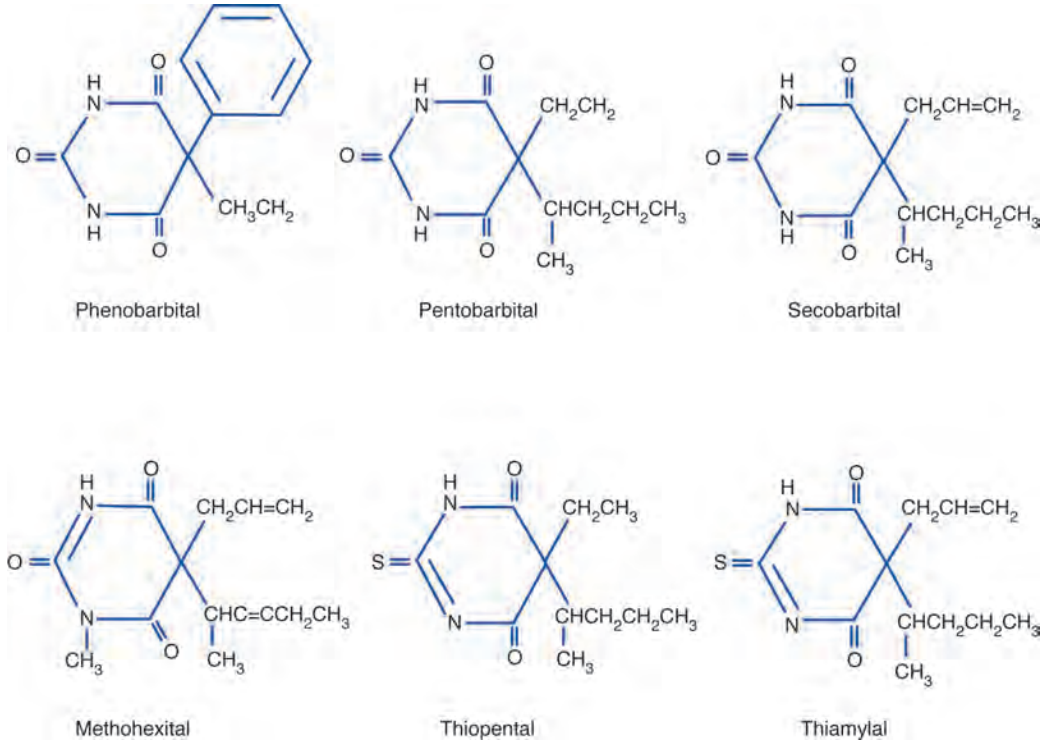


FIGURE 3-5. Chemical structure of etomidate.

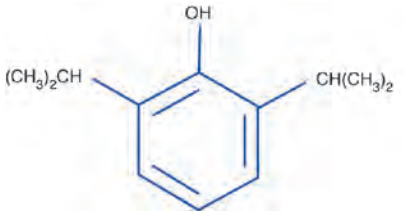


FIGURE 3-6. Chemical structure of ketamine.

and recovery that is faster than with thiopental but slower than with methohexital or propofol.

Myoclonus is common in over 50% of patients and may be partially prevented with preadministration of a benzodiazepine or opioid. Many patients experience pain on injection secondary to the propylene glycol. Etomidate has been associated with adrenocortical suppression, but this is less profound when only a single induction dose is administered.

Ketamine

Ketamine is a phencyclidine derivative (Figure 3-6) that induces a state of "dissociative anesthesia." This is characterized as a "dissociation" between the thalamocortical and the limbic systems, producing a cataleptic state during which the patient may appear awake but does not respond to commands.[10] The eyes may be open and nystagmic. Ketamine does produce anterograde amnesia, and unlike other induction agents, it can produce intense analgesia.

Unlike other hypnotic agents, ketamine does not interact with GABA receptors. The exact mechanism of action is unclear, but ketamine is a nonselective antagonist of supraspinal *N*-methyl-D-aspartate receptors, a subset of excitatory glutamate receptors. Inhibition of these receptors decreases neuronal signaling and is likely responsible for some analgesic effects. Ketamine may also interact with pain receptors in the spinal cord as well as opioid receptors, which may also account for analgesia.[11]

Ketamine is highly lipid soluble and redistributes quickly, which accounts for its rapid onset of action and relatively short duration. It is metabolized by hepatic enzymes and has an active metabolite, norketamine. Ketamine does have a significant abuse potential, and chronic use can lead to enzyme induction.

The cardiovascular effects of ketamine reflect its indirect activation of the sympathetic nervous system. Ketamine causes an increase in norepinephrine by inhibiting reuptake at postganglionic sympathetic neurons. Sympathetic stimulation increases heart rate and systemic blood pressure. Ketamine should, therefore, be used with caution in patients with uncontrolled hypertension or in whom tachycardia should be avoided. However, ketamine may be chosen for induction of general anesthesia at 1 to 2 mg/kg intravenously when cardiovascular stimulatory effects are desired, as in emergent trauma surgery. Practitioners should note that ketamine is actually a direct myocardial depressant, an effect normally masked by the indirect sympathetic stimulation. In severely compromised patients, however, catecholamine stores may be exhausted and hypotension secondary to myocardial depression can become significant.

Respiratory depression is not significant with ketamine, although apnea may still occur with rapid bolus administration. Upper airway reflexes remain largely, but not reliably, intact; aspiration is still possible, especially as ketamine increases salivary secretions and PONV. Ketamine does not cause histamine release and is a potent bronchodilator secondary to sympathetic activation as well as direct bronchial smooth muscle relaxation.

In oral surgical practice, one primary indication for ketamine is IM injection for uncooperative adult patients, such as the mentally challenged or those with severe psychiatric illness, or for children who will not tolerate IV placement. The IM dose for induction of general anesthesia is 3 to 7 mg/kg, whereas 2 to 3 mg/kg is usually sufficient to obtain adequate control for IV placement. A water-soluble benzodiazepine like midazolam is commonly added to reduce the possibility of uncomfortable dreaming associated with ketamine. An anticholinergic medication like glycopyrrolate can also given to reduce the production of salivary secretions secondary to ketamine. Glycopyrrolate may be preferred over atropine or scopolamine for its superior antisialagogue effects, less pronounced cardiac effects, and poor CNS penetration.

The other main use in oral surgical practice is in a planned IV deep sedation technique. Moderate sedation is first achieved with a benzodiazepine with or without an opioid, followed by subanesthetic doses of 10 to 30 mg of ketamine until a state similar to deep sedation is achieved. Because ketamine is quite analgesic, some surgeons omit opioid administration. Alternatively, if a standard deep sedation technique with baseline moderate sedation and propofol does not provide adequate operating conditions without high enough doses of propofol to induce apnea, and intubation or laryngeal mask airway inhalation anesthesia is not an option, "rescue" with ketamine is oftentimes effective to continue a nonintubated style deep sedation/general anesthesia.

"Emergence delirium" can occur during awakening. The patient may experience visual and auditory hallucinations that can be perceived as either pleasant (euphoria) or unpleasant (dysphoria), lasting for up to several hours. Delirium occurrence is less common in children and with IV doses less than 2 mg/kg. It may be attenuated with prior or concurrent administration of benzodiazepines, which should be routine when IV sedation techniques with ketmine are used.

BARBITURATES

Barbiturates are sedative-hypnotic medications that have long been employed as induction agents of general anesthesia. Recently, with the introduction of propofol, the use of IV ultrashort-acting barbiturates has decreased significantly in both oral surgery and hospital anesthesia practice. Barbiturates produce sedation, loss of consciousness, and amnesia. These drugs do not provide analgesia and may actu-

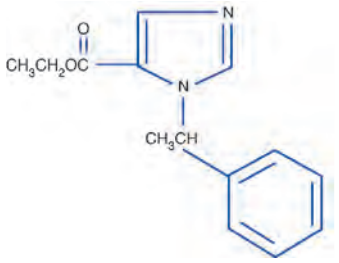


FIGURE 3-7. Chemical structure of barbituric acid.

ally reduce pain threshold at lower doses. Several barbiturates such as IV pentobarbital and oral phenobarbital are commonly used as anticonvulsants for both prevention and treatment of seizures. High doses of any IV barbiturate can also suppress acute seizure activity.

Barbiturates are derivatives of barbituric acid (Figure 3-7). The characteristics of the individual barbiturate are determined by the side chains attached to the barbiturate ring (Figure 3-8). For example, sulfur substitution on the no. 2 carbon in thiobarbiturates increases the lipid solubility of these drugs and, hence, decreases onset of action and duration of activity. The methyl group attached to the nitrogen atom of the ring in methohexital results in a more rapid onset for this oxybarbiturate and increased susceptibility to cleavage, producing a shorter duration than other oxybarbiturates.

Barbiturates act on GABA receptors at a specific binding site (different from benzodiazepines), causing the chloride channel to remain open for a longer duration. The increased negative inward flow hyperpolarizes the membrane, decreasing neuronal transmission.

Awakening from IV barbiturates is dependent on redistribution from the brain. These medications are metabolized by hepatic enzymes without the formation of active metabolites and are then cleared renally. Because these drugs are highly protein-bound, hypoproteinemia secondary to liver failure or malnutrition increases the plasma concentration of free drug. Chronic use of barbiturates can cause induction of liver enzymes. Barbiturates are also contraindicated in patients with acute intermittent porphyria because they may precipitate an attack.

Barbiturates are associated with a dose-dependent decrease in respiratory rate and tidal volume with apnea observed at higher doses. Centrally mediated peripheral vasodilation leads to a transient drop of 10% to 30% in systemic blood pressure, particularly when a full induction dose is administered. This is partially attenuated by a compensatory increase in heart rate as baroreceptor reflexes remain intact. Hypotension is more evident in the elderly or medically compromised, hypovolemic patients. Thiopental can cause histamine release, which is clinically insignificant with methohexital.

Intra-arterial injection of barbiturates causes painful spasm of the vessel from precipitation of barbiturate crystals, which damage the endothelium and may result in occlusion of the artery. At worst, decreased distal perfusion may result in tissue necrosis of a limb or nerve damage and must be addressed immediately. The IV catheter should be left in place, IV cardiac lidocaine or procaine (without epinephrine) administered, and the patient should be transported to an emergency department where medications or regional blockade may be given to relieve the spasm and reduce the occlusion. Although also uncommon, venous irritation and thrombosis secondary to crystal formation is also possible with concentrations of barbiturates above 1% methohexital and 2.5% thiopental.

These medications are stored in powder form and reconstituted in saline before use as sodium salts. The alkalinity of the solutions prevents bacterial growth and ensures a longer refrigerated shelf life of up to 2 weeks for thiopental and 6 weeks for methohexital.

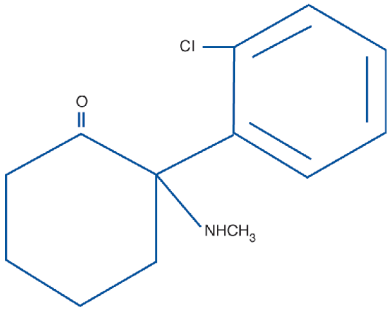


FIGURE 3-8. Chemical structure of barbiturates.

Thiopental

Thiopental is an ultrashort-acting barbiturate that is commonly used at 3 to 5 mg/kg intravenously in a 2.5% solution to induce loss of consciousness for general anesthesia before endotracheal intubation. It is associated with a longer recovery than methohexital owing to its decreased plasma clearance and is generally not used as a continuous infusion to maintain anesthesia owing to significant storage in multiple drug compartments. Thiopental can release histamine, which is a concern in asthmatic patients.

Methohexital

Methohexital is an ultrashort-acting barbiturate that was commonly employed for outpatient oral surgical procedures, primarily for its more rapid recovery compared with thiopental and its lower cost compared with propofol. As the cost of propofol has significanlty decreased, methohexital is now used by many fewer oral surgeons. As an oxybarbiturate, methohexital is less lipid soluble than thiopental but is associated with a more rapid awakening because of its increased hepatic clearance.[12] Psychomotor function returns more quickly with methohexital than thiopental, allowing for earlier discharge after an outpatient procedure.

Methohexital is reconstituted into a 1% solution and given at 1.5 to 2 mg/kg intravenously for induction of general anesthesia. With these doses, blood pressure may drop by up to 35% and heart rate increases up to 40% of baseline. In a common deep sedation technique used in oral surgical practice,

10- to 30-mg increments of methohexital are periodically administered after obtaining baseline conscious sedation with a benzodiazepine and opioid to produce a state of deep sedation for local anesthetic administration and other stimulating portions of dentoalveolar surgery.

Methohexital is associated with involuntary movements such as myoclonus and hiccuping. These excitatory phenomena are dose dependent and may be reduced by prior administration of opioids. Low doses of methohexital can activate seizure foci and should be used cautiously, if at all, for epileptic patients. Shivering upon awakening is also common after methohexital anesthesia. Methohexital exhibits clinically insignificant histamine release.

Pentobarbital

Pentobarbital is an IV short-acting barbiturate with a duration of action of 2 to 4 hours. It is generally used for conscious sedation in doses of 100 to 300 mg, combined with opioids and possibly benzodiazepines, for longer operative procedures. Cardiovascular effects are more modest than with the ultrashort-acting agents.

INHALATION ANESTHETICS

Inhalation anesthetics include nitrous oxide (N_2O) as well as the potent volatile halogenated agents, such as isoflurane, sevoflurane, and desflurane. N_2O alone is commonly used in dental offices for anxiolysis and minimal sedation, but it is also used in combination with other medications to induce and maintain both sedation and general anesthesia. The halogenated agents are extremely potent and are used for induction and maintenance of general anesthesia. They are complete anesthetics providing hypnosis, amnesia, analgesia, and muscle relaxation.

The pharmacokinetics of these anesthetic agents differ from those of IV medications. These drugs are inhaled and cross from the alveoli into the pulmonary vasculature, entering the general circulation. They are able to cross the blood-brain barrier and exert anesthetic effects within the brain. These agents are minimally metabolized and are subsequently excreted unchanged back into the alveoli. Once exhaled, these gases are deposited into the anesthesia circuit and eventually scavenged.

Plasma concentrations of the inhaled anesthetics are dependent on the concentration of the gas within the alveoli, solubility characteristics of the individual gases, and cardiac output.[13,14] Cardiac output influences the rate of uptake from the alveoli in that low cardiac output increases pulmonary venous uptake by increasing the amount of time any given volume of blood is in contact with the alveolar membrane. Main factors affecting alveolar gas concentration include the inspired concentration of gas, alveolar ventilation, and the total gas flow rate. Administering a higher concentration of gas will increase intra-alveolar concentration, whereas altering the total gas inflow or alveolar ventilation (respiratory rate, tidal volume) will affect how quickly the concentration of gas within the alveoli changes.

Each agent varies in its solubility in blood and other tissues such as the brain and fat, and these characteristics determine the ease with which the gas crosses into the different tissues. Of these, the blood-gas solubility coefficient (Table 3-1) is the most useful in describing the onset and offset of action of an anesthetic gas. The blood-gas solubility coefficient expresses the extent to which the anesthetic gas molecules from the alveolar spaces will dissolve into plasma before the plasma solution becomes saturated. Conceptually, a lower coefficient means that the gas is less soluble in blood and will saturate the plasma compartment quickly. Additional "overflow" molecules will then be free to move into other highly vascular tissues such as the brain, where the CNS anesthetic effect takes place. A lower blood-gas coefficient therefore translates into faster onset of action at the brain. Once the gas is discontinued and the alveolar and plasma concentrations decrease, the gas molecules move down their concentration gradient from the tissues back into the bloodstream and then into the alveoli. Gases with lower blood-gas coefficients will likewise "offload" from the bloodstream into alveoli more quickly and can translate into a faster offset of action.

TABLE 3-1. **Different Properties of Nitrous Oxide and Potent Volatile Agents**

	Nitrous Oxide	Isoflurane	Halothane*	Desflurane	Sevoflurane
General Properties of Inhalation Anesthetics—Adults					
Molecular weight	44	184.5	197.4	168	218
Vapor pressure 20°C	Gas	238	243	664	160
MAC in O_2	105	1.2	0.77	6.0	2.0
% Recovered metabolites	0	0.2	20	0.02	3
Partition Coefficients at 37°C					
Blood:gas	0.47	1.46	2.5	0.42	0.69
Brain:blood	1.1	1.6	1.9	1.3	1.7
Muscle:blood	1.2	2.9	3.4	2	3.1
Fat:blood	2.3	45	51	27	48

MAC = minimum alveolar concentration.
*Not available in the United States.

Unlike IV medications, these inhaled drugs are not administered in doses of milligrams per kilogram. The rough equivalent of the effective dose 50% (ED_{50}) of inhaled anesthetic agent is the minimum alveolar concentration (MAC). The MAC value of any given agent is the inhaled concentration (volume %) of that agent required to prevent movement in 50% of patients to a surgical stimulus. MAC values for different agents are given in Table 3-1. MAC values provide a useful dosage guide for anesthetic gases. In adults, a level of 1.3 MAC will prevent movement in 95% of patients, whereas 1.5 MAC (MAC_{BAR}) will block an adrenergic response in 95% of patients. Below 0.3 MAC (MAC_{AWAKE}), patient awareness is more likely. MAC values are additive; for example, if 0.5 MAC of N_2O and 1.0 MAC of isoflurane are given simultaneously, the total MAC of anesthetic agent administered to the patient is 1.5 MAC. It should be noted that MAC values are general guidelines and individual anesthetic requirements can be influenced by a variety of factors such as age or medical status. Neonates have the lowest MAC requirement, whereas children have the highest requirement. MAC requirements subsequently decrease in the elderly patient. MAC values are typically listed for adult (30- to 40-yr-old) patients at 1 atm pressure and 20°C.

The exact mechanism of action of inhaled anesthetic agents at the CNS is still controversial. Earlier theories have suggested that anesthetic molecules insert into and disrupt the lipid bilayer of neuronal cell membranes, thus interfering with the cellular function. More current theories suggest that anesthetic molecules may instead directly interact with cellular proteins, possibly with membrane ion channels or even specific receptors.

Whereas N_2O has mild or minimal sympathomimetic effects, all of the halogenated agents produce generalized cardiovascular depressant effects. The potent volatile agents block peripheral vasoconstriction, thus lowering mean arterial blood pressure. At lower doses below 1 MAC, the baroreceptor sympathetic reflex is activated, which leads to a compensatory increase in heart rate.

At usual doses, N_2O does not appreciably affect respiration. However, the halogenated agents produce a characteristic "rapid and shallow" spontaneous breathing pattern. A decrease in tidal volume is accompanied by an increase in the frequency of breathing, but the faster respiratory rate does not fully compensate for the smaller tidal volumes. Therefore, minute ventilation is reduced and arterial CO_2 levels will be elevated in patients spontaneously breathing while under general anesthesia with these agents. The halogenated agents also cause a dose-dependent decrease in airway resistance and produce bronchodilation. Hypoxic pulmonary vasoconstriction is attenuated at over 1 MAC for all volatile agents.

Although hepatic blood flow decreases with these agents, hepatic damage, if any, resulting from hypoxia is usually subclinical and transient. Renal blood flow and urine output are reduced secondary to the decreased mean arterial pressure. The release of fluoride from the halogenated gases does not appear to cause clinically significant damage to renal tissues. With sevoflurane, fresh gas flows of at least 2 L/min to minimize compound A accumulation in the CO_2 absorber, which can lead to very rare hepatic or renal damage.

Malignant hyperthermia (MH) is another rare but very dangerous reaction triggered by the halogenated agents as well as SCh. N_2O, nondepolarizing neuromuscular blockers, opioids, benzodiazepines, and other IV anesthetic agents do not trigger MH. Exposure to triggering medications causes an abnormal receptor in skeletal muscle cells to release excessive intracellular calcium, leading to uncontrolled muscle contractions. As a result, CO_2 production increases quickly and exhaled CO_2 rises sharply. Initial signs include tachycardia and tachypnea, along with muscle stiffness. Metabolic acidosis and hyperkalemia develop next and cardiac arrest is a possibility. Increasing body temperature is a relatively late sign. The halogenated agent must be discontinued at once and 100% O_2 given, preferably through a different circuit and machine. Dantrolene at 2.5 to 10 mg/kg intravenously must be given as soon as possible. Cooling measures including cooled IV fluids should be instituted. Emergency help must be obtained immediately and the patient will require medical management and monitoring for at least 24 hours after the episode. Reemergence of the reaction is common, requiring readministration of dantrolene, and acute renal failure is the most common morbidity secondary to myoglobinemia. A mortality rate of 10% is associated with an acute MH episode, even with immediate proper management.

Nitrous Oxide

N_2O is commonly administered in dental offices for anxiolysis and mild sedation. It is a colorless and odorless gas, available in blue cylinders in the United States. In the dental setting, it is commonly administered with a nasal hood and appropriate scavenger system. Concentration ratios of N_2O/O_2 range up to 70:30 on most dental N_2O and anesthesia machines. High levels of N_2O/O_2 alone can produce sedation and significant analgesia. Unexpected respiratory depression or airway obstruction can occur when N_2O is added to other sedative agents.

N_2O in O_2 is likely the most commonly used sedative agent in dental offices and enjoys the unique advantage of not requiring an escort after completion of the procedure, provided adequate recovery time has elapsed. The drug can be titrated, usually starting at 20% N_2O and gradually increasing to 50% as needed. Doses above that level are associated with increased nausea and dysphoria, although the brief application of doses higher than 50% is useful during local anesthetic administration and other short stimulating surgical episodes. At the conclusion of N_2O sedation, 3 to 5 minutes of 100% O_2 is administered to maintain scavenging and prevent diffusion hypoxia. This is a rare occurance in the healthy individual if doses of N_2O at or below 50% are delivered for short times and if room air gas instead of 100% O_2 is used at the conclusion of sedation. It is possible that the

rapidly exiting N_2O can dilute the O_2 concentration in the alveoli to hypoxic levels during recovery.

With a low blood-gas solubility coefficient of 0.47, N_2O has a very quick onset and recovery. Whereas N_2O lacks the potency of the halogenated agents at a MAC value of 105, it also lacks the respiratory and cardiovascular side effects. During general anesthesia, it can be administered to an intubated patient in combination with other medications such as halogenated gases and opioids. Using this combination can reduce the dose required of each drug if given singly and will lessen the incidence of potential adverse effects. N_2O is also inexpensive and can reduce the total cost of administered drugs. Recently, N_2O has fallen out of favor for intubated general anesthesia due to the documented increased incidence of PONV; the use of higher concentrations of O_2, which may benefit wound healing; as well as any pharmacokinetic advantage being negated by the newer potent halogentated agents.

There are a few contraindications for the use of N_2O. It can enter closed spaces faster than nitrogen can exit, leading to distention of the closed space.[13] In oral surgical practice, the implication of this property is to avoid N_2O use in patients with current otitis media, sinus infections, and COPD (chronic obstructive pulmonary disease; emphysema with blebs that may rupture). Other contraindications of N_2O use include current respiratory disease and a history of severe PONV.

Several precautions should be exercised when using N_2O. It has been implicated in producing sexual hallucinations in some patients, predominantly young women. An additional person such as an assistant should always be present when this gas is being administered. Patients with preexisting psychiatric disorders may experience exacerbated symptoms while undergoing N_2O sedation. Because low levels of N_2O in room air have been demonstrated to increase spontaneous abortion rates in pregnant anesthesia providers, proper scavenging is essential to minimize room air levels so that surgical personnel are not at increased risk. Frequent recreational use of N_2O has been reported to lead to peripheral neuropathy and other deleterious effects. As with all anesthetic agents, anesthesia providers must never use these drugs for personal use and should be alert to potential misuse by other providers of these drugs.

Potent Inhalation Agents

The halogenated inhalation agents commonly in use today in the United States include isoflurane, sevoflurane, and desflurane. As seen in Figure 3-9, all are derivatives of ether with halothane, an ethane, added for historical reference. Unlike the original anesthetic gas, diethyl ether, these agents are halogenated and nonflammable. The newer halogenated agents, sevoflurane and desflurane, are unique in that all of the side chain halogen atoms are fluorine. The gases are stored and released by gas-specific vaporizers that control the concentration (volume %) allowed into the anesthesia circuits and into the patient. They must also be scavenged effectively so that room air levels do not affect health care personnel.

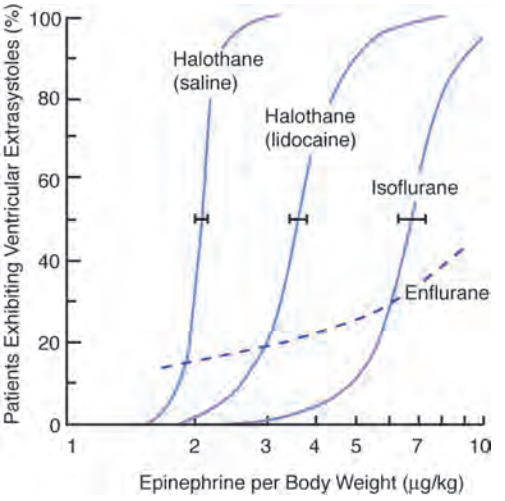


FIGURE 3-9. Chemical structure of inhalation agents.

Isoflurane

Isoflurane has an intermediate potency (MAC 1.2) and blood-gas partition coefficient (1.46). With the newer halogenated agents having improved pharmacokinetics, isoflurane is a less common choice for maintenance of anesthesia, because recovery time is longer and there is less ability to quickly not titrate the drug intraoperatively. Isoflurane is not a good choice for inhalation induction because it frequently leads to gagging and coughing when used. Isoflurane is also much more cost-effective for longer periods of anesthesia than two other popular agents, sevoflurane and desflurane; its cost per equivalent MAC administration is significantly lower than the other available agents.

Isoflurane may be associated with an increase in coronary steal phenomena, leading some practitioners to avoid using this anesthetic in patients with significant atherosclerotic cardiac disease. Otherwise, contraindications for using isoflurane are few.

Sevoflurane

Sevoflurane is nonpungent and a common choice for inhalation induction. It has an intermediate potency (MAC 2.0), and at higher doses, induction will be rapid. Recovery from sevoflurane after a short anesthetic (<1 hr) is more rapid than either isoflurane owing to the lower blood gas solubility coefficient (0.69). For longer procedures, however, the advantage of faster recovery is offset by the much greater cost of sevoflurane compared with isoflurane. The recovery time is also not significantly improved compared with isoflurane, because both gases similarly redistribute into fat during longer anesthesia periods, and offset of these gases from fat storage is not different.

All of the side chain halogen atoms in sevoflurane are fluorine, contributing to its low blood-gas solubility and recovery profile. Unlike earlier inhaled agents, the small

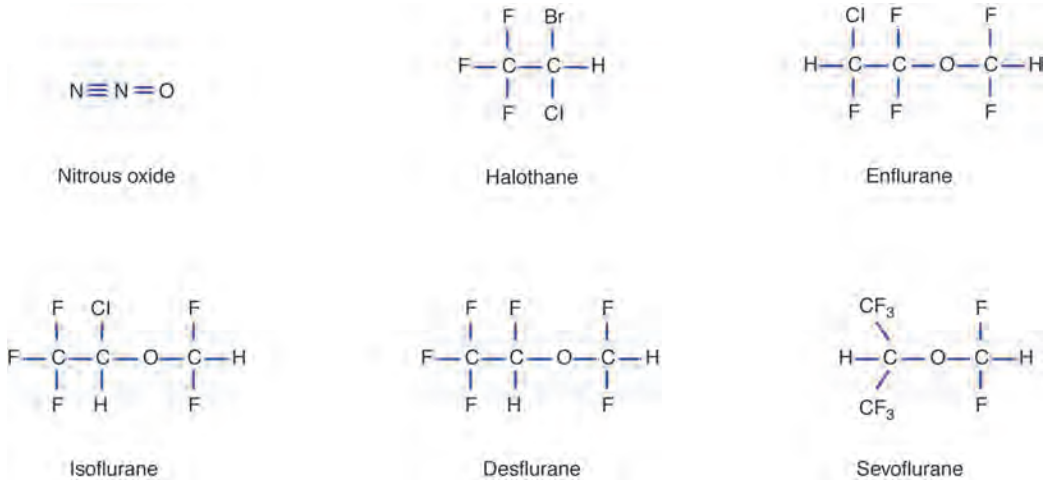


FIGURE 3-10. Halothane sensitizes the myocardium to dysrhythmias after administration of epinephrine in saline. Addition of 0.5% lidocaine to the epinephrine solution decreases the incidence of dysrhythmias, but the incidence is still higher than during isoflurane use. (Adapted from Johnston RR, Eger EI, Wilson C. A comparative interaction of epinephrine with enflurane, isoflurane, halothane in man. Anesth Analg 1976;55:709–712.)

amount of inorganic fluorine released during sevoflurane use has not been associated with renal damage.[15] Sevoflurane and CO_2 absorbers (soda lime, barium lime) produce a degradation product called "compound A," an olefin, which is nephrotoxic in rats but has not been associated with significant permanent renal damage in humans. Regardless, sevoflurane may not be the agent of choice for patients with renal disease. Sevoflurane is fequently used at a total gas flow of at least 2 L/min to reduce the production of compound A.

Desflurane

Desflurane is extremely pungent and can be so irritating to nonanesthetized airways that it may precipitate coughing and laryngospasm. It is to be avoided for inhalation inductions. During initial administration of desflurane, tachycardia can also occur until deeper levels of anesthesia are realized.

Desflurane is delivered from specially heated vaporizers because its vapor pressure is close to atmospheric pressure. It also possesses only fluorine substitutions that, like sevoflurane, confer a low blood-gas solubility. In fact, desflurane has the lowest blood-gas solubility coefficient (0.43) of any inhalation agent, lower than even N_2O. This confers a quick onset and offset and easy titratability, and recovery can be very rapid after a short anesthetic with desflurane. Like sevoflurane, desflurane is the most expensive of the potent agents based on equivalent MAC administration.

Halothane

For historical perspective, since the 1950s, halothane, an ethane, was widely used, particularly in pediatric anesthesia, owing to its a sweet, nonpungent odor that did not irritate the airway mucosa. This was the only inhalation inducton agent available for many years until the introduction of sevoflurane. In 2000, the FDA changed the prescribing information for halothane, causing the agent to eventually no longer be available in the U.S. market. Concerns included increased incidence of cardiac dysrythmias and myocardial depression, epineprhine- and hypercarbia-induced dysrythmia, drug-induced hepatitis particularly with repeated use, and potent triggering of MH, among other issues (Figure 3-10). A significant decrease in pediatric perioperative cardiac arrests resulted.[16] Halothane is very potent, with a MAC value at 0.75 but a relatively high blood-gas solubility of 2.54. Around the world, halothane is still commonly used because its cost is low and inhalation induction well tolerated.

PERIOPERATIVE ANALGESIC MEDICATIONS

Opioid medications, which have been discussed previously, are the mainstay intraoperative and postoperative analgesic medications. In the operating room, opioids are often given concurrently with other anesthetic agents in a balanced technique to supplement intraoperative analgesia. An opioid with a long duration of action such as morphine or hydromorphone is commonly administered by the practitioner before the end of the procedure, in anticipation of postoperative pain. During the initial phase of postoperative care, these medications are usually given by the nursing staff but once initial recovery has occurred, patient administration can also occur via computer-aided patient-controlled analgesia pumps.

Another option is ketorolac tromethamine, currently the only available IV NSAID medication in the United States. This agent can provide effective analgesia for adults for many dentoalveolar procedures at an IV dose of 30 mg and IM doses of 30 to 60 mg. For pediatric patients, 0.5 mg/kg is used, although off-label one-time doses of 1 mg/kg can be considered. Onset time is 10 to 15 minutes, with an analgesic duration of approximately 6 hours. Ketorolac 30 mg intramuscularly is the analgesic equivalent of 10 mg of parenteral morphine and does not produce opioid-related respiratory

depression, nausea, or sedation.[3] NSAID use does have several cautions, however. Because of possible NSAID-induced inhibition of platelet aggregation, the drug is normally administered after bleeding has been controlled and should be avoided for surgeries associated with postoperative hemorrhage. Patients with bleeding-related disorders (e.g., gastrointestinal ulcers, inflammatory bowel disease, blood dyscrasias, liver failure) should not be given ketorolac. Life-threatening bronchospasm can also occur with NSAIDs, particularly in those with a history of asthma or aspirin allergy. Because NSAIDs block prostaglandin production, patients who depend on renal prostaglandins for adequate renal function should be administered ketorolac cautiously. Patients with congestive heart failure, hypovolemia, or cirrhosis may have impaired renal perfusion and NSAID administration can result in acute fluid retention. This drug is also associated with a higher cost than other analgesic medications.

The most commonly used agents for postoperative pain control in office-based oral surgery are likely the local anesthetics. Long-acting local anesthetics, like bupivacaine and etidocaine, provide several hours of analgesia for inferior alveolar nerve block anesthesia as well as soft tissue anesthesia in the maxilla. Lidocaine with epinephrine given intraoperatively can also provide adequate analgesic duration until postoperative oral NSAIDs or opioid/acetaminophen combinations can achieve reliable plasma levels for generally predictable postoperative pain control.

ALPHA 2 AGONISTS

Alpha 2 agonists have sedative, analgesic, and muscle relaxing properties in addition to their inhibition of CNS sympathetic outflow and, thus, their antihypertensive effects. They are one of the very few drug classes known to decrease anesthetic requirements. Clonidine, the prototypical alpha 2 agonist, can be used as an anxiolytic premedication with the added benefit of cardiovascular stability intraoperatively. Dexmedetomidine, an IV agent approved for United States use in 1999, is the dextro-isomer of medetomidine and eight times more selective for the alpha 2 receptor than clonidine. It was intially approved for use in the intensive care unit for sedation for intubated pateints, but in 2008, after extensive use in anesthesiology, the FDA approved the agent for procedural sedation in nonintubated patients. Dexmedetomidine has proved useful in awake endotracheal intubation and some preipheral procedures in which good local anesthesia can be obtained. It can also be used in a continuous infusion as an adjunct to general anesthesia to reduce anesthetic requirements. Some practitioners use a single bolus dose before extubation for smooth emergence from anesthesia.

NEUROMUSCULAR BLOCKING MEDICATIONS

Skeletal muscle relaxation is often required during surgery when patient movement interferes with procedures involving anesthesia or surgery. For example, paralysis may be required to facilitate tracheal intubation, relax abdominal wall muscles for access during gastrointestinal surgery, or completely inhibit patient movement during ocular surgery. Whereas relaxation can be achieved with deeper anesthetic levels or appropriate peripheral neural blockade, neuromuscular blocking agents are commonly used to provide the necessary amount and duration of relaxation.

The potential of these drugs during anesthesia and surgery was not recognized until the middle of the 20th century. Many of the current neuromuscular blocking agents used are derivatives of curare, one of the oldest paralyzing agents, used by ancient hunters to paralyze prey. All are competitive antagonists that bind to the nicotinic ACh receptors located at the postsynaptic membrane of the neuromuscular junction of skeletal muscle, thus interfering with proper contraction of the muscle.

Neuromuscular blocking agents can be classified as either *depolarizing* or *nondepolarizing* and, within the latter group, can be divided based on structure, speed of onset, duration of action, and metabolism.

Succinylcholine

SCh, two joined ACh molecules, was introduced for surgical muscle relaxation in the 1950s and is the only depolarizing agent used today. Once SCh binds to the ACh receptor, the postsynaptic membrane depolarizes, an action potential is generated, and the muscle contracts. Subsequent muscle contractions are delayed until SCh dissociates from the receptor and is metabolized by pseudocholinesterase.

SCh has the fastest onset (30–60 sec) and shortest duration (5–10 min) of the neuromuscular blocking agents and is typically used to treat laryngospasm not relieved with positive pressure (20–40 mg or 0.1–0.2 mg/kg). It is also given to facilitate tracheal intubation (1–1.5 mg/kg intravenously) or when emergent tracheal intubation is required to treat laryngospasm. It is no longer used to maintain intraoperative paralysis.

SCh has several notable side effects. Tachycardia can result upon initial administration but bradycardia may develop, especially with repeated administration. Widespread muscle contractions can result in postoperative myalgia, which can at times be prevented by prior administration of a small dose of a nondepolarizing muscle blocker. The contractions may increase intraocular and intragastric pressure and can also cause a transient elevation in plasma potassium levels by 0.5 mEq/L. Plasma potassium levels may rise even higher than 0.5 mEq/L in patients with certain neuromuscular disorders, stroke, spinal cord injury, or significant burn injury 24 hours after injury. SCh is, therefore, contraindicated in these patients. SCh is a trigger for MH (see "Malignant Hyperthermia," earlier). Its use should also be avoided in patients with pseudocholinesterase abnormalities, because the recovery from this drug will be prolonged.

Nondepolarizing Agents

All of the remaining neuromuscular blocking agents are nondepolarizing and do not initiate muscle contraction upon

TABLE 3-2. **Common Neuromuscular Blocking Medications and Their Properties**

	Intubating Dose (mg/kg)	Time to Intubate (min)	25% Twitch Recovery (min)	Metabolism and Elimination	Histamine Release	Vagolysis
Depolarizing						
Succinylcholine	1	1	5–10	Plasma cholinesterase	±	0
Nondepolarizing						
Aminosteroids						
Rocuronium	0.6–1.2	1–1.5	40–150	Liver; kidney	0	0
Vecuronium	0.1–0.12	2–3	25–30	Liver; kidney	0	0
Pancuronium	0.08–0.1	3–5	80–100	Liver; kidney	0	+
Pipecuronium	0.07–0.085	3–5	50–120	Liver; kidney	0	±
Benzylisoquinolines						
Atracurium	0.4–0.5	2–3	25–30	Hofmann elimination	+	0
Cisatracurium	0.15–0.2	1.5–2	50–60	Hofmann elimination	0	0
Doxacurium	0.05–0.08	4–5	100–160	Liver; kidney	0	0
D-Tubocurarine	0.5–0.6	3–5	80–100	Liver; kidney	++	0

administration. The chemical structures of these drugs fall into two classes: benzyliso-quinolines and aminosteroids.[13] Characteristics of currently available nondepolarizing muscle relaxants are outlined in Table 3-2.

Although it is not as rapid in onset as SCh, rocuronium has the fastest onset of the nondepolarizing agents, with paralysis occurring at approximately 1 minute with higher doses. It is often chosen for facilitating intubation when SCh cannot be used, particularly in an emergent situation. Onset time for most other agents is approximately 3 minutes.

Drug selection for maintenance of muscle relaxation is often based upon the anticipated need for continued paralysis. Pancuronium has the longest duration, whereas vecuronium or rocuronium has the shortest depending on the dose administered. With any of these agents paralysis, will last longer than that produced with SCh and controlled ventilation must be provided. Return of skeletal muscle function is usually monitored by a nerve stimulator, and the degree of paralysis is gauged by the number of muscle contractions (twitches) produced by stimulation of specific muscles, such as adductor pollicis and orbicularis orbis. Paralysis may need to be reversed by an anticholinesterase to ensure adequate recovery of airway and respiratory muscle function before extubation.

Adverse effects may also affect the choice of neuromuscular blocking agent and can be categorized by structure. The benzylisoquinoline compounds may trigger histamine release, thus causing flushing and peripheral vasodilation. Aminosteroid structures may block vagal activity, causing a noticeable increase in heart rate. Histamine release may be undesirable in asthmatic patients. Increased heart rate can be problematic in patients with cardiovascular disease.

Many of the nondepolarizing agents are metabolized by the liver and excreted by the kidney but some rely more on renal excretion. Atracurium and cisatracurium are removed by Hofmann elimination, whereby the drug spontaneously degrades at body pH and temperature.

ANTICHOLINESTERASES

Anticholinesterases, or anti-acetylcholinesterases, block the action of acetylcholinesterase, the enzyme that breaks down ACh. In anesthesia, anticholinesterases such as neostigmine, edrophonium, and pyridostigmine are used to reverse the effects of nondepolarizing muscle relaxants once partial muscle function has returned and paralysis is no longer necessary, usually at the conclusion of surgery. By increasing the amount of ACh available at the neuromuscular junction, more of the neurotransmitter can bind to nicotinic ACh receptors, overcoming the competitive inhibition of the neuromuscular blocker and aiding in the return of muscle function.

Increased ACh will also bind to muscarinic ACh receptors at the heart, lungs, salivary glands, and smooth muscle. This can lead to undesirable side effects including bradycardia, bronchospasm, abdominal cramping, and excessive salivation.[17] To prevent these effects, anticholinergic medications such as atropine or glycopyrrolate, which block muscarinic but not nicotinic ACh receptors, are given together with anticholinesterases. The anticholinesterase and anticholinergic medications are paired according to similar time of onset and duration. Glycopyrrolate is generally administered with neostigmine, and atropine is more commonly used with edrophonium. Doses of these agents are listed in Table 3-3.

ANTICHOLINERGIC MEDICATIONS

ACh is a neurotransmitter that binds to two types of receptors. Nicotinic receptors are located at autonomic ganglia and the neuromuscular junctions of skeletal muscle. Muscarinic receptors are found at postganglionic sites of the parasympathetic nervous system at the heart, salivary glands, and smooth muscle. Anticholinergic medications specifically block muscarinic receptors but do not affect nicotinic receptors.

Clinical uses in anesthesia of atropine, glycopyrrolate, and scopolamine are defined by their varied effect at the

TABLE 3-3. **Reversal Doses of Acetylcholinesterase and Anticholinergic Medications***

Cholinesterase	Cholinesterase Dose (mg/kg)	Anticholinergic	Anticholinergic Dose (mg/mg of cholinesterase)
Neostigmine	0.4–0.8	Glycopyrrolate	0.2
Edrophonium	0.5–1.0	Atropine	0.014

*The two most commonly used acetylcholinesterase medications are listed. Acetylcholinesterase and anticholinergic medications are given in recommended combinations according to similar onset time and duration of action of the two types of drugs. The maximum dose of cholinesterase is not always necessary, but should be given based on the degree of recovery from muscle relaxation. The dose of the anticholinergic drug is determined by the amount of cholinesterase given.

muscarinic receptor sites of different organs (Table 3-4). Atropine has the fastest onset of increasing heart rate by blocking vagal nerve receptors at the heart and is used to treat emergent bradycardia. Both atropine and glycopyrrolate are used to counteract bradycardia secondary to anticholinesterase use during reversal of muscle relaxation. All three anticholinergic medications decrease salivary secretions. Glycopyrrolate is a quaternary ammonium compound, which cannot cross the blood-brain barrier. Atropine and scopolamine, both tertiary amines, can cross the blood-brain barrier and cause sedation. Scopolamine is also used for management of nausea and prevention of motion sickness.

Central anticholinergic syndrome is a concern with higher doses of centrally acting anticholinergic medications, manifesting as restlessness and confusion. It may be reversed by physostigmine, an anticholinesterase that can cross the blood-brain barrier.

ANTIEMETIC MEDICATIONS

PONV is one of the most common complaints after surgery. Certain groups of patients (female, obese, previous history of nausea and vomiting) appear to be more susceptible. Certain surgeries (ear, ocular, tonsillar, gynecologic) are likewise associated with increased PONV. Nausea and vomiting after oral surgery are not uncommon. Swallowed blood and secretions stimulate the gag reflex and are potent gastric irritants. Drugs used during sedation and anesthesia, such as N_2O, potent inhalation anesthetics, opioids, and ketamine, may trigger nausea postoperatively. Other "nonchemical" triggers of nausea include smell, gastric distention, motion, and even stress.

Chemical triggers in the bloodstream come into contact with an area in the medulla lacking an intact blood-brain barrier called the *chemoreceptor trigger zone (CTZ).*[18] The CTZ (Figure 3-11) contains receptors for serotonin, histamine, muscarinic ACh, and dopamine. Opioids, toxins, and chemotherapy agents, as well as input from the middle ear, also stimulate this area. Stimulation of the CTZ will activate vomiting.

Many antiemetic medications act by blocking these receptors at the CTZ. Medications that block the dopamine receptor include phenothiazines (e.g., prochlorperazine) and butyrophenones (e.g., droperidol). They effectively reduce PONV but are associated with adverse effects such as sedation and extrapyramidal reactions. 5-Hydroxytryptamine (serotonin; $5\text{-}HT_3$) antagonists including ondansetron, dolasetron, and granisetron produce less sedation and other adverse effects than the dopamine antagonists. Antihistamines such as promethazine (which also possesses a phenothiazine structure) and diphenhydramine can cause significant sedation. Scopolamine, an anticholinergic medication, can be used in a patch behind the ear to reduce PONV, although its original use is for motion sickness. Ideally, the patch should placed at least 1 hour or more before anesthesia with the effects lasting up to 3 days. Other anticholinergics such as hydroxyzine and benadryl also have some antiemetic properties. The newest antiemetic agent is an NK_1 receptor antagonist, aprepitant, which appears especially useful for delayed phase PONV. The natural ligand for the NK_1 receptor is substance P.

Recently, dexamethasone has been shown to decrease the incidence of PONV when given shortly after induction of general anesthesia. A minimum adult dose of 8 mg intravenously appears to be required for this effect to be realized.[19]

Selection of anesthetic agents may help prevent PONV. Propofol appears to have antiemetic effects as well, particularly when administered for maintenance of anesthesia. Additional antiemetic treatment may be unnecessary following the use of propofol infusions, even in patients with a previous

TABLE 3-4. **Varied Effects of Anticholinergic Medications**

	ANTICHOLINERGIC MEDICATION CHARACTERISTICS			
	Tachycardia	Bronchodilation	Sedation	Antisialagogue
Atropine	≠≠≠	≠≠	≠	≠≠
Glycopyrrolate	≠≠	≠≠	0	≠≠≠
Scopolamine	≠	≠	≠≠≠	≠≠≠

≠ = mild effect; ≠≠ = moderate effect; ≠≠≠ = strong effect.

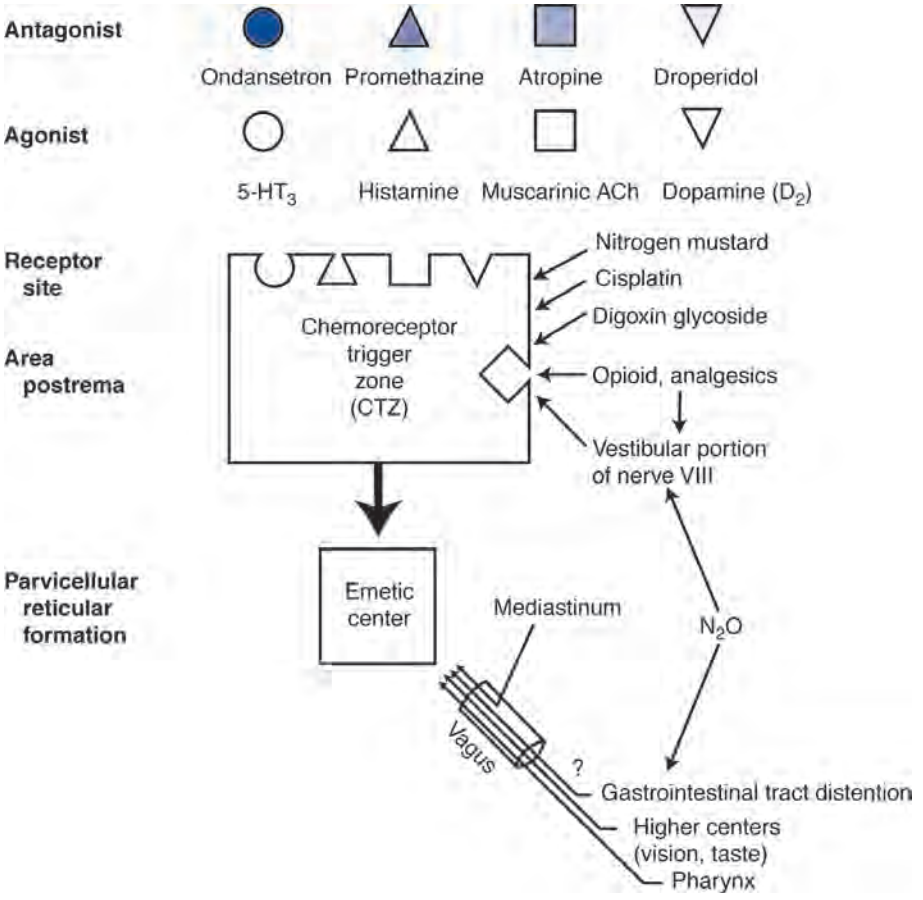


FIGURE 3-11. Diagrammatic representation of the chemoreceptor trigger zone (CTZ). 5-HT_3 = 5-hydroxytryptamine (serotonin); N_2O = nitrous oxide. (Adapted from Watcha MF, White PF. Postoperative nausea and vomiting. Anesthesiology 1992;77:162–184.)

history of PONV. Avoidance of known nausea triggering agents such as N_2O, ketamine, and longer-acting opioid medications may also reduce PONV.

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CHAPTER 4

Outpatient Anesthesia

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Anesthesia is an integral component of office-based oral and maxillofacial surgery. In regards to office-based dental surgery, it is what distinguishes the oral and maxillofacial surgeon. The goal is to provide anxiolysis, amnesia, sedation, and/or hypnosis to establish a comfortable and cooperative patient who is hemodynamically stable. This chapter reviews the various principles of anesthesia.

LEVEL OF SEDATION

Sedation and analgesia include a continuum of states of consciousness ranging from minimal sedation (anxiolysis) to general anesthesia. Table 4-1 lists the definitions of levels of sedation as defined and adopted by the American Society of Anesthesiologists (ASA).[1] These levels of sedation are not dependent on the route of drug administration nor the specific anesthetic agent or combination of agents administered.

GOALS OF SEDATION

When considering an anesthetic plan, it must be customized to the patient in order to achieve certain goals. These include (1) provide an optimal environment for completion of the surgical procedure, (2) minimize patient anxiety and optimize patient comfort, (3) control the patient's behavior and movement and optimize patient cooperation, (4) optimize analgesia and minimize patient discomfort and pain, (5) maximize the potential for amnesia, and (6) optimize patient safety, while maintaining respiratory and hemodynamic stability. One must consider both the patient and the specific

TABLE 4-1. **American Society of Anesthesiologists' Definitions of General Anesthesia and Levels of Sedation/Analgesia**

	Minimal Sedation (Anxiolysis)	Moderate Sedation/ Analgesia (Conscious Sedation)	Deep Sedation/Analgesia	General Anesthesia
Responsiveness	Normal response to verbal stimulation	Purposeful response to verbal or tactile stimulation	Purposeful response after repeated or painful stimulation	Unarousable, even with painful stimulus
Airway	Unaffected	No intervention required	Intervention may be required	Intervention often required
Spontaneous ventilation	Unaffected	Adequate	May be inadequate	Frequently inadequate
Cardiovascular function	Unaffected	Usually maintained	Usually maintained	May be impaired

Minimal sedation: Also known as ***anxiolysis.*** A drug-induced state during which the patient responds normally to verbal commands. Cognitive function and coordination may be impaired. Ventilatory and cardiovascular functions are unaffected.
Moderate sedation/analgesia (conscious sedation): A drug-induced depression of consciousness during which the patient responds purposefully to verbal command, either alone or accompanied by light tactile stimulation. No interventions are necessary to maintain a patent airway. Spontaneous ventilation is adequate. Cardiovascular function is usually maintained.
Deep sedation/analgesia: A drug-induced depression of consciousness during which the patient cannot be easily aroused, but responds purposefully* after repeated or painful stimulation. Independent ventilatory function may be impaired. The patient may require assistance to maintain a patent airway. Spontaneous ventilation may be inadequate. Cardiovascular function is usually maintained.
General anesthesia: A drug-induced loss of consciousness during which the patient is not arousable, even to painful stimuli. The ability to maintain independent ventilatory function is often impaired. Assistance is often required in maintaining a patent airway. Positive-pressure ventilation may be required due to depressed spontaneous ventilation or drug-induced depression of neuromuscular function. Cardiovascular function may be impaired.
From American Society of Anesthesiologists. ASA Standards, Guidelines and Statements, October 2007. Available at http://www2.asahq.org/publications/p-106-asa-standards-guidelines-and-statements.aspx

surgical procedure to determine the depth or level of sedation required to achieve these goals.

PATIENT ASSESSMENT

The oral and maxillofacial surgery office is presented with a diverse patient population. In a prospective outcome study, Perrot and coworkers[2] provided some characteristics of this diverse population. An overwhelming number of patients were classified as ASA 1 (72.2%) with the remainder being ASA 2 (24.5%), ASA 3 (3.1%), and ASA 4 or 5 (0.1%). The mean ages of the patients were 35.2 ± 20.7 and 28.0 ± 16, and they received conscious sedation or deep sedation/general anesthesia, respectively. This represents a relatively healthy population and is consistent with what one would anticipate as a significant percentage of the typical oral and maxillofacial surgeons practice in the extraction of impacted third molars. However, this study did demonstrate that greater than 25% of the patients were ASA 2 or higher. The percentage of ASA 3 patients is relatively low because of the large number of patients managed in the outpatient office with ambulatory anesthesia and, therefore, does not reflect the absolute number of patients with significant medical conditions who are actually treated.

The oral and maxillofacial surgeon must complete a comprehensive preprocedural history and physical examination to identify the various issues that could potentially modify the anesthetic plan. Components of this form include age, airway assessment, last menstrual cycle/pregnant, weight/height/body mass index (BMI), substances, nil per os (NPO) status, medical history, medications, history of prior anesthetic (self or family), and adverse events. This chapter cannot discuss the multitude of diseases that our patients have; however, we do review aspects that are pertinent to the items listed previously.

Airway Assessment

Airway assessment is critical when determining an anesthetic plan. The most common mechanism of patient injury under anesthesia is secondary to airway compromise, with studies reporting that almost half of these events result in permanent brain injury or death. There does not appear to be a significant difference in the incidence of adverse events (usually due to inadequate oxygenation/ventilation) between monitored anesthesia care and general anesthesia. In order to avoid such patient injuries, it is critical to evaluate the patient's airway and identify the patient with a potentially difficult airway. There are several ways to determine the presence of a difficult airway, including both a history and a physical examination.

A patient's history should inquire as to difficulties with airway management during previous anesthetics, presence of obstructive sleep apnea (OSA), and previous surgeries on the airway. The physical examination focuses on several potentially contributing anatomic abnormalities, which have been shown to correlate well with increasing difficulty with intubation:

- Modified Mallampati test—based on the ability to visualize the uvula and facial pillars with the mouth open wide, tongue maximally protruded, neck extended forward into the sniffing position, while the patient phonates (Figure 4-1).
- Thyromental distance—a measurement from the mandibular menton to the prominence of the thyroid cartilage, with the neck fully extended. The distance should be at least 6 cm or approximately three ordinary fingerbreadths. A short thyromental distance equates with an anterior larynx that is at a more acute angle and also results in less space for the tongue to be compressed into by the laryngoscope blade.
- Mandibular retrognathia.
- Interincisal opening less than 3 cm (reduced temporomandibular joint [TMJ] mobility, trismus, scarring, fibrosis).
- Short neck.
- Neck circumference greater than 17 cm.
- Enlarged tonsils (grade 3–4).
- Limited neck flexion or head extension (e.g., rheumatoid arthritis, ankylosing spondylitis).
- Prominent upper incisors.
- Abnormal oropharyngeal/neck masses.
- Congenital, developmental, or acquired facial deformities (e.g., craniofacial syndromes, burns to the head and neck).

Used independently, none of these tests is sufficient to consistently predict the difficulty of airway intubation. However, when several different tests (i.e., Mallampati and thyromental distance) are combined, the sensitivity and positive predictive value of ease of intubation improves significantly.

The typical office-based oral and maxillofacial surgery patient is nonintubated, and therefore, the ability to ventilate the patient is considered far more practical and concerning. The incidence of being unable to ventilate a patient has been reported as high as 5% of the general adult population. Five independent predictors have been associated with impossible

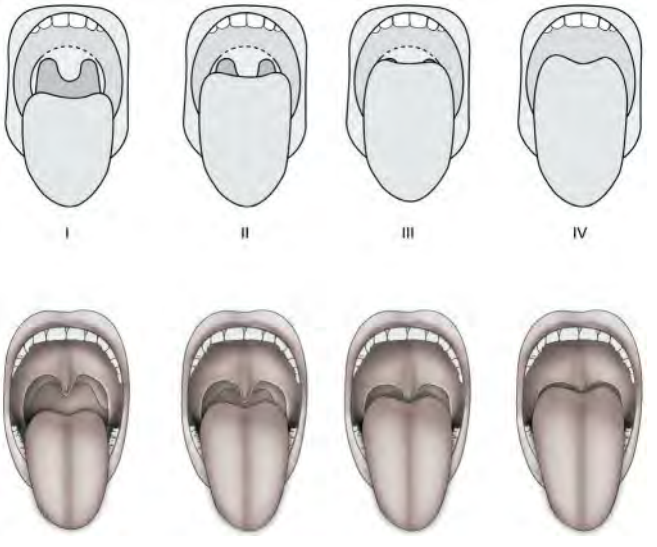


FIGURE 4-1. Mallampati classification.

mask ventilation: neck radiation changes, male sex, OSA, Mallampati 3 or 4, and presence of a beard. Certain features such as obesity, snoring, and lack of teeth were reported to potentially increase the difficulty of mask ventilation but could be overcome using various maneuvers and were not found to be predictors of impossible mask ventilation.[3]

The nonintubated patient during intraoral surgery is always potentially at risk owing to the proximity of the surgical site (oral cavity), resulting in airway obstruction from multiple contributing factors (mandibular position, head-neck flexion-extension, tongue position). It is only with the consideration of the previous historical and physical examination criteria that an overall anesthetic plan may be determined, taking into consideration the ease of potential airway embarrassment with a given level of anesthesia. Once all contributing criteria have been evaluated, the surgeon may decide to manage the patient with significant findings either with a minimal sedation in the office or plan on using advanced airway techniques (either a laryngeal mask airway [LMA] or intubation) either in the office or hospital setting. If the potential for a difficult airway is recognized, the appropriate preparations can be made to increase the likelihood of a good outcome.

Smoking

Approximately one quarter of the American adult population smoke cigarettes.[4] Cigarette smoking is associated with increased perioperative morbidity, likely secondary to its association with ischemic heart disease and chronic obstructive pulmonary disease (COPD). Other influencing factors on both the respiratory and the cardiovascular systems are the effects of cigarette's constituents, namely nicotine and carbon monoxide.

Cigarette smoking increases airway irritability, mostly through mucous hypersecretion and impaired mucociliary clearance. As a result, laryngospasm and airway obstruction are more common. A ventilation-perfusion mismatch can result from an increased closing capacity, secondary to small airway narrowing. The oxyhemoglobin dissociation curve undergoes a leftward shift, resulting in less oxygen released to the peripheral tissues. In addition, the blood carbon monoxide level increases, increasing the carboxyhemoglobin levels (1–10%), resulting in a lower oxyhemoglobin concentration. The net effect is decreased peripheral oxygen delivery.

Schwilk and colleagues,[5] in a large survey, compared specific respiratory events such as reintubation, laryngospasm, bronchospasm, aspiration, hypoventilation, and hypoxemia during anesthesia in smokers and nonsmokers. The incidence was found to be 5.5% in smokers compared with 3.3% in nonsmokers. They calculated the relative risk of these events occurring during anesthesia and found that the risk in all smokers was 1.8 times greater than in nonsmokers; in young smokers, it was 2.3 times; and in obese smokers, it was 6.3 times the normal. It was significantly noted that smokers have a significantly high risk of having bronchospasms during anesthesia. This risk was higher in female smokers, and the risk was 25.7 times normal in young smokers with chronic bronchitis. The incidence of adverse respiratory events, such as oxygen desaturation, has also been shown to occur in pediatric patients as a result of passive smoking. The adverse events are related to the cumulative number of cigarettes the child is exposed to.[6]

Patients are usually counseled to stop smoking during the perioperative period. To achieve the greatest benefit from this counseling, the period of smoking cessation should be at least 6 to 8 weeks. The benefit from abrupt cessation before surgery is related to the half-life of nicotine and carboxyhemoglobin, which are 2 hours and 4 hours, respectively. The elimination of nicotine and carboxyhemoglobin decreases adrenergic stimulation minimizing adverse cardiovascular effects and increasing both the oxygen-carrying ability of the blood and the amount of oxygen released to the peripheral tissues resulting in increased tissue oxygenation, respectively. Overnight abstinence of smoking is beneficial, however, because the half-life of both nicotine and carboxyhemoglobin can be prolonged during sleep; a 24-hour "cigarette smoke–free" period is preferable.

There has been controversy over smoking cessation beyond the initial 24 hours and a 6- to 8-week period. This controversy focuses on a paradoxical effect in which there is a transient increase in airway secretions and airway irritability associated with smoking cessation. Warner and associates[7] reported pulmonary complications such as purulent sputum, atelectasis, and pleural effusion in those that had stopped smoking for 8 weeks or more before surgery to be only 14.5%, compared with 57.9% in those who stopped smoking less than 8 weeks before surgery, which is a four times higher incidence of complications in those who stopped less than 8 weeks from surgery.[5] Mitchell and coworkers[8] reported the incidence of postoperative purulent sputum to be 25% more for those who stopped smoking 8 weeks before surgery and 50% more in those who stopped smoking less than 8 weeks before surgery than in nonsmokers. Warner and associates[7] found that if patients stopped smoking for 6 months or more before surgery, the incidence was the same as that in nonsmokers. Bluman and colleagues[9] found that risks were seven times higher in those who reduced smoking near surgery than for those who smoked continuously up to the time of surgery. These results indicate that smoking cessation the day before surgery is beneficial but cessation between this immediate 24-hour period and 8 weeks before surgery may not be beneficial with regard to postoperative pulmonary morbidity.

The polycyclic hydrocarbons present in cigarette smoke are potent inducers of liver enzymes. Smoking cessation decreases these metabolic enzymes, potentially increasing blood levels of drugs that interact with the cytochrome P-450 pathway. Coumadin is one drug that is metabolized by the affected enzymes, which may result in increased postoperative bleeding. Interestingly, postoperative nausea and vomiting (PONV) is less in smokers. This may be attributed to the induction of liver enzymes. These enzymes may be responsible for the metabolization and excretion of various chemicals that may affect PONV.

Obesity

Obesity warrants further comment because its incidence is rapidly increasing in the United States with an incidence in children and adolescents that exceeds 15% and in adults that is up to 40%. Clinically, obesity is associated with several diseases, including type 2 diabetes, hypertension, and coronary artery disease. Physiologically, the metabolic rate is proportional to body weight, resulting in an increased O_2 demand and increased CO_2 production and alveolar ventilation. Anatomically, obesity is associated with restrictive lung disease, secondary to (1) decreased chest wall compliance (which increases the work of breathing) and (2) decreased lung volumes of which the cephalad displacement of the diaphragm contributes. This is exacerbated by reclining the patient. The obese patient's functional residual capacity (FRC) may fall below the closing capacity, leading to alveolar collapse and a resultant ventilation/perfusion mismatch. Obesity is associated with relative hypoxemia and occasionally hypercapnea.

Several considerations are necessary when determining the anesthetic plan for the obese patient. The obese patient is up to four times more likely to develop intraoperative and postoperative pulmonary complications. Airway maintenance may be more difficult. There is a decrease in cervical and mandibular range of motion, there is an excess of lateral pharyngeal fat, and the airway is more likely to collapse. Intraoperatively, a relatively high fractional concentration of inspired oxygen (FiO_2) may be needed, while limiting respiratory depressant use. It is controversial if obesity is associated with increased difficulty in intubation. Recent literature has negated the concept of increased gastric content and decreased pH in the obese patient; however, difficult airway management may increase the risk of pulmonary aspiration.

Establishing venous access to administer intravenous drugs may also be more difficult. When administering intravenous agents, there is an increased volume of distribution of lipid-soluble drugs. Understanding the alteration in the pharmacokinetics in obesity is critical because it will affect the dosage administered and the duration of the effect on the patient. For midazolam and fentanyl, the loading dose is determined by the total body weight and the maintenance dose is determined by the patient's ideal body weight. Recovery from the drug is also affected. Because of the increased volume of distribution, the terminal elimination half-life of the drug is greater. For midazolam, this can result in an elimination half-life that is up to three times longer in the obese patient than in the ideal body weight patient. A propofol (sedative) infusion may be a good choice for the obese patient, because it has been shown not to accumulate.[10] The loading dose has been reported to be calculated based on ideal body weight plus 0.4 times total body weight with a maintenance infusion based on total body weight. Ketamine is another agent that has become popular in office-based anesthesia. The administration of this agent is based on ideal body weight.

There is a higher incidence of OSA in the obese patient. It is estimated that OSA is undiagnosed in 80% of patients who have the condition. Various assessment tools may aid the practitioner in identifying the patient with OSA. The STOP-BANG scoring model[11] consists of a combination of four subjective and four objective data points: (1) *s*noring—do you snore loudly? (2) *t*ired—do you feel tired, fatigued, or sleepy during daytime? (3) *o*bserved—has anybody observed you stop breathing during your sleep? (4) blood *p*ressure—do you have or have you been treated for high blood pressure? (5) *B*MI greater than 35 kg/m^2, (6) *a*ge older than 50 years, (7) *n*eck circumference greater than 40 cm, and (8) male *g*ender. It is reported to have a sensitivity of 83.6% for identifying mild OSA, 100% sensitivity for identifying severe OSA, and a negative predictive value of 90%. Identifying the patient with OSA is critical because it can be associated with adverse complications that may extend into the postoperative period. The ASA guidelines suggest that the "OSA patient be monitored for a median of three hours longer than their non-OSA counterparts before discharge from the facility." Further discussion is warranted. A recent publication sought to identify patients who would be at risk for postoperative problems.[12] These authors screened patients preoperatively using various parameters including neck circumference and snoring for OSA. Critical to the management of the patients was a postoperative observation period of 90 minutes (three 30-min intervals) in which the patient was monitored for apnea, bradypnea, desaturations, and pain-sedation mismatch. If the patient had any of these findings in at least two of the three 30-minute intervals, he or she had an incidence of postoperative problems over the next 48 hours up to 33%. If the patient was without any adverse events in only one or none of the 30-minute intervals, the incidence of a postoperative problem over the next 48 hours was as low as 1% for the patient with a low OSA score and up to 2% for the patient with a high OSA score.[12]

Nil per Os

The basic premise of preoperative fasting is to minimize the volume and the acidity of the residual gastric volume that, if regurgitated and aspirated during anesthesia, can result in laryngospasm, airway obstruction, and/or aspiration pneumonitis. Establishing parameters that minimize the risk of particulate gastric contents as well as decrease the quantity and acidity (pH < 2.5) of residual gastric fluids can decrease the incidence of this morbidity.

A basic knowledge of gastric-emptying times facilitates an understanding of these modifications. Gastric emptying for solid food is variable. A surgeon should be aware that approximately 50% of the contents of an ingested solid meal will have remained in the stomach after 2 hours. A 6- to 8-hour fast from solids is recommended to allow gastric emptying and minimize the risk of particulate aspiration. Eight hours remains the recommended minimum interval for

"fatty" foods. Alternatively, gastric-emptying time for clear liquids is approximately 10 to 15 minutes. After a 1-hour fast of clear liquids, approximately 80% of the consumed liquid is usually absorbed from the stomach. Numerous studies have shown that consumption of unlimited volumes of clear liquids by patients up to 2 hours before surgery does not significantly increase the quantity of gastric volume or gastric acidity.[13,14] Guidelines have, thus, been established for healthy patients that allow unlimited amounts of clear liquids to be consumed up to 2 to 3 hours before surgery. This recommendation avoids the need for an extended fast. Whereas beneficial in the adult, the ability to allow fluids up to 2 to 3 hours before the anesthetic is very beneficial in the pediatric patient because it can minimize patient irritability and discomfort and can decrease the incidence of hypotension secondary to dehydration. For the pediatric patient, it still may be simplest to state that the patient should be NPO after midnight and to schedule the procedure as the first case in the morning. Patients who are scheduled in the afternoon may have a light breakfast at least 6 hours before the surgery.

A preoperative evaluation assessing for co-morbidities may result in a modification of the general guidelines of 2 hours NPO for clear liquids and 6 hours NPO for a "light meal." Numerous situations may delay gastric emptying: high sympathetic tone as a result of pain and anxiety, opioid use, cigarette smoking, cannabinoids, and functional dyspepsia.

Emergency Treatment: Full Stomach

Patients may present to the office or emergency room requiring urgent care. The injury or the patient's ability to cooperate may be such that the necessary treatment cannot be completed on the patient while she or he is awake and nonmedicated, despite the fact that the patient is not NPO. The duration between the last food ingestion and the injury is the critical time period that is important in assessing a patient's risk of gastric aspiration. Each patient and situation must be assessed individually. If sedation or general anesthesia is required, patient management may necessitate the placement of an endotracheal tube to minimize the risk of gastric aspiration.

The following interventions may minimize the risk of aspiration and/or the ensuing injury that may result from gastric aspiration: an H_2 antagonist to decrease gastric acidity, a clear antacid such as sodium citrate to decrease gastric acidity, and metoclopramide to promote gastric emptying and increase the tone of the lower esophageal sphincter.

Pregnancy Testing

Because of the severity of the potential consequences of anesthetizing a pregnant patient, it is important to reliably detect a pregnancy. An accurate and reliable history in the educated patient can be effective tool to identify this possibility.[15] Typically, this entails asking female patients about their last menstrual cycle and whether they are pregnant or could be pregnant. The inquiry must be made to all females of childbearing age because the incidence of pregnancy detected by routine universal testing in the ambulatory surgical adolescent between 12 and 21 years of age has been reported to be approximately 0.5%.[16] Assessment must be tactfully done when working with an adolescent because patients in this age group may not provide an accurate history, especially in the presence of their family.

Routine pregnancy testing is done in some hospitals but is not standard of care in oral and maxillofacial practices. If routine testing is implemented, there is the potential for a false-positive test result, which may have significant emotional consequences. The issue remains controversial.

Substances

Substance abuse is a problem affecting all ages. This section makes general comments and focuses on the young patient, which is a significant percentage of ambulatory oral and maxillofacial surgery. Substance abuse among children and teens has reached epidemic proportions, regardless of socioeconomic status. In 2001, an estimated 15.9 million Americans ages 12 or older were current illicit drug users, meaning they had used an illicit drug during the month before the survey interview. This estimate represents 7.1% of the population ages 12 years old or older. Among youths ages 12 to 17 years, approximately10% were current illicit drug users. Data from 1999 to 2001 identify marijuana as the most popular abused drug, with a use approximating 7% of this population. Other abused substances included psychotherapeutic agents (~3%), cocaine (~0.5%), hallucinogens (~1%), and inhalants (~1%). An adequate history taking before anesthesia regarding substance use and abuse is, therefore, mandatory with all patients. This history allows for a safer selection of anesthetic agents and improved management of any perioperative complications.

Marijuana

Marijuana is the most commonly used nonalcohol illicit drug. Approximately 56% of current illicit drug users consumed only marijuana, 20% used marijuana and another illicit drug, and the remaining 24% used an illicit drug but not marijuana in the past month. Patients who use marijuana may present with anxiety, panic attacks, and sympathetic discharge. Adverse effects of marijuana include immunodeficiency and upper airway hyper-reactivity. Cases of laryngospasms within 36 hours of its use have been reported.[17] A beta 2 adrenergic agonist such as albuterol may be considered to treat this increased airway reactivity. Other perioperative considerations include that marijuana potentiates opioid-induced respiratory depression, and barbiturate and ketamine recovery time may be prolonged. Marijuana increases heart rate by 20% to 100% shortly after smoking. This effect can last up to 3 hours. Myocardial depression can occur, and the threshold for sympathomimetic-induced dysrhythmias is lowered. A 4.8-fold increase in the risk of heart

attack in the first hour after smoking marijuana has been reported. This adverse effect may be due to the increased heart rate as well as the induced dysrhythmias.[18]

Alcohol

Alcohol is the most commonly used and abused substance (including teenagers). Most alcohol use by U.S. teenagers is in the form of binge drinking. Most long-term systemic effects of chronic alcohol abuse, including hepatic injury, pancytopenia, and the neurotoxic effects (seizures, Wernicke-Korsakoff syndrome), are not present in the preadult abuser. Nonetheless, laboratory examinations may reveal elevation of Υ-glutamyltransferase, which is usually the first liver enzyme to increase as a result of heavy ethanol ingestion. Hepatic damage owing to alcohol frequently results in an aspartate transaminase–to–alanine aminotransferase ratio greater than 1. A mean corpuscular volume greater than 100 is strong confirmatory evidence of alcoholism. Aspiration risk is significantly increased in the chronic alcoholic because alcohol stimulates gastric acid secretion and delays gastric-emptying time. In addition, the alcoholic patient may consume alcohol the morning of the procedure to quell the signs of withdrawal, thus negating the NPO status. Cardiovascular changes associated with chronic alcohol abuse result in alcoholic cardiomyopathy, with resultant tachycardia and unexplained atrial or ventricular ectopy. Alcohol abuse influences the choice of anesthetic agents used in an outpatient setting. Tolerance to anesthetic agents appears to develop in the chronic alcoholic. Altered liver function results in an increased toxicity with anesthetic agents that undergo hepatic metabolism. Prolonged activity and increased serum levels of both succinylcholine and local anesthetic agents are the result of decreased activity of plasma cholinesterase. Nondepolarizing paralytics are also prolonged in chronic alcohol abuse secondary to an increased level of acetylcholine. Intravenous agents should also include a benzodiazepine that compensates for the lack of GABAergic stimulation.

Amphetamine

Amphetamine, a racemic mixture of β-phenylisopropylamine, is an indirect sympathomimetic drug. It is a powerful central nervous system (CNS) stimulant with peripheral alpha and beta actions. The CNS mechanism of amphetamine appears dependent on the local release of biogenic amines such as norepinephrine from storage sites in nerve terminals. Acute amphetamine use dramatically increases anesthetic requirement and has been implicated in a case of severe intraoperative intracranial hypertension.[19,20] Chronic amphetamine use is associated with a markedly diminished anesthetic requirement.[21] This results from chronic stimulation of adrenergic nerve terminals in the peripheral nervous system and CNS that depletes CNS catecholamines. Refractory hypotension can result both intra- and postoperatively, requiring prompt pharmacologic intervention. There can be a diminished pressor response to ephedrine after chronic amphetamine use. This is due to catecholamine depletion in central and peripheral adrenergic neurons.

Cocaine

Cocaine is an alkaloid derived from the leaves of a South American shrub. The drug is snorted (intranasal), injected (intravenous), or smoked (inhaled). Its administration provides an intense euphoria. The medical effects from cocaine result from both acute intoxication as well as chronic use. CNS stimulation, hypervigilance, anxiety, and agitation are common in the acutely intoxicated individual. Cardiovascular effects may include tachycardia, arrhythmias, hypertension, and ischemia. Ischemic myocardial injury may occur, even in the young patient. These effects result from the inhibition of neural reuptake of dopamine, serotonin, and tryptophan; increased adrenergic activity; and blockade of the sodium conduction channels. Chronic cocaine abuse has been associated with ventricular hypertrophy, myocardial depression, and cardiomyopathy. Long-term use may also lead to contraction band necrosis. This phenomenon is associated with hypermetabolic conditions, such as cocaine abuse, hyperthyroidism, and pheochromocytoma resulting from continuous catecholamine concentration elevation. This condition predisposes the patient to dysrhythmias.[22] Patients may also manifest neurologic effects. A decrease in seizure threshold has been demonstrated in young adults. Ischemic cerebral vascular accidents may result from the hypertensive crisis potentiated by the cerebral vasoconstriction resulting from the increased serotonin levels. Respiratory complications associated with intranasal administration include sneezing, sniffing, and acute rhinitis. Pulmonary complications associated with inhalational administration include cocaine-induced asthma, chronic cough, pulmonary edema, and pneumopericardium. Acute intoxication may result in hypoxia owing to pulmonary vasculature vasoconstriction. High levels of cocaine may persist for 6 hours after nasal administration. Elective anesthetic management should be deferred for at least 24 hours after the patient has last used cocaine. Electrocardiographic monitoring is recommended in all patients owing to the potential for silent ischemia and arrhythmias. Anesthetic management may include control of preoperative anxiety with benzodiazepines. Consideration should be given to avoiding adrenergic stimulants such as ketamine and epinephrine-containing local anesthetics.

"Ecstasy"

Ecstasy (3,4-methylenedioxymethamphetamine [MDMA]) is a stimulant that has psychedelic effects that can last for 4 to 6 hours and is usually taken orally in pill form. The psychological effects of MDMA include confusion, depression, anxiety, sleeplessness, drug craving, and paranoia. Adverse physical effects include muscle tension, involuntary teeth clenching, nausea, blurred vision, feeling faint, tremors, rapid eye movement, and sweating or chills. There is also an added risk involved with MDMA ingestion by people with circulatory problems or heart disease because of MDMA's ability to increase heart rate and blood pressure.

The principle constituent of MDMA can produce robust deleterious effects on serotonergic functioning in animals, including serotonin depletion and the degeneration of sero-

tonergic nerve terminals.[23] Although MDMA has been characterized as a hallucinogenic amphetamine because of its structural similarity to mescaline and amphetamine, it rarely induces hallucinatory experiences, nor is it as potent a psychostimulant as amphetamine. Whether neurotoxicity also occurs in humans is unknown, but emerging evidence indicates that repeated ecstasy exposure results in performance decrements in neurocognitive function, which may be a manifestation of neurotoxicity.

Most ecstasy tablets contain MDMA; other commonly identified ingredients include ketamine, methylenedioxyamphetamine, amphetamine, dextromethorphan, and combinations of these drugs. Some tablets contain inert ingredients, whereas others contain phencyclidine hydrochloride (PCP).

Perioperative management may involve addressing several complications, the most common being syndrome of inappropriate antidiuretic hormone, and hyperthermia. Other less common but well-known potential complications include tachycardia, agitation, and nausea and vomiting. Monitoring for the stigmata of hyponatremia and hyperthermia supplements a well-performed preoperative history to determine which patients are at risk.

Inhalational Substances

Inhalation substance abuse is a problem usually associated with young patients including preteens. The 1997 Monitoring the Future nationwide survey reported that inhalant use is most common in the eighth grade, in which 5.6% of students used inhalants on a past-month basis and 11.8% on a past-year basis.[24] They may present with photophobia, eye irritation, diplopia, tinnitus, sneezing, anorexia, chest pain, and dysrhythmia. Before administering anesthesia, one must take into consideration hepatic, renal, bone marrow, and other organ pathology caused by halogenated and impure chemicals.

Lysergic Acid Diethylamide

Lysergic acid diethylamide (LSD), also known as "acid," is odorless and colorless, has a slightly bitter taste, and is usually taken by mouth. Often, LSD is added to absorbent paper such as blotter paper and divided into small decorated squares, with each square representing one dose. The effects of LSD are unpredictable. They depend on the amount taken; the user's personality, mood, and expectations; and the surroundings in which the drug is used. Usually, the user feels the first effects of the drug 30 to 90 minutes after taking it. Physical manifestations include mydriasis, hyperthermia, tachycardia, hypertension, diaphoresis, anorexia, and tremors. Extreme emotional variability may occur, with extreme delusions and visual hallucinations. LSD effects are prolonged, typically lasting for greater than 12 hours. "Flashbacks" with auditory and visual hallucinations may recur suddenly without reuse of the drug and may occur within a few days or more than a year after LSD use. Flashbacks usually occur in people who have used hallucinogens chronically or who have an underlying personality problem. However, otherwise healthy people who use LSD may also experience flashbacks. Long-term effects of chronic LSD include psychiatric disorders (schizophrenia, severe depression). It is difficult to determine the extent and mechanism of the LSD involvement in these illnesses. Perioperative anesthetic practice involves recognition of the potential psychiatric effects of LSD on patients and avoidance of potentially aggravating agents.

Phencyclidine Hydrochloride

PCP is a dissociative anesthetic that originally was synthesized for intravenous use. Because of its postoperative emergence reactions (i.e., hallucinations, prolonged abnormal level of consciousness, agitation), it fell out of favor, and its use as an anesthetic in humans was discontinued in 1963. PCP subsequently emerged as an oral drug of abuse. PCP is a commonly abused street drug that is sold under many different names and in various forms. It may be sold on the street in tablet or capsule form, as a powder, or as a solution. The PCP content in each form differs widely, commonly from 10% to 30%. "Angel dust," the powdered form of PCP, generally has a higher PCP content, occasionally reaching 100%. Angel dust may be sniffed, smoked, ingested, or injected intravenously. Percutaneous absorption also has been reported to occur in individuals handling PCP (e.g., law enforcement officers). Smoking remains the desired method of use; the substance commonly is sprinkled onto dried leaf material (e.g., marijuana, tobacco, oregano, mint) and then smoked. Perioperative anesthetic considerations include its sympathomimetic effects, similar to its congener, ketamine, with the potential for tachycardia, tachyarrhythmias, and a true hypertensive emergency. Maintaining normotension and avoiding sympathomimetics, which may exacerbate PCP's effects, are the standard for anesthetic management.

PATIENT ASSESSMENT—UNDERSTANDING THE CHANGES WITH AGE

The purpose of a preoperative evaluation is to compile information about the patient to establish the most optimal treatment plan. One needs to assess the psychological and behavioral development of the patient, obtain a medical history that identifies both acute and chronic disease processes, and determine the patient's preparation for surgery (e.g., cardiovascular status), while performing an appropriate physical examination dictated largely by the patient's medical history and age.

The Pediatric Patient

The anesthetic management of the pediatric patient presents the oral and maxillofacial surgeon with unique and different challenges from those with an adult patient. The surgeon must be aware of anatomic and physiologic differences, different pharmacokinetics and pharmacodynamics of most medications, and the unique psychological development of the child and his or her corresponding ability to cope with the

stress of the surgical experience. As the child matures, changes in these parameters occur; therefore, an understanding of the growth and maturation of the pediatric patient dictates the selection of the anesthetic technique and medications used in the patient's management.

Anatomic Considerations in the Pediatric Patient

Pediatric Respiratory System

Much of the uniqueness regarding anesthetic management of children in oral and maxillofacial surgery is focused on anesthesia delivered during intraoral procedures in which the patient is not intubated. Intraoral surgery in the anesthetized nonintubated patient presents a formidable and unique challenge. The foremost concern is that the surgical site—the oral cavity—is in close proximity to the pharynx, thereby rendering the patient susceptible to airway obstruction and irritation. These factors can result in a significant degree of hypoxia.[25] Such effects can be exacerbated by a decreased minute ventilation and airway tone secondary to sedative medication used during the anesthetic administration.

Anatomic differences unique to the pediatric upper airway increase the risk of airway obstruction. In the young child, the tongue is large relative to the size of the oral cavity. It is positioned higher in the oral cavity impinging on the soft palate secondary to the rostrally positioned larynx. Lymphoid hypertrophy with enlargement of the tonsils and adenoids between the ages of 4 and 10 years can also contribute to upper airway obstruction.

The lower airway, consisting of the trachea, bronchi, and alveoli, also differs between pediatric and adult patients. The trachea and bronchi are conduits in which gas is transported from the environment to the alveoli. The pediatric airway diameter is relatively smaller than that of the adult. Because resistance is inversely proportional to the radius of the lumen to the fourth power, there is an increased resistance. Narrowing of the airway secondary to secretions or edema will have a more profound adverse effect on airway exchange. The pediatric trachea is also more compliant. The increased compliancy makes the airway susceptible to collapse secondary to increased negative inspiratory pressure. This is significant because of the potential for airway obstruction in the nonintubated patient. When patients become obstructed, they attempt to overcome the obstruction by increasing the respiratory effort. In the child, an attempt to compensate for upper airway obstruction with increasing respiratory effort can cause collapse of the trachea and bronchial passages, which may paradoxically worsen the obstruction. The frightened child may already be at risk for airway collapse because crying tends to increase negative inspiratory pressure.

Anatomic differences between pediatric and adult patients diminish the efficacy of ventilation. In the child, each rib is angled more horizontally relative to the vertebral column; adults' ribs have a caudal slant. In addition, the accessory muscles are less developed in the child. This results in a less effective thoracic expansion and a greater dependence on diaphragmatic breathing. Upper airway obstruction in the young child occurring with sedation can result in a paradoxical chest wall movement, characterized by an inward movement of the chest opposing the expansile downward movement of the diaphragm. Greater energy is required, which can lead to fatigue and subsequent hypoxia.

Exchange of gas takes place within the alveoli. Closing volume, the volume of the lung at which dependent airways begin to close, is greater in the pediatric patient. The increased closing volume in the pediatric patient results in increased dead space ventilation. Thus, more energy must be expended to adequately ventilate the alveoli. The alveoli are also both smaller and fewer in number in the pediatric patient. The alveoli increase in number until around 8 years of life and continue to increase in size until full adult growth is reached. The number of alveoli may increase more than 10-fold from infancy to adulthood, with a resultant increase in surface area that can be as great as 60-fold.[26]

FRC is the volume of gas in the lung after a normal expiration and is related to the surface area of the lung. The pediatric patient has a diminished FRC expressed on a basis of weight.[27] This is illustrated by a minute ventilation–to–FRC ratio of approximately 5:1 in a 3-year-old and 8:1 in a 5-year-old compared with approximately 2:1 in an adult. FRC decreases further in the sedated patient. The FRC provides a pulmonary oxygen reserve. Because children have a higher metabolic demand and greater oxygen consumption, the decreased FRC results in a more rapid desaturation of hemoglobin during periods of respiratory depression. One model comparing the child with the adult concluded that an apneic period of 41 seconds in the pediatric patient would result in an arterial oxyhemoglobin saturation of 85%, compared with an apneic period of 84 seconds in the adult.[28]

Endotracheal Intubation There are also anatomic differences between the pediatric and the adult airways that influence intubation. A large tongue, rostral larynx, and long and narrow epiglottis make laryngoscopy and visualization of the glottic opening more difficult in the pediatric patient. Adenoidal hypertrophy can also result in hemorrhage or obstruction of an endotracheal tube, particularly during nasal intubation.

The narrowest part of the trachea in the pediatric patient is the cricoid cartilage, in contrast with the glottis in the adult. It is not until the age of approximately 10 to 12 years that the pediatric airway matures to that of the adult. In the pediatric patient, care must be taken when placing and securing an endotracheal tube to prevent impingement of the tip of the tube on the narrow subglottic region. Such impingement of the endotracheal tube on the tracheal mucosa can result in edema and tracheal narrowing causing increased airway resistance postextubation. Uncuffed tubes are used by most anesthesiologists for patients younger than 8 to 10 years. The arguments against cuffed tubes are that they increase the risk of airway mucosal injury and that an appropriately sized uncuffed endotracheal tube can provide an adequate seal at the level of the cricoid cartilage. Formulas exist for calculat-

ing the appropriate size of endotracheal tube ([age (yr) +16]/4) and the appropriate length of endotracheal insertion ([age (yr)/2 + 12]).[29] However, 28% of the time, the initially selected uncuffed endotracheal tube does not provide an adequate seal, and re-intubation may be necessary. An additional benefit in using the uncuffed tube is that a larger tube may be inserted, which causes less airway resistance and less breathing work. The argument for a cuffed endotracheal tube is that the fit can be adjusted and it can protect against aspiration. Ensuring that the cuff pressure does not exceed 25 cmH_2O, which is believed to be the mucosal capillary pressure, can minimize injury to the mucosa. When using an uncuffed tube, an air leak of 25 cmH_2O should be allowed.

The trachea is also shorter in the pediatric patient. It is not uncommon that head position is frequently changed during an oral and maxillofacial surgery procedure; this can cause the tube to become displaced out of the trachea or pass farther into the trachea and impinge on the mucosa overlying the cricoid cartilage. Change in head position, use of an endotracheal tube that is too large, and patient age between 1 and 4 years are three factors contributing to the reported 1% incidence of postintubation croup.[30]

Certain congenital anomalies are well recognized for their altered anatomy. Some of the most commonly encountered disorders are Crouzon's syndrome (hypoplastic maxilla—obligate mouth breather), Goldenhar's syndrome (micrognathia, vertebral anomalies), hemifacial microsomia (hypoplasia of mandibular condyle and ramus), Möbius sequence (micrognathia and limited mandibular movement), Pierre Robin's anomalad (micrognathia, glossoptosis), and Treacher-Collins syndrome (mandibular hypoplasia). These craniofacial anomalies may complicate ventilation and/or endotracheal intubation. For example, maxillary or mandibular hypoplasia may increase the difficulty in achieving a satisfactory mask fit. Anatomic differences in the nasal cavity may impair nasal ventilation. This can potentiate respiratory obstruction during an intraoral procedure in which a pharyngeal curtain is placed and the patient is dependent on nasal respiration. The tongue may be displaced posteriorly by either maxillary or mandibular hypoplasia, increasing the potential for obstruction.

Upper Respiratory Infection It is not uncommon for children to present for surgery with a runny nose. Reports of children presenting to surgery with or having recently had such symptoms state incidences as high as 22.3% and 45.8%, respectively.[31] Rhinitis is not a contraindication to general anesthesia. Alternatively, a child with a severe upper respiratory infection (URI; symptoms include a productive cough, fever, and mucopurulent discharge) should not be anesthetized. However, it is unclear whether a child with a mild URI or a child recovering from a URI should be anesthetized; therefore, it is important to differentiate between the diagnosis of rhinitis and an infective process.

Pathophysiologic changes in the pulmonary system secondary to a URI include increased nasal and lower airway secretions, increased airway edema and inflammation, and increased airway tachykinins. These pathophysiologic changes can result in laryngospasm, bronchospasm, severe coughing, airway hyperactivity, breath holding, diminished diffusion capacity, increased closing volumes, atelectasis, and postintubation croup. The elevated hyperactivity with associated bronchoconstriction and the increased closing volume compounded by a greater oxygen uptake (secondary to the inflammatory response of the infection) and a decreased FRC (which normally occurs with general anesthesia) increases the risk of hypoxemia.[32] Oxygen desaturation can occur both intraoperatively and postoperatively; the latter indicates the need for continued postoperative monitoring. URIs have also been demonstrated to cause respiratory muscle weakness that can persist for up to 12 days.[33] The pathophysiologic changes that contribute to these adverse respiratory events can persist for 4 to 6 weeks after the URI.

Traditional office-based ambulatory anesthesia in oral and maxillofacial surgery is dependent on spontaneous ventilation in the nonintubated patient. This is significant because the incidence of adverse respiratory events is less in a patient anesthetized with a facemask or LMA than in those with an endotracheal tube. However, surgery involving the airway has been shown to increase the risk of adverse respiratory events. Although intraoral surgery is not truly airway surgery, it encroaches on the airway and can cause airway irritability. The nonintubated patient undergoing oral or maxillofacial surgery is also susceptible to periods of hypoventilation and apnea, which cannot be corrected without interrupting the surgery. Kinouchi and associates[34] demonstrated that a patient with an active or recent URI requires approximately 30% less apneic time to desaturate than does a healthy patient.

In conclusion, the patient who presents for elective surgery with allergic rhinitis or a mild URI that is not of acute onset may be anesthetized in the office without an endotracheal tube. If the patient has a significant URI, the procedure should be rescheduled. Traditional guidelines suggest that the procedure should be rescheduled for 4 to 6 weeks later if the patient is to be intubated, but because many children have several URIs per year, trying to reschedule the surgery for a date when the child is without symptoms may be difficult.[35] Considering this, a delay of 2 weeks is probably acceptable before performing a short office-based minor dentoalveolar procedure in which the patient is not intubated.

Pediatric Cardiovascular System

The pediatric cardiovascular system has some significant differences compared with that of the adult. Each relevant physiologic difference is outlined later.

Cardiac Output Perfusion is dependent on cardiac output and peripheral resistance. Cardiac output is dependent on heart rate and stroke volume. The pediatric heart has less compliance than that of the adult, with minimal ability to alter stroke volume. Thus, pediatric cardiac output is largely dependent on heart rate (Table 4-2).

TABLE 4-2. **Means and Ranges of Normal Cardiovascular Function**

	AGE (YR)		
Function	2–6	7–13	14–18
Heart rate (bpm)	100 (80–120)	90 (70–110)	80 (55–95)
Systemic arterial pressure (mmHg)	75–115/50–75	95–125/60–80	105–140/65–85
Cardiac output (mL/kg/min)	150–170	100–140	90–115

Neural Innervation The myocardium is innervated by both the sympathetic and the parasympathetic nervous systems, with the parasympathetic nervous system having a greater influence in the pediatric patient than in the adult. In one retrospective study, the incidence of bradycardia during anesthesia was reported to be age related. The incidence of bradycardia was approximately threefold less in the 3- to 4-year-old than in the 2- to 3-year-old.[36]

Blood Pressure Blood pressure is the product of cardiac output and peripheral vascular resistance. The pediatric patient has less ability to alter peripheral vascular resistance; therefore, blood pressure is largely dependent on cardiac output. A bradycardia with resultant decreased cardiac output thus results in a decrease in blood pressure because the child cannot compensate by increasing peripheral vascular resistance.

Summary

These fundamental concepts clearly illustrate the increased potential risks associated with sedating the pediatric patient:

- The airway is more susceptible to obstruction, and the patient has less ventilatory reserve; these result in a more rapid oxygen desaturation (and hypoxia causes bradycardia).
- The pediatric patient has increased parasympathetic innervation, resulting in a more rapid onset of bradycardia (which may be influenced indirectly by respiratory impairment or directly by the sedative drugs).
- There is less cardiovascular compensatory ability, which results in hemodynamic instability.

Psychological Assessment

The perioperative period can be very stressful for a child. The child is confronted with an unfamiliar environment, unfamiliar people, apprehension about the unknown, and loss of control. The child fears separation from the parents, the threat of needles, the perception of impending pain, and the fear of mutilation. Younger children frequently cannot verbalize these concerns. Behavioral manifestations of perioperative anxiety may include hyperventilation, trembling, crying, agitation, and/or physical resistance. Children younger than 6 years of age frequently cannot comprehend the need for or benefits of the surgical procedure. Children older than 6 years or those who have better-developed social skills (e.g., acquired from day care programs) may be more capable of understanding the situation and expressing their concerns. If possible, an older child should be allowed to participate in determining the anesthetic treatment and should be exposed to the various induction techniques: intravenous, intramuscular, oral, and inhalation.

Adolescents may be more capable of comprehending the planned surgery and anesthetic management. However, they are not adults. They have the ability to demonstrate myriad behaviors and rapid mood changes. A paradoxical reaction to sedation in which the adolescent appears to become agitated after the administration of anxiolytic medication may necessitate a deeper level of anesthesia than what may have originally been planned. Another concern in the adolescent patient is the use of illicit substances. This has reached epidemic proportions with an estimated 10.8% of 12- to 17-year-old youths reported to be current illicit drug users in 2001.[37]

The presence of parents during the administration of the sedative agent may reduce the stress of the procedure and improve the child's cooperation. Conversely, a parent's anxiety may be sensed by the child, further exacerbating the child's own level of anxiety. Clear, simple, and succinct explanations appropriate for the age of the child may minimize adverse behavior.

The Geriatric Patient

The number of elderly patients is increasing as defined by chronologic age. From a chronologic perspective, older adults have been classified as young-old (65–74 yr), mid-old (75–84 yr) and the oldest-old (≥85 yr).[38] This grouping is supposed to indicate increased risk among the oldest-old. It is founded on the principle that intrinsic biologic or physiologic aging results in a progressive impairment of function that occurs essentially in every organ system with age. This impairment results in diminished reserves. The diminished reserves may not be manifested under normal circumstances. However, under stressful conditions, which may occur during surgery and anesthesia, the diminished reserves may prevent a compensatory response. Therefore, chronologic age may suggest an increased vulnerability.

Chronologic age as a determinant of anesthetic risk, however, is limited because many older healthy patients may do better than their younger counterpart who is in poor health with chronic disease. This heterogeneity among individuals is because chronologic age does not take into consideration that many individuals have co-morbidities. With age, individuals develop chronic disease and various health habits (e.g., smoking). Certain diseases become more prevalent with age. These chronic diseases and extrinsic factors also contribute to diminished reserves in older adults. Each component of the aging process is not independent of the other. Physiologic aging may hasten the progression of the chronic disease and the chronic disease may hasten the physiologic aging. The presence and number of coexisting diseases, and their severity, are better predictors of adverse outcome rather than age alone.[39,40]

Surgical morbidity and mortality in the geriatric patient also varies widely dependent on surgical procedure. Emergency surgery, major vascular surgery, or surgery with significant blood loss or fluid shifts is associated with increased risk. Office-based dentoalveolar surgery is categorized as low risk by the current guidelines for the cardiovascular evaluation of patients undergoing noncardiac surgery.[41]

Cardiovascular

The surgeon must be cognizant of alterations in both anatomy and physiologic function, as well as the pathogenic age-related diseases that affect the cardiovascular system.[42] There is a higher prevalence of pathogenic age-related cardiovascular diseases in the geriatric patient such as coronary artery disease, prior myocardial infarction, congestive heart failure, hypertension, and arrhythmias (including atrial fibrillation). Of these, a history of congestive heart failure is associated with the highest incidence of perioperative complications. The surgeon must appreciate the physiologic changes associated with each disease and the impact that it may have on anesthetic management. For example, tachycardia, which increases the work of the heart and decreases oxygen delivery, may be secondary to anxiety (inadequate anxiolysis) or an adverse drug effect (e.g., epinephrine in the local anesthesia or a sympathomimetic effect of ketamine). Selected drugs may require modified doses or be contraindicated because of underlying diseases. Pathogenic age-related diseases are discussed in more detail in Chapter 2.

This section focuses on changes associated with aging. With aging, there is a decrease in the distensibility of the aorta and systemic arteries. The reduced vascular elasticity results in an increase in systolic blood pressure and an increase in afterload and left ventricular hypertrophy. There is a decline in autonomic function and the patient has a diminished heart rate response. There is also a diminished ability to increase ejection fraction in response to stress. This is partially secondary to the ventricular filling being more dependent on atrial contribution. The decrease in compliance of the capacitance vessels, a contracted intravascular volume, and diminished autonomic function can contribute to intraoperative blood pressure lability. Decreased cardiac output also contributes to a decrease in the delivery of drug. Clinically, this results in a slower onset of the drug's effect and a slower redistribution.

Pulmonary

Pathogenic age-related diseases or habits affect the respiratory system of the geriatric patient also. Common comorbid conditions that are more prevalent in the geriatric patient include COPD, smoking, and Parkinson's disease. Parkinson's disease has several clinical manifestations. One component is an impaired cough reflex and dysphagia, which increases the risk of aspiration. Functional changes of the respiratory system with aging include the conducting airways, the pulmonary mechanics, the lung parenchyma, gas exchange, and control of ventilation.

The functional changes of the respiratory system have several potential implications on the anesthetic management of the geriatric patient. The geriatric patient has decreased pharyngeal muscular support and the integrity of the upper airway from the nose to the trachea is predisposed to obstruction. Opening and/or maintaining the airway, which is frequently achieved with neck extension, is limited due to cervical arthritis. The geriatric patient is at increased risk for aspiration. Several factors contribute to this. There is an increased incidence of regurgitation secondary to a depressed lower esophageal sphincter tone and diminished gastric-emptying time.[43] The protective laryngeal reflexes are diminished, requiring a greater stimulation to trigger a response, and the patient has a decreased cough reflex.[44] A decreased number and activity of respiratory cilia additionally compromise the ability to maintain airway protection.[45]

As the patient ages, there are changes in chest wall mechanics. Calcified articulations of the costal cartilage, sternum, rib, and vertebrae result in a progressive decrease in compliance. There is also a narrowing of the intervertebral disk space and vertebral fractures associated with osteoporosis, which both decrease chest wall compliance and flatten the diaphragm. Muscle contraction is less efficient with a flattened diaphragm. This is compounded by a decrease in respiratory muscle strength with age, which is further reduced with malnutrition. When comparing the healthy geriatric and young adult male patient, the diaphragm strength is reduced by about 25%.[46] The effect is up to a 30% increase in effort to expand the lungs in a 60-year-old compared with a 20-year-old.[47]

Structural changes occur in the alveolus to impede efficient gas exchange. The alveolar surface area decreases 15% by age 70.[48] Changes in the elastic recoil forces in the lung parenchyma result in an increase in closing volume, which produces air trapping and ventilation-perfusion mismatches. The net effect is a progressive decrease in arterial oxygen with age.

Ventilation is controlled centrally in the brainstem and peripherally within the carotid and aortic bodies. Physiologic changes occur with aging that decrease their sensitivity and responsiveness. The geriatric patient has an impaired response to hypoxia and hypercarbia. This decreased sensitivity is associated with irregular breathing patterns and apneic spells, which may occur even during sleep. Exaggerated responses may occur with anesthetic agents.[49]

Urinary and Hepatic System

The renal and hepatic systems have functional decline in the geriatric patient. These changes in turn impact the bioavailability, pharmacokinetic, and pharmacodynamic effects of the anesthetic drugs.

Many drugs are bound to plasma proteins produced by the liver. For example, acidic drugs such as diazepam bind to albumin. An alteration in these protein concentrations results in a change in the percentage of bound and free drug. Structural changes may occur in the proteins that reduce their

efficiency to bind drugs. An increase in the amount of free drug may result in an increase in pharmacologic effect. This initial effect may be offset by the increased amount of free drug in the plasma that is available to be cleared.

The liver is also involved in the metabolism of drugs. The hepatic metabolism of drugs is classified as phase 1 or phase 2. Phase 1 drug metabolism is achieved by oxidation, reduction, and hydrolysis reactions. It occurs within the liver in what is referred to as the *cytochrome P-450 enzyme system.* Phase 2 drug metabolism is achieved by conjugation and acetylation reactions. Phase 1 drug metabolism has been reported to decline with age; however, this is controversial and some studies have shown no alteration in the cytochrome P-450 enzyme system.[50] The dysfunction, however, may be more related to co-morbidities, such as tobacco, alcohol, sedentarity, or environmental factors. These factors may contribute to the variability of hepatic metabolism in the elderly patient rather than parenchymal changes in the liver. Phase 2 drug metabolism is believed to be unaltered with age.

Clearance of drugs is also affected by hepatic blood flow. This is dependent on the ability of the liver to remove a drug. Drugs that are removed rapidly by the liver or those that are classified as having a high extraction ratio are removed by the liver at a rate comparable with hepatic blood flow. Drugs that are removed slowly by the liver or those that are classified as having a low extraction ratio have their removal influenced more by intrinsic factors within the liver. With aging, there is a decline in liver blood flow, such that by the age of 65 years, the reduction in hepatic blood flow approximates 40% of what it is in an individual 25 years of age.[51] Disease states that alter hepatic blood flow, such as congestive heart failure, will also affect hepatic drug clearance. Therefore, drugs that have a high hepatic extraction ratio will have a reduction in clearance and those with a low hepatic extraction ratio will not have a decrease in clearance secondary to a decrease in hepatic blood flow. Drugs common to office-based anesthetic practice that have a high extraction ratio include fentanyl, meperidine, ketamine, and propofol; drugs with an intermediate hepatic extraction ratio include morphine and triazolam; and those with a low extraction hepatic extraction ratio include diazepam

There is about a 1% to 2% decline in blood flow to the kidney per year of age and a significant loss of functional nephrons. By the age of 70 years, a patient may have a 50% reduction in renal function.[52] Several drugs have active metabolites that are eliminated by the kidneys. These drugs may have prolonged duration in the elderly.

ANESTHETIC CONCEPTS

Monitoring

Monitoring is defined as a continuous observation of data to evaluate physiologic function. The rationale for such is to permit prompt recognition of a deviation from normal so that corrective therapy can be implemented before morbidity ensues. Various professional organizations have established guidelines for anesthetic monitoring. Standard parameters that are monitored during office-based anesthesia in the oral and maxillofacial surgeon's office include respiratory and cardiovascular function and depth of sedation.

Respiratory Monitoring

The role of respiratory monitoring is to maintain arterial oxygenation and adequacy of ventilation. Arterial oxygenation monitoring is not readily available and arterial oxygenation is inferred by monitoring oxyhemoglobin saturation using pulse oximetry. Pulse oximetry is highly accurate and provides a continuous account of peripheral arterial oxyhemoglobin saturation. The device is capable of rapidly detecting changes in oxyhemoglobin saturation. Although the device will rapidly detect changes, there are limitations to its rapidity in detecting clinical changes. Most anesthesiologists and oral and maxillofacial surgeons place the oximetry probe on the finger. The circulation of blood from the pulmonary system to the finger where the probe is located takes a finite time. This circulatory time results in an approximately 25- to 35-second delay in the monitor alerting the anesthetic team to the physiologic changes that may have occurred within the central circulation. The delay is longer in patients who have slower circulation, such as the geriatric patient.

The relationship between oxyhemoglobin saturation and arterial oxygen tension (Pa_{O_2}) also limits the efficacy of pulse oximetry. The following will provide insight into this limitation. Administration of supplemental oxygen is recommended for deep sedation and general anesthesia. For office-based oral and maxillofacial surgery, oxygen is delivered by either a nasal cannula or a nasal hood. An oxygen Fi_{O_2} of 0.4 in a patient with a 5% shunt should result in a Pa_{O_2} content of approximately 180 to 200 mmHg. The pulse oximeter monitor will register an oxygen saturation of 100% for this Pa_{O_2} content. Anesthetic agents depress respiratory drive and can cause upper airway obstruction, resulting in a potential for impaired ventilatory exchange. Because of the horizontal plateau of the oxyhemoglobin dissociation curve, the pulse oximeter will not detect the initial change in arterial oxygen content until the Pa_{O_2} sustains a significant decrease from the approximate 180 to 200 mmHg to approximately 100 mmHg. This point should not negate the importance and benefit that pulse oximetry provides but to emphasize that it is not the sole component of respiratory monitoring critical for the care of the patient.

Ventilatory monitoring is the other component of respiratory monitoring. Monitoring of ventilations can be accomplished with observation, auscultation of breath sounds (typically with a pretracheal stethoscope), plethysmography, and/or capnography. Whereas each of these has their benefit, it is the author's opinion that either auscultation or capnography should be routine instrumentation. The pretracheal stethoscope consists of a weighted stethoscope bell, conducting tubing, and a customized monaural earpiece. A wireless

modification of the stethoscope allows more than one person to monitor the volume-controlled breath sounds. The wireless technology also allows the breath sounds to be broadcast on a speaker, which would allow all involved in the surgery to be cognizant and participate in monitoring the patient's breathing. Auscultation of breath sounds allows the practitioner to monitor respiratory rate and the character of the air exchange (e.g., differentiating between normal unobstructed air exchange, partial obstruction, and bronchospasm).

Capnographic monitoring consists of capnography and capnometry. End-tidal CO_2 sampling can be achieved in a nonintubated patient (open system) using a nasal cannula (in which one prong delivers oxygen and the other prong samples end-tidal CO_2) or inserting a sampling tube into a nasal hood (which is used to administer an oxygen/nitrous oxide mixture). Capnometry is the numerical measurement of end-tidal CO_2. In the open system, there is moderate potential for dilution of the gases resulting in an artifactual decrease in end-tidal CO_2 values. Alternatively, elevated end-tidal CO_2 values should be noted and appropriate action taken based on their interpretation. In the open system, capnography provides a visual representation of the patient's ventilatory pattern. This graphic display provides the respiratory rate and a depiction of various respiratory patterns, which may suggest unobstructed ventilations, partial airway obstruction, hypoventilation, bronchospasm, and/or apnea.

Ventilatory monitoring is critical because changes in ventilation are the most likely contributing factor to a decrease in Pao_2. Most commonly, it is anticipated that the patient's ventilation will be decreased and that, with the administration of supplemental oxygen, the patient will maintain an adequate Pao_2 and oxygen saturation. However, ventilatory monitoring provides the practitioner with the information so that the anesthetic team can identify changes in ventilatory pattern (either rate or air exchange) and determine whether any action is required. In prior publications and unpublished data (Dembo and Bennett), it was suggested that the use of ventilatory monitoring would have prevented anesthetic morbidity and mortality.

Fluids

Fluid administration provides the ability to correct the preexisting fluid deficit, to replace ongoing fluid losses, and to compensate for perioperative fluid shifts between the intracellular and the extracellular spaces. Expansion of the intravascular compartment of the extracellular space may also be beneficial, in that it will compensate for the vasodilatory effects of many anesthetic agents. In minor surgery, there is minimal concern about fluid loss or fluid shift, both of which can be associated with major surgery. Nevertheless, there exists a preexisting fluid deficit secondary to the preoperative fasting guidelines. Variations in these fasting guidelines now allow clear fluid intake up to 2 to 3 hours before surgery. However, for the morning-scheduled patient, many surgeons and anesthesiologists still adhere to a restricted intake after midnight. The typical 70-kg adult patient may thus have a fluid deficit of between 1 and 2 L, which is a deficit primarily of free water as well as of a small quantity of sodium, potassium, and chloride electrolytes.

In the ambulatory setting, intravenous access is frequently established just before the commencement of minor surgery. Any of the various crystalloid solutions (e.g., 5% dextrose in water [D_5W], D_5/0.45%NS, D_5/0.9%NS, 0.45%NS, and 0.9%NS) could be used for the healthy ambulatory patient scheduled for minor surgery. However, it is important for the surgeon-anesthetist to understand the concept of fluid compartmentalization so that the solution most appropriate for infusion can be selected. Total body water exists in two major compartments, intracellular and extracellular. The intracellular compartment represents approximately two thirds of the total body water, and the extracellular compartment represents approximately one third of the total body water. The extracellular compartment is further subdivided into intravascular (one fourth) and extravascular fluid (three fourths) compartments. Among all of these compartments, there is a constant redistribution of free water dictated by ionic and osmotic pressure. The primary extracellular electrolytes are sodium and chloride. These electrolytes are not freely diffusible between compartments. Any alteration in the concentration of these electrolytes in a specific compartment will result in a compensatory shift of free water.

One possible physiologic choice for replacement of the fluid deficit is 0.45% saline solution. Infused over a 24-hour period, this would provide the daily sodium requirement (1–2 mEq/kg/day). The advantage of an isotonic solution (e.g., 0.9% NaCl) is the preferential distribution of the fluid within the extracellular space. This fluid will remain within the intravascular space for the first 15 to 30 minutes before equilibration within the extracellular compartment. This provides the advantage of countering the vasodilation secondary to anesthetic agents. D_5W, a hypotonic solution, is rapidly redistributed and leaves the intravascular space. It thus does not provide a physiologic benefit comparable with that of the saline solutions. In addition, the normal hormonal response to surgery produces an increase in blood glucose. The infusion of a dextrose solution may intensify this hyperglycemia, which may worsen neurologic injury in the face of hypoxemia.[53,54] The potential for such problems is unlikely in office dentoalveolar anesthesia. Inasmuch as many patients who have had dentoalveolar surgery may have a diminished intake for several hours, the inclusion of dextrose in the infusion fluid cannot be considered inappropriate. However, the dextrose should be combined with a solution such as 0.45% or 0.9% saline.

Dentoalveolar surgery is considered minor surgery. Intravenous access is often used solely as a route for drug administration, and the infusion may be limited to a low rate to maintain patency of the intravenous catheter. Despite the fluid deficit discussed previously, such a patient typically has a stable intraoperative course. However, attention to perioperative fluid rehydration may decrease postoperative

morbidity and improve the "quality" of recovery after office-based anesthesia in the nonintubated patient, with less residual sedation, headaches, drowsiness, fatigue, dizziness, and nausea and vomiting.[55]

It is important to understand some potential complications associated with fluid administration. Rapid and excessive administration of a hypotonic solution (e.g., D_5W) to a pediatric patient can result in cerebral edema and death. This can be avoided by administering an isotonic solution. Intravenous access provides the route to administer fluids and anesthetic drugs, but it also provides access to administer many emergency drugs. When emergency access is desired, it is most critical for it to be in place. Attention to some minor points will optimize the outcome if such an emergency were to occur. Intravenous access should be established with a catheter that is firmly secured. Once intravenous access is established there should be no rush to remove it once the surgery is completed.

Patient Positioning

The positioning of the patient has potentially significant physiologic consequences when under anesthesia. FRC is the volume of air present in the lungs at the end of passive expiration. FRC is the sum of expiratory reserve volume (ERV) and residual volume (RV) and measures approximately 2400 mL in a 70 kg, average-sized male. The FRC acts as an oxygen reserve in the event of impaired ventilation. In the erect patient, the thoracic expansion during inspiration is dependent upon the movement of the rib cage, the diaphragm, and the abdomen. Conversely, in the supine position, the diaphragmatic contraction is lessened owing to the pressure of the abdominal contents, as well as decreasing ability of the abdomen to expand during inspiratory efforts. This decreases the FRC by approximately 0.5 L. Several studies have demonstrated the benefit of head-up positioning of varying degrees on minimizing oxygen desaturation after preoxygenation and induction of anesthesia.[56,57] In addition to nonerect patient positioning, several other factors decrease FRC including obesity, pregnancy, restrictive pulmonary disease, and short stature.

Most oral and maxillofacial surgeons utilize, in an office-based setting, a seated patient positioning of approximately 35 to 60 degrees. This maximizes surgical access and ergonomic positioning for the staff and physiologically minimizes the decrease in FRC.

SEDATIVE TECHNIQUES—CONSIDERATIONS BASED ON AGE

Sedative Techniques in the Pediatric Patient

It is generally agreed that managing the anxious, uncomfortable, and uncooperative pediatric patient is one of the more difficult anesthetic tasks. The primary goals in the management of the pediatric patient include reducing anxiety, establishing cooperation, ensuring comfort, establishing amnesia and analgesia, and ensuring hemodynamic stability. Although the goals of sedation are similar for both the child and the adult, reducing anxiety in the adult may enhance cooperation, whereas in the child it may not. To achieve a satisfactory result and facilitate completion of the planned surgical procedure, the child may require a greater depth of sedation.

Sedation should be accomplished in as nonthreatening a manner as possible. Because some children may be intensely afraid of needles, establishing intravenous access may not be possible. The surgeon must be familiar with alternative techniques that allow for a safe satisfactory induction and recovery from anesthesia. Each case must be considered individually to select both the most appropriate drug and the route of administration. The surgeon must take into consideration the following factors in developing the anesthetic plan: (1) the age of the patient, (2) the level of anxiety and ability to cooperate with medical/dental staff, (3) the medical history of the patient, (4) the patient's prior surgical or anesthetic experience, (5) the infringement of the procedure on the airway, and (6) the duration of procedure. The selected technique should ideally be painless, be accepted by the patient and parents, be rapid in onset, be appropriate in duration with rapid recovery, and have minimal side effects and a broad margin of safety. If drug administration is associated with pain or adverse memories, the benefit of the sedation may be decreased. The anesthetic must also provide an environment in which the procedure can be completed. In certain clinical situations, a moderate degree of movement may be acceptable, whereas in other situations, no movement is acceptable. Also, the induction agent may establish a depth such that the treatment may be completed, but in other cases, the goal of the induction agent may be to establish sufficient sedation to allow intravenous access and maintenance of anesthesia with intravenous agents. Lastly, and of extreme importance, one is cautioned not to sedate a young child who will be transported in a car seat before arrival in the office. The respiratory depressant effect of the medication combined with the positioning of the unattended child in the car can result in unrecognized upper airway obstruction or respiratory impairment, with resultant death or significant neurologic impairment.[58]

Routes of Administration

Sedative medication may be administered by many routes, including oral, intranasal, transmucosal, rectal, intramuscular, inhalational, and intravenous. The advantage of the intravenous route is that it results in the most rapid onset, rapid offset, and predictable effect. The disadvantage is that it entails establishing intravenous access. A percentage of children do not cooperate and allow an intravenous catheter to be inserted. Many children report the needle puncture from either intravenous placement or intramuscular injection as the worst part of their care.

Even with a cooperative or an anesthetized child, gaining peripheral intravenous access can present a challenge. Proper

knowledge of venous anatomy with a controlled organized approach gives the best chance for success. Commonly accepted sites for venous cannulation include the dorsum of the hand, volar aspect of the wrist, antecubital fossa, and greater saphenous vein. Even when an alternative route (e.g., inhalation or intramuscular) is used to induce the anesthetic, we recommend the establishment of intravenous access. This can be achieved once the child is sedated. Even if the procedure can be accomplished without the administration of an intravenous agent, an established intravenous line can be used to administer intravenous agents if needed to augment the initial anesthetic agent or to prolong the duration of the anesthesia. The line can additionally be used to administer other medications required to manage adverse events.

In an emergent situation, if the traditional peripheral cannulation technique is not successful, the clinician has two possible access sites that allow for a high degree of predictability. These sites are the femoral vein and intraosseous access, which are associated with a higher incidence of morbidity. The femoral vein usually requires a 20- or 22-gauge angiocatheter. The intraosseous needle is recommended primarily for children younger than 6 years of age because they still have red bone marrow.(Figure 4-2). In this technique, a bone marrow needle or a no. 14 through 18 Cook intraosseous infusion needle is percutaneously inserted into the flat portion of the proximal tibia. Entry is made in the tibial plateau 1.5 cm below the knee joint and 2 cm medial to the tibial tuberosity. The special bone marrow styletted needle is inserted with a rotary motion into the bone until the cavity is reached. The depth of the needle insertion should be planned. If it is advanced too far, the needle penetrates the posterior cortex and does not allow infusion. The needle should be firmly set in the bone. Often, bone marrow may be aspirated to confirm the placement. A syringe or intravenous line can be attached; if it runs easily, placement is confirmed. Slight extravasation around the placement site should not prevent the use of the needle. The catheter can serve as a conduit for all intravenous fluids and drugs.

The inhalational induction of anesthesia with a potent anesthetic agent also provides rapid onset, rapid offset, and a predictable effect. The advantage of this technique, similar to the intravenous route, is the option to use short-acting agents, enabling the anesthetic state to be rapidly terminated at the end of the procedure.

The traditional inhalation induction is accomplished by administering oxygen or a mixture of oxygen (minimum concentration of 30%) and nitrous oxide using a full facemask. Induction can be achieved using one of two techniques. The potent vapor agent can be increased gradually every few breaths until the induction is complete. Alternatively, the patient may be immediately administered a high concentration of the potent inhalational agent. A modification of the latter technique is to ask the patient to exhale completely and then take a deep inspiration of the vapor

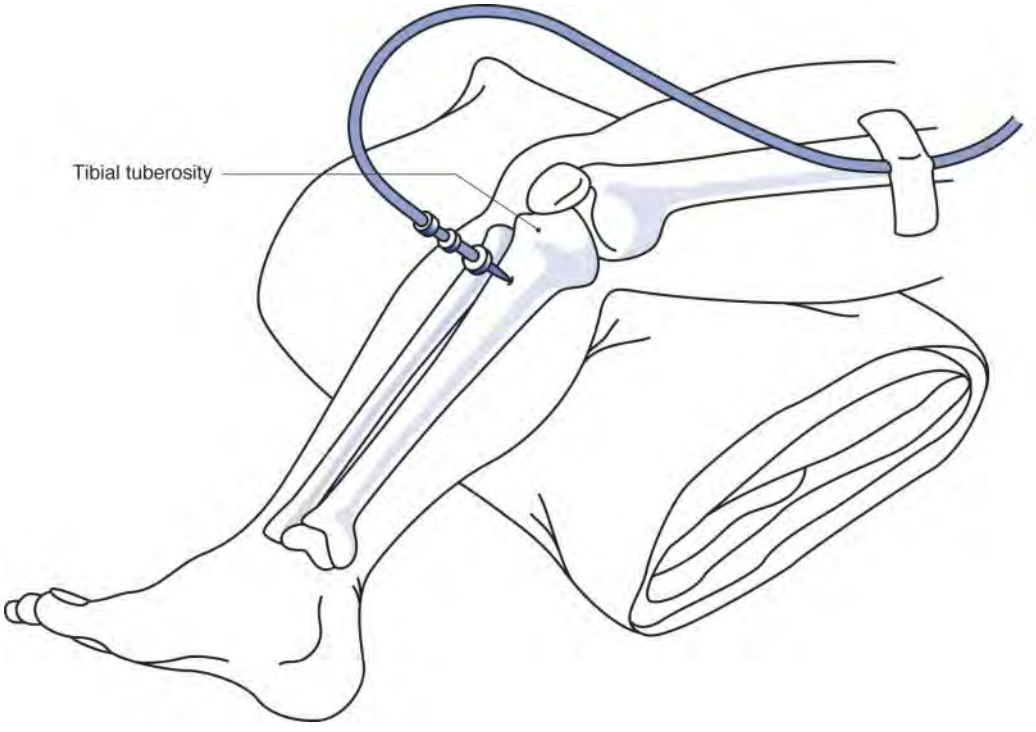


FIGURE 4-2. For intraosseous infusions a bone marrow needle or specially made intraosseous needle is inserted into the tibial plateau (just medial and down from the tibial tuberosity). The catheter is then secured and intravenous solutions and medications may be administered. Adapted from American Heart Association. Textbook of pediatric advanced life support. Dallas: American Heart Association; 1994.

agent and hold her or his breath. Induction will be achieved with a single breath, and spontaneous ventilation will resume once a state of general anesthesia is achieved. For brief procedures (e.g., extraction of a deciduous tooth), once general anesthesia is achieved, the facemask can be removed, the procedure performed, the facemask reapplied, and the patient allowed to awaken breathing 100% oxygen. Some clinicians advocate maintaining the general anesthesia by continuing the administration of the potent vapor agent via a traditional nasal hood. This can result in the delivery of a diluted concentration of anesthetic agent to the alveoli, resulting in a lightening of the patient's anesthetic depth. Such an occurrence would necessitate the interruption of the procedure to replace the full facemask to increase the alveolar concentration of the inhalational agent. Although the continued administration of the vapor agent via a nasal hood is not contraindicated, it may result in excessive environmental pollution, even with a scavenger device that is a component of the nasal hood. A circuit that scavenges the vapor agent must also be used with the facemask. To avoid these potential problems, especially for longer procedures, the establishment of intravenous access is recommended. The vasodilatory effects of the potent agent may optimize conditions for establishing intravenous access. Once access is set, anesthetic depth can be maintained with intravenous anesthetic agents.

There are a few disadvantages to inhalation induction. The vapor agent has a scent that may be objectionable to some. Applying a scent (e.g., scented lip gloss) selected by the child to the facemask may alter the odor of the agent. The odor may also be minimized if the child breathes through the nose as opposed to the mouth. In addition, inhalation induction is also dependent on the child accepting the facemask. Techniques such as asking the child to inflate a balloon may be employed to distract the child. Any need for mild restraint should be explained to the parent and may be used to facilitate induction in the younger child. However, in older children or extremely uncooperative children, the technique is dependent on the child's acceptance of the facemask. If excessive physical restraint is necessary, an alternative technique should be considered.

The intramuscular route of administration approximates the rapidity and predictability of onset of intravenous administration. Its primary disadvantage is the discomfort associated with the injection. However, for the uncooperative child, it may be the least traumatic method of inducing anesthesia. Four anatomic regions are used for intramuscular administration of drugs: the deltoid muscle, the vastus lateralis muscle, the ventrogluteal area, and the superior lateral aspect of the gluteus maximus muscle. These sites have been identified because they have minimal numbers of nerves and large blood vessels as well as adequate bulk to accommodate the volume of the injected medication. The rapidity of onset of the drug is dependent upon the perfusion of the muscle. Absorption and onset are also affected by the ionization of the drug and the vehicle in which it is dissolved.

Oral administration is considered by many to be the least threatening induction technique. Children are generally familiar with and readily accept oral medications. Oral administration also is generally well accepted by the mentally impaired or autistic patient. However, oral techniques have limitations. In one study of children between the ages of 20 and 48 months, one third of the children required that the medication be administered into the back of their throat with a needle-free syringe.[59] Although frequently used as a sole sedative agent by many surgeons, an oral sedative agent can be used as a premedicant before establishing intravenous access or inducing general anesthesia by a different route (e.g., inhalation or intramuscular). The limited volume of fluid administered with the oral medication is not associated with an increased risk for aspiration pneumonitis.

The primary disadvantages of oral sedation are the slow onset, variable response, and prolonged recovery. Injecting a sedative agent into the back of the throat with a needle-free syringe (when the child does not otherwise accept the medication) has also been associated with adverse consequences. It has been theorized that the drug intended for orogastric administration can be inadvertently aspirated by the crying child. Bronchial absorption can result in an excessive plasma level of drug.

The intranasal route was initially proposed for pediatric sedation because it was believed to avoid first-pass degradation, be rapid in onset, and be less traumatic than the other routes that possessed these same benefits. Medications administered intranasally do result in a rapid rise in the plasma level of a drug. This occurs because the nasal cavity, which functions to warm and cleanse nasal respirations, has a relatively extensive surface area with a thin nasal mucosa and an abundance of capillaries that facilitate the absorption of drug. The nasal mucosa also provides a direct connection to the CNS through the cribriform plate. Medication may be absorbed through the cribriform plate directly into the CNS through the capillary beds or the olfactory neurons or directly into the cerebrospinal fluid. Rhinitis or a URI may impair the absorption of a drug via this route.

The intranasal route, although initially thought to be less traumatic than alternative routes, is frequently not well accepted by children. The volume of medication used frequently results in a portion passing into the pharynx and being swallowed. Therefore, the unpleasant taste of the medication is not avoided, and the drug is subject to first-pass hepatic degradation. Midazolam is the most commonly intranasally administered medication, but the acidic pH is irritating to the nasal mucosa.

Transmucosal absorption has also been considered. The oral epithelium is thin with a rich vascular supply. The minimum epidermal barrier and the vascular supply provide an environment that promotes relatively rapid absorption of drugs. Oral transmucosal administration of a drug also has the advantage of avoiding hepatic first-pass degradation. Transmucosal administration requires cooperation of the patient to keep the drug in contact with the oral mucosa. The

medication may be administered as a solution placed sublingually or as a lozenge. At the present time, the only available lozenge that has an acceptable flavor and is commercially available is fentanyl citrate. Other sedative medications are bitter. Palatability can be improved by mixing these medications with a flavored solution that increases their volume; thus, the solution will be bitter or the volume will be excessive, neither of which is advantageous for the transmucosal administration of a liquid/solution. Many, if not most, pediatric patients expectorate the medication or prematurely swallow the liquid medication that is placed within the oral cavity as opposed to keeping it there.

Rectal drug administration has been used for the administration of antiemetics, antipyretics, and analgesics to both adults and pediatric patients. Many sedative drugs that are usually administered intravenously, intramuscularly, or orally can be administered rectally. Rectal administration may also be used in the management of emergencies. For example, rectal administration of diazepam is an acceptable route for the treatment of seizures.

The rectum is a flat organ that is usually empty. Its blood supply is derived from the inferior rectal arteries and is drained via the superior, middle, and inferior rectal veins. The superior rectal vein drains into the hepatic portal circulation via the inferior mesenteric vein. The middle and inferior rectal veins drain into the internal iliac vein. The internal iliac vein drains into the vena cava, thus bypassing the hepatic-portal circulation and avoiding first-pass metabolism by the liver.

The absorption of a drug that is administered per rectum is affected by several factors. The variable absorption of the drug may be partially influenced by the venous drainage of the rectum. Therefore, some individuals feel that absorption and subsequent peak plasma level of medication is dependent on the location of deposition of drug within the rectum. However, there are significant anastomoses between the three rectal veins, and peak drug blood level has not clearly been shown to be dependent on the location of agent deposition within the rectum. Solutions are absorbed more rapidly than are suppositories. A more dilute solution with greater volume provides more rapid onset and prolonged duration. Stool within the rectal vault as well as expulsion of an unmeasurable quantity of drug results in delayed or decreased absorption. Alteration in the integrity of the mucosa or the presence of hemorrhoids results in greater absorption. If a child is uncooperative, he or she may tightly close the anal sphincter during any aspect of the administration process. Excessive force in placing or removing the catheter may result in a laceration of the mucosa and cause a greater absorption of drug.

Pharmacologic Agents for the Pediatric Patient

The objective in selecting a pharmacologic agent is to choose an agent that establishes an appropriate environment to complete the surgical procedure. The effects sought in the pediatric patient include anxiolysis, amnesia, analgesia, immobilization, sedation, and hypnosis. Numerous agents are currently used by oral and maxillofacial surgeons and other practitioners. In this section, we discuss what we feel to be the most appropriate anesthetic agents and the routes by which they should be delivered.

Ketamine

Ketamine is a pharmacologic agent that induces a distinct anesthetic state that resembles catalepsy. The patient appears awake but is noncommunicative. Nonpurposeful movements may occur but are not disruptive. The eyes are commonly open with a blank stare and intact corneal and light reflexes.[60] A lateral nystagmus is also very characteristic. Ketamine also produces amnesia and analgesia. The clinical effect created by ketamine results from a dissociation between the thalamoneocortical and the limbic systems, which disrupts the brain from interpreting visual, auditory, and painful stimuli.[61] The analgesic effect, which occurs at subanesthesia plasma levels, is partially mediated by ketamine binding to the μ opioid and *N*-methyl-D-aspartate (NMDA) receptors. This is significant because the effect persists into the postoperative period and may decrease the need for postoperative analgesia.[62] Ketamine is also unique in its effects on the respiratory system. In clinical doses commonly used in oral and maxillofacial surgery, ketamine usually preserves upper airway musculature tone, spontaneous respirations, and FRC. This minimizes the incidence of upper airway obstruction and hypopneas/apneas and maintains the pulmonary oxygen reserve.[63,64] In contrast, most other anesthetics contribute to a decrease in muscular tone, respirations, and FRC. In addition to maintaining upper airway muscular tone, ketamine tends to better maintain the pharyngeal and laryngeal airway reflexes. This allows the patient to maintain the ability to swallow and cough, which minimizes the risk of pulmonary aspiration. Ketamine has also been shown to relax bronchial smooth muscle and cause bronchial dilation. It has been used in the management of wheezing during anesthesia.[65] Despite these benefits, the practitioner must respect the inherent dangers associated with the anesthetic management of a patient. Respiratory depression characterized by a decrease in respiratory rate and tidal volume can occur with ketamine. Respiratory arrest has been reported in a 4-year-old child following the intramuscular administration of ketamine 4 mg/kg.[66] However, respiratory depression is not common, and the occurrence of apnea is more likely to occur in infants or with the rapid intravenous infusion of an induction dose greater than 2 mg/kg. Slow intravenous infusion over 30 to 60 seconds of doses between 0.5 mg/kg and 1 mg/kg should minimize the incidence of significant respiratory depression. Aspiration of gastric contents can also occur despite the fact that ketamine better preserves the protective airway reflexes, allowing a patient the ability to swallow and cough.[67,68] The protective reflexes, although less impaired than with other drugs, are diminished. We feel that a patient who is considered not to have an empty stomach should not be sedated, and disagree with those who feel that preservation of the airway reflexes justifies sedating such patients.[69] The preservation of the laryngeal reflexes is a protective mechanism; this may

also contribute to airway complications. Ketamine produces an increase in salivary and tracheobronchial secretions, and the preservation of the laryngeal reflexes may predispose the patient to laryngospasm.

Ketamine has both direct and indirect effects on the cardiovasculature. The direct myocardial depressant effects are generally not seen in the healthy patient anesthetized in the office. The indirect effects, which are a result of a sympathetic stimulation, produce an increase in heart rate and blood pressure. The former may be more common in the pediatric patient. These effects are well tolerated in the healthy pediatric patient. These hemodynamic changes may be reduced when ketamine is combined with an anesthetic agent that tends to blunt sympathetic stimulation (e.g., benzodiazepines, propofol).

A disadvantage of ketamine is its stimulation of dreams and hallucinations described as "out of body" experiences, sensations of floating, and delirium.[70] Although the incidence is less in children younger than 16 years of age, the incidence may be as high as 10%.[71,72] Ketamine is also contraindicated in patients who may have a globe or intracranial injury because ketamine increases both intraocular and intracranial pressure.

Ketamine can be administered intravenously, intramuscularly, orally, intranasally, and rectally. We discuss only the intravenous, intramuscular, and oral administrations of ketamine. The advantage of intramuscular administration is that it does not require patient cooperation. The mild distress associated with the injection is brief because the drug has a rapid onset, within 3 to 5 minutes. Dosing recommendations up to 10 mg/kg intramuscularly have been described in various papers and texts. The larger dose clearly produces a general anesthetic state. For office-based or emergency department procedures performed by oral and maxillofacial surgeons, however, a dose of 4 to 5 mg/kg intramuscularly should provide effective dissociation. One investigation prospectively assessed pediatric patients requiring sedation for minor procedures in an emergency department and found that a 4-mg/kg dose provided effective sedation and immobilization for 86.1% of the children. A satisfactory quality of sedation was achieved with adjunctive local anesthesia for 97.2% of these patients, although 3.7% required mild restraint despite adequate sedation and an absent withdrawal response to pain. Only 2.8% of the patients required a repeat dose secondary to inadequate sedation.[73] Local anesthesia is an important component of any sedative technique used by oral and maxillofacial surgeons. Although this study demonstrated that it is not always required, incorporation of local anesthesia into the anesthetic plan minimizes the amount of other anesthetic agents required. The working time achieved from a 4-mg/kg dose of ketamine was 15 to 30 minutes. A disadvantage of intramuscular ketamine is that recovery is variable and can be quite long. Although the mean recovery time in this study was 82 minutes, recovery from injection to discharge at times took up to 3 hours.

Benzodiazepines can be administered concomitantly with ketamine. The purpose for coadministering a benzodiazepine is to reduce the amount of ketamine administered, reduce the incidence of ketamine-induced hallucinations, attenuate the cardiovascular effects of ketamine, and provide additional amnesia.[74] Coadministration of a benzodiazepine with ketamine may prolong recovery.[75] Midazolam produces a better reduction in unpleasant dreams than does diazepam.[76] The favorable pharmacokinetics of midazolam compared with diazepam also provides a more rapid recovery. In a prospective investigation, ketamine 3 mg/kg with midazolam 0.5 mg/kg was administered to pediatric patients requiring sedation for minor surgical procedures in the emergency department.[77] Although 30% of the patients who received this regimen manifested "intermittent crying," only 14% required additional medication to establish a satisfactory anesthetic state to allow completion of the planned treatment. Recovery for this regimen was at times prolonged. The level of sedation and immobilization is dependent on the planned procedure. Although the intent is to provide an atraumatic experience for the child, a mildly dissociative sedative and analgesic state compared with a deeper dissociative anesthetic state may be acceptable for a brief dentoalveolar procedure. The intent is to modify the patient's perception of the procedure. In this situation, the patient is not profoundly sedated and the practitioner has to tolerate some movement and possibly some vocalization. Ketamine 2 to 3 mg/kg intramuscularly should provide this desirable sedative depth. The lower dose of 2 mg/kg is advantageous in that recovery from injection to discharge approximates 60 minutes. For many children, the low intramuscular dose of ketamine provides a depth of sedation that allows the placement of an intravenous line. If necessary, the depth of sedation can then be modified using intravenous medications. Incremental doses of ketamine 5 to 10 mg intravenously can be administered to the sedated patient, with onset occurring within 30 to 60 seconds. The duration of sedation is 10 to 15 minutes. Although we have found that ketamine 2 mg/kg generally facilitates intravenous placement, one study reported that 31% of the children resisted intravenous placement with a dose of 3 mg/kg.[78] For the patient who remains combative and for whom intravenous access cannot be established, an additional dose of ketamine 1 to 2 mg/kg intramuscularly can be administered. If the child allows placement of an intravenous catheter (without any premedicant), a dose of ketamine 0.5 to 1 mg/kg intravenously administered over 30 to 60 seconds will establish dissociation. An anticholinergic agent (e.g., glycopyrrolate or atropine) is frequently coadministered with ketamine to decrease hypersalivation. Tachycardia and postoperative psychomimetic effects are problems associated with ketamine. Atropine, when combined with ketamine, produces a significantly higher heart rate compared with the effect of glycopyrrolate. As a tertiary amine, atropine crosses the blood-brain barrier and can, itself, produce postoperative delirium. A higher incidence of adverse emergence phenomenon, however, was not identified in studies comparing glycopyrrolate with atropine.[79,80] Both drugs can be mixed in the same syringe with ketamine for an intramuscular injection.

The peak effect of intramuscular glycopyrrolate occurs within 30 minutes, at which time the procedure is frequently completed and the patient is in the recovery phase of treatment. If an intravenous line is to be established after the onset of sedation, glycopyrrolate can be administered intravenously with a peak effect in approximately 1 minute. The dose of atropine is 0.1 to 0.2 mg/kg, with a minimum dose of 0.1 mg and a maximum dose of 0.6 mg. Glycopyrrolate is twice as potent as atropine. The dose is the same for both drugs regardless of the route of administration.

Ketamine can also be administered orally.[81] Bioavailability is approximately 17% after oral administration compared with 93% after intramuscular administration.[82,83] Onset of sedation occurs in approximately 20 minutes. Although doses reported have ranged from 3 to 10 mg/kg, a more consistent effect is achieved with doses greater than 6 mg/kg. In one investigation, oral ketamine 6 mg/kg was administered for sedating anxious pediatric dental patients with a mean duration of sedation of 36 minutes.[84] The quality of sedation was reported as good for 65% of the patients, and 100% of the treatment was completed. Mean recovery time was 56 minutes, with one child sleeping for 3 hours. Creating a state of deep sedation is dependent on using larger doses of medications. Ketamine 10 mg/kg orally was used as a premedicant in the management of pediatric patients undergoing invasive oncologic procedures. Approximately 50% of the patients were unresponsive at 60 minutes. This dose was ineffective in fewer than 10% of the patients.[85] Recovery, however, generally took 2 to 4 hours, with 20% of the patients being deeply sedated at 120 minutes after administration. Several authors have shown that the anxiolytic and sedative properties of midazolam 0.5 mg/kg result in a more clinically effective sedation than does ketamine 5 or 6 mg/kg.[86,87] The combination of oral midazolam and ketamine has also been described. This drug combination may provide effective sedation when oral midazolam has been ineffective. One study that demonstrated a greater efficacy with this combination used ketamine 4 mg/kg with midazolam 0.4 mg/kg.[88] The reported dosing regimens have varied from ketamine 4 to 10 mg/kg with midazolam 0.25 to 0.5 mg/kg. Situations may occur in the management of a mentally impaired, autistic, or older child in whom an intravenous line or an intramuscular injection cannot be administered without harm to the patient or the health care provider and who will not accept a facemask. Oral ketamine alone or combined with oral midazolam can be used to establish a cataleptic state, facilitating treatment of the combative patient.[89,90] It may be helpful to solicit assistance from the patient's caregiver or parent, because these individuals may be aware of strategies to ensure that the full oral dose is taken. Atropine or glycopyrrolate can be orally administered with ketamine; however, the time to peak decrease in salivation is 2 hours.[91]

Regardless of the route of administration, ketamine can establish a clinical effect described as a "chemical straight jacket." The catatonic state created by ketamine is different from that with other general anesthetic agents; ketamine, when used at the doses discussed previously, may not be considered to be a true general anesthetic. However, the anesthetic depth created by ketamine is not consistent with conscious sedation, and airway problems can occur. Therefore, appropriate anesthetic standards for deep sedation or general anesthesia must be followed.

Midazolam

Midazolam is a water-soluble short-acting benzodiazepine. As a class of agents, the benzodiazepines provide anxiolysis, sedation, and amnesia. Midazolam can be administered intravenously, intramuscularly, orally, sublingually, intranasally, or rectally. Because of its water solubility, intramuscular injection of midazolam is pain free, and absorption is predictable. Unlike ketamine, however, as a single agent, there is no unique anesthetic benefit to the intramuscular administration of midazolam. Intranasal administration of midazolam was popular in the past. It was once the most common intranasally administered medication. However, because of an acidic pH, it produces irritation to the nasal mucosa. The medication, if administered slowly, is discomforting, and if administered rapidly, passes through the nose into the nasopharynx and is swallowed. In a study that compared oral with intranasal administration of midazolam, children were found to be less tolerant of the intranasal administration.[92] Oral midazolam is probably the most widely used premedicant in children. The recommended dose of midazolam is 0.5 to1.0 mg/kg to a maximum of 20 mg. Midazolam 0.5 mg/kg achieves anxiolysis in 70% to 80% of patients. The anesthetic depth may be potentiated by the administration of nitrous oxide. The combined administration of 40% nitrous oxide with midazolam 0.5 mg/kg has produced deep sedation in 12% of patients.[93] Unlike ketamine, midazolam causes loss of airway muscle tone. Although airway obstruction is not common with doses of 0.5 to 1.0 mg/kg, airway obstruction has been reported after 0.5 mg/kg oral midazolam.[94] The incidence of airway obstruction may increase with the administration of nitrous oxide. In one study, the combined administration of 50% nitrous oxide and 0.5 mg/kg oral midazolam resulted in a 56% incidence of upper airway obstruction in children with enlarged tonsils.[95] With maintenance of airway patency, however, oral midazolam doses of 0.5 to 0.75 mg/kg generally do not result in a change in oxygen saturation, heart rate, or blood pressure.[96] The onset of effect of oral midazolam is within 20 minutes, and the duration of sedation is 20 to 40 minutes. Patients can generally be discharged within 60 to 90 minutes from the time the medication is administered. Midazolam is metabolized by the cytochrome oxidase system. Oral midazolam is subject to hepatic first-pass metabolism. Erythromycin, clarithromycin, protease inhibitors, azole antifungal medications, fluvoxamine maleate, and grapefruit juice alter this cytochrome oxidase system and result in a higher and a more sustained midazolam plasma level.[97,98] Higher doses of oral midazolam (0.75–1.0 mg/kg) are associated with a greater incidence of side effects. These include loss of head control, blurred vision, and/or dysphoria. A

paradoxical reaction may also occur in which the patient becomes more excited as opposed to sedated. This is more common in children and adolescents.[99]

Induction Agents

Methohexital and propofol are rapid-onset, short-acting agents that are effective for induction and maintenance of anesthesia. These are the primary anesthetic agents for general anesthesia in oral and maxillofacial surgery performed in an office. The pharmacology of these agents is discussed in Chapter 3. There are some important points to make relative to their use in the pediatric patient.

Methohexital is an ultrashort-acting oxybarbiturate. It can be administered rectally, intramsucularly, and intravenously. The advantage to the rectal administration of methohexital is that the drug is administered in the presence of the parents, and thus, the child is asleep before parental separation. Rectal administration, however, can be distressing, as discussed previously. Methohexital can also be administered intramuscularly. Administration is quite painful, and there is no advantage to its use in office-based anesthesia compared with other available intramuscular agents. Neither rectal nor intramuscular administration is generally employed in ambulatory oral and maxillofacial surgery offices. Most frequently, methohexital is administered intravenously. Interestingly, despite years of safe administration in this environment, the manufacturer's package insert states that the use of methohexital in the pediatric patient is not adequately studied and, thus, not recommended.

Propofol is an alkylphenol. Its characteristics include rapid onset and short duration of clinical effect, similar to those of methohexital. Its high clearance rate and minimal tendency for drug accumulation make it a more ideal anesthetic agent for ambulatory surgery in both adult and pediatric patients. In one study comparing propofol with methohexital for anesthesia in pediatric patients undergoing procedures in a dental chair, propofol was associated with a 9% incidence of ventricular arrhythmias compared with a 32% incidence associated with methohexital.[100] Clinical trials and case series have demonstrated propofol's efficacy in pediatric patients.[101–107] The proprietary formulation of propofol (Diprivan) is licensed by the U.S. Food and Drug Administration (FDA) for use in children older than 3 years in the surgical setting. Transient pain at the site of injection is reported in approximately 10% to 20% of patients given propofol. In the pediatric patient, this discomfort may result in gradations of movement, which may require restraint of the patient until induction is fully achieved. Propofol may also cause hypotension and bradycardia. The incidence is reported to be higher in the pediatric patient (17%) compared with that in the adult patient (3–10%). This usually is not detected in the adult oral and maxillofacial surgery patient when a relatively low initial dose (<1 mg/kg) is typically used to achieve deep sedation or general anesthesia. Pediatric patients frequently need to be more profoundly anesthetized. This requires the administration of a greater dose of propofol, which may result in a higher occurrence of hypotension or bradycardia in pediatric oral and maxillofacial surgery patients. Propofol may also cause excitatory movement or myoclonus, the incidence of which is greater in the pediatric patient (17% vs. 3–10%).The greatest potential concern with the use of propofol in the pediatric patient is that cases of fatal metabolic acidosis and cardiac failure, termed *propofol infusion syndrome*, have been reported in over a dozen children.[108–112] These incidents have all been associated with prolonged intubation and propofol infusions. A review by the FDA concluded that propofol had not been shown to have a direct link to any pediatric deaths.[113] Although the causal relationship between propofol and metabolic acidosis remains unproved, clinicians should be aware of the risk for this reaction in children and limit the dose and duration of propofol therapy accordingly.

TABLE 4-3. **Inhalational Anesthetic Agents**

		MAXIMUM ACCEPTABLE CONCENTRATION (%)	
Agent	Blood Gas Solubility	1–12 Yr	Adult
Nitrous Oxide	0.47	—	105.00
Halothane	2.40	0.87	0.76
Sevoflurane	0.69	2.5	1.70
Desflurane	0.42	7.98–8.72	7.30
Isoflurane	1.40	1.60	1.20

Adapted from Cauldwell CB. Induction, maintenance and emergence. In Gregory GA, editor. Pediatric Anesthesia. 2nd ed. New York: Churchill Livingstone; 1989.

Inhalational Agents

The origin of anesthesia is rooted within dentistry. The first anesthetic was nitrous oxide. Nitrous oxide has anxiolytic, analgesic, amnestic, and sedative effects.[114,115] Although not a potent anesthetic agent, nitrous oxide possesses a wide margin of safety and has few, if any, residual side effects. Another advantage of nitrous oxide is its low solubility. An anesthetic agent that has low solubility has rapid equilibration between the alveoli and the blood and the blood and the brain. This results in both rapid onset and anesthetic emergence. Also, nitrous oxide may be combined with other anesthetic agents. A deep sedative or general anesthetic state may be established with the co-administration of nitrous oxide and an oral or parenteral agent. This may result in respiratory impairment.

Although nitrous oxide may potentiate the effect of another agent, the discontinuance of it can, likewise, reverse the anesthetic depth and promote a more rapid emergence.[116–118] Although nitrous oxide lacks sufficient potency to solely induce general anesthesia, halothane, sevoflurane, desflurane, andisoflurane have sufficient potency to induce and maintain general anesthesia (Table 4-3). The primary benefit of an inhalational agent is for mask induction, and of the potent inhalational agents, only halothane and sevoflurane are nonpungent. These agents can be administered to an awake patient with minimal respiratory complications (e.g., coughing, breath holding, laryngospasm), whereas desflurane and isoflurane tend to irritate the airway if used for mask induction.[119–121]

The blood and tissue solubility of an inhalational agent are also important. These properties influence the speed of induction and emergence from anesthesia. Agents that have a low solubility in blood have a more rapid induction and shorter emergence time. The blood-gas solubility coefficients of desflurane, nitrous oxide, sevoflurane, isoflurane, and halothane are 0.42, 0.47, 0.6, 1.4, and 2.3, respectively. These figures imply a more rapid onset and emergence for desflurane, sevoflurane, and nitrous oxide.

Because all anesthetic agents affect the pulmonary and cardiovascular systems, it is important to understand these effects. All potent inhalational agents depress minute ventilation in a dose-dependent manner, with a resulting increase in partial pressure of carbon dioxide in arterial blood ($PaCO_2$). Clinically the practitioner will observe a decrease in tidal volume and a slight increase in respiratory rate. Although acceptable respiratory parameters can be maintained during spontaneous ventilations, of the two agents used for mask induction, halothane produces less respiratory depression than does sevoflurane.[122] Not all respiratory effects are detrimental. All inhalational agents are beneficial in that they produce bronchial dilation and are advantageous in the management of the patient with bronchospastic disease. All potent inhalational agents have myocardial depressant effects. The cardiovascular depressant effects are greatest with halothane use, which can result in hypotension and bradycardia. However, of greater significance is the ability of halothane to sensitize the heart to catecholamines with resultant dysrhythmias. One study reported that 48% of pediatric patients anesthetized with halothane had arrhythmias compared with 16% of those induced with 8% sevoflurane. Patients who had an incremental induction of sevoflurane had even fewer arrhythmias. Furthermore, of the arrhythmias associated with halothane, 40% were ventricular arrhythmias (consisting of ventricular tachycardia, bigeminy, and couplets); with sevoflurane, only 1% were ventricular arrhythmias (consisting of single ventricular ectopic beats).[123] The occurrence of these arrhythmias may also be associated with the administration of local anesthetics containing epinephrine. Halothane is the only inhalational agent that is associated with arrhythmias with clinical doses of epinephrine. A limit of 1 μg/kg of epinephrine in patients receiving halothane is recommended.[124–126]

Use of inhalational agents is advantageous in the oral and maxillofacial surgeon's office because they provide a general anesthetic state without intravenous access. Therefore, only agents that are pleasant and nonirritating to the airway can be used. Halothane has traditionally been the agent used by both anesthesiologists in the operating room and oral and maxillofacial surgeons in their offices. Sevoflurane appears to have the characteristics that most approximate the ideal inhalational agent, in that it is of sufficient potency, is nonpungent, has a low blood and tissue solubility, and has limited cardiorespiratory effects. Sevoflurane has replaced halothane in the operating rooms.

There are several variations in mask induction techniques. First, the inhalational agent may be administered with a combination of nitrous oxide and oxygen or 100% oxygen. The combination of nitrous oxide with the potent vapor agent decreases the percentage of vapor agent required to achieve an anesthetic depth. The decrease in minimum alveolar concentration (MAC) for halothane is significantly clinically greater than for sevoflurane. This most likely is related to the difference in solubility of the two potent inhalational agents. Another variation in mask induction pertains to the concentration of inhalational agent administered. The practitioner may administer an incrementally increasing concentration of an agent (e.g., increasing an agent by 0.5–1% after a few breaths) or a high initial concentration of an agent (e.g., sevoflurane 8%). Although one would expect that sevoflurane would have a more rapid speed of induction, the differences between sevoflurane and halothane have not been consistently demonstrated.[121,127] The difference in speed of induction appears to be less distinguishable when a high concentration of halothane is used.

Similar to speed of induction, anesthetic emergence is dependent on several variables. Agents that have a low blood solubility coefficient should have a shorter emergence time. Several studies have shown that desflurane, which has the lowest blood solubility coefficient, has a very rapid anesthetic emergence (5–7 min), and halothane, which has the highest blood solubility coefficient, has a more prolonged recovery (10–21 min).[128–132] Sevoflurane has been shown, although not consistently, to have a more rapid anesthetic emergence for intermediate- and long-duration anesthetics compared with halothane. However, typically, the required state of anesthesia for a pediatric dental procedure in the office is brief, lasting less than 10 minutes. Recovery from anesthesia is also dependent on the duration of the anesthesia. Clinical studies comparing sevoflurane and halothane for pediatric dental extractions lasting between 4 and 6 minutes have not demonstrated a more rapid recovery with sevoflurane.[133] In one study, in which children were subject to a 4-minute anesthesia, time to eye opening was 102 seconds with halothane and167 seconds with sevoflurane.[134]

The last factor that needs to be considered both in comparing sevoflurane and halothane and in selecting an anesthetic agent for the office is the toxicity of each drug. Halothane is metabolized in the liver to a trifluoroacetylated product, which binds liver proteins promoting an immunologic response that can result in hepatic injury.[135,136] The incidence, which may be as high as 1 in 6000 cases of anesthesia in adults, is significantly lower in the pediatric population. Sevoflurane, although not associated with liver toxicity, has been associated with the potential for renal toxicity.[137,138] The drug undergoes hepatic metabolism, which produces inorganic fluoride. However, the rapid elimination of sevoflurane minimizes the renal fluoride exposure, which probably accounts for the lack of clinical renal dysfunction, despite some reports of serum fluoride levels greater than 50 μmol. Renal injury has also been associated with the formation of compound A, which is a product of the reaction between sevoflurane and CO_2 absorbents. Most of the data,

however, suggest that compound A does not induce renal toxicity in humans.

Other Medications

Chloral hydrate is an alcohol-based sedative. It produces a sleep from which one is easily roused, in which the cardiorespiratory effects are consistent with those that occur with natural sleep. The onset of chloral hydrate is slow (30–60 min), its duration is variable (2–5 hr), and it lacks the anxiolytic effects of benzodiazepines. The sedative effect of chloral hydrate does not produce as favorable a work environment as the anxiolytic effect of a benzodiazepine.[139] Another disadvantage of chloral hydrate is that it is a gastric irritant and is associated with nausea and vomiting.

Antihistamines are commonly used in medicine and dentistry for their antipruritic and antiemetic effects. When used for these conditions, sedation is frequently an unwanted side effect. However, the sedative effects can be used to advantage, and antihistamines such as promethazine and hydroxyzine are frequently combined with other drugs such as chloral hydrate and meperidine to potentiate the sedative effect of the primary anesthetic agent and to provide antiemetic effects. The sedative effects of antihistamines may last between 3 and 6 hours and, when used alone, do not provide anxiolysis.

The oral transmucosal administration of a sedative medication is appealing. Fentanyl citrate is available as a lozenge on a stick. The recommended dose is between 10 and 20 μg/kg. Bioavailability is between 33% in children and 50% in adults.[140]

The difference in bioavailability results from the amount of drug swallowed and the amount of drug absorbed through the oral mucosa. The drug provides both analgesia and sedation. Onset of analgesia precedes the onset of sedation. Analgesia also lasts for 2 to3 hours, providing some postoperative pain control. Adverse side effects associated with the fentanyl lozenge include a high incidence of nausea and vomiting and pruritus. The major adverse effect associated with the use of fentanyl citrate is a higher incidence of respiratory depression than that seen with other sedative medications. The respiratory depression associated with the fentanyl lozenge may last beyond the sedative effect.[141]

Pharmacology of Intravenous Drugs in the Geriatric Patient

Pharmacokinetics is defined as what the patient does to the drug. *Pharmacodynamics* is defined as what the drug does to the patient. Various changes in the elderly will affect the pharmacology of anesthetic agents.

Drugs that are taken orally may have increased bioavailability because of a reduced hepatic first-pass effect. The distribution of drugs is modified secondary to a change in body composition consisting of a reduction in intracellular water and a gain in adipose tissue. The gain in adipose tissue results in a reservoir for lipid-soluble drugs. For lipid-soluble drugs, the volume of distribution is increased, resulting in a lower plasma concentration and delayed elimination. For less lipid-soluble drugs, the volume of distribution is decreased, resulting in a higher than expected initial plasma drug concentration and a greater pharmacologic effect. A smaller volume of distribution will also result in a quicker decrease in plasma concentration. In addition, most anesthetic agents are bound to plasma proteins. Plasma protein concentration, changes in anesthetic drug binding, and competition with other pharmacologic agents tend to increase the unbound fraction of drugs, which most frequently results in increased pharmacologic effect. Pharmacodynamic changes also contribute to the elderly patient requiring lower doses of anesthetic agents.

The elderly patient has an increased sensitivity to propofol, which results in a higher incidence of apnea and hypotension. A more profound effect may also result in too-rapid administration because of a lack of appreciation for the slow onset secondary to the slower circulation time. Other common drugs, such as midazolam, fentanyl, and ketamine, have prolonged durations.

PERIOPERATIVE COMPLICATIONS

Laryngospasm

Intraoral surgery in the anesthetized nonintubated patient renders the patient susceptible to airway obstruction and airway irritation. Such irritation can result in laryngospasm, which is the apposition of the supraglottic folds, false vocal cords, and true vocal cords. The laryngospasm may be sustained and may become progressively worse as the supraglottic tissues fold over the vocal cords during forceful inspiratory efforts. The incidence of laryngospasm is 8.7 per 1000 patients in the total population and 17.4 per 1000 in patients younger than 9 years of age.[142]

The treatment of laryngospasm depends on whether the airway obstruction is complete or incomplete. The single diagnostic feature that distinguishes complete from incomplete airway obstruction is simply the absence or presence of sound. If there are inspiratory or expiratory squeaks, sounds, grunts, or whistles, chances are the child has incomplete airway obstruction. Airway obstruction of either type requires initial treatment with a patency-preserving maneuver such as the jaw-thrust/chin-lift maneuver.

Because incomplete airway obstruction may rapidly become complete, signs and symptoms of obstruction (e.g., tracheal tug, paradoxical respiration) should be treated aggressively. The first maneuver is to apply gentle continuous positive airway pressure with 100% O_2 by facemask. An effective technique to deliver gentle positive pressure is to "flutter the bag." In this technique, the reservoir bag is very rapidly squeezed and released in a staccato rhythm, similar to what one would see with an atrial flutter of the heart. In essence, one performs a manual high-frequency oscillatory ventilation with this technique. If the patient improves, anesthesia and normal ventilation may be resumed. Overuse

of the high-pressure flush valve to fill the breathing circuit and anesthetic bag may dilute potent anesthetic gases (if being used) and lead to a lighter plane of anesthesia. In addition, high pressure applied to the airway may force gas down the esophagus and into the stomach, reducing ventilation even more. Positive airway pressure cannot "break" laryngospasm in the presence of complete airway obstruction and may, in fact, worsen laryngospasm by forcing supraglottic tissues downward to occlude the glottic opening.

For the laryngospasm refractory to continuous positive airway pressure, a neuromuscular blocking agent should be administered. The ideal agent should have rapid onset. For the nonintubated patient, rapid recovery is also desirable. Succinylcholine is the only neuromuscular blocking agent that provides these effects.

Succinylcholine

If intravenous access is available, succinylcholine 0.5 to 1.0 mg/kg is administered. If intravenous access is not available, succinylcholine (4 mg/kg) may be administered intralingually or intramuscularly.[143] Succinylcholine may cause bradycardia secondary to its muscarinic effect. When administering succinylcholine to a child who is hypoxemic, atropine 0.02 mg/kg should precede it to prevent a bradycardia secondary to the muscarinic effect of succinylcholine. The administration of atropine should also be considered before a repeat dosing of succinylcholine in the adult patient to prevent bradycardia.

Several potential complications are associated with the use of succinylcholine. These include myalgias, malignant hyperthermia, masseter muscle rigidity, and hyperkalemic cardiac arrest in patients with undiagnosed myopathies. In some children, the administration of succinylcholine can result in masseter muscle spasm. Masseter muscle spasm may indicate a susceptibility to malignant hyperthermia, but it can also be isolated and not progress to malignant hyperthermia. The anesthetic team needs to differentiate between an isolated spasm and a prodromal sign of an impending emergency to make a decision regarding the continuation of the anesthetic and surgical course. In a tertiary environment with appropriate monitoring, the anesthesia may be continued with observation for the development of other systemic signs reflective of the hypermetabolic state of malignant hyperthermia. Tachycardia is usually the earliest sign, whereas end-tidal CO_2 is the most sensitive sign of malignant hyperthermia.[144,145]

Another potential life-threatening complication following the administration of succinylcholine is hyperkalemic cardiac arrest. Hyperkalemic cardiac arrest follows the administration of succinylcholine in patients with undiagnosed myopathies; succinylcholine induces rhabdomyolysis, which causes hyperkalemia leading to bradycardia/asystolic rhythm. Several case reports have appeared in the literature emphasizing this potential risk in the pediatric patient, which exists because Duchenne's and Becker's muscular dystrophies may go undiagnosed until the ages of 6 and 12 years, respectively.[146,147]

Alternative neuromuscular agents have been developed that can provide rapid onset and should be used for elective situations. Rocuronium may be used when succinylcholine is contraindicated. Its onset is rapid, however, with a considerably longer duration. The administration of lidocaine topically to the vocal cords may also be effective. Succinylcholine remains the most ideal drug for the management of laryngospasm and emergent tracheal intubation and is the essential drug for managing laryngospasm in the oral and maxillofacial surgery office.

Nausea and Vomiting

PONV is a cause of morbidity in pediatric patients. Even mild PONV is associated with delayed discharge, decreased parental satisfaction, and increased use of resources. More severe complications associated with PONV include dehydration and electrolyte disturbances or hypoxemia secondary to airway obstruction or aspiration. The incidence is variable depending on age of the patient, the sex of the patient (there is a greater incidence in females > 13 yr), the anesthetic agents used, and the surgical procedure. Fortunately, severe or intractable PONV is less common, occurring in 1% to 3% of patients.[148]

Anesthetic drug selection can have an effect on the incidence of PONV. Preoperative midazolam has been associated with reduced PONV.[149] Subsedative doses of propofol also provide antiemetic effects. This contrasts with methohexital, which is associated with a higher incidence of PONV than is propofol. Premedication with opioid analgesics increases the risk of PONV. Oral transmucosal fentanyl citrate in doses of 5 to 20 µg/kg is associated with PONV in almost all patients.[150] As discussed previously, ketamine is an excellent agent for pediatric sedation. An unfortunate adverse effect associated with ketamine is a reported incidence of PONV as high as 50%. Nitrous oxide also has emetic effects. However, concentrations lower than 40% are less likely to cause PONV.

Vomiting is a complicated response mediated by the emetic center located in the lateral reticular formation of the medulla. This center receives input from several areas within the CNS, including the chemoreceptor trigger zone, vestibular apparatus, cerebellum, higher cortical and brainstem centers, and solitary tract nucleus. These structures are rich in dopaminergic, muscarinic, serotoninergic, histaminic, and opioid receptors. Blockade of these receptors is the mechanism of the antiemetic action of drugs. At the present time, there are no drugs known that act directly on the emetic center. Routine administration of antiemetic agents to all patients undergoing surgery is not justifiable as the majority do not experience PONV or have, at most, one or two episodes.

Phenothiazines

The phenothiazines are believed to exert their antiemetic effects primarily by antagonism of central dopaminergic receptors in the chemoreceptor trigger zone. Low doses of chlorpromazine, promethazine, and perphenazine are effective

in preventing and controlling PONV. These drugs are frequently combined with opioids (when administered orally by pediatric dentists) to decrease the emetic effect of the opioid. All phenothiazines are capable of producing extrapyramidal symptoms and sedation, which may complicate postoperative care. The degree of sedation varies among phenothiazines, with little sedation produced by perphenazine compared with the other phenothiazines.

Benzamides

The benzamide derivative metoclopramide has antiemetic and prokinetic effects and is the most effective antiemetic of this class. Its antiemetic effects are mediated by antagonism of central dopaminergic receptors, and at high doses, it also antagonizes serotonin-3 receptors. In the gastrointestinal tract, metoclopramide has significant dopaminergic and cholinergic actions and increases motility from the distal esophagus to the ileocecal valve. High doses of metoclopramide are well tolerated by adults, but children are prone to dystonic reactions. For this reason, metoclopramide is combined frequently with diphenhydramine to decrease this incidence (in the pediatric patient). Although metoclopramide has been used successfully to reduce the incidence of PONV in high-risk children, it is not as effective as droperidol or the newer serotonin antagonists.[151]

Histamine Antagonists

The histamine receptor antagonists are weakly antiemetic drugs with profound sedative effects, which make them less suitable for use in postoperative patients. They are frequently combined with other anesthetic agents (for pediatric patients) in an oral cocktail for their sedative and antiemetic effects. These drugs may be useful for controlling emesis resulting from vestibular stimulation, as occurs in patients with motion sickness or after middle ear surgery. They also counteract the extrapyramidal effects of the more efficacious dopamine receptor antagonists.

Muscarinic Receptor Antagonists

The vestibular apparatus and the nucleus of the tractus solitarius are rich in muscarinic and histaminic receptors. Muscarinic receptor antagonism is effective in preventing emesis related to vestibular stimulation, which may be the mechanism of morphine-induced PONV. In adults, the use of glycopyrrolate, a drug that does not cross the blood-brain barrier, has been associated with three times the need for rescue antiemetic therapy compared with atropine.[152] Transdermal scopolamine has been used successfully to reduce PONV in patients receiving morphine but is associated with a significant increase in sedation and dry mouth.[153] Other potential side effects include dysphoria, confusion, disorientation, hallucinations, and visual disturbances.

Serotonin Receptor Antagonists

Serotonin antagonists were discovered serendipitously when compounds structurally related to metoclopramide were found to have significant antiemetic effects but lacked dopamine receptor affinity. These drugs produce pure antagonism of the serotonin-3 receptor. Ondansetron was the first drug of this class to become available for clinical use in 1991. Since that time, granisetron and dolasetron have been introduced. This class of pure serotonin-3 receptor antagonists is not associated with the side effects of dopamine, muscarinic, or histamine receptor antagonists. The most serious side effects of ondansetron are rare hypersensitivity reactions. Gastric emptying and small bowel transit time were not affected by ondansetron. Asymptomatic brief prolongation of the P-R interval and the QRS complex of the electrocardiogram have been reported in adults, but rapid intravenous infusion of ondansetron in children was not associated with changes in heart rate, arterial pressure, or oxyhemoglobin saturation.[154] Psychomotor and respiratory function were unaffected by ondansetron. Prophylactic ondansetron intravenously or orally reduced the incidence of PONV in patients after a variety of surgical procedures.[155]

Glucocorticoids

Glucocorticoids (dexamethasone, methylprednisolone) exert antiemetic properties by a mechanism as yet unknown. These drugs have been used successfully in the postoperative setting to prevent PONV. Dexamethasone in doses of 8 mg in adults and up to1 mg/kg (maximum dose 25 mg) in children intravenously has antiemetic effect.[156] This class of drugs is better used in combination with another antiemetic than as the sole agent to prevent PONV.

POSTOPERATIVE RECOVERY AND DISCHARGE

The concept of office-based surgery is dependent on short surgery with a focus on rapid recovery. Whereas concepts are similar for the nonintubated versus the patient with advanced airway intervention (e.g., LMA or intubation), this section focuses on the nonintubated patient. Recovery is a period of transition from administration of the last anesthetic agent and/or termination of surgery to discharge. The first stage in this transition is when the patient emerges from sedation and opens her or his eyes and responds to voice. Anesthetic monitoring during this time interval is a continuum from the perioperative period. The following parameters are assessed: ability to maintain independent airway patency, adequate ventilations, adequate oxygenation, and hemodynamic stability. At an appropriate time, the supplemental oxygen delivery should be discontinued and the patient observed for the ability to maintain adequate oxygenation without supplemental oxygen. Intravenous access should be maintained and suction (with a Yankauer tip) should be available.

The surgeon should develop a process that can be used to assess the patient before discharge. Objective data for such a process should be established. The modified Aldrete scoring system uses the following parameters: activity level, respira-

tion, blood pressure, level of consciousness, and oxygen saturation.[157] These values should be within acceptable parameters compared with the patient's baseline. Another parameter common among health care facilities is to wait a minimum time interval from the last anesthetic drug before discharge.

Inadequate pain and/or PONV are factors that may delay discharge. Good local anesthesia (with a long-acting local anesthetic agent, if appropriate) is critical and is beneficial in managing postoperative pain as well as minimizing sympathetic stimulation, which can adversely affect hemodynamic stability and contribute to PONV. Ensuring adequate intake is unnecessary in the typical healthy patient. However, attention to replacing the fluid deficit with intravenous fluids during the perioperative period is beneficial considering the patient may have impaired postoperative oral consumption. Ensuring that the medically compromised patient (e.g., diabetic) can take and maintain oral fluids has benefit.

SPECIAL CONSIDERATIONS

Oral and maxillofacial surgeons treat a diverse group of patients. This section reviews the clinical presentation and anesthetic management of some patients with special considerations.

Attention Deficit Hyperactivity Disorder

Attention deficit hyperactivity disorder (ADHD) is defined as a persistent severe pattern of inattention or hyperactivity impulsivity symptoms compared with other children at a comparable developmental level. Three subtypes of ADHD are identified: a predominantly hyperactive/impulsive type, a predominantly inattentive type, and a combined type. ADHD is estimated to affect up to 5% of children. Medical therapy frequently includes psychostimulants such as methylphenidate, dextroamphetamine, or pemoline. Methylphenidate is the most commonly prescribed drug for ADHD. In addition to its use in the management of ADHD, 1% to 2% of the U.S. high school population without a diagnosed medical condition is reported to abuse this drug.[158] These drugs increase the bioavailability of neurotransmitters. The drugs tend to cause an increase in blood pressure and heart rate. Adverse effects are similar to that of other sympathomimetic agents. CNS effects include restlessness, dizziness, tremor, hyperactive reflexes, weakness, insomnia, delirium, and psychosis. Cardiovascular effects may include headaches, palpitations, arrhythmias, hypertension followed by hypotension, and circulatory collapse. Perioperative management of a patient on a psychostimulant (such as methylphenidate) includes recognizing signs and symptoms suggestive of inappropriate use. If there is a suggestion regarding overdose of the medication, the surgery should be postponed. However, when the medication is used appropriately, it is generally well tolerated. If there are no indications of adverse events, the medication should be continued throughout the perioperative period. The anesthetic management of these patients is dependent on the level of cooperation of the patient. Preoperative sedatives may be used. Many of these individuals allow the placement of an intravenous catheter. However, for the patient in whom intravenous access cannot be established, ketamine (with or without midazolam) administered orally or intramusculary is effective and not contraindicated owing to the chronic use of a psychostimulant.

Autism

Autism is a complex developmental disability that typically appears during the first 3 years of life. The result of a neurologic disorder that affects the functioning of the brain, autism is the third most common developmental disability in the United States and occurs in approximately 2 to 4 per 10,000 live births.[159] Autism is four times more prevalent in boys than in girls and knows no racial, ethnic, or social boundaries. Family income, lifestyle, and educational levels do not affect the chance of autism's occurrence. Autism affects the normal development of the brain in the areas of social interaction and communication skills. Children and adults with autism typically have difficulties in verbal and nonverbal communication, social interactions, and leisure or play activities. The disorder makes it difficult for them to communicate with others and relate to the outside world.[160,161] In some cases, aggressive and/or self-injurious behavior may be present. Persons with autism may exhibit repeated body movements (hand flapping, rocking), unusual responses to people, or attachments to objects and resistance to changes in routines. Children with autistic disorders may include a subgroup of individuals with associated psychiatric symptoms, including aggression, self-abusive behavior, and violent tantrums, and oftentimes necessitate the use of psychiatric medications; antipsychotics are the most prevalently prescribed medications in this group.[162] The autistic patient may also be prescribed medications similar to those prescribed for ADHD. Management of these patients in the oral and maxillofacial surgery setting requires respect for the autistic child's need for ritualistic behavior, which may result in tantrum-like rages with any disruptions of routine. Providing a calm environment with minimal stimulation and consideration of all associated pharmacologic influences aids in the management of these patients. Premedication with a benzodiazepine may be beneficial. However, establishing an intravenous access still may not be possible and an alternative technique may be required. A mask induction with a potent vapor agent or intramuscular ketamine may be considered; however, the individual may be too physically strong and combative for these techniques. An alternative that should be considered (even in the noncombative individual) is oral administration of a premedicant of ketamine or ketamine and midazolam.[163] Alterations in management must be carried over into the postoperative period, in which many patients with behavioral or mental impairments are more agitated. Restraint may be necessary to prevent premature removal of the intravenous line, wound disturbance, or self-injury.

Cerebral Palsy

Cerebral palsy is a group of neurologic disorders characterized by impaired control of movement. The clinical manifestations are variable and are dependent on the site and extent of injury. There are four classifications: spastic, athetoid, ataxic, and mixed. Spastic cerebral palsy is the most common form and affects up to 80% of the patients. Patients with spastic cerebral palsy present with muscle hypertonicity, hyperreflexia, muscle contractures, muscle rigidity, and muscle weakness. The pattern of dysfunction can be further classified into monoplegia (one limb), diplegia (both arms or both legs), hemiplegia (unilateral), triplegia (three limbs), and quadriplegia (all limbs). The severity of the contractures may result in spinal deformities such as scoliosis. Athetoid or dyskinetic cerebral palsy is characterized by choreiform, tremor, dystonia, and hypotonia. The involuntary movements seen with athetoid cerebral palsy often increase with emotional stress. Ataxic cerebral palsy is characterized by poor coordination and jerky movements.

Associated medical conditions include mental retardation, speech abnormalities, seizures, drooling, dysphagia, and gastroesophageal reflux.[164] Mental impairment is most common in patients with spastic cerebral palsy. It is important to recognize that greater than 50% of patients with cerebral palsy do not demonstrate mental impairment. Dysarthria or speech abnormalities secondary to a lack of coordination in muscle movement of the mouth can be seen in athetoid cerebral palsy. This muscle abnormality should not be confused with mental impairment. Seizures are seen in up to 35% of patients with spastic cerebral palsy. The lack of muscle coordination contributes to drooling and dysphagia. The inability to handle the secretions and the incompetent pharyngeal swallow reflex increases the risk of laryngospasm. Individuals with impaired neurologic function may also have an increased incidence of gastroesophageal reflux. Several factors must be taken into consideration in treating these patients. The spasticity and lack of coordination can contribute to a hyperactive gag reflex. Anxiety can aggravate the involuntary movements. Nitrous oxide sedation may be effective in reducing these responses.[165] Severe contractures may make positioning the patient difficult. Contractures, which may result in scoliosis, can result in a restrictive lung disorder. The patient's hypotonia may necessitate stabilization of the head (even for the nonsedated patient). If the patient is to be sedated, muscle weakness may predispose the patient to impaired respirations. This may be compounded by medications prescribed to control the spasticity or seizure disorder. Conscious sedation may be contraindicated because of the inability to handle oral secretions and the risk of gastroesophageal reflux. It may be necessary to protect the airway with the placement of an endotracheal tube. In the event that the airway requires emergent intubation, the use of succinylcholine is not contraindicated.[166]

It is important for the surgeon to remember that physical disabilities are not always associated with mental impairments. The health care provider must avoid treating these patients as if they were mentally impaired because of their inability to communicate normally.

Down Syndrome

Down syndrome, or trisomy 21, is a common chromosomal disorder occurring at a rate of 1.5 per 1000 live births and is usually characterized by mild to moderate mental retardation, cardiovascular abnormalities, and craniofacial abnormalities. Craniofacial abnormalities that have an impact on the anesthetic management of these patients include macroglossia, micrognathia, and a short neck, putting these patients at increased risk for airway obstruction during sedation. Enlargement of the lymphoid tissue may also place these patients at risk for upper airway obstruction. In addition, these patients have generalized joint laxity that may be associated with subluxation of the TMJ during airway manipulation. Intubation is usually not difficult, but subglottic stenosis, which is present in up to 25% of Down syndrome individuals, may necessitate a smaller-diameter endotracheal tube. Atlantoaxial instability occurs in approximately 20% of patients with Down syndrome, and airway maneuvers, such as neck positioning during anesthesia for airway opening or intubation, may induce a serious cervical injury (C1–2 subluxation). This cervical spine instability is a contraindication for routine treatment until both the patient and the treatment risks are fully evaluated. Sequelae to neurologic injury are usually characterized by significant symptoms or declining neurologic function without other neurologic disorder. Specific symptoms may include a positive Babinski sign, hyperactive deep tendon reflexes, ankle clonus, neck discomfort, and gait abnormalities. Down syndrome is associated with congenital heart disease in approximately 40% of its patients, and consideration of these abnormalities (endocardial cushion defect, ventricular septal defect, tetralogy of Fallot, patent ductus arteriosus, and atrial septal defect) in conjunction with their primary care physician is mandatory before proceeding with a surgical procedure.

Muscular Dystrophy

Muscular dystrophy is a group of diseases of genetic origin, characterized by the progressive loss of skeletal muscle function. There are nine types of muscular dystrophies, the most common and dramatic being Duchenne's disease (pseudohypertrophic muscular dystrophy). Symptoms typically begin between the ages of 2 and 5 years, often with the patient becoming wheelchair-bound by age 12 years. Death usually occurs between ages 15 and 25 years, usually secondary to pneumonia or congestive heart failure. Becker's muscular dystrophy is the next most common form of muscular dystrophy. Its manifestations are similar to, although milder than, those of Duchenne's disease. Its onset is later, and the progression of the disease is slower. Time to onset of disease, being wheelchair-bound, and death are 12, 30, and 42 years,

respectively.[167] The anesthetic management of these patients is complicated by muscle weakness contributing to poor respiratory function. Atrophy of the paraspinal muscles also leads to kyphoscoliosis (restrictive lung disease), which further restricts respiratory function. Pulmonary function tests should be considered as part of the preoperative assessment. Patients with functional vital capacities less than 35% of normal are at increased risk. Muscle weakness also contributes to obtunded laryngeal reflexes and an inability to clear tracheobronchial secretions. Patients are at increased risk for aspiration secondary to the obtunded laryngeal reflexes and delayed gastric emptying. Patients with muscular dystrophy may also have cardiovascular disorders. These include degenerative cardiomyopathy, cardiac arrhythmias, and mitral valve prolapse. It is frequently difficult to assess cardiovascular function in these patients because they are usually wheelchair-bound and not sufficiently stressed. However, cardiac compromise must be considered, especially in an older individual. Anesthetic considerations must take into consideration the potential for underlying respiratory and cardiovascular disease. Succinylcholine is contraindicated because it can cause rhabdomyolysis with a resultant hyperkalemia. Although all patients may have a slight increase in extracellular potassium after the administration of succinylcholine, the increase in a patient with muscular dystrophy can cause hyperkalemic cardiac arrest. The avoidance of succinylcholine and volatile inhalational agents is also recommended because of the association of Duchenne's disease with increased malignant hyperthermia. Nondepolarizing muscle relaxants may be used; however, a prolonged recovery time is seen in patients with muscular dystrophy. The response to reversal agents is also variable. In addition, patients are susceptible to an unexplained late respiratory depression. Ambulatory surgery may be unadvisable but at a minimum requires prolonged observation before discharge.[168]

SUMMARY

Ambulatory anesthesia in the patient can be safely achieved in the oral and maxillofacial surgery office. The surgeon has an array of techniques that are available. A technique has to be selected that is appropriate for the patient, the planned procedure, and the specific office.

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SECTION 2

Dentoalveolar and Implant Surgery

5

CHAPTER

Impacted Teeth

Gregory M. Ness, DDS

INTRODUCTION

Evaluation and management of impacted teeth are among the most frequent services offered by oral and maxillofacial surgeons. Although the mandibular third molar is the most commonly impacted tooth, any other tooth may be impacted. Extensive training, skill, and experience are necessary to treat these conditions with minimal trauma: when the surgeon is untrained and/or inexperienced, the incidence of complications rises significantly.[1–3] Determining the need for removal of asymptomatic teeth may also be problematic. Contemporary medical and dental practice demands evidence-based decision making and the surgeon is increasingly called upon to justify surgical procedures, including the removal of impacted teeth, by scientific data. In many situations, this decision is clear-cut based on available scientific data; in others, the decision remains based on clinical experience and professional judgment.

This chapter reviews and discusses the etiology of impacted teeth, indications and contraindications for their exposure or removal, impaction classification and determination of the degree of difficulty of surgery, parameters of perioperative patient care, and the likely complications and their management after third molar surgery.

Impacted versus Unerupted Teeth

Not all unerupted teeth are impacted. A tooth is considered impacted when it has failed to fully erupt into the oral cavity within its expected developmental time period and can no longer reasonably be expected to do so. Consequently, diagnosing an impaction demands a clear understanding of the usual chronology of eruption as well as the factors that influence eruption potential.

INCIDENCE AND ETIOLOGY

The incidence of impacted permanent teeth has been addressed in several studies with comparable findings. Generally speaking, the order of impaction frequency is the reverse of eruption order. Maxillary and mandibular third molars are the most likely teeth to become impacted, followed by maxillary canines, mandibular premolars, maxillary premolars, and second molars. Impaction of first molars or incisors is uncommon in both arches.[4–7] True impaction of deciduous teeth, as opposed to ankylosis and subsequent submersion or defects in permanent successor tooth eruption, is exceedingly rare.[8,9]

Impactions Other than Third Molars

A number of large-scale epidemiologic studies have examined the incidence of dental impactions. Grover and Lorton[4] examined 5000 army recruits and found a high frequency of impacted teeth (Figure 5-1). Although maxillary and mandibular third molars were the teeth most commonly impacted,



FIGURE 5-1. Multiple impacted teeth.

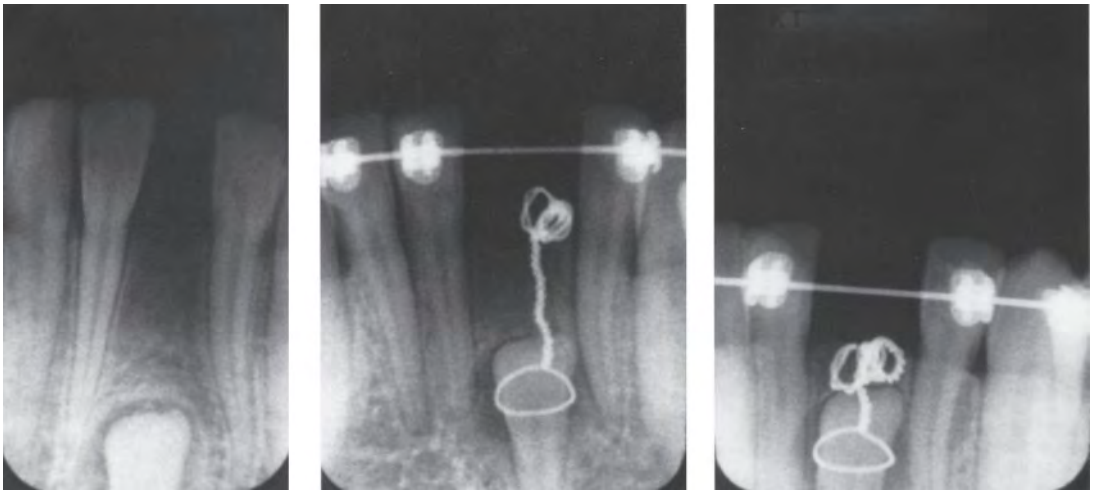


FIGURE 5-2. Unusual case of an impacted mandibular incisor. (Reproduced with permission from Zeitler D. Management of impacted teeth other than third molars. Oral Maxillofac Surg Clin North Am 1993;5:95–103.)

212 other impacted teeth were found. Among these teeth, maxillary canines were the most common, although impactions of every permanent tooth, except mandibular incisors and first molars, were identified. Thilander and Myrberg[5] examined more than 6000 Swedish school children and found a 5.4% prevalence of non–third molar impacted teeth. In an evaluation of 3874 full-mouth radiographs, Dachi and Howell[6] found the incidence of impacted canines in the maxilla to be 0.92% and of other non–third molar teeth to be 0.38%. In addition, in a study of middle-aged and older Swedish women, Grondahl[7] found approximately 25 non–third molar impacted teeth in 1418 women evaluated. Again, the canine tooth was the most frequent non–third molar impaction identified, followed by premolars and second molars. The incidence of impaction was lower in this study of older patients, presumably because symptomatic teeth and those with pathologic findings were removed earlier in life (Figure 5-2).

Permanent teeth may become impacted because of systemic or local factors. Impaction of teeth in the hereditary syndrome of cleidocranial dysplasia (Figure 5-3) is more properly termed *primary retention.*[10] Endocrine deficiencies (hypothyroidism and hypopituitarism), febrile diseases, Down syndrome, and irradiation are other systemic factors that may influence impaction of permanent teeth.[11,12] Multiple teeth are often involved in these conditions.

More commonly, local factors are the cause of permanent tooth impaction. These factors include prolonged deciduous tooth retention, malposed tooth germs, arch-length deficiency, supernumerary teeth, odontogenic tumors, abnormal eruption path, and cleft lip and palate.[10,13,14]

Because maxillary canines are impacted relatively often, they have been studied extensively in an effort to identify the causes of impaction. Usually, they are displaced to either the palatal or the labial side of the alveolus, and Jacoby[13] distinguishes between these positions in his conclusions on impaction's etiology. Labially unerupted canines tend to show a degree of arch-length deficiency, whereas palatally impacted canines do not. He stated that a canine might appear in a palatal position if extra space is available in the maxillary bone owing to either excessive growth, agenesis or peg shape of the lateral incisor, or stimulated eruption of a lateral incisor or first premolar.[13] In his review of impacted maxillary canines, Bishara[11] echoes this theory, stating that the presence of the lateral incisor root with normal length at the normal time is important to guide the canine in a proper eruptive direction.

Impacted second molars have also been studied to determine the cause of these impactions. Although maxillary second permanent molars are infrequently impacted, in a study of these cases, Ranta[15] found that the third molar was generally positioned occlusally and palatally in relation to the second molar, acting as an obstruction (Figure 5-4). In a similar study, Levy and Regan[12] identified the most probable cause of impaction of developing maxillary second molars as malposition of the tooth germs of the third molars. A typical finding in their series of cases was deformation of the mesial



FIGURE 5-3. Multiple impacted teeth in a case of cleidocranial dysplasia.

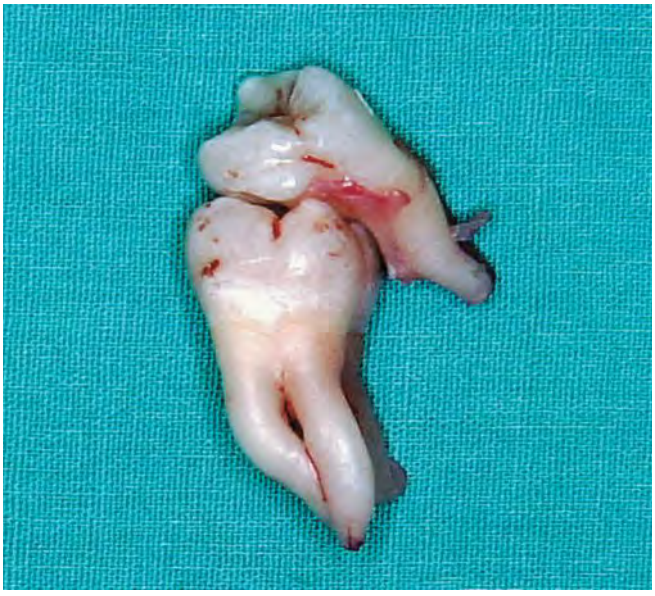


FIGURE 5-4. Impacted maxillary second and third molars.

surfaces of the crowns and roots of the third molars. Raghoebar and colleagues[10] stated that impaction of first molars is often diagnosed as ectopic eruption, whereas impaction of second molars is usually associated with arch-length deficiency. Other studies have found association of impaction with inadequate space as high as 90%.[16] However, a more recent study by Bondemark and Tsiopa[17] examined impacted second molars including those which were erupted but not completely, having stopped short of the occlusal plane. Among impacted teeth in this population, 75% had no clinically or radiographically apparent physical barrier to eruption, indicating that inadequate space is only one potential etiology of this condition. None of the teeth with disturbed eruption in Bondemark and Tsiopa's study were associated with pathologic conditions such as cysts, tumors, ankylosis, or jaw hypoplasia/dysplasia associated with syndromes, all of which may cause dental impactions.

IMPACTED THIRD MOLARS

The age range when the third molars, the final teeth to erupt, may appear is broader than that for other teeth. Whereas eruption of lower third molars is often complete at the average age of 20 years, it can occur as late as 24 years. A tooth that appears impacted at age 18 years may have as much as a 30% to 50% chance of erupting fully by age 25 years, according to several longitudinal studies.[18–21] It is fairly well established that the position of retained third molars does not change substantially after age 25 years,[14] although there is some evidence of continued movement as late as the fourth decade.[18] Many patients are evaluated for third molar removal in their mid to late teens, and the surgeon must, therefore, attempt to discern the probable outcome of the eruption process based on more than tooth position alone.

A number of longitudinal studies have clearly defined the development and eruption pattern of the third molar.[22–25] The mandibular third molar tooth germ is usually visible radiographically by age 9 years, and cusp mineralization is completed approximately 2 years later. At age 11 years, the tooth is located within the anterior border of the ramus with its occlusal surface facing almost directly anteriorly. The level of the tooth germ is approximately at the occlusal plane of the erupted dentition. Crown formation is usually complete by age 14 years, and the roots are approximately 50% formed by age 16 years. During this time, the body of the mandible grows in length at the expense of resorption of the anterior border of the ramus. As this process occurs, the position of the third molar relative to the adjacent teeth changes, with the third molar assuming a position at approximately the root level of the adjacent second molar. The angulation of the crown becomes more horizontal also. Usually, the roots are completely formed with an open apex by age 18 years.

The change in orientation of the occlusal surface from a straight anterior orientation to a straight vertical inclination occurs primarily during root formation. During this time, the tooth rotates from horizontal to mesioangular to vertical. Therefore, the normal development and eruption pattern, assuming the tooth has sufficient room to erupt, brings the tooth into its final position by age 20 years. By age 24 years, 95% of all third molars that will erupt have completed their eruption.

Most third molars do not complete this eruption sequence and, instead, become impacted teeth. Approximately half do not assume the vertical position and remain as mesioangular impactions. There are several possible explanations for this. The Belfast Study Group suggested that there may be differential root growth between the mesial and the distal roots, which causes the tooth to either remain mesially inclined or rotate to a vertical position, depending on the amount of root development.[25,26] In their studies, they found that underdevelopment of the mesial root results in a mesioangular impaction. Overdevelopment of the same root results in overrotation of the third molar into a distoangular impaction. Overdevelopment of the distal root, commonly with a mesial curve, is responsible for severe mesioangular or horizontal impaction. They go on to note that, whereas the expected normal rotation is from horizontal to mesioangular to vertical, failure of rotation from the mesioangular to the vertical position is also common. To a lesser extent, they documented worsening of the angulation from mesioangular to horizontal impaction and overrotation from mesioangular to distoangular. These overrotations from mesioangular to horizontal and from mesioangular to distoangular occur during the terminal portion of root development.

A second major reason for the failure of the third molar to rotate into a vertical position and erupt involves the relation of the bony arch length to the sum of the mesiodistal widths of the teeth in the arch. Several studies have demonstrated that when bony length is inadequate, a higher proportion of impacted teeth is seen.[27–29] In general, patients with impacted

teeth almost invariably have larger-sized teeth than do those without impactions.[29] Even when the tooth-bone relationship is favorable, a lower third molar that is abnormally positioned laterally almost always fails to erupt,[27] perhaps influenced in part by the dense bone present in the external oblique ridge.

A final factor that seems to be associated with an increased incidence of third molar impaction is retarded maturation. When dental development of the tooth lags behind the skeletal growth and maturation of the jaws, there is an increased incidence of impaction. This is most likely a result of a decreased influence of the tooth on the growth pattern and resorption of the mandible. This phenomenon results in the rather counterintuitive observation that by age 20 years, an impacted third molar with partially developed roots is less likely to erupt than a similarly positioned tooth with fully developed roots.

CLINICAL EVALUATION

Diagnosis of impacted permanent teeth is straightforward, involving clinical inspection that discloses the absence of the tooth in its normal position combined with the radiographic assessment showing the position of the unerupted tooth. When multiple or uncommonly impacted teeth are found to be impacted, the surgeon should evaluate for an underlying systemic cause.

Radiographic assessment of the impacted teeth is important in the preparation for surgical or orthodontic treatment. The optimal age for evaluating an ectopically positioned canine has been suggested to be 10 to 13 years, depending on individual development.[30] Most techniques for localization of an impacted tooth have been studied primarily with maxillary canines, but these can be generalized to other sites in the oral cavity. Ericson and Kurol[31] have studied the radiographic appearance of ectopically erupting maxillary canines and have found that a palpable canine generally erupts in a relatively normal position and that axial or panoramic films were less useful than conventional periapical radiographs. A study comparing plain film radiography with computed tomography (CT) showed CT to be superior in showing tooth and root shape, crown-root relationship, and tooth inclination.[32] The growing availability of in-office cone beam imaging makes this an increasingly important diagnostic adjunct, particularly in difficult cases.

Standard radiographic techniques to establish three-dimensional tooth location include the tube shift method, the buccal object rule, and the periapical occlusal method (Figure 5-5).[33] All of these methods make use of what is commonly known as the *SLOB principle*, for same-lingual, opposite-buccal. The tube shift method uses two periapical radiographs, shifting the tube horizontally between exposures. If the unerupted tooth moves in the same direction as the tube was shifted, it is localized on the lingual or palatal side. A facial or buccally located tooth moves in the opposite direction to the tube shift. The buccal object rule uses two radiographs taken with different vertical angulations of the x-ray beam, with the same results. The periapical occlusal method uses the periapical radiograph taken with a standard technique and an occlusal radiograph to give two different views of the impacted tooth.



FIGURE 5-5. Panoramic films can be used to localize maxillary canines.

Numerous studies have evaluated the influence of various factors on the eruption potential of a lower third molar. Two radiographically determined factors consistently emerge as most prognostic: angulation of the third molar and space available for its emergence.[34–38] By age 18 to 20 years, lower third molars that are horizontal or strongly mesioangular have much less eruption potential than do those oriented more vertically. Distoangular teeth are intermediate in their likelihood to erupt fully. However, the strongest hope of future eruption lies with those third molars that can be seen radiographically to have space at least as wide as their crown between the distal of the second molar and the ascending mandibular ramus. At age 20 years, unerupted lower third molars that are nearly vertical and have adequate horizontal space are more likely to erupt than to remain impacted. However, if the crown-to-space ratio is greater than 1, or if the tooth orientation diverges substantially from vertical, the tooth is unlikely to ever erupt fully.

TREATMENT OF AN IMPACTED TOOTH

An impacted tooth can cause the patient mild to serious problems if it remains in the unerupted state. Not every impacted tooth causes a problem of clinical significance, but each does have that potential. A body of information has been collected based on extensive clinical experience and clinical studies from which indications for removal of impacted teeth have been developed. For some indications, there is lack of evidence-based data gained from long-term prospective longitudinal studies.

For many—if not most—non–third molar impactions, the most desirable treatment outcome is eruption of the tooth into its normal, functional position in the dental arch. This is generally accomplished through a combination of orthodontic and surgical treatment, with close consultation between specialists determining the timing and sequencing of treatment steps. For teeth whose eruption potential is poor, where

space cannot be created to accommodate them, that have associated pathologic lesions, and for nearly all third molars, treatment will include removal of the impacted tooth. There is extensive discussion of the strength of evidence supporting removal of impacted third molars, and the contemporary oral and maxillofacial surgeon must be closely familiar with the current literature on this topic.

Indications for Removal of Impacted Third Molars

Most clinicians agree that if a patient presents with one or more of the pathologic problems or symptoms described later, the involved third molar should be removed. It is much less clear what should be done prophylactically with third molars that are impacted but have not yet caused these problems. Most of the symptomatic pathologic problems that result from third molars occur as a result of a partially erupted tooth. There is a lower incidence of problems associated with a complete bony impaction.

Periodontitis

Perhaps the most important and complete new knowledge regarding the retained third molar is its influence on initiation and progression of periodontal disease. The potential for partially erupted third molars to contribute to early advanced periodontitis has been recognized for many years and was the primary reason for removal of approximately 5% of impacted third molars in studies from the 1980s.[21,39–42] More recent evidence demonstrates that even young patients in otherwise good general periodontal health have a significant increase in periodontal pocketing, attachment loss, pathogenic bacterial activity, and inflammatory markers at the distal of the second molar and around the third molar.[43–45] Periodontal probing depths distal to the second molar of 5 mm or more are twice as likely in the presence of a visible third molar.[46] Young patients with visible third molars are significantly more likely to have moderate to severe periodontal disease.[47] Furthermore, retained visible third molars are assocated with progressively deepening periodontal pockets over time,[48] particularly in the common presence of pathogenic periodontal microflora[49] (“red” complex bacteria) as described originally by Socransky and coworkers.[50,51] This is observed even when the third molar continues to erupt to the level of the mandibular occlusal plane.[48] Periodontal pocketing worsens not only in the third molar area but in adjacent teeth as well.[52] Finally, dental prophylaxis is less effective at lowering periodontal pathogen counts in patients with visible third molars present.[53]

This evidence points strongly to the impacted third molar’s important role in the onset, severity, and progression of periodontitis in otherwise young and healthy patients. Removal of these impacted teeth before the age of 25 years usually results in improved periodontal attachment at the distal of the second molar[54–56] and reduces the incidence of periodontal pockets of 4 mm or more in other teeth as well.[57] Current knowledge of the biology of periodontal pathology, applied to the third molar, makes obsolete the concept that third molar “pseudopocketing” is a benign phenomenon.[58]

Pericoronitis

When a third molar, usually the mandibular third molar, partially erupts through the oral mucosa, the potential exists for the establishment of a mild to moderate inflammatory response similar to gingivitis and periodontitis. The bacteria that are most commonly associated with pericoronitis are *Peptostreptococcus*, *Fusobacterium*, and *Porphyromonas*.[59–61] Initial treatment of pericoronitis is usually aimed at débridement of the periodontal pocket by irrigation or by mechanical means, disinfection of the pocket with an irrigation solution such as hydrogen peroxide or chlorhexidine, and surgical management by extraction of the opposing maxillary third molar and, occasionally, of the offending mandibular third molar. Severe cases of pericoronitis with systemic symptoms may warrant antibiotic therapy. In uncommon situations, the patient may develop a severe infection requiring vigorous medical and surgical treatment.

Prevention of recurrent pericoronitis is usually achieved by removal of the involved mandibular third molar. Approximately 25% to 30% of impacted mandibular third molars are extracted because of pericoronitis or recurrent pericoronitis.[21,39–42] Pericoronitis is the most common reason for removal of impacted third molars after age 20 years. With increasing age, the incidence of pericoronitis as an indication for removal of impacted teeth also increases.

Dental Caries

Dental caries can occur in the mandibular third molar or in the adjacent second molar, most commonly at the cervical line. Owing to the patient’s inability to effectively clean this area and because the third molar is inaccessible to the restorative dentist, caries in the second and third molars are responsible for extraction of impacted third molars in approximately 15% of patients.[21,39–42] Occlusal caries in the partially erupted third molar are common but not as likely as periodontal pathology.[58] As with pericoronitis, the presence of caries and eventual pulpal necrosis are responsible for an increasing percentage of extractions with age.

Orthodontic Considerations

The presence of the impacted third molar, especially in the mandible, may be responsible for several orthodontic problems. These problems fall into three general areas, which are outlined here.

Crowding of Mandibular Incisors

Perhaps one of the most controversial issues regarding mandibular third molars in the past was the issue of their influence on anterior crowding of mandibular incisor teeth, especially after orthodontic therapy. More recent literature includes longitudinal reviews of orthodontically treated patients in larger numbers,[62,63] and the preponderance of evidence now suggests that impacted third molars are not a

significant cause of postorthodontic anterior crowding. In fact, anterior incisor crowding is associated with deficient arch length, not the presence of impacted teeth.

Obstruction of Orthodontic Treatment

In some situations, the orthodontist attempts to move the molar teeth distally, but the presence of an impacted third molar may inhibit or even prevent this procedure. Therefore, if the orthodontist is attempting to move the buccal segments posteriorly, removal of the impacted third molar may facilitate treatment and allow predictable outcomes.

Interference with Orthognathic Surgery

When maxillary or mandibular osteotomies are planned, presurgical removal of the impacted teeth may facilitate the orthognathic procedure. Delaying removal of third molars until mandibular osteotomy, especially in mandibular advancement surgery, substantially reduces the thickness and quality of lingual bone at the proximal aspect of the distal segment, where fixation screws are usually applied. If third molars are to be removed in advance, sufficient time must be allowed for the extraction site to fill with mature bone. Conversely, following maxillary down-fracture, a deeply impacted upper third molar is often easily approached from above through the maxillary sinus and may be safely removed in this manner without compromising the soft tissue vascular pedicle of the maxilla. Although these circumstances involve a small percentage of all impacted third molars, the surgeon must plan well in advance (6–12 mo) for patients undergoing these procedures.

Prevention of Odontogenic Cysts and Tumors

In the impacted third molar that is left intact in the jaw, the follicular sac that was responsible for the formation of the crown may undergo cystic degeneration and form a dentigerous cyst. The follicular sac may also develop an odontogenic tumor or, in quite rare cases, a malignancy. These possibilities have frequently been cited as a reason for removal of asymptomatic teeth; although rare, when pathology occurs, it may pose a serious health threat.[64] The general incidence of neoplastic change around impacted molars has been estimated to be approximately 3%.[65,66] In retrospective surveys of large numbers of patients, between 1% and 2% of all third molars that are extracted are removed because of the presence of odontogenic cysts and tumors.[21,39–42] These pathologic entities are usually seen in patients younger than 40 years, suggesting that the risk of neoplastic change around impacted third molars may decrease with age.

Root Resorption of Adjacent Teeth

Third molars in the process of eruption may cause root resorption of adjacent teeth. The general view is that misaligned erupting teeth may resorb the roots of adjacent teeth, just as succedaneous teeth resorb the roots of primary teeth during their normal eruption sequence. The actual occurrence of significant root resorption of adjacent teeth is not clear, although it may be as high as 7%.[67] If root resorption is noted on adjacent teeth, the surgeon should consider removing the third molar as soon as it is convenient. In most cases, the adjacent tooth repairs itself with the deposition of a layer of cementum over the resorbed area and the formation of secondary dentin. However, if resorption is severe, both teeth may require removal.

Teeth under Dental Prostheses

Before construction of a removable or fixed prosthesis, the dentist should make sure that there are no impacted teeth in the edentulous area that is being restored. If such teeth are present, the general recommendation is that they be removed before the final placement of the prosthesis. Teeth that are completely covered with bone, that show no pathologic changes, and that are in patients older than 40 years are unlikely to develop problems on their own. However, if a removable tissue-borne prosthesis is to be constructed on a ridge where an impacted tooth is covered by only soft tissue or 1 or 2 mm of bone, it is highly likely that in time the overlying bone will be resorbed, the mucosa will perforate, and the area will become painful and often inflamed. If this occurs, the impacted tooth will likely then need to be removed and the dental prosthesis either altered or refabricated.

The risks and benefits of removing the impacted tooth must be given careful individual consideration. In older patients with tooth- or implant-borne fixed prostheses, asymptomatic deeply impacted teeth can be safely left in place. However, if a removable prosthesis is to be made and the bone overlying the impacted tooth is thin, the tooth should probably be removed before the final prosthesis is constructed.

Prevention of Jaw Fracture

Patients who engage in contact sports, such as football, rugby, or martial arts, should consider having their impacted third molars removed to prevent jaw fracture during competition. An impacted third molar presents an area of lowered resistance to fracture in the mandible and is, therefore, a common site for fracture.[68–70] In addition, the presence of an impacted third molar in the line of fracture may cause increased complications in the treatment of the fracture.

Management of Unexplained Pain

Occasionally, patients complain of jaw pain in the area of an impacted third molar that has neither clinical nor radiographic signs of pathology, and other sources of pain are ruled out. In these situations, removal of the impacted third molar frequently results in resolution of this pain. At this time, there is no plausible explanation as to why this relief of pain occurs. Approximately 1% to 2% of mandibular third molars that are extracted are removed for this reason.[21,39–42] The patient must be informed that removal of the third molar may not relieve the pain completely.

Contraindications to Treatment of Impacted Teeth

The decision to remove a given impacted tooth must be based on a careful evaluation of the potential benefits versus risks. In situations in which pathology exists, the decision to

remove the tooth is uncomplicated because it is necessary to treat the disease process. Likewise, there are situations in which removal of impacted teeth is contraindicated because the surgical complications and sequelae outweigh the potential benefits. The general contraindications for removal of impacted teeth can be grouped into three primary areas: advanced patient age, poor health, and surgical damage to adjacent structures.[71]

Extremes of Age

Healing generally occurs more rapidly and more completely in younger patients; however, surgical removal of unerupted third molars in the very young is contraindicated. Historically, some clinicians reported that removal of the tooth bud of the developing third molar at age 8 or 9 years can be accomplished with minimal surgical morbidity.[72] However, the contemporary consensus is that this is not a prudent approach. The original view was based on the belief that accurate growth predictions could be made and, therefore, that an accurate determination could be established regarding whether a given tooth would be impacted. If such a determination were the case, then the tooth bud could be removed relatively atraumatically in the very young patient. The evidence at this time, however, is contradictory to that opinion, and the general consensus is that removal of the tooth bud at this stage may, in fact, be unnecessary because the involved third molar may erupt into proper position.

At the other end of the age spectrum, with increasing age, healing response decreases,[73] which may result in a greater bony defect postoperatively than was present because of the impacted tooth. In addition, the surgical procedure grows more and more difficult as the patient ages owing to more densely calcified bone, which is less flexible and more likely to fracture. With aging, a patient's response to surgical insult is less tolerant and the recuperation period grows longer. There is overwhelming clinical evidence to support the fact that the number of days missed from work and other normal activity after third molar extraction is much higher in the patient older than 40 years compared with patients younger than 18 years.

As a general rule, if a patient has a fully impacted third molar that is completely covered with bone, has no obvious potential source of communication with the oral cavity, and has no signs of pathology such as an enlarged follicular sac, and if the patient is older than 40, the tooth probably should not be removed. Long-term follow-up by the patient's dentist should be performed periodically, with radiography performed every several years to ensure that no adverse sequelae are occurring. If signs of pathology develop, the tooth should be removed. If the overlying bone is very thin and a removable denture is to be placed over that tooth, the tooth should probably be removed before the final prosthesis is constructed.

Compromised Medical Status

Patients who have impacted teeth may have some compromise in their health status, especially if they are elderly. As age increases, so does the incidence of moderate to severe cardiovascular disease, pulmonary disease, and other health problems. Thus, the combination of advanced age and compromised health status may contraindicate the removal of impacted teeth that have no pathologic processes.

Other factors may compromise the health status of younger people, such as congenital coagulopathies, asthma, and epilepsy. In this group of patients, it may be necessary to remove impacted teeth before the incipient pathologic process becomes fulminant. Thus, not only in the older compromised patient but also in the younger compromised patient, the surgeon occasionally needs to remove asymptomatic as well as symptomatic third molars. The compromised medical status remains a relative contraindication and may require the surgeon to work closely with the patient's physician to manage the patient's medical problems.

Surgical Damage to Adjacent Structures

Occasionally, an impacted tooth is positioned such that its removal may seriously compromise adjacent nerves, teeth, and other vital structures (e.g., sinus), making it prudent to leave the impacted tooth in situ. The potential complications must be weighed against the potential benefits of surgical removal of the tooth. In this clinical setting, many clinicians advocate removing only the crown of the impacted tooth and intentionally leaving the roots in place; scientific literature offers substantial support for this approach, particularly for mandibular third molars.[74–76] The risks of pathology due to the impacted tooth may be reduced with less exposure to the complications associated with root proximity to vital structures.

In older patients, the surgical extraction of impacted third molars can result in significant bony defects that may not heal adequately and, in fact, may result in the eventual loss of adjacent teeth rather than the improvement or preservation of periodontal health. This may also be viewed as a contraindication to removal of the impacted tooth.

SURGERY AND PERIOPERATIVE CARE FOR IMPACTIONS OTHER THAN THIRD MOLARS

Exposure versus Exposure and Bonding

Surgical exposure is a procedure intended to permit natural eruption of impacted teeth. Typically, the crowns of the teeth are surgically exposed with removal of tissues in the direction most appropriate for crown movement. The wound is then packed until it is totally epithelialized. Öhman and Öhman[77] studied 542 impacted teeth in 389 patients treated using this technique. The teeth were allowed to erupt for up to 24 months or until the greatest diameter of the crown reached the level of the mucosal surface. Of 542 teeth, only 16 were failures (failure to erupt after 24 mo or with other complications). This study found that the teeth tended to show a change of inclination of the longitudinal access by rotation along the root. Age did not appear to be a factor in success, although most patients were younger than 19 years.

More commonly, the technique of surgical exposure is combined with attachment of an orthodontic appliance to the tooth, allowing active guidance of the impacted tooth into an ideal position. Important factors in this technique are prior orthodontic treatment to provide both anchorage and adequate space within the dental arch for the impacted tooth. Many methods have been suggested for making attachment to the impacted tooth, including polycarbonate crowns, attachments such as rare earth magnets, or pins inserted into the structure of the tooth. These techniques are rarely used. The most common method of orthodontic attachment to an impacted tooth is the placement of a bonded orthodontic bracket.[78] This can usually be done with conservative exposure of the tooth, removing only enough soft tissue and bone to place the bonded bracket and avoiding exposure of the cementoenamel junction.[11] This exposure, especially when combined with use of a wire ligature around the neck of the tooth, has been shown to increase the risk of root resorption, ankylosis, periodontal inflammation, and other complications and is contraindicated.

Studies have compared simple exposure and packing to maintain a gingival path for eruption with exposure and bonding of a bracket. In a study of impacted premolars, Thilander and Thilander[79] showed that surgical exposure alone resulted in eruption, provided that space was present in the arch. However, mesially tipped premolars had a poor prognosis and required orthodontic guidance. Iramaneerat and associates[80] found that there was no difference in total orthodontic treatment time for the two techniques. Pearson and colleagues[81] found that bracketing was more costly and more likely to require reoperation. Nonetheless, placing a bracket is the more popular technique, perhaps owing to orthodontist preference and patient comfort. Laskin and Peskin[82] believe that if exposure of teeth is to result in successful spontaneous eruption, it should be done as soon as it is determined that the tooth is not going to erupt spontaneously. When treating molar teeth, it is important to remove third molars that are often obstructing the second molars' normal eruption (Figure 5-6). Ranta[83] stated that it is typical for impacted second molars to erupt normally when the offending third molar is removed. Vig[84] also recommends routine removal of the third molar when a second molar is impacted. Although removal of the second molar to allow eruption of the third molar into the second molar position may occasionally have a satisfactory outcome in the maxilla, this is not likely to happen in the mandible.[85] Malformed, severely tipped, or deeply impacted second molars may need to be extracted.



FIGURE 5-6. Third molar in path of second molar eruption.

Surgical Technique

Exposure and Bonding

The most common type of non–third molar impaction is the palatally positioned maxillary canine. Typical surgical exposure involves reflection of a full-thickness palatal flap, conservative exposure of the tooth, and bonding of a bracket to its palatal surface (Figure 5-7). If the tooth is near the free edge of the flap, soft tissue may be removed to leave the

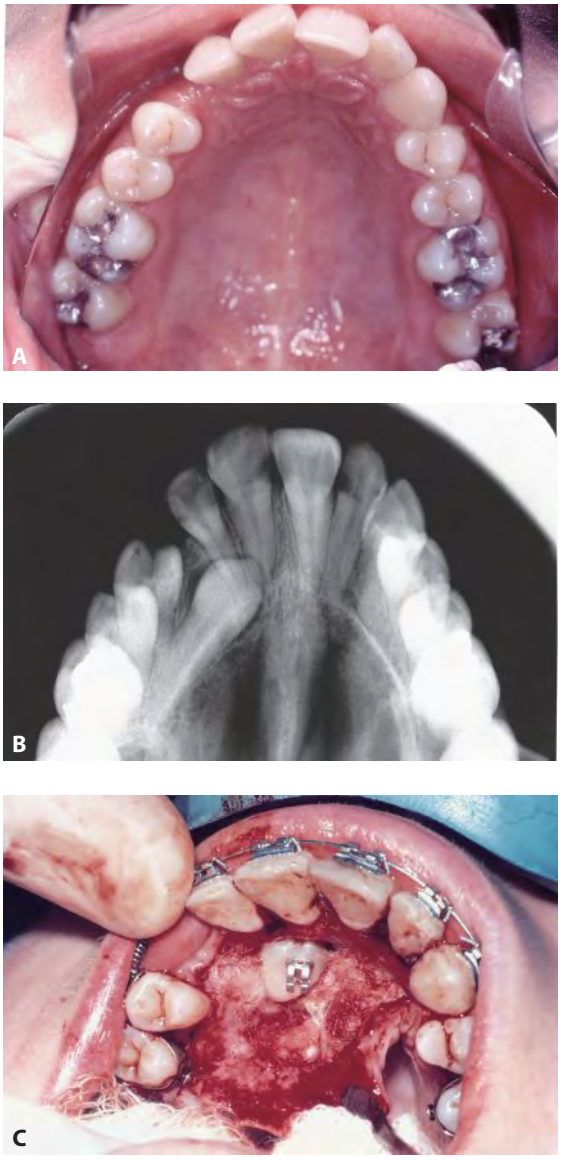


FIGURE 5-7. **A,** Right maxillary canine is unerupted. **B,** Radiograph shows impacted canine. **C,** Bracket placed. (**A–C,** Reproduced with permission from Zeitler D. Management of impacted teeth other than third molars. Oral Maxillofac Surg Clin North Am 1993;5:95–103.)

crown exposed; the wound is then packed gently during the initial healing period. If the tooth is deeply impacted, it may be more appropriate to replace the soft tissue flap, bringing a wire attached to the bonded bracket through the soft tissues near the crest of the ridge. Alternatively, if making attachment to the canine is deemed unnecessary or not possible, the flap may be returned to its original position. A full-thickness soft tissue window is then created over the canine crown, which is then packed open and allowed to epithelialize, leaving the crown visible in the mouth for spontaneous eruption or attachment at a later date. The technique of replacing the flap has been examined for its periodontal consequences. The clinical outcomes show minimal effects of the closed eruption technique on the periodontium.[86]

Management of the labially positioned impacted canine follows similar principles to the palatal impaction, with the added concern of maintaining keratinized, attached mucosa adjacent to the cervical line of the tooth when eruption is complete. Adequate space in the arch must be established by preliminary orthodontic treatment before canine exposure. A full-thickness mucoperiosteal flap is elevated with mesial and distal releasing incisions extending along the sides of the canine crown. It is often useful to make these incisions more closely parallel than in other circumstances, because widely divergent releasing incisions make repositioning the flap apically more difficult. The crown of the tooth is uncovered, avoiding dissection beyond the cervical line of the tooth. A bonded bracket is attached and the flap is repositioned and secured to cover the cementoenamel junction of the tooth. The bonded bracket helps to support the attached gingiva in this apical relationship (Figure 5-8). When the tooth is too far superior to allow an apically repositioned flap, the wire or chain secured to the bracket is brought out from under the flap at the crest of the ridge and the flap is returned to its original position. As the tooth is orthodontically moved into position, an adequate band of keratinized gingiva either migrates with the tooth or remains in place at the alveolar crest and the tooth is guided to erupt through it. Techniques that involve removal of the attached gingiva, leaving alveolar mucosa surrounding the cervical area of the tooth, are to be avoided.

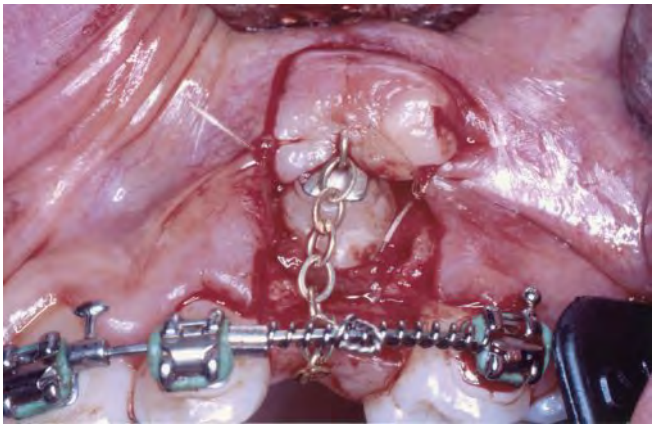


FIGURE 5-8. Labially impacted canine exposed using an apically repositioned flap.

These basic principles of exposure of canines can be generalized to many other impacted teeth. Exposure and orthodontic attachment of maxillary and mandibular premolars can be treated much like maxillary canines. Often, mandibular premolars are located relatively centrally in the alveolar process. This may also be true of mandibular molars. When this is the case, exposure from the coronal aspect of the tooth may be indicated. A bonded bracket may be placed on the occlusal surface of the tooth and orthodontic forces applied in a relatively vertical direction until the tooth is exposed sufficiently to place the orthodontic bracket in a more traditional position.

Impacted second molars with angulation, root morphology, and available space deemed adequate may be orthodontically uprighted after surgical exposure and bracketing. A conventional button with gold chain may be bonded to the tooth, although a molar bracket with buccal tube has been reported to offer shorter treatment times with excellent outcomes.[87]

Surgical Uprighting

Uprighting of teeth has been applied most commonly to impacted second molars. Impacted lower second molars with incomplete apical closure may be exposed and surgically uprighted according to a method nicely described by Pogrel.[88] The optimal time for uprighting a molar tooth is when two thirds of the root has formed; molars with fully formed roots have a poor prognosis.[10] The tooth must not be buccally or lingually tipped because intact cortical plates are necessary adjacent to the tooth for stability. The technique begins with removal of the third molar (Figure 5-9). This generally creates the necessary space for posterior tipping of the second molar. If no third molar is present, it will likely be necessary to remove bone posterior to the second molar. When doing so, it is important to avoid damage to the cementoenamel junction of the second molar. The tooth should not be tipped more than 90 degrees and is splinted into place. An extremely important part of this surgical procedure is ensuring that there are no occlusal forces on the repositioned second molar. Terry and Hegtvedt[89] describe a similar but self-stabilizing technique. Recently, Sohn and coworkers[90] have described uprighting an impacted second molar using miniscrew anchorage.

Transplantation

Transplantation of teeth has been advocated as an alternative to other methods of treatment of impacted teeth. Although contemporary dental implant therapy has largely overtaken this technique, transplantation remains useful for those patients who are not candidates for implant dentistry. It may be appropriate for the adult patient who cannot undergo conventional orthodontic movement of a canine or premolar. The advocated technique is a careful wide exposure of the impacted tooth. The tooth is then moved into its position

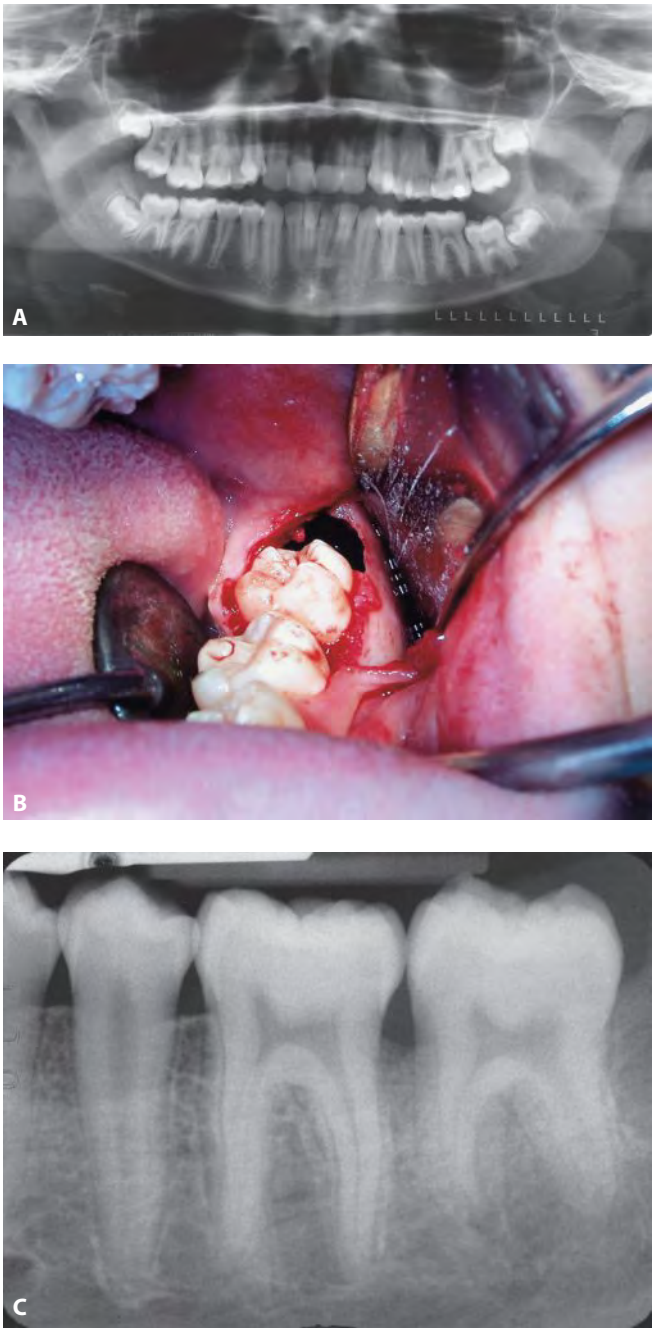


FIGURE 5-9. **A,** Impacted second molar. **B,** Second molar lifted into position. **C,** Six-month follow-up radiograph of repositioned second molar.

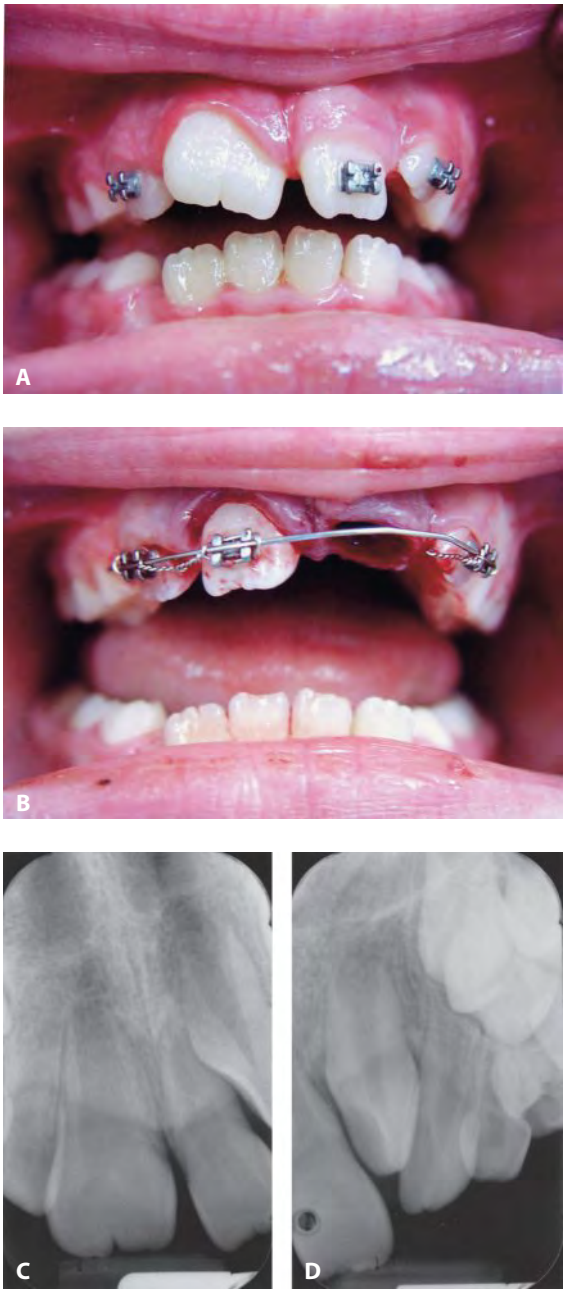


FIGURE 5-10. **A,** Geminated tooth no. 8. **B,** After removal of abnormal tooth no. 8 and transplantation of erupted tooth no. 9, the unerupted tooth no. 9 is expected to erupt. **C,** Radiograph of geminated tooth no. 8. **D,** Radiograph of duplicated tooth no. 9.

within the dental arch and stabilized with a segmental orthodontic appliance. Endodontic treatment begins with calcium hydroxide paste 6 to 8 weeks after the surgical procedure. Conventional root canal filling is performed at 1 year after surgery. Sagne and Thilander[92] studied 47 patients with 56 canines that were surgically transplanted. This study showed a successful outcome in 54 of 56 transplanted canines. Their concluding recommendation is to perform conventional orthodontic treatment for impacted canines in children and young individuals. However, when extraction would otherwise be performed, they recommend transalveolar transplantation as a sound alternative (Figure 5-10).[91]

Removal

Surgical removal of impacted permanent teeth may be performed when other methods of treatment are unavailable. Basic surgical principles of radiographic assessment and careful surgical technique must be followed. Conservation of bone through conservative exposure and removal with sectioning of the tooth should be considered, and care must be

taken to avoid injury to the roots of adjacent teeth. Impacted teeth should be approached from the surface of the maxilla with which they are most closely associated. Labially impacted teeth are frequently removed with an elevator technique, but palatal impactions generally require removal of the crown followed by sectioning of the root. Longitudinal sectioning of the root of the palatal canine often is useful and may conserve bone. When a large palatal flap has been reflected, maintaining a palatal splint to support the soft tissues for several days prevents hematoma formation and may improve patient comfort.

Mandibular premolars are generally approached from the labial surface of the mandible. Care must be taken to preserve the integrity of the mental nerve when the impacted tooth is nearby. When the impacted lower premolar is lingually positioned, it is sometimes useful to identify the tooth through a lingual exposure; a labial flap then may be raised and a small hole placed in the labial surface of the bone to allow the premolar to be pushed through to the lingual. Removal of impacted molars is similar to removal of impacted third molars.

SURGERY AND PERIOPERATIVE CARE FOR IMPACTED THIRD MOLARS

Determining Surgical Difficulty

Preoperative evaluation of the third molar, both clinically and radiographically, is a critical step in the surgical procedure for removal of impacted teeth. The surgeon pays particular attention to the variety of factors known to make the impaction surgery more or less difficult. A variety of classification systems have been developed to aid in the determination of difficulty. The three most widely used are angulation of the impacted tooth, the relationship of the impacted tooth to the anterior border of the ramus and the second molar, and the depth of the impaction and the type of tissue overlying the impacted tooth.

It is generally acknowledged that the mesioangular impaction, which accounts for approximately 45% of all impacted mandibular third molars, is the least difficult to remove. The vertical impaction (40% of all impactions) and the horizontal impaction (10%) are intermediate in difficulty, whereas the distoangular impaction (5%) is the most difficult.

The relationship of the impacted tooth to the anterior border of the ramus is a reflection of the amount of room available for the tooth eruption as well as the planned extraction. If the length of the alveolar process anterior to the anterior border of the ramus is sufficient to allow tooth eruption, the tooth is generally less difficult to remove. Conversely, teeth that are essentially buried in the ramus of the mandible are more difficult to remove.

The depth of the impaction under the hard and soft tissues is likewise an important consideration in determining the degree of difficulty. The most commonly used scheme for determining difficulty involves consideration of the soft tissues and partial or complete bony impaction. It is widely employed in part because it may be the most useful indicator of the time required for surgery and, perhaps even more important, because it is the system required to classify and code impaction procedures to all commercial insurance carriers. Surprisingly, factors such as the angulation of impaction, the relationship of the tooth to the anterior border of the ramus, and the root morphology may have little influence on the time that surgery requires.[92]

Other factors have been implicated in making the extraction process more difficult. Roots can be either conical and fused roots or separate and divergent, with the latter being more difficult to manage. A large follicular sac around the crown of the tooth provides more room for access to the tooth, making it less difficult to extract than one with essentially no space around the crown of the tooth.

Another important determinant of difficulty of extraction is the age of the patient. When impacted teeth are removed before age 20 years, the surgery is almost always less difficult to perform. The roots are usually incompletely formed and, thus, less bone removal is required for tooth extraction. There is usually a broader pericoronal space formed by the follicle of the tooth, which provides additional access for tooth extraction without bone removal. Because the roots of the impacted teeth are incompletely formed, they are usually separated from the inferior alveolar nerve.

In contradistinction, removal of impacted teeth in patients of older age groups is almost always more difficult. The roots are usually completely formed and are thus longer, which requires more bone removal, and closer to the inferior alveolar canal, which increases the risk of postsurgical anesthesia and paresthesia. The follicular sac almost always degenerates with age, which makes the pericoronal space thinner; as a result, more bone must be removed for access to the crown of the tooth. Finally, there is increasing density and decreasing elasticity in the bone, necessitating greater bone removal to deliver the tooth from its socket.

In summary, the degree of difficulty of the surgery to remove an impacted tooth is determined primarily by two major factors: (1) the depth of impaction and type of overlying tissue and (2) the age of the patient. Full bony impactions are always more difficult to remove than are soft tissue impactions, and given two impactions of the same depth, the impaction in the older patient is always more difficult than the one in the younger patient.

A corollary of surgical difficulty is difficulty of recovery from the surgery. As a general rule, a more challenging and time-consuming surgical procedure results in a more troublesome and prolonged postoperative recovery. It is more difficult to perform surgery in the older individual, and it is harder for these patients to recover from the surgical procedure.

Technique

The technique for removal of impacted third molars must be learned on a theoretical basis and then performed repeatedly

to gain adequate experience. There is more variety in presentation of impacted third molars than in any other condition requiring a dental surgical procedure. Therefore, extensive experience is required to master their removal. Other textbooks describe in detail the techniques for removal of the different types of impactions.[93,94]

In general, the surgeon's approach must gain adequate access to the underlying bone and tooth through a properly designed and reflected soft tissue flap. Bone must be removed in an atraumatic, aseptic, and non–heat-producing technique, with as little bone removed and damaged as possible. The tooth is then divided into sections and delivered with elevators, using judicious amounts of force to prevent complications. Finally, the wound must be thoroughly débrided mechanically and by irrigation to provide the best possible healing environment in the postoperative period.

The initial step in removing impacted teeth is to reflect a mucoperiosteal flap that is adequate in size to permit access. The most commonly used flap is the envelope flap, which extends from just posterior to the position of the impacted tooth anteriorly to approximately the level of the first molar (Figure 5-11A and B). If the surgeon requires greater access to remove a deeply impacted tooth, the envelope flap may not be sufficient. In that case, a release incision is done on the anterior aspect of the incision, creating a three-cornered flap (see Figure 5-11C and D). The envelope incision is usually associated with fewer complications and tends to heal more rapidly and with less pain than the three-cornered flap. The buccal artery is sometimes encountered when creating the releasing incision; this may be bothersome during the early portion of the surgery.

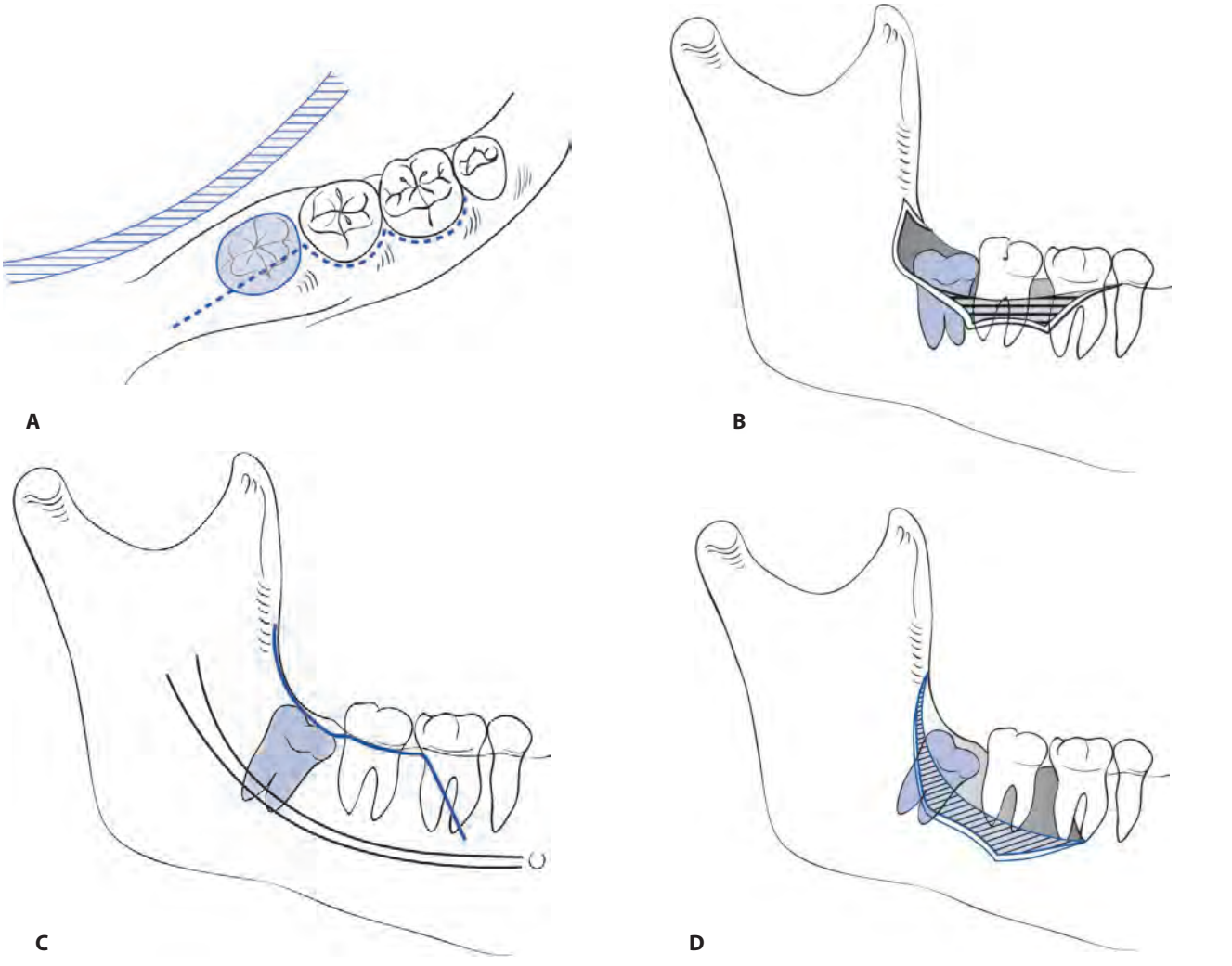


FIGURE 5-11. **A,** The envelope incision is most commonly used to reflect the soft tissue of the mandible for removal of an impacted third molar. Posterior extension of the incision should diverge laterally to avoid injury to the lingual nerve. **B,** The envelope incision is reflected laterally to expose bone overlying impacted tooth. **C,** When a three-cornered flap is used, the release incision is made at the mesial aspect of the second molar. **D,** When the soft tissue flap is reflected by means of a release incision, greater visibility is possible, especially at the apical aspect of the surgical field. (**A–D,** Adapted from Peterson LJ. Principles of management of impacted teeth. In: Peterson LJ, Ellis E III, Hupp JR, Tucker MR, editors. Contemporary Oral and Maxillofacial Surgery. 4th ed. St. Louis: CV Mosby; 2003; p. 207.)

The posterior extension of the incision must extend to the lateral aspect of the anterior border of the mandibular ramus. The incision should not continue posteriorly in a straight line because the mandibular ramus diverges laterally. If the incision were to be extended straight, the blade might damage the lingual nerve. Miloro and associates[95] used high-resolution magnetic resonance imaging to demonstrate that the lingual nerve may be intimately associated with the lingual cortical plate in the third molar region in 25% of cases and above the lingual crest in 10%. The mucoperiosteal flap is reflected laterally to the external oblique ridge with a periosteal elevator and held in this position with a retractor such as an Austin or Minnesota.

The most commonly used incision used for the maxillary third molar is also an envelope incision (Figure 5-12A and B). It extends posteriorly from the distobuccal line angle of the second molar and anteriorly to the first molar. A releasing incision is rarely necessary for the maxillary third molar (see Figure 5-12C and D), although it may be useful when the occlusal surface of the third molar is at or superior to the midportion of the second molar root.

The second major step is bone removal from around the impacted tooth. Most surgeons use a high-speed, high-torque air- or nitrogen-driven handpiece, although a few surgeons still choose to use a chisel for bone removal. It is essential that the handpiece exhaust the air pressure away from the surgical site to prevent tissue emphysema or air embolism and that the handpiece can be sterilized completely, usually in a steam autoclave. High-speed high-torque electric drills have gained wide acceptance and, like the nitrogen-driven instruments, greatly reduce the time required for bone removal and tooth sectioning.

The bone on the occlusal, buccal, and (cautiously) distal aspects of the impacted tooth is removed down to the cervical line. The amount of bone that must be removed varies with the depth of the impaction. It is advisable not to remove any bone on the lingual aspect because of the likelihood of damage to the lingual nerve (Figure 5-13). A variety of burs can be used to remove bone, but the most commonly used are the no. 8 round bur and the 703 fissure bur.

For maxillary teeth, bone removal is done primarily on the lateral aspect of the tooth down to the cervical line to expose the entire clinical crown. Frequently, the bone on the buccal aspect is thin enough that it can be removed with a periosteal elevator or a chisel using manual digital pressure.

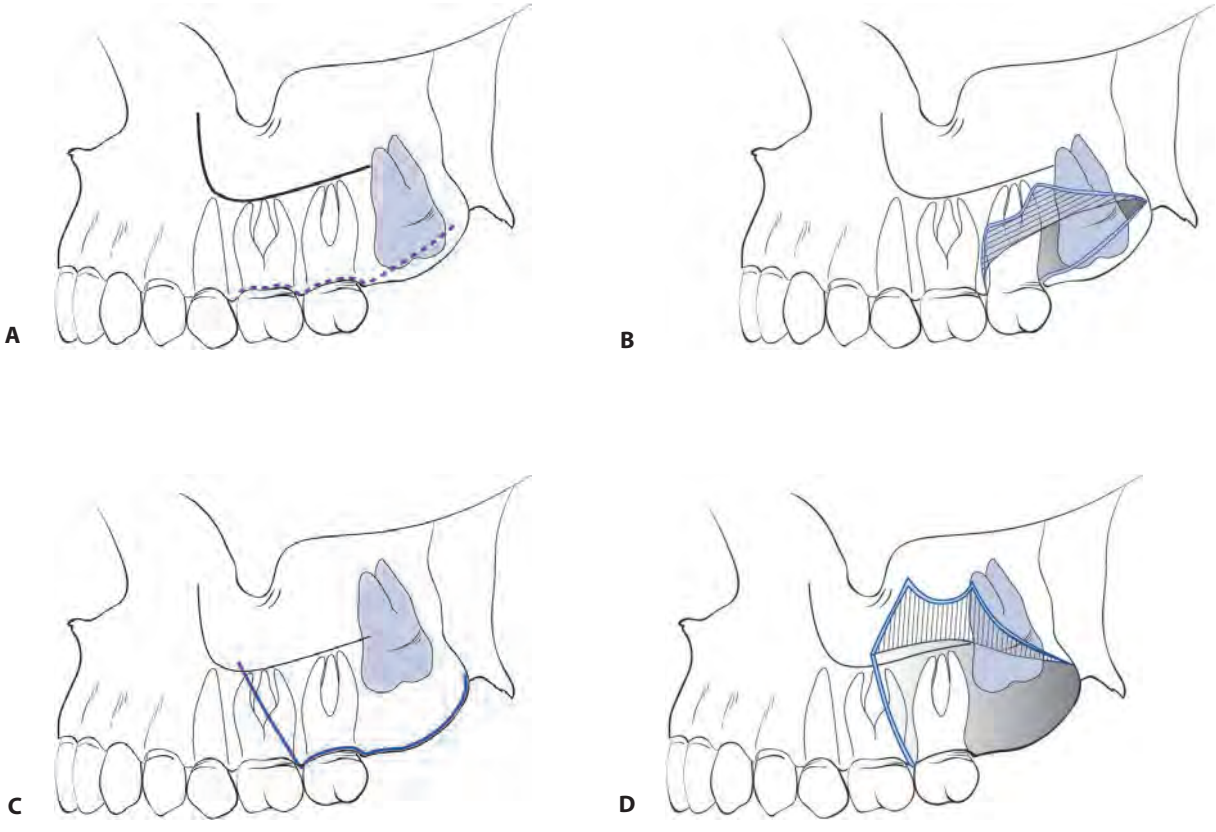


FIGURE 5-12. **A,** The envelope flap is the most commonly used flap for the removal of maxillary impacted teeth. **B,** When soft tissue is reflected, the bone overlying the third molar is easily visualized. **C,** If the tooth is deeply impacted, a release incision can be used to gain greater access. **D,** When the three-cornered flap is reflected, there is greater visibility of bone's more apical portions. (**A–D,** Adapted from Peterson LJ, Ellis E III, Hupp JR, Tucker MR, editors. Contemporary Oral and Maxillofacial Surgery. 4th ed. St. Louis: CV Mosby; 2003, p. 207.)

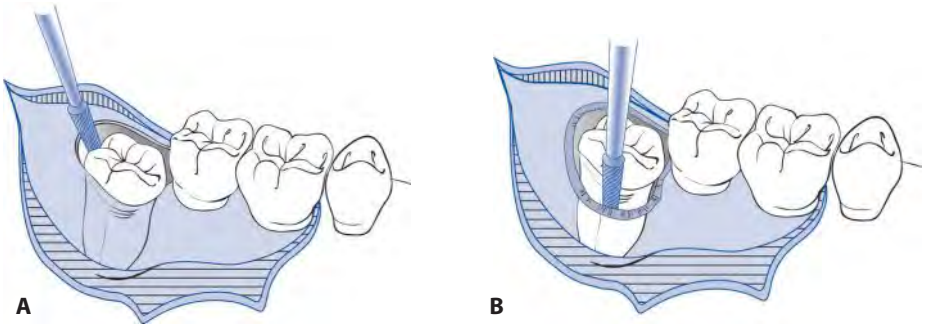


FIGURE 5-13. **A,** After the soft tissue has been reflected, the bone overlying the occlusal surface of tooth is removed with a fissure bur. **B,** Bone on the buccal and distal aspects of impacted tooth is then removed with bur. (**A** and **B,** Adapted from Peterson LJ, Ellis E III, Hupp JR, Tucker MR, editors. Contemporary Oral and Maxillofacial Surgery. 4th ed. St. Louis: CV Mosby; 2003, p. 208.)

Once the tooth has been sufficiently exposed, it is sectioned into appropriate pieces so that it can be delivered from the socket. The direction in which the impacted tooth is divided is dependent on the angulation of the impaction. Tooth sectioning is performed with either a bur or a chisel, but with the advent of high-speed drills, the bur is most commonly used because it provides a more predictable plane of sectioning. The tooth is usually divided three quarters of the way through to the lingual aspect and then split the remainder of the way with a straight elevator or a similar instrument. This prevents injury to the lingual cortical plate and reduces the possibility of damage to the lingual nerve.

The mesioangular impaction is usually the least difficult to remove. After sufficient bone has been removed, the distal half of the crown is sectioned off from the buccal groove to just below the cervical line on the distal aspect of the tooth. This portion of the tooth is delivered, and the remainder of the tooth is removed with a small straight elevator placed at a purchase point on the mesial aspect of the cervical line (Figure 5-14). An alternative is to prepare a purchase point in the tooth with the drill and use a crane pick or a Cryer elevator in the purchase point to deliver the tooth.

The horizontal impaction usually requires the removal of more bone than does the mesioangular impaction. The crown of the tooth is usually sectioned from the roots and delivered with a Cryer elevator. The roots are then displaced into the socket that was previously occupied by the crown and are delivered into the mouth. Occasionally, they may need to be sectioned into separate portions and delivered independently (Figure 5-15).

The vertical impaction is one of the more difficult ones to remove, especially if it is deeply impacted. The procedure for bone removal and sectioning is similar to that for the mesioangular impaction in that occlusal, buccal, and judicious distal bone is removed first. The distal half of the crown is sectioned and removed, and the tooth is elevated by applying a small straight elevator at the mesial aspect of the cervical line (Figure 5-16). The option of preparing a purchase point in the tooth is also frequently used, as for the mesioangular impaction.

The most difficult tooth to remove is one with a distoangular impaction. After the removal of bone, the crown is usually sectioned from the roots just above the cervical line and delivered with a Cryer elevator. A purchase point is then prepared in the tooth, and the roots are delivered together or sectioned and delivered independently with a Cryer elevator (Figure 5-17). Extraction of this impaction is more difficult because more distal bone must be removed and the tooth tends to be elevated posteriorly into the ramus portion of the mandible.

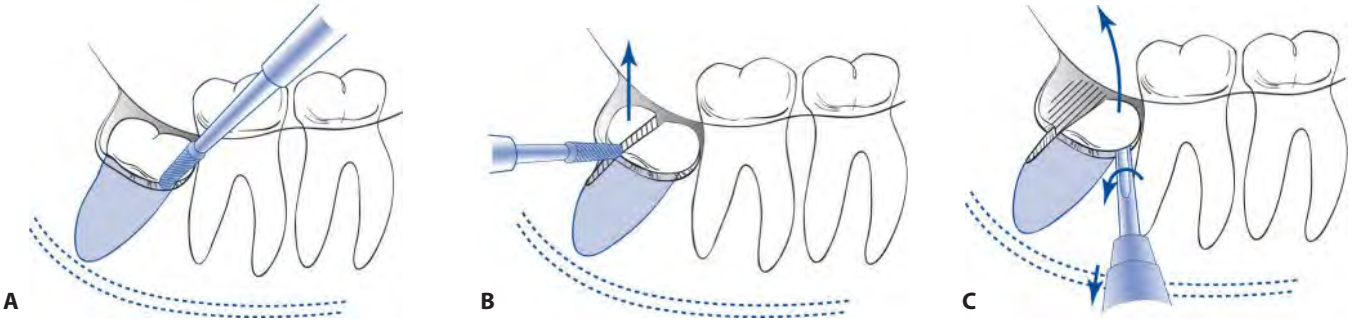


FIGURE 5-14. **A,** When removing a mesioangular impaction, buccal and distal bone are removed to expose crown of tooth to its cervical line. **B,** The distal aspect of the crown is then sectioned from tooth. Occasionally it is necessary to section the entire tooth into two portions rather than to section the distal portion of crown only. **C,** After the distal portion of crown has been delivered, a small straight elevator is inserted into the purchase point on mesial aspect of third molar, and the tooth is delivered with a rotational and level motion of elevator. (**A–C,** Adapted from Peterson LJ, Ellis E III, Hupp JR, Tucker MR, editors. Contemporary Oral and Maxillofacial Surgery. 4th ed. St. Louis: CV Mosby; 2003, p. 208.)

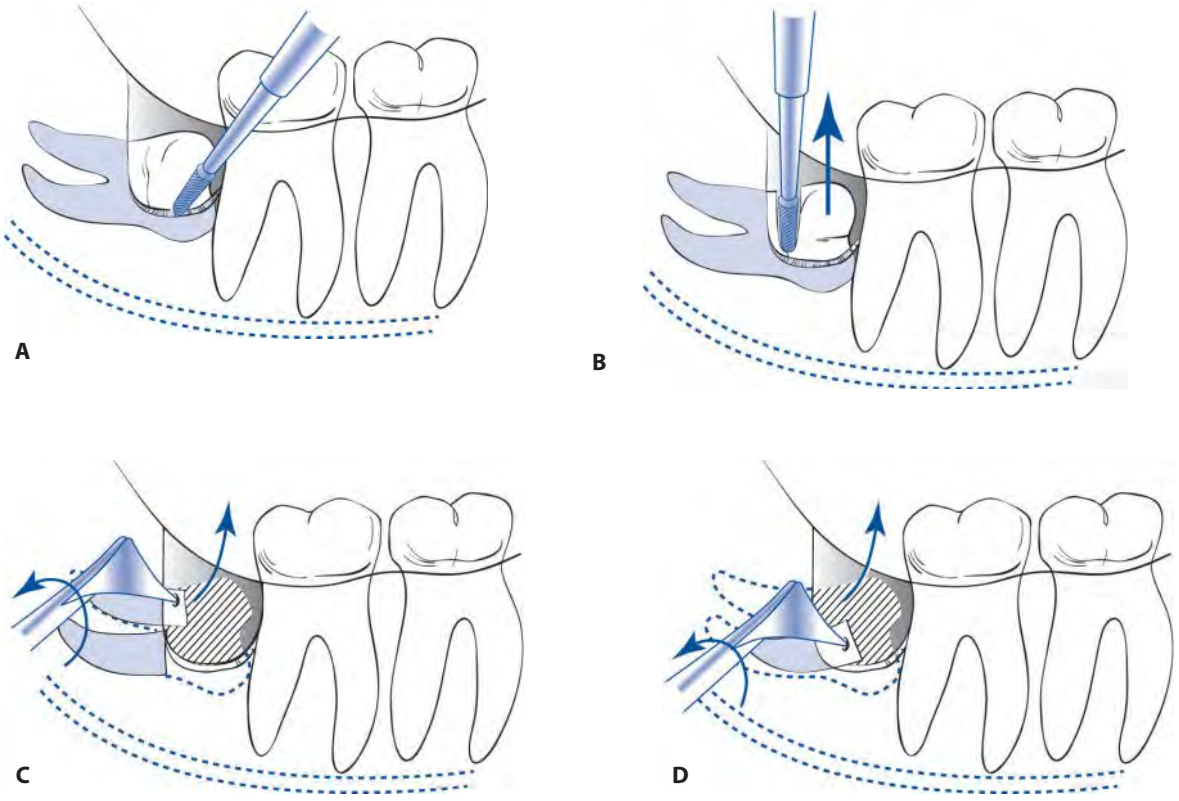


FIGURE 5-15. **A,** During the removal of a horizontal impaction, the bone overlying the tooth—that is, the bone on the distal and buccal aspects of tooth—is removed with a bur. **B,** The crown is sectioned from the roots of the tooth and is delivered from socket. **C,** The roots are delivered together or independently with a Cryer elevator used with a rotational motion. The roots may need to be separated into two parts: occasionally, the purchase point is made in the root to allow the Cryer elevator to engage it. **D,** The mesial root of the tooth is elevated in similar fashion. (**A–D,** Adapted from Peterson LJ, Ellis E III, Hupp JR, Tucker MR, editors. Contemporary Oral and Maxillofacial Surgery. 4th ed. St. Louis: CV Mosby; 2003, p. 209.)

Impacted maxillary third molars are rarely sectioned because the overlying bone is thin and relatively elastic. In patients with thicker bone, the extraction is usually accomplished by removing additional bone rather than by sectioning the tooth. The tooth should never be sectioned with a chisel because it may be displaced into the maxillary sinus or infratemporal fossa when struck with the chisel (Figure 5-18).

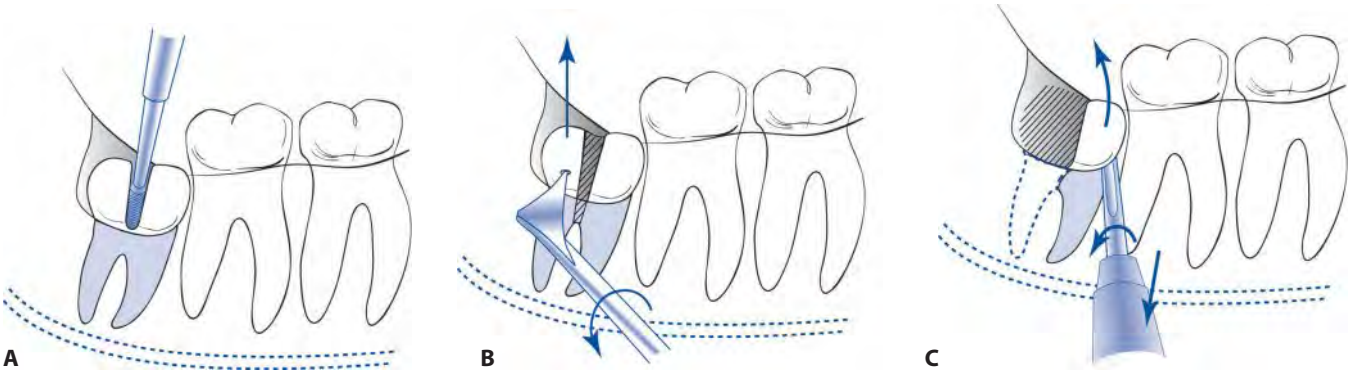


FIGURE 5-16. **A,** When removing a vertical impaction, the bone on the occlusal, buccal, and distal aspects of the crown is removed, and the tooth is sectioned into mesial and distal portions. If the tooth has a fused single root, the distal portion of the crown is sectioned off in a manner similar to that depicted for a mesioangular impaction. **B,** The posterior aspect of the crown is elevated first with a Cryer elevator inserted into a small purchase point in the distal portion of the tooth. **C,** A small straight no. 301 elevator is then used to lift the mesial aspect of the tooth with a rotary and levering motion. (**A–C,** Adapted from Peterson LJ, Ellis E III, Hupp JR, Tucker MR, editors. Contemporary Oral and Maxillofacial Surgery. 4th ed. St. Louis: CV Mosby; 2003, p. 210.)

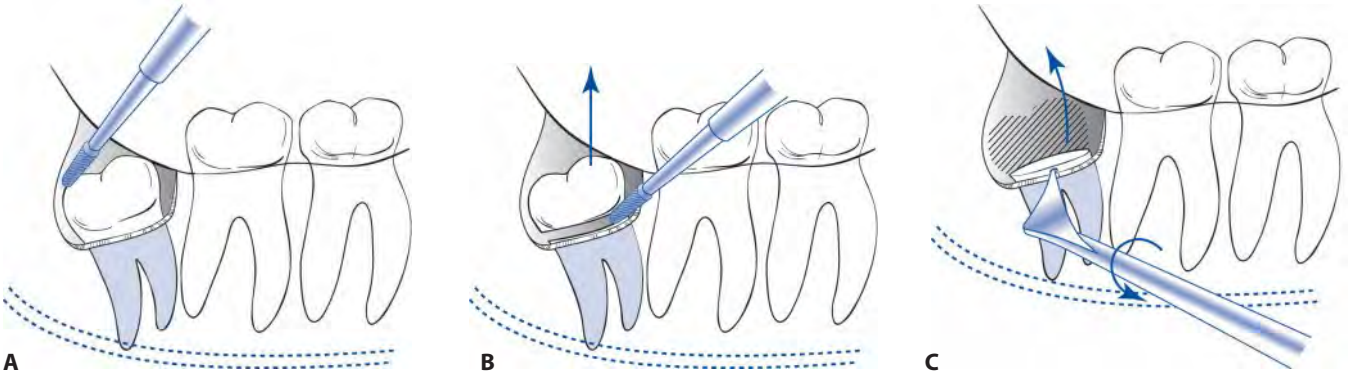


FIGURE 5-17. **A,** For a distoangular impaction, the occlusal, buccal, and distal bone is removed with a bur. It is important to remember that more distal bone must be taken off than for a vertical or mesioangular impaction. **B,** The crown of the tooth is sectioned off with a bur and is delivered with straight elevator. **C,** The purchase point is put into the remaining root portion of the tooth, and the roots are delivered by a Cryer elevator with a wheel-and-axle motion. If the roots diverge, it may be necessary in some cases to split them into independent portions. (**A–C,** Adapted from Peterson LJ, Ellis E III, Hupp JR, Tucker MR, editors. Contemporary Oral and Maxillofacial Surgery. 4th ed. St. Louis: CV Mosby; 2003, p. 211.)

Once the impacted tooth is delivered from the alveolar process, the surgeon must pay strict attention to débriding the wound of all particular bone chips and other debris. The best method to accomplish this is to mechanically débride the socket and the area under the flap with a periapical curette. A bone file should be used to smooth any rough sharp edges of the bone. A mosquito hemostat is usually used carefully to remove any remnant of the dental follicle. Finally, the socket and wound should be thoroughly irrigated with saline or sterile water (30–50 mL is optimal).[96] Within certain limitations, the more irrigation that is used, the less likely the patient is to have a dry socket, delayed healing, or other complications.

The incision should usually be closed by primary intention. The flap is returned to its original position, and the initial resorbable suture is placed at the posterior aspect of the second molar. Additional sutures are placed as necessary.

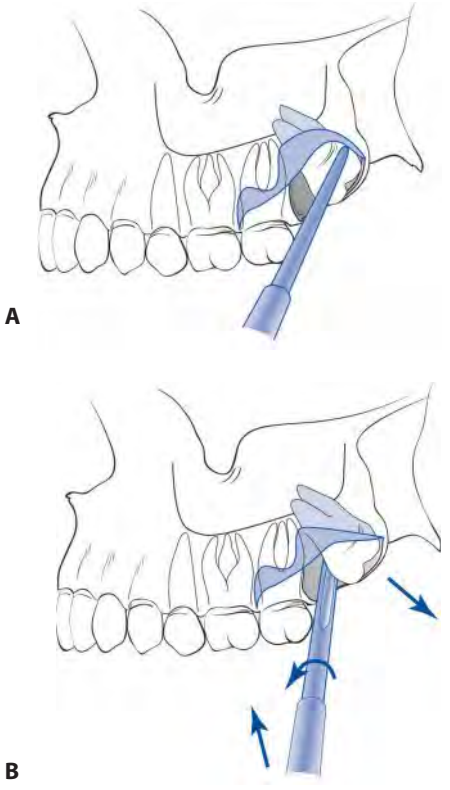


FIGURE 5-18. Delivery of an impacted maxillary third molar. **A,** Once the soft tissue has been reflected, a small amount of buccal bone is removed with a bur or a hand chisel. **B,** The tooth is then delivered by a small straight elevator with rotational and lever types of motion. The tooth is delivered in the distobuccal and occlusal direction. (**A** and **B,** Adapted from Peterson LJ, Ellis E III, Hupp JR, Tucker MR, editors. Contemporary Oral and Maxillofacial Surgery. 4th ed. St. Louis: CV Mosby; 2003, p. 212.)

Use of Perioperative Systemic Antibiotics

One of the primary goals of the surgeon in performing any surgical procedure is to prevent postoperative infection as a result of surgery. To achieve this goal, prophylactic antibiotics are necessary in some surgical procedures. Most of these procedures fall into the clean-contaminated or contaminated categories of surgery. The incidence of postoperative infections in a clean surgery is related more to operator technique than to the use of prophylactic antibiotics.

Surgery for the removal of impacted third molars clearly fits into the category of clean-contaminated surgery; however, the exact incidence of postoperative infection is unknown. In the usual sense of the word, infection probably is a rare occurrence after third molar surgery. This means that it is unusual to see pain, swelling, and a production of purulence that requires incision and drainage or antibiotic therapy. The incidence of such infections is very low for most surgeons. In general, a competent, experienced surgeon would expect to have an infection rate in the range of 1% to 5% for all third molar procedures.[97] It is difficult, and probably impossible, to reduce infection rates below 5% with the use of prophylactic antibiotics. Therefore, it is unnecessary to use prophylactic antibiotics in third molar surgery to

prevent postoperative infection in the normal healthy patient. Although the literature contains many articles that discuss the use of prophylactic perioperative antibiotics, there is essentially no report of their usefulness in the prevention of infection after third molar surgery.[98,99]

A more subtle type of wound healing problem that occurs after the surgical removal of the impacted mandibular third molar is so-called alveolar osteitis or dry socket. This disturbance in wound healing is most likely caused by the combination of saliva and anaerobic bacteria. The use of prophylactic antibiotics in third molar surgery does, in fact, reduce the incidence of dry socket. Other techniques that reduce bacterial contamination of the socket, such as copious irrigation, preoperative rinses with chlorhexidine, and placement of antibiotics in the extraction socket, are also effective.[100–107] Once again, the issue of risks versus benefits becomes important. Although systemic antibiotics are effective in the reduction of postoperative dry socket, they are no more effective than local measures. The increase of antibiotic-related complications, such as allergy, resistant bacteria, gastrointestinal side effects, and secondary infections, is not outweighed by the benefits. Therefore, the routine administration of perioperative systemic antibiotics does not seem to be valid.

Use of Perioperative Steroids

Just as the oral and maxillofacial surgeon desires to minimize the incidence of infection after third molar surgery, he or she also has a major interest in reducing the perioperative morbidity. The use of corticosteroids to help minimize swelling, trismus, and pain has gained wide acceptance in the oral and maxillofacial surgery community. The method of usage, however, is extremely variable, and the most effective therapeutic regimen has yet to be clearly delineated.

There is little doubt that an initial intravenous dose of steroid at the time of surgery has a major clinical impact on swelling and trismus in the early postoperative period. However, if the initial intravenous dose is not followed up with additional doses of steroids, this early advantage disappears by the second or third postoperative day. Maximum control of swelling requires that additional steroids be given for 1 or 2 days after surgery. The two most widely used steroids are dexamethasone and methylprednisolone. Both of these are almost pure glucocorticoids, with little mineralocorticoid effect. In addition, these two appear to have the least depressing effect on leukocyte chemotaxis. Common dosages of dexamethasone are 4 to 12 mg intravenously at the time of surgery. Additional oral dosages of 4 to 8 mg twice a day on the day of surgery and for 2 days afterward result in the maximum relief of swelling, trismus, and pain. Methylprednisolone is most commonly given 125 mg intravenously at the time of surgery followed by significantly lower doses, usually 40 mg orally three or four times a day, later on the day of surgery and for 2 days after surgery.

High-dose short-term steroid use is associated with minimal side effects. It is contraindicated in the patient with gastric ulcer disease, active infection, and certain types of psychosis. The administration of perioperative steroids may increase the incidence of alveolar osteitis after third molar surgery, but the data are lacking as to the precise degree of increase.[108–112]

EXPECTED POSTOPERATIVE COURSE

Surgical removal of impacted third molars is associated with a moderate incidence of complications, approximately 10%.[113,114] These complications range from the expected and predictable outcomes, such as swelling, pain, stiffness, and mild bleeding, to more severe and permanent complications, such as inferior alveolar nerve anesthesia and fracture of the mandible. The overall incidence of complication and the severity of these complications are associated most directly with the depth of impaction, that is, whether it is a complete bony impaction, and to the age of the patient.[115–117] Because of factors already discussed, removal of impacted teeth in the older patient is associated with a higher incidence of postoperative complications, especially alveolar osteitis, infections, mandible fracture, and inferior alveolar nerve anesthesia. The removal of complete bony impactions is likewise associated with increased postoperative pain and morbidity and an increase in the incidence of inferior alveolar nerve anesthesia.

Another determinant of the incidence of complications of third molar surgery is the relative experience and training of the surgeon. The less experienced surgeon will have a significantly higher incidence of complications than the trained experienced surgeon.[1,2] After the surgical removal of an impacted third molar, certain normal physiologic responses occur. These include such things as mild bleeding, swelling, stiffness, and pain. All of these are interpreted by the patient as being unpleasant and should, therefore, be minimized as much as possible.

With experience, most oral and maxillofacial surgeons develop a clear understanding of third molar surgery's impact on their patients' lives. However, despite its extreme importance, this topic has received little significant study. Several authorities have published data on the short-term impact of third molar removal on quality of life.[118,119] As expected, third molar removal often has a profoundly negative impact for the first 4 to 7 days after surgery, but longer follow-up reveals improved quality of life, mostly resulting from the elimination of chronic pain and inflammation (usually pericoronitis). Large population studies of post–third molar removal health-related quality of life have provided detailed information on the consequences of impaction surgery and have shed light on predictors of delayed recovery.[120–123] The performing surgeon must be familiar with this rapidly expanding knowledge if she or he is to provide proper preoperative counseling.

Bleeding

Bleeding can be minimized by using a good surgical technique and by avoiding the tearing of flaps or excessive

trauma to the overlying soft tissue. When a vessel is cut, the bleeding should be stopped to prevent secondary hemorrhage after surgery. The most effective way to achieve hemostasis after surgery is to apply a moist gauze pack directly over the site of the surgery with adequate pressure. This is usually done by having the patient bite down on a moist gauze pad. In some patients, immediate postoperative hemostasis is difficult. In such situations, a variety of techniques can be employed to help secure local hemostasis, including oversuturing and the application of topical thrombin on a small piece of absorbable gelatin sponge into the extraction socket. The socket can also be packed with oxidized cellulose. Unlike the gelatin sponge, oxidized cellulose can be packed into the socket under pressure. In some situations, microfibrillar collagen can be used to promote platelet plug formation. Patients who have known acquired or congenital coagulopathies require extensive preparation and preoperative planning (e.g., determination of International Normalized Ratio, factor replacement, hematology consultation) before third molars are removed surgically.

Swelling

Postsurgical edema or swelling is an expected sequela of third molar surgery. As discussed earlier, the parenteral administration of corticosteroids is frequently employed to help minimize the swelling that occurs. The application of ice packs to the face may make the patient feel more comfortable but has no effect on the magnitude of edema.[124] The swelling usually reaches its peak by the end of the second postoperative day and is usually resolved by the fifth to seventh day.

Stiffness

Trismus is a normal and expected outcome after third molar surgery. Patients who are administered steroids for the control of edema also tend to have less trismus. Like edema, jaw stiffness usually reaches its peak on the second day and resolves by the end of the first week.

Pain

Another postsurgical morbidity expected after third molar surgery is pain. The postsurgical pain begins when the effects of the local anesthesia subside and reaches its maximum intensity during the first 12 hours postoperatively.[125] A large variety of analgesics are available for management of postsurgical pain. The most common ones are combinations of acetylsalicylic acid or acetaminophen with codeine and its congeners, and the nonsteroidal anti-inflammatory analgesics. Women may be more sensitive to postoperative pain than men[126]; thus, they require more analgesics. Analgesics should be given before the effect of the local anesthesia subsides. In this manner, the pain is usually easier to control, requires less drug, and may require a less potent analgesic. The administration of nonsteroidal anti-inflammatories before surgery may be beneficial in aiding in the control of postoperative pain.

The most important determinant of the amount of postoperative pain that occurs is the length of the operation. Neither swelling nor trismus correlates with the length of time of the surgery. There is, however, a strong correlation between postoperative pain and trismus, indicating that pain may be one of the principal reasons for the limitation of opening after the removal of impacted third molars.[127]

POSTOPERATIVE COMPLICATIONS AFTER THIRD MOLAR SURGERY

Infection

An uncommon postsurgical complication related to the removal of impacted third molars is infection. The incidence of infection after the removal of third molars is very low, ranging from 1.7% to 2.7%.[128] Infection after removal of mandibular third molars is almost always a minor complication. Approximately 50% of infections are localized subperiosteal abscess-type infections, which occur 2 to 4 weeks after a previously uneventful postoperative course. These are usually attributed to debris that is left under the mucoperiosteal flap and are easily treated by surgical débridement and drainage. Of the remaining 50%, few postoperative infections are significant enough to warrant surgery, antibiotics, and hospitalization. Infections occur in the first postoperative week after third molar surgery approximately 0.5% to 1% of the time. This is an acceptable infection rate and would not be decreased with the administration of prophylactic antibiotics.

Tooth Fracture

One of the most frequent problems encountered in removing third molars is the fracture of a portion of the root, which may be difficult to retrieve. In these situations, the root fragment may be displaced into the submandibular space, the inferior alveolar canal, or the maxillary sinus. Uninfected roots left within the alveolar bone have been shown to remain in place without postoperative complications.[129] The pulpal tissues undergo fibrosis, and the root becomes totally incorporated within the alveolar bone. Aggressive and destructive attempts to remove portions of roots that are in precarious positions seem to be unwarranted and may cause more damage than benefit. Radiographic follow-up may be all that is required.

Alveolar Osteitis

The incidence of alveolar osteitis or dry socket after the removal of impacted mandibular third molars varies between 3% and 25%. Most of the variation is most likely a result of the definition of the syndrome. When *dry socket* is defined in terms of pain that requires the patient to return to the surgeon's office, the incidence is probably in the range of 20% to 25%.[2,130,137]

The pathogenesis of alveolar osteitis has not been clearly defined, but the condition is most likely the result of lysis of a fully formed blood clot before the clot is replaced with granulation tissue. This fibrinolysis occurs during the third and fourth days and results in symptoms of pain and malodor after the third day or so following extraction. The source of the fibrinolytic agents may be tissue, saliva, or bacteria.[130] The role of bacteria in this process can be confirmed empirically based on the fact that systemic and topical antibiotic prophylaxis reduces the incidence of dry socket by approximately 50% to 75%. The periodontal ligament may also play a role in the development of alveolar osteitis.

The incidence of dry socket seems to be higher in patients who smoke and in female patients who take oral contraceptives.[132,133] Its occurrence can be reduced by several techniques, most of which are aimed at reducing the bacterial contamination of the surgical site. Presurgical irrigation with antimicrobial agents such as chlorhexidine reduces the incidence of dry socket by up to 50%.[2] Copious irrigation of the surgical site with large volumes of saline is also effective in reducing dry socket.[96] Topical placement of small amounts of antibiotics such as tetracycline or lincomycin may also decrease the incidence of alveolar osteitis.[133–136]

The goal of treatment of dry socket is to relieve the patient's pain during the delayed healing process. This is usually accomplished by irrigation of the involved socket, gentle mechanical débridement, and placement of an obtundent dressing, which usually contains eugenol. The dressing may need to be changed on a daily basis for several days and then less frequently after that. The pain syndrome usually resolves within 3 to 5 days, although it may take as long as 10 to 14 days in some patients. There is some evidence that topical antibiotics such as metronidazole may hasten resolution of the dry socket.[137]

In summary, alveolar osteitis is a disturbance in healing that occurs after the formation of a mature blood clot but before the blood clot is replaced with granulation tissue. The primary etiology appears to be one of excess fibrinolysis, with bacteria playing an important but yet ill-defined role. Antimicrobial agents delivered by perioperative mouthrinses, topically placed in the socket, or administered systemically all help to reduce the incidence of dry socket. Mechanical débridement and copious saline irrigation of the surgical wound also are effective in reducing the incidence of dry socket. A rational approach may be to provide preoperative chlorhexidine rinses for approximately 1 week before surgery, irrigate the wound thoroughly with normal saline at the conclusion of surgery, place a small square of gelatin sponge saturated with tetracycline in the socket, and continue chlorhexidine rinses for 1 additional week. This combination approach should substantially reduce the incidence of dry socket.

Nerve Disturbances

Surgical removal of mandibular third molars places both the lingual and the inferior alveolar branches of the third division of the trigeminal nerve at risk for injury. The lingual nerve is most often injured during soft tissue flap reflection, whereas the inferior alveolar nerve is injured when the roots of the teeth are manipulated and elevated from the socket. The generally accepted incidence of injury to the inferior alveolar and lingual nerves after third molar surgery is approximately 3%.[114–118,138–140] Only a small proportion of these anesthesia and paresthesia problems remain permanent. However, there is a significant incidence of some minor alterations of sensation after injury caused by third molar surgery. As many as 45% of nerve compression injuries, which are typical in third molar surgery, result in a permanent neurosensory abnormality.[141]

Inferior alveolar nerve injury is most likely to occur in specific situations. The first and most commonly reported predisposing factor is complete bony impaction of mandibular third molars. The angulation classifications most commonly involved are usually mesioangular and vertical impaction. In some cases, nerve proximity to the root is indicated by an apparent narrowing of the inferior alveolar canal as it crosses the root or severe root dilaceration adjacent to the canal. Other well-documented radiographic signs are diversion of the path of the canal by the tooth, darkening of the apical end of the root indicating that it is included within the canal, and interruption of the radiopaque white line of the canal.[142] In surgically verified inferior alveolar nerve injuries, the presence of more than one of these signs was highly sensitive but not highly specific for the risk of injury, whereas the absence of all of these signs had a strong negative predictive value.[143] When they are noted on a preoperative evaluation of the radiograph, the surgeon should take extraordinary precautions to avoid injury to the nerve, such as additional bone removal or sectioning of the tooth into extra pieces, and the patient should be counseled in advance regarding his or her increased risk of nerve injury.

When an injury to the lingual or inferior alveolar nerve is diagnosed in the postoperative period, the surgeon should begin long-term planning for its management including consideration of referral to a neurologist and/or microneurosurgeon. These issues are dealt with elsewhere in this book.

Rare Complications

The complications already discussed are the more common occurrences, accounting for the great majority of complications in surgery to remove impacted third molars. Several additional complications occur only rarely and are mentioned briefly.

Maxillary third molars that are deeply impacted may have only thin layers of bone posteriorly separating them from the infratemporal fossa or anteriorly separating them from the maxillary sinus. Small amounts of pressure in an errant direction can result in displacement of the maxillary third molar into these adjacent spaces. When a maxillary third molar is displaced posteriorly into the infratemporal fossa, the surgeon should try to manipulate the tooth back into the socket with

finger pressure placed high in the buccal vestibule near the pterygoid plates. If this is unsuccessful, the surgeon can attempt to recover the tooth by placing the suction tip into the socket and aiming it posteriorly. If both of these maneuvers are unsuccessful in recovering the tooth, the most effective technique is to allow the tooth to undergo fibrosis and to have the patient return 2 to 4 weeks later to have it removed. If the tooth is asymptomatic, is not causing any restriction in jaw movement, infection, or pain, the surgeon should consider leaving the tooth in place. If the decision is made to remove the tooth, three-dimensional localization of the tooth should be done before surgery is initiated.

If the tooth is displaced into the maxillary sinus, retrieval is usually done by a Caldwell-Luc procedure at the same appointment. The surgeon should localize the tooth with at least a one-dimensional radiographic view and preferably a three-dimensional study before performing the retrieval surgery.[145]

Fracture of the mandible during the removal of impacted mandibular third molars is a rare occurrence. The typical situation is a deeply impacted third molar, most commonly in an older individual with dense bone. The surgeon places excessive pressure on the tooth with an elevator in an attempt to deliver the tooth or tooth section into the mouth; the fracture occurs, and the remaining portion of the tooth is easily retrieved. The surgeon should then perform an immediate reduction and fixation of the fracture. If the surgeon has the experience and the armamentarium available, rigid internal fixation with miniplates is an excellent choice in this unfortunate situation. Wire fixation and application of intermaxillary fixation is an acceptable alternative. Late mandible fractures usually occur 4 to 6 weeks after extraction in patients older than 40 years.

PERIODONTAL HEALING AFTER THIRD MOLAR SURGERY

Among the important reasons for removing impacted third molars is preserving periodontal health or, in some situations, treating a periodontitis that already exists.[145] A relative contraindication to the removal of impacted third molars is a situation in which there is good periodontal health and a complete bony impaction in an older patient. Removal is contraindicated because the healing response in older patients would likely result in a large persistent postsurgical defect.

After third molar surgery, the bone height distal to the second molar usually remains at the preoperative level,[54,146,147] although some studies have indicated a net gain in bone level after surgery.[148] If the bone level on the distal aspect of the mandibular second molar is compromised by the presence of the third molar, it usually remains at that level after healing of the bone. There is universal agreement that bone healing is better if surgery is done before the third molar resorbs the bone on the distal aspect of the second molar and while the patient is young.[55,56,149] The greatest bony defect occurs in situations in which the third molar has resorbed extensive amounts of bone from the second molar in an older patient, which compromises bony repair and bone healing.

The other periodontal parameter of importance is attachment level or, less accurately, sulcus or pocket depth. As with bone levels, if the preoperative pocket depth is great, the postoperative pocket depth is likely to be similar. In most studies, the attachment level has been found to be at essentially the same level as it is preoperatively.[150–152] In older patients with complete bony impactions, pocket depth and attachment levels may be significantly lower than preoperative levels. However, in patients younger than age 19 years, removal of complete bony impactions results in no compromise in attachment level or pocket depth. Initial healing after third molar surgery usually results in a reduction in pocket depth in young patients.[54] The long-term healing in this group continues for up to 4 years after surgery, with continuing reduction in probable pocket depths.[148] However, long-term follow-up of older patients clearly demonstrates that this long-term healing does not occur.[55,148]

Usually, the surgeon makes an attempt to mechanically débride the distal aspect of the second molar root area with a curette to encourage improved bone regeneration after third molar extraction. In recent years, attention has turned to placing graft material in the third molar socket after its removal. Many studies have shown that routine use of adjunctive techniques to regenerate bone in the lower third molar socket, including use of graft materials, is not warranted.[153–155] Dodson[156–158] has studied this question exhaustively and has identified a population of patients in whom bone grafting the third molar socket appears to have a predictable benefit. These are patients who are older than 25 years, have preexisting periodontal defects at the second molar such as probing depths greater than 4 mm or attachment levels greater than 3 mm, and have horizontal or mesioangular third molar impaction. Some studies suggest that patients with two of these risk factors may also have increased risk for persistent periodontal defects on the distal of the second molar,[159,160] although evidence supporting bone grafting or other interventions remains inconclusive.

In summary, periodontal healing after third molar surgery is clearly best when the impacted tooth is removed before it becomes exposed in the mouth, before it resorbs bone on the distal aspect of the second molar, and when the patient is as young as possible.[54,55,146,149,151,152] If the third molar is partially impacted and is partially exposed in the mouth, it should be removed as soon as possible. The reason for this is that there is already a deep and potentially destructive periodontal lesion that is difficult for the patient to maintain hygienically Even if the patient is asymptomatic, the impacted tooth should be removed as soon as possible to allow the best possible periodontal healing after surgery. In these situations, the periodontal healing is compromised because of the fact that there was already a destructive lesion caused by the presence of the partially impacted third molar.

The completely impacted third molar in a patient older than 35 years should be left undisturbed unless some

pathology develops. Removal of asymptomatic completely impacted third molars in these older patients results in pocket depths that are significant and the potential loss of alveolar bone on the posterior aspect of the second molar.

SUMMARY

The issue of whether to remove impacted third molars has generated much controversy since the 1980s. The reason for this controversy has been the lack of long-term prospective studies that have followed up large groups of patients with impacted teeth to determine the eventual outcome of leaving impactions in situ. Recently, there has been intense interest in establishing clear scientifically valid evidence regarding the role of third molar removal in patient health care, especially with respect to predicting the likelihood of eruption or the risk of future pathology in asymptomatic patients. Ongoing studies are already greatly improving our knowledge in these areas, and significant advances will continue to appear in the scientific literature.

Clearly, impacted third molars associated with or contributing to adjacent pathology require removal as early as is reasonably possible. The major controversy regarding proper care centers around asymptomatic unerupted third molars. It is clear that, although incompletely erupted mandibular third molars will continue to erupt beyond age 18 or 20 years, in the vast majority of these situations, there will be a soft tissue or bone tissue flap over the distal aspect of the erupted third molar, which has the potential to cause recurrent pericoronitis. In fact, the tooth that is most likely to be involved in pericoronitis is the erupted vertically positioned third molar with a soft tissue flap (operculum) over the distal aspect of the tooth. Although attempts at very early prediction of impaction and removal of tooth buds at age 8 or 9 years have now been largely abandoned, it is likely that by age 16 or 18 years the dentist and surgeon can reasonably predict whether there will be adequate room for the tooth to erupt with sufficient clearance of the anterior ramus to prevent soft tissue overgrowth (as in patients with large arch length and relatively small teeth).

Soft tissue and bone tissue healing will occur at a maximum level if the surgery to remove impacted third molars is done as early possible. By age 16 years, if the diagnosis of inadequate room for functional eruption can be made, then the asymptomatic third molar should be removed. Even though the tooth may be completely covered with soft and hard tissue, removing the third molar at that age will eliminate the future pathologic potential and maximize the periodontal health of the second molar; these are important goals of the oral and maxillofacial surgeon.

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6

CHAPTER

Preprosthetic and Reconstructive Surgery

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Preprosthetic surgery in the 1970s and early 1980s involved methods to prepare or improve a patient's ability to wear complete or partial dentures. Most procedures were centered around soft tissue corrections that allowed prosthetic devices to fit more securely and function more comfortably. In severe cases, bony augmentation was incorporated and included such procedures as cartilage grafts, rib grafts, alloplastic augmentation, visor osteotomies, and sandwich grafts. Patients who were poor candidates for surgery were often left with less than satisfactory results both functionally and aesthetically.

In the late 1970s, Brånemark and colleagues[1] demonstrated the safety and efficacy of the implant-borne prosthesis. In the 1990s, implantology, distraction osteogenesis, and guided tissue regeneration significantly expanded the capabilities of today's reconstructive and preprosthetic surgeon. Genetically engineered growth factors will soon revolutionize our thoughts about reconstructive procedures. As a result, more patients are able to tolerate procedures because they are given increased freedom and satisfaction with regard to their prosthetic devices and, in many cases, undergo less invasive techniques.

In spite of the fact that routine dental care has improved over the past century, approximately 10% of the population is either partially or completely edentulous and more than 30% of patients older than 65 years are completely edentulous.[2] Although these figures are predicted to decrease over the next several decades, the treatment of partial and total edentulism will never be completely eliminated from the oral and maxillofacial surgeon's armamentarium.

Because the primary goal in preprosthetic reconstructive surgery is to eliminate the condition of edentulism, one must consider the etiology of the edentulous state when evaluating patients and planning treatment. In many cases, the etiology of a patient's edentulism has a major bearing on the reconstructive and restorative plan. Edentulism arising from neglect of the dentition and/or periodontal disease often poses different reconstructive challenges than does that resulting from trauma, ablative surgery, or congenital defects. Although restoration of a functional dentition is the common goal, each specific etiology poses its own unique set of challenges. The goal of preprosthetic and reconstructive surgery in the 21st century is to establish a functional biologic platform for supportive or retentive mechanisms that will maintain or support prosthetic rehabilitation without contributing to further bone or tissue loss. This environment will allow for a prosthesis that restores function, is stable and retentive, preserves the associated structures, and satisfies aesthetics.[3]

CHARACTERISTICS OF ALVEOLAR BONE IN THE EDENTULOUS PATIENT

Native alveolar bone responds to the functional effects (or lack thereof) caused by edentulism. Increased resorption owing to traditional methods of oral rehabilitation with complete and partial dentures often results in an overall acceleration of the resorptive process. The mandible is affected to a greater degree than the maxilla owing to muscle attachments and functional surface area.[4] As a result, there is proportionally a qualitative and quantitative loss of tissue, resulting in adverse skeletal relationships in essentially all spatial dimensions (Figure 6-1).

General systemic factors, such as osteoporosis, endocrine abnormalities, renal dysfunction, and nutritional deficiencies, play a role in the overall rate of alveolar atrophy. Local factors, including jaw function, vascular changes, increased physical demands owing to decreased mandibular plane

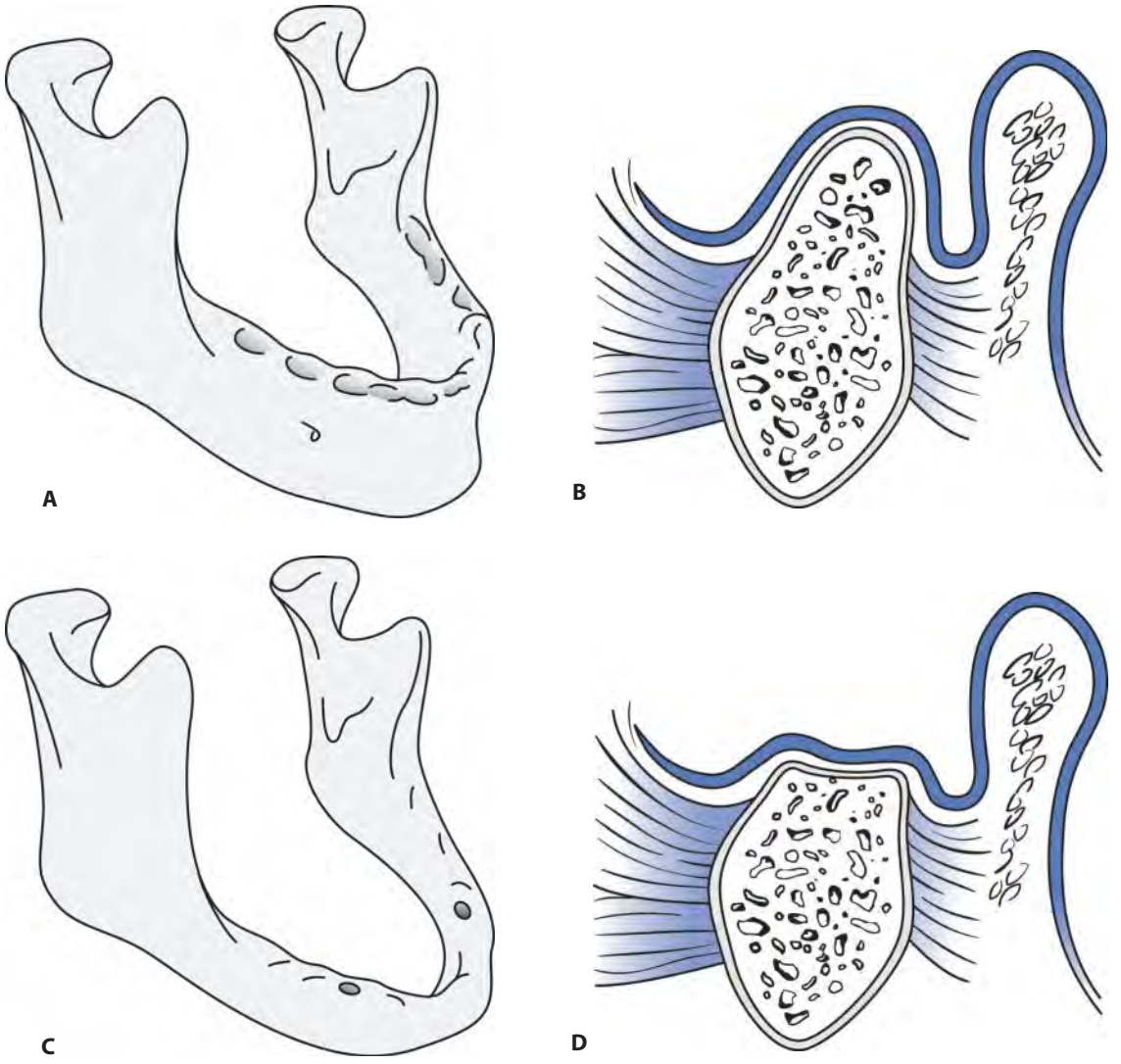


FIGURE 6-1. The diagrams show patterns and varying degrees of severity of mandibular atrophy. **A,** Mandible shows minimal alveolar bone resorption. **B,** Cross-section of large alveolar ridge including mucosal and muscular attachments. **C,** Mandible shows severe loss of alveolar bone that has resulted in residual basal bone only. **D,** Cross-section shows resorbed alveolar ridge, with muscular attachments. (**A–D,** Adapted from Tucker MR. Ambulatory preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; p. 1104.)

angle, adverse prosthetic loading, mucosal inflammation, vascular changes, and the number and extent of previous surgeries involving mucoperiosteal elevation, also contribute to progressive alveolar bone loss.[5]

Although the factors contributing to bone loss and the resulting patterns are well understood, the rate of bone loss varies significantly from individual to individual. The consistent factor is the overall duration of the patient's edentulous state.

FUNCTIONAL EFFECTS OF EDENTULISM

The maxillomandibular relationship is altered in all spatial dimensions as a result of the loss of physiologic function and teeth. There is a progression toward decreased overall lower facial height, leading to the typical overclosed appearance, decreased alveolar support for traditional prostheses, encroachment of muscle and tissue attachments to the alveolar crest resulting in progressive instability of conventional soft tissue–borne prosthetic devices, neurosensory changes secondary to atrophy, and an overall reduction in size and form in all three dimensions. These changes result in an overall decrease in fit and an increase in patient discomfort with the use of conventional dentures. The prolonged effects of edentulism compounded with systemic factors and functional physical demands from prosthetic loading produce atrophy that, in severe cases, places the patient at significant risk for pathologic fracture. As a result of these effects, a

goal-oriented approach to treatment is the most appropriate. The overall objectives include[6]

1. Eliminating preexistent or recurrent pathology.
2. Rehabilitating infected or inflamed tissue.
3. Reestablishing maxillomandibular relationships in all spatial dimensions.
4. Preserving or restoring alveolar ridge dimensions (height, width, shape, and consistency) conducive to prosthetic restoration.
5. Achieving keratinized tissue coverage over all load-bearing areas.
6. Relieving bony and soft tissue undercuts.
7. Establishing proper vestibular depth and repositioning of attachments to allow for prosthetic flange extension if necessary.
8. Establishing proper notching of the posterior maxilla and palatal vault proportions.
9. Preventing or managing pathologic fracture of the atrophic mandible.
10. Preparing the alveolar ridge by onlay grafting, corticocancellous augmentation, sinus lift, or distraction osteogenesis for subsequent implant placement.
11. Satisfying facial aesthetics, speech requirements, and masticatory challenges.

To satisfy these goals, a treatment plan directly addressing each existing condition is indicated. Such a plan should include correction of maxillomandibular relationship; restoration of ridge form and soft tissue relationship including histologic type and condition, bone and/or soft tissue grafting/repositioning; options regarding implant-supported or -stabilized prosthetic treatment; immediate versus delayed implant placement; preservation of existing alveolar bone with implants; and correction or minimization of the effects of combination syndrome in cases involving partially edentulous patients.

Before developing a plan, one must consider the amount and source of bone loss. Common causes of primary bone loss include trauma; pathology such as periodontal disease, destructive cysts or tumors; and bone loss associated with extraction and alveoloplasty. Secondary bone loss, if not prevented, can follow all of the primary types listed previously. Secondary maxillary/mandibular bone loss is an insidious regressive remodeling of alveolar and even basal bone that is a sequela of tooth loss. This secondary process is referred to as *edentulous bone loss* and varies in degree based on a number of factors. The pathophysiology of edentulous bone loss relates to an individual's characteristic anatomy, metabolic state, jaw function, and prior use of and type of prosthesis. Anatomically, individuals with long dolichocephalic faces typically have greater vertical ridge dimensions than do those with short brachycephalic faces. In addition, those with shorter faces are capable of a higher bite force. Metabolic disorders can have a significant impact on a patient's potential to benefit from osseous reconstructive surgery. Nutritional or endocrine disorders and any associated osteopenia, osteoporosis, and especially osteomalacia must be addressed before beginning bone reconstruction.[5] Mechanical influences on the maxilla and mandible have a variable effect on the preservation of bone. The normal nonregressive remodeling of bone essentially represents a balance between breakdown and repair that maintains bone osteons, the functional unit of bone, and consequently, the viability of bone shape and form. Bone requires stimulation often referred to as "the minimum essential strain" to maintain itself. Both insufficient strain and excessive loads can lead to regressive remodeling of bone, with the classic example being denture compression leading to an anteroposterior and transverse deficient maxilla opposing a wide mandible that is excessive in its anteroposterior dimension.

Residual ridge form has been described and classified by Cawood and Howell[7] (Figures 6-2 and 6-3) as follows:

- Class I—dentate
- Class II—postextraction
- Class III—convex ridge form, with adequate height and width of alveolar process
- Class IV—knife-edge form with adequate height but inadequate width of alveolar process
- Class V—flat-ridge form with loss of alveolar process
- Class VI—loss of basal bone that may be extensive but follows no predictable pattern

Modifications to this classification that may be relevant to contemporary reconstructive methods include subclassifications in II and VI: class II—no defect, buccal wall defect, or multiwall defect or deficiency; and class VI—marginal resection defect or continuity defect.

MEDICAL CONSIDERATIONS

During the patient evaluation process, particular attention to the patient's chief complaint and concerns is imperative; a thorough understanding of the past medical history is mandatory in the treatment and evaluation of any patient. A current or previous history regarding the patient's success or failure at maintaining previous prosthetic devices is also necessary. Careful attention to patient's functional, cognitive, and physical ability to participate with the reconstructive plan is crucial to the success of future restorations and overall patient satisfaction. The evaluation process should include a comprehensive workup of the patient's predilection for metabolic disease, including serum calcium, phosphate, albumin, alkaline phosphatase, and calcitonin levels.[5] Decreased renal function and the presence of a vitamin D deficiency should also be ruled out. The maintenance of bone mass requires a balanced calcium metabolism, a functional endocrine system, and physiologic loading of bone tissue. Secondary medical complications affecting edentulous patients include candidiasis, hyperkeratosis, fibrous inflammatory hyperplasia, dysplasia, papillomatosis, breathing changes, and diet compromise away from natural foods high in fiber and toward an increase in processed foods.

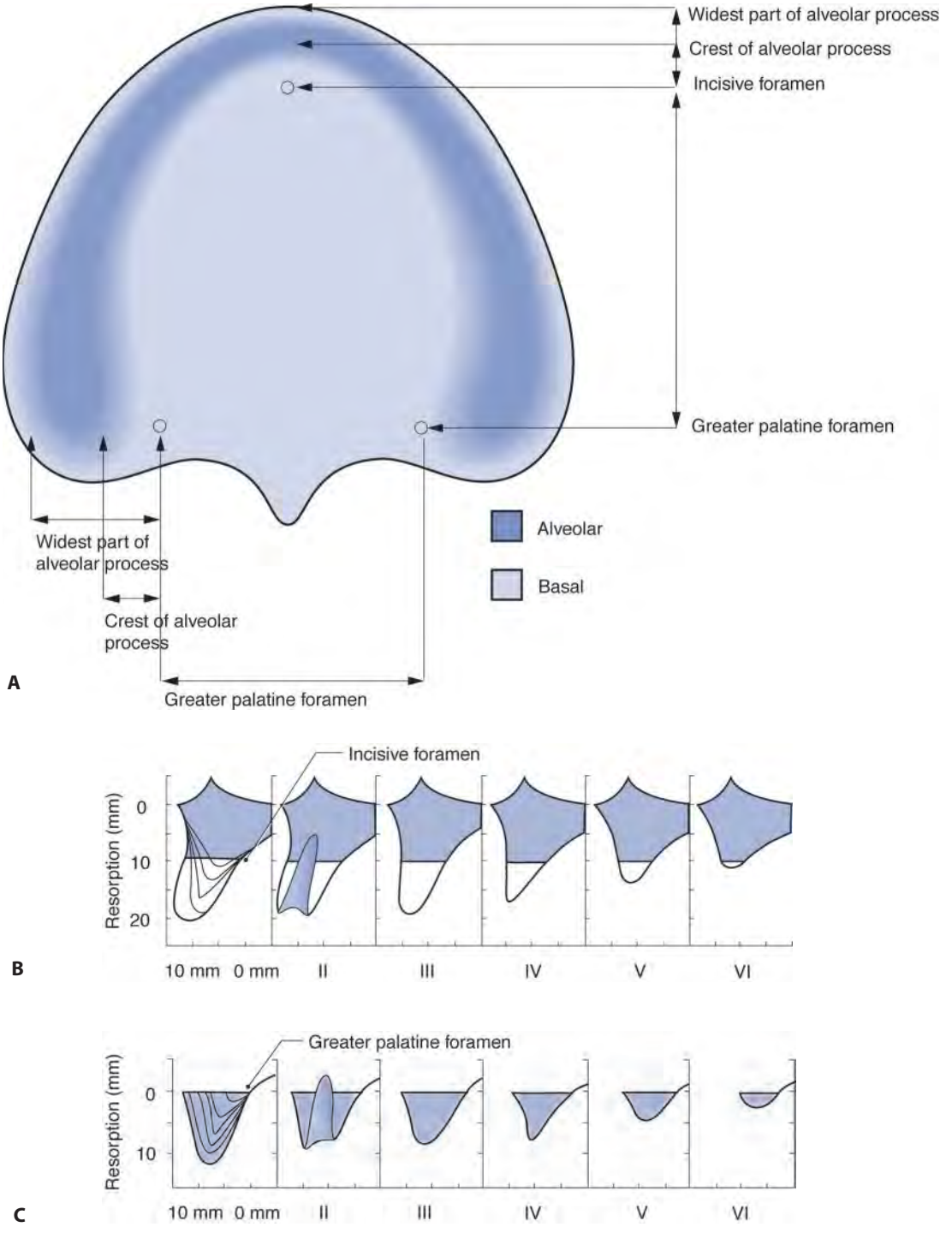


FIGURE 6-2. **A,** Maxillary horizontal measurements. Classification of resorption of maxillary alveolar ridge: anterior **(B)** and posterior **(C).** (**A–C,**Adapted from Cawood JI, Howell RA. A classification of the edentulous jaws. Int J Oral Maxillofac Surg 1988;17:232–236.)

Hard and Soft Tissue Examination

A problem-oriented physical examination should include evaluation of the maxillomandibular relationship; existing alveolar contour, height, and width; soft tissue attachments; pathology; tissue health; palatal vault dimension; hamular notching; and vestibular depth. Identification of both soft tissue and underlying bone characteristics and/or deficiencies is essential to formulate a successful reconstructive plan. This plan should be defined and presented to the patient both to educate the patient and to allow him or her to play a role in the overall decision-making process with all members of the dental team.

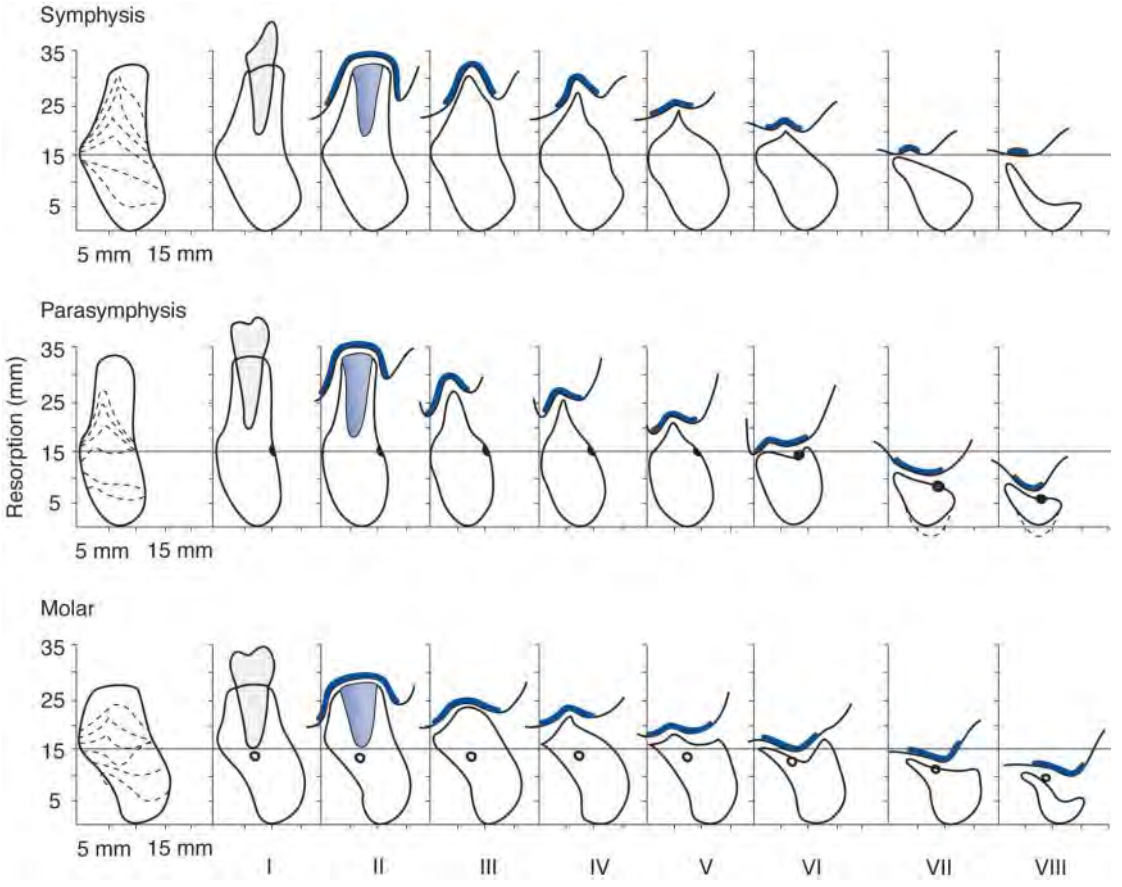


FIGURE 6-3. Modified Cawood and Howell classification of resorption. The *thicker line* illustrates the amount of attached mucosa, which decreases with progressive resorption. (Adapted from Cawood JI, Howell RA. A classification of the edentulous jaws. Int J Oral Maxillofac Surg 1988;17:232–236.)

The soft tissue evaluation should involve careful visualization, palpation, and functional examination of the overlying soft tissue and associated muscle attachments (Figure 6-4). Retraction of the upper and lower lips help one identify muscle and frenum attachments buccally. A mouth mirror can be used lingually to tent the floor of mouth to evaluate the mylohyoid–alveolar ridge relationship. Careful palpation with manipulation of both upper and lower alveolar ridges is the

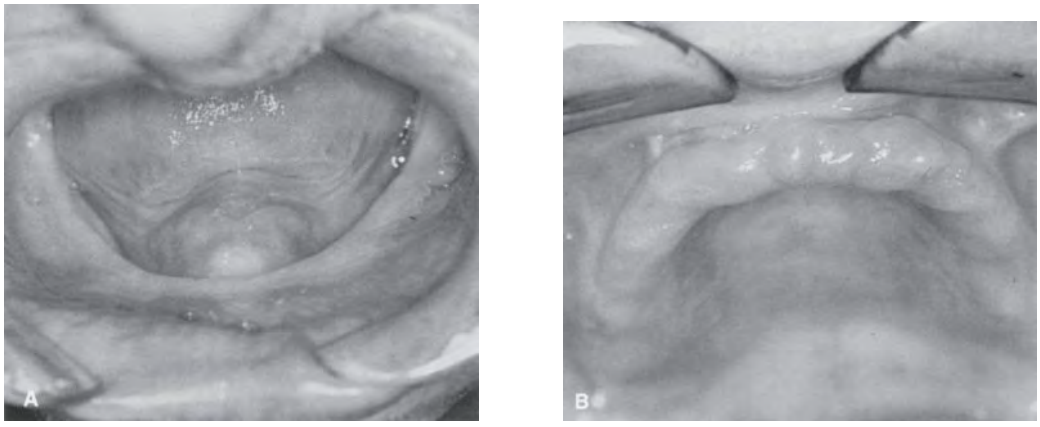


FIGURE 6-4. **A,** Example of mandible with muscular attachments at or near the crest of the ridge. Also note the absence of fixed keratinized tissue over the alveolar ridge area. **B,** Example of maxilla with inadequate vestibular depth anteriorly, frenal attachments near the crest of the alveolar ridge, and flabby soft tissue over the alveolar ridge crest. (**A** and **B,** Reproduced with permission from Tucker MR. Ambulatory preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; p. 1107.)

best diagnostic determinant of loose and excessive soft tissue. One must be aware of occult bony abnormalities obscured by soft tissue excess, especially in cases in which adequate alveolar ridge height and width are imperative for implant placement (Figure 6-5). Such abnormalities can lead to embarrassing and unexpected changes in the restorative plan at the time of mucoperiosteal reflection of the overlying soft tissue. If conventional prosthetic restorations are planned, attention to bony and soft tissue undercuts that oppose the prosthetic path of insertion must be addressed. Critical attention should be given to deficiencies in the palatal vault or buccal/lingual vestibule, defects in the alveolar ridge, and the presence of buccal, palatal, or lingual exostoses. During this evaluation process, final decisions should be made regarding the prognosis of any existing teeth and their role in the overall rehabilitation and contribution to the long-term success of the treatment plan. Finally, careful neurosensory evaluation of the patient with severe regressive remodeling may play a significant role in the determination of future grafting or repositioning procedures aimed at maintaining proper neurosensory function in conjunction with prosthetic rehabilitation.

Radiographic Evaluation

To date, the panoramic radiograph provides the best screening source for the overall evaluation and survey of bony structures and pathology in the maxillofacial skeleton. From examination radiographs, one can identify and evaluate pathology, estimate anatomic variations and pneumatization of the maxillary sinus, locate impacted teeth or retained root tips, and gain an overall appreciation of the contour, location, and height of the basal bone, alveolar ridge, and associated inferior alveolar neurovascular canal and mental foramina.[8]

Calibration of radiographs for magnification is necessary to determine the spatial dimensions needed to plan implant restorations adjacent to neurovascular structures or the maxillary sinus, to determine defect size and shape in distraction osteogenesis, and to predict the necessary dimensions of planned augmentation materials.

Posteroanterior and lateral cephalometric radiographs can be used to evaluate interarch space, relative and absolute skeletal excesses or deficiencies existing in the maxilla or mandible, and the orientation of the alveolar ridge between arches. These are exceptionally useful when the presence of skeletal discrepancies may necessitate orthognathic correction to provide acceptable functional relationships for prosthetic rehabilitation. Cephalometric analysis in combination with mounted dental models helps one establish the planned path of insertion of future prosthetic devices as well as identify discrepancies in interarch relationships that affect the restorative plan (Figure 6-6).[9]

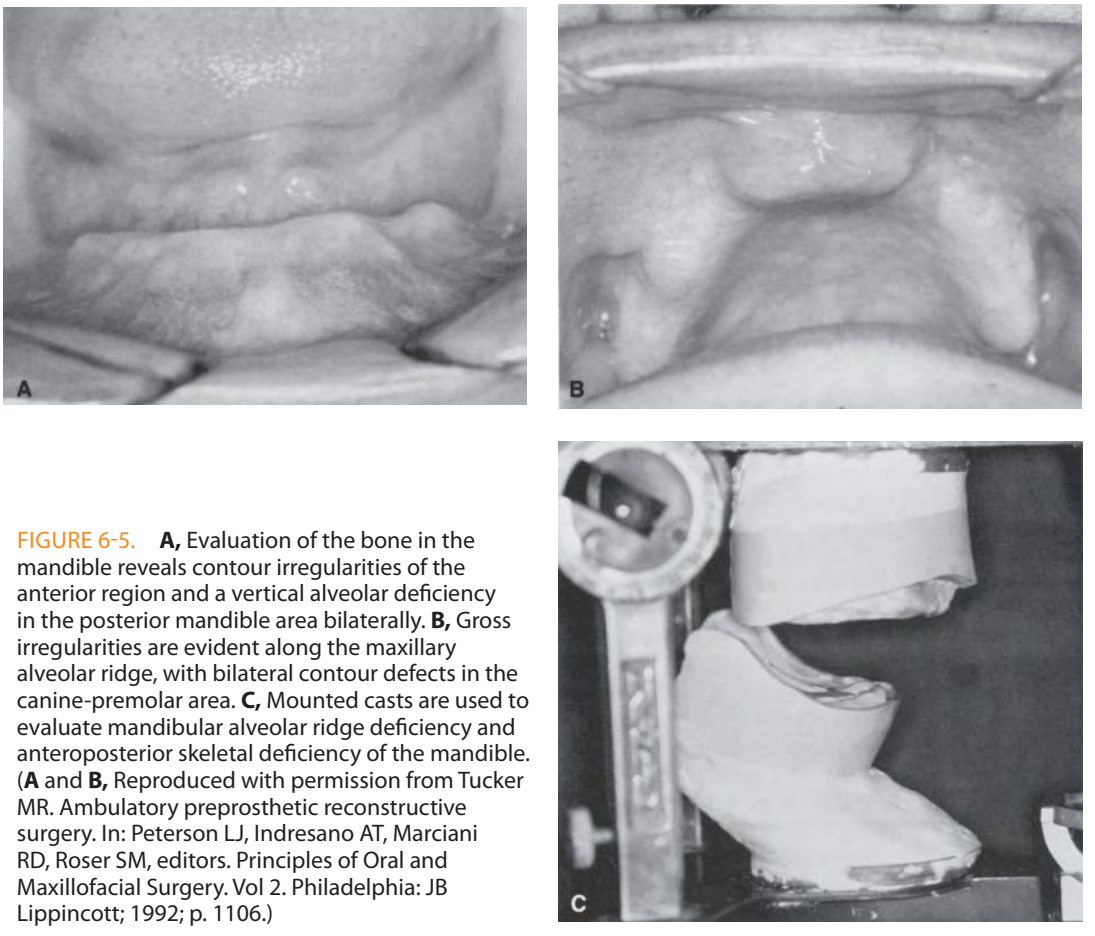


FIGURE 6-5. **A,** Evaluation of the bone in the mandible reveals contour irregularities of the anterior region and a vertical alveolar deficiency in the posterior mandible area bilaterally. **B,** Gross irregularities are evident along the maxillary alveolar ridge, with bilateral contour defects in the canine-premolar area. **C,** Mounted casts are used to evaluate mandibular alveolar ridge deficiency and anteroposterior skeletal deficiency of the mandible. (**A** and **B,** Reproduced with permission from Tucker MR. Ambulatory preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; p. 1106.)

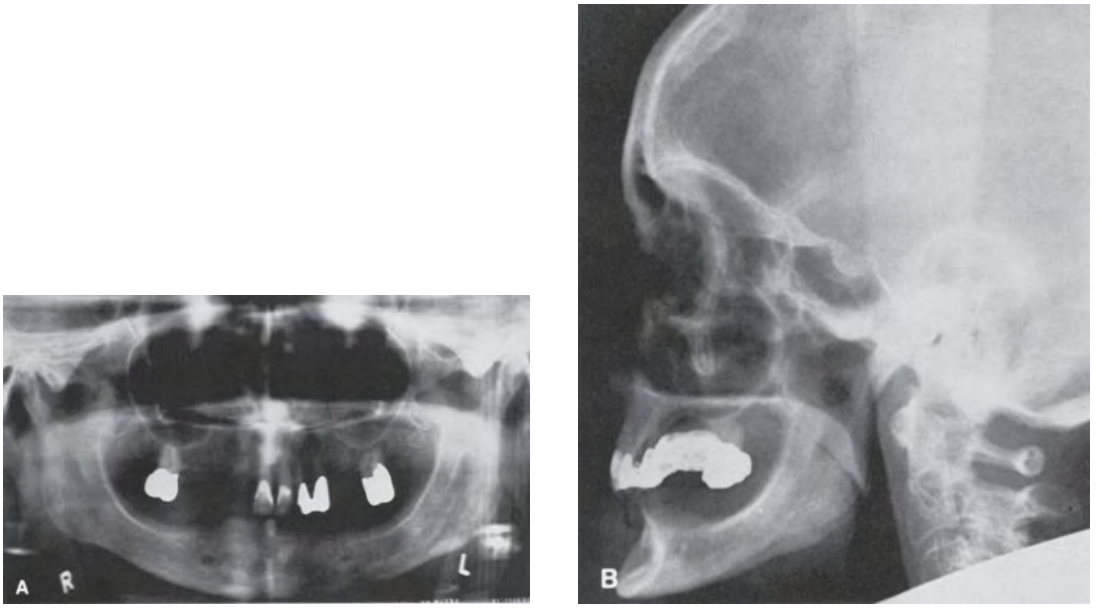


FIGURE 6-6. **A,** Panoramic radiograph shows an apparently adequate alveolar ridge height. **B,** Lateral cephalometric radiograph of the same patient shows a concavity in the anterior area of the alveolar ridge, which produces a knife-edge ridge crest. This type of alveolar ridge deformity cannot be fully appreciated except on a cephalometric radiograph. (**A** and **B,** Reproduced with permission from Tucker MR. Ambulatory preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; p. 1107.)

In recent years, computed tomography (CT) has played an increased role in the treatment planning of complex cases. Detailed evaluation of alveolar contour, neurovascular position, and sinus anatomy is available for the subsequent planning of advanced implant applications. Zygomatic implants that obviate the need for sinus lifting can be used in cases involving edentulous atrophic maxillary sinuses (Figure 6-7). Careful evaluation of the path of insertion is easily accomplished using coronal CT examination of the maxillary sinuses. CT can also provide the clinician with information regarding bone quantity and volume as well as density (Figure 6-8).

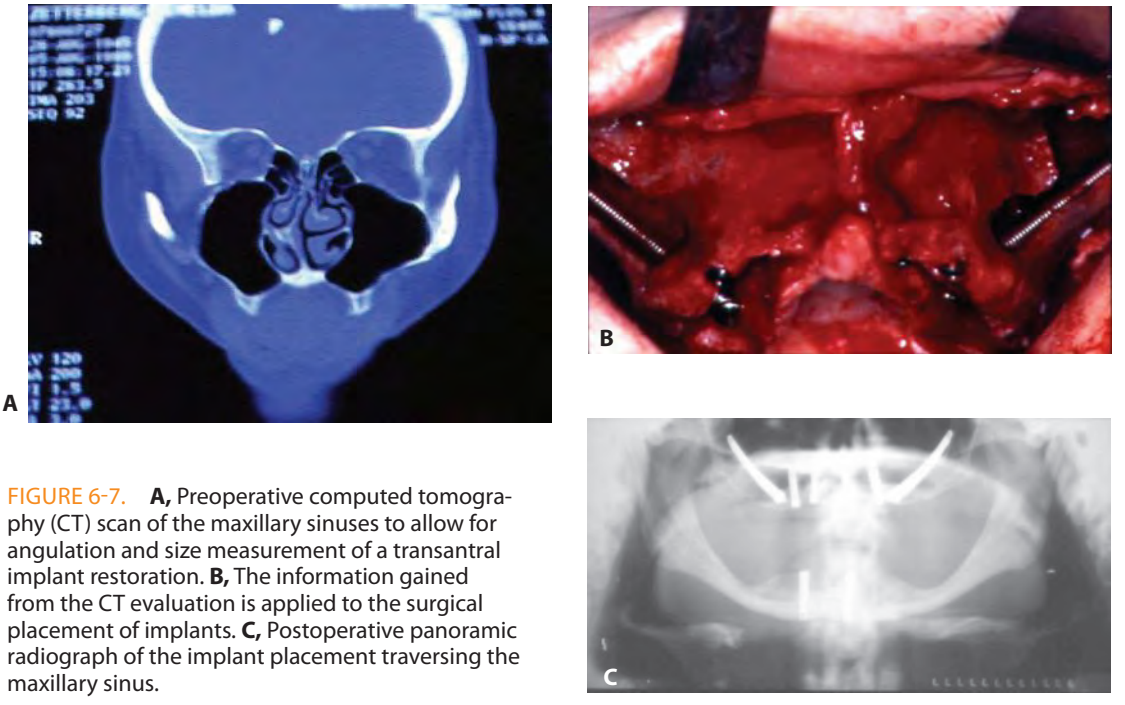


FIGURE 6-7. **A,** Preoperative computed tomography (CT) scan of the maxillary sinuses to allow for angulation and size measurement of a transantral implant restoration. **B,** The information gained from the CT evaluation is applied to the surgical placement of implants. **C,** Postoperative panoramic radiograph of the implant placement traversing the maxillary sinus.

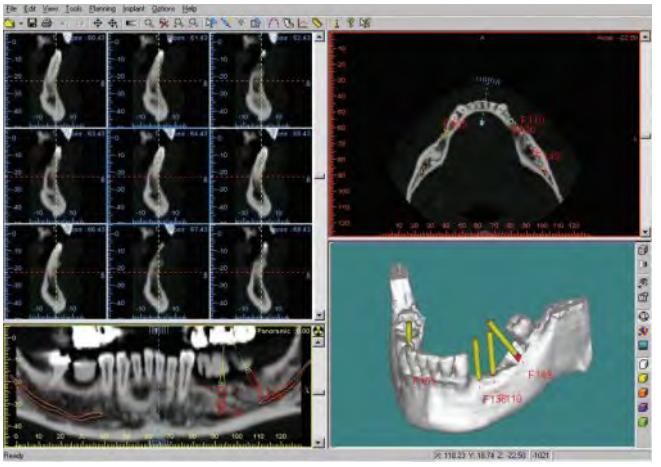


FIGURE 6-8. CT-based imaging used to evaluate bone density, quality, contour, and volume. This information, which has cross-sectional tomographic and three-dimensional components, can be used to plan treatments for complex cases of implant placement. (Courtesy of SimPlant Technologies.)

In many cases, the combination of imaging modalities and mounted models with diagnostic wax-ups can be helpful in determining the reconstructive plan. These elements are also useful in the fabrication of surgical stents guiding implant placement or grafting procedures. Surgical stents fabricated from CT-based models combine aesthetic and surgical considerations; bridge the gap between the model surgery and the operation; and allow cooperation between the surgeon, the laboratory technician, the periodontist, the prosthodontist, and the orthodontist, which results in a cost-effective prosthetic reconstruction with improved aesthetic results (Figure 6-9). In addition, accuracy of the surgical procedure can be greatly increased with an overall decrease in the duration of the procedure.

TREATMENT PLANNING CONSIDERATIONS

The conventional tissue-borne prosthesis has given way to implant-borne devices that have proved superior in providing increased patient function, confidence, and aesthetics. Preprosthetic surgical preparation of areas directly involved with device support and stability are of primary importance and should be addressed early in the treatment plan.

Overlying soft tissue procedures need not be attempted until satisfactory positioning of underlying bony tissues is complete. As a general rule, one should always maintain excessive soft tissue coverage where available until the final bony augmentation is complete. Complications such as dehiscence, loss of keratinized mucosa, and obliteration of vestibular depth can be avoided if respect is given to overlying soft tissue. Once bony healing is complete, if the overlying tissue is clearly excessive, removal of the excess soft tissue can proceed without complication. Using the classification of edentulous jaws according to Cawood and Howell,[7]

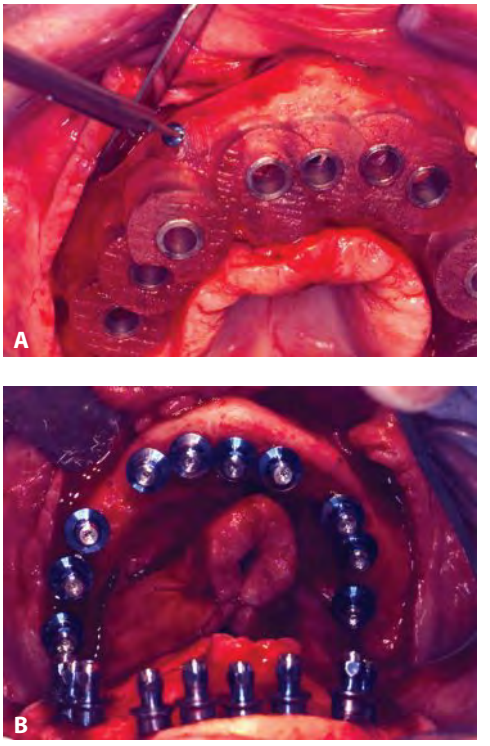


FIGURE 6-9. **A,** Computer-generated surgical stent for implant placement. **B,** Clinical photograph of implants placed with the use of a computer-generated surgical stent.

the reconstructive surgeon can plan treatment for her or his patients accordingly.

Many excellent reconstructive plans achieve less than satisfactory results because of inadequate anesthetic management of the patient during the procedure. Although many procedures can be accomplished under local anesthesia or sedation, the clinician must have a low threshold to provide general anesthesia in a controlled operative setting to allow for appropriate manipulation of the surgical site to achieve the necessary goals of the surgical procedure. Patient desires, health issues, surgeon comfort, and the magnitude of the deformity should all be considered when making decisions regarding anesthetic type.

The loss of maxillary and mandibular bone can have mild to severe effects on an individual's well-being. Interestingly, the size of the defect does not always correlate with the level of debilitation perceived by the patient. Individuals missing a single anterior tooth with an associated buccal wall defect can feel quite compromised, whereas, although it is rare, we have encountered totally edentulous patients who live and function without even a removable denture. This variability underscores the need for the dental team to understand the patient's chief complaint and desired restorative goals. After obtaining a medical dental history and diagnostic database, time spent educating the patient about his or her problem may help the patient refine goals and make it easier to develop a satisfactory treatment plan. Because acceptable prosthetic reconstruction can be achieved with a variety of

treatments that vary in complexity, invasiveness, time to completion, simplicity of maintenance, functional attributes, aesthetic attributes, and cost, it is reasonable to develop more than one treatment plan that can address the patient's needs.

The patient's overall health status, compliance potential, patience, and ability to maintain the final prosthesis/prostheses must be considered when planning reconstructive preprosthetic surgical procedures as well as future prosthetic rehabilitation. Moreover, a multidisciplinary approach involving the patient's input is imperative for long-term success and patient satisfaction.[3]

PRINCIPLES OF BONE REGENERATION

Many approaches are available for reconstructing a deficiency or defective osseous anatomy of the alveolar portions of the facial skeleton. These include biologically viable autogenous bone grafts, nonviable homologous allogeneic or heterogeneic bone implants, recombinant human bone morphogenetic protein-2 (rhBMP-2), and tissue regeneration by distraction histogenesis. These techniques can be used alone or in combination and often are enhanced by the application of adjunct procedures such as rigid fixation and guided bone regeneration. The choice of a reconstructive technique is influenced by many variables, including the location, ridge relationships, dimensions of the defect, dimensions of underlying bone stock, soft tissue availability and viability, and aesthetic goals.

Beyond choosing a reconstructive technique, one must also consider inherent properties of facial bone and its natural growth and remodeling characteristics. For bone to grow or regenerate in direct pressure areas, it must go through an endochondral replacement process such as that in active long bones. Areas of the skeleton that are under pressure must be covered by cartilage—a tissue adapted to this function because it grows interstitially; is minimally calcified, avascular, turgid, and nourished by diffusion; and does not require a membrane for nutrition. In contrast, bone cannot withstand significant pressure because of compression closure of the vascular bed in the periosteum. Because bone matrix is calcified, it must be vascularized to grow, regenerate, or be sustained. In addition, calcification of the matrix precludes interstitial growth, so bone can grow only by the appositional activity of its membranes. Periosteum has a dense connective tissue component and is structurally adapted to transfer tensile forces that are generated by muscles, tendons, and ligaments to bone.

The majority of the facial skeleton is not under load during development; thus, it does not require an endochondral phase, so it develops by an intramembranous process. In the natural state, alveolar bone is protected from load by the dentition and is actually stimulated by strain forces transferred to the alveolus via the periodontal ligament. Although technology to date has not been able to exactly replicate this interface, osseointegrated implants have a similar protective effect on underlying bone, native or reconstructed, and thus should be a component of all alveolar bone reconstructive plans.

Another aspect of facial bone growth and development relevant to reconstruction that needs to be clearly understood is the regional differences in periosteum activity that exist in association with facial bones. It is a misconception that the cortices of growing facial bones are produced only by periosteum. In fact, at least half of the facial bone tissue is formed by endosteum, the inner membrane lining the medullary cavity. Of great significance to the placement of alveolar ridge or alveolar defect bone grafts are the findings that about half of the periosteal surfaces of facial bones are resorptive in nature and half are depository. These properties exist because facial growth is a complex balance between deposition and resorption that adds to the size and shape of a bone while it is being displaced to achieve its final position and relationships to the bones of the facial cranial skeleton. One can study the works and diagrams of Enlow and coworkers[10] to gain a better understanding of these concepts and the regional variations of naturally resorptive and depository surfaces of the facial skeleton. This understanding should help one better determine the most efficacious location for graft placement. For example, the anterior surface of the maxillary and mandibular alveolar ridges are resorptive and, thus, are best treated by the placement of interpositional grafts in association with the endosteal aspects of these bones, as seen in Figure 6-10. Interestingly, the periosteal lining of the maxillary sinus is also mostly resorptive. Successful bone grafting via the sinus lift technique has been demonstrated by numerous authors using a variety of graft techniques. It has been our experience that sinus lift grafts of autogenous cancellous bone, and bone induced to grow by rhBMP-2, secondarily treated with osseointegrated implants remodel over time. A follow-up of greater than 10 years of some of our patients has shown that the grafts become scalloped over the surfaces of the implants, similar to the relationship seen when natural roots extend above the floor of a pneumatized sinus. This finding suggests that the capacity for remodeling by the periosteal membrane exists even after the face is mature and that viable bone established by autogenous grafts or rhBMP-2-mediated induction responds to this process.[11]

Another concept of facial growth that bears relevance to contemporary methods of reconstruction is the functional matrix concept that has largely been described by Moss.[12] This concept states that bone itself does not regulate the rate of bone growth. Instead, the functional soft tissue matrix related to bone actually directs and determines the skeletal growth process. The vector and extent of bone growth are secondarily dependent on the growth of associated soft tissue. Bone, by virtue of its matrix maturity, gives feedback to this process by either inhibiting it or allowing it to accelerate. Thus, the volume of bone generated is based on genetic properties of the soft tissue and a mechanical equilibration between bone and its soft tissue matrix. These principles are visited when distraction forces are applied to osteotomized bone.

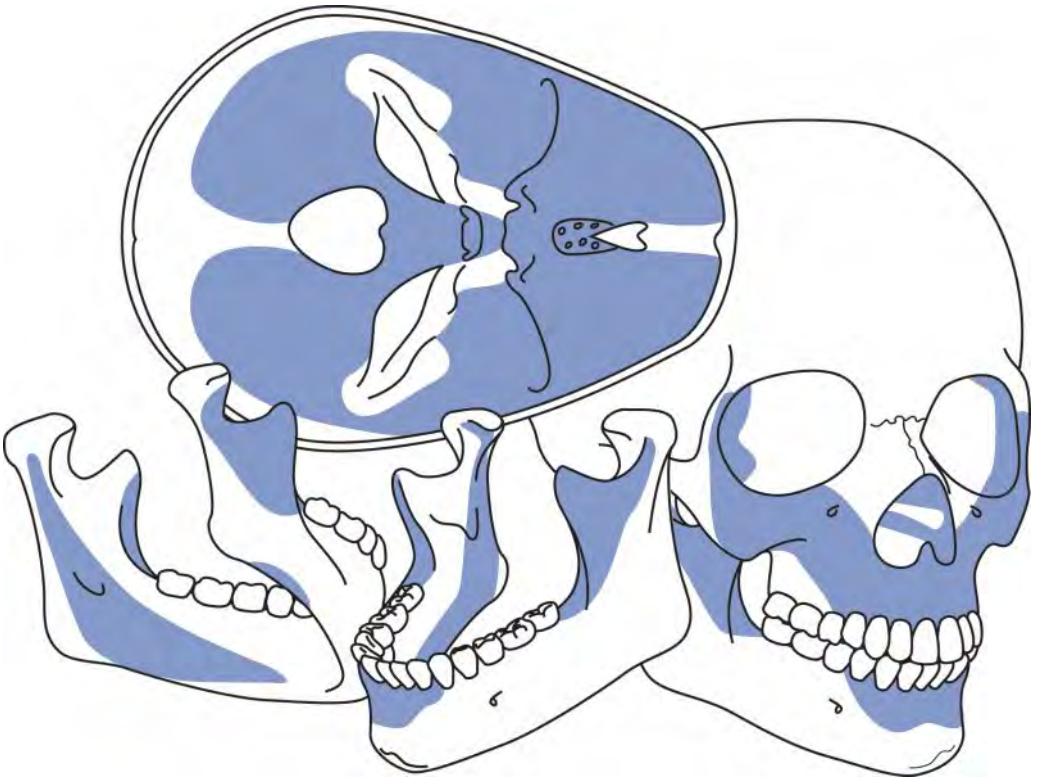


FIGURE 6-10. Growth and remodeling field of the craniofacial skeleton. Resorptive fields are *shaded* and depository fields are *free of shading.*

In 1989, Ilizarov[13,14] forwarded the theory of tension-stress applied to bone as a mechanism of lengthening bone. He stated that controlled, mechanically applied tension-stress allows bone and soft tissue to regenerate in a controlled, reliable, and reproducible manner. During the latency phase of distraction, there is a periosteal and medullary revascularization and recovery. Simultaneously, a relatively hypovascular fibrous interzone develops that is rich in osteoprogenitor cells and serves as a pseudo–growth plate. Adjacent and connected to the interzone are areas of hypervascular trabeculae aligned in the direction of the distraction. Osteoprogenitor cells in the interzone differentiate into osteoblasts and line the trabeculae. As distraction progresses, appositional bone growth enlarges the trabeculae. This underscores the idea that mechanical stress applied to the soft tissue matrix of osteotomized bone can reactivate these native growth processes.

It is interesting to note that if the distraction device lacks sufficient mechanical stability or if the rate of distraction progresses too rapidly, the tissue established may mature very slowly or not at all. Conversely, if distraction progresses too slowly, the regenerate may mature prematurely or there may be increased pain during the procedure. We have found that if there is recurrent pain associated with the activation of a distractor, a slight increase in the rate of distraction usually reduces the pain. In many ways, distraction histogenesis recapitulates the process of native bone growth directed by the influence of the soft tissue matrix. Premature maturation of the matrix increases resistance to distraction necessitating increased distraction force and the perception of pain by the patient. This suggests that even the feedback role of the bone matrix is active during this process. In most cases, the net result of distraction histogenesis is the formation of a bone ossicle that is vascular and rich in osteolysis, has a shape similar to the native bone, and has an appropriate soft tissue envelope. Often, distraction histogenesis alone is sufficient to regenerate deficient alveolar ridge anatomy. In other cases, distraction can be used in association with bone grafting, especially when the associated bone stock is of less than ideal shape or volume. In some cases, particularly in the posterior mandible, the distraction osteotomy can be extended beyond the area of intended implants so that the distraction process actually grows the bone needed for the graft. Bone grafts placed adjacent to regenerate typically mature very rapidly owing to the vascularity, cellularity, and high concentration of natural bone morphogenetic protein (BMP) in regenerate.

BONE GRAFTS

Bone graft principles are discussed in several chapters throughout the book. Nonetheless, some of the characteristics of grafts and bone implants pertinent to preprosthetic surgery are examined here. By far the most common graft type is the free autogenous viable bone graft. Because these

grafts are from the patient, they do not elicit an immune-rejection response. Common areas for procurement include the maxilla, mandible, cranium, tibial plateau, iliac crest, and rib. The shape, form, and volume of the graft procured are linked to the defect to be reconstructed. These grafts are used as corticocancellous blocks or particulate cancellous grafts compacted and shaped by various membranes or trays. In many instances, purely cancellous blocks or cancellous particulate bone is used again with membranes or trays or sandwiched in unloaded osteotomies or defects. A third form includes purely cortical grafts, primarily used to form a wall or strut in association with a defect that is simultaneously packed with particulate cancellous bone. Cortical grafts revascularize very slowly and have minimal to no cell survival; thus, they are not ideal for implant placement.[15]

Cancellous grafts have the greatest concentration of adult mesenchymal stem cells that can be induced to form osteoblasts. The particulate form of these grafts has the greatest percentage cell survival owing to better diffusion and rapid revascularization. These grafts must completely undergo a two-phase mechanism of graft healing.[16] Mesenchymal stem cells that survive transplantation proliferate and differentiate into osteoblasts to form osteoid. This process is active in the first 2 to 4 weeks, and the definitive amount of bone formed is related to the quantity of osteoid formed in phase one. Phase two starts around the second week after grafting, and although it peaks in intensity at approximately 4 to 6 weeks, it continues until the graft matures. The initiation of phase two is marked by osteoclastic cell activity within the graft. Osteoclasts remove mineral, forming Howship's lacunae along the trabeculae. This resorptive process exposes the extracellular matrix of bone, which is the natural location of the bone-inductive glycoprotein BMP. Exposure of BMP initiates an inductive process characterized by chemotaxis of mesenchymal stem cells, proliferation of cells in response to mitogenic signals, and differentiation of cells into osteoblasts.[17] Inducible cell populations may be local or distant from the graft site. Examples of local cell populations that may contribute to the graft include osteoprogenitor cells in the graft endosteum, stem cells of the transplanted marrow, or cells in the cambium layer of adjacent periosteum. Additional inducible pluripotent cells may arrive at the graft site with budding blood vessels. During phase two, there is progressive osteoclastic resorption of phase one osteoid and nonviable graft trabeculae; this continues to expose BMP, which perpetuates the differentiation of osteoblasts, leading to the formation of mature vascular osteocyte-rich bone.

This two-phase bone graft healing process is the one that most reliably and quickly can regenerate bone with characteristics suitable for implant placement. When choosing a bone graft, one must consider its ultimate purpose; because most grafts associated with preprosthetic surgery are designed to support implants, these grafts must provide the biologic environment necessary for osseointegration. Osseointegration is a biologic process, and its long-term success requires vascular osteocyte-rich bone.

Another adjunct to preprosthetic bone reconstruction is the use of allogeneic bone. Because these grafts are nonviable, they are technically implants. Allogeneic bone is procured in a fresh sterile manner from cadavers of genetically unrelated individuals. American Association of Tissue Bank standards require that all donors be screened, serological tests be performed, and all specimens be sterilized and verified by culture before release. Processing of allogeneic bone is designed to achieve sterility and reduce immunogenicity. Bone cell membranes have both class I and II major histocompatibility complexes on their surfaces. These are the main sources of immunogenicity within allogeneic bone grafts. Allogeneic bone implants are processed to remove the organic matrix and retain only the mineral components; architecture is generally considered to be nonimmunogenic. Implants retaining both mineral and organic components or demineralized implants with only the organic component are washed and then lyophilized to reduce immunogenicity. In most cases, this process reduces the immune response to clinically insignificant levels. In addition to this treatment, allogeneic bank bone is irradiated with gamma rays, a process that assures sterility and further reduces antigenicity. Unfortunately, this requires 2 to 3 Mrad per radiation dose, which destroys BMP and, thus, the ability of these implants to be osteoinductive.[18]

Common applications of allogeneic bone implants for preprosthetic surgery include mandibles, iliac crest segments, and calcified or decalcified ribs that can be prepared and used as biologic trays for the placement and retention of cancellous bone grafts. Additional uses include mineral matrix or demineralized particulate implants used as osteoconductive graft extenders or for extraction site shape and form preservation. Research on particle size suggests that particles in the range of 250 to 850 μm are the most useful. Although the current carrier system used for rhBMP-2 bone induction is a collagen membrane, Becker and associates[19] showed that BMP extracted from the bone can be added to particulate 200 to 500 μm demineralized freeze-dried bone allografts obtained from four American tissue banks; this resulted in the transformation of noninductive particles to particles with osteoinductive properties. Heterogeneous bone grafts, or xenografts, are specimens transferred from one species to another. Implants of this type contain an organic component that would elicit a strong immune response; thus, they are not used in contemporary practice. Bovine implants that have undergone complete deproteinization to remove the organic component have been shown to be nonimmunogenic. These implants remain as an inorganic mineral scaffold that can be used for their osteoconductive properties as graft extenders or for extraction site preservation.

The previous discussion has identified two reconstructive methods that can reliably restore bone with the characteristics necessary for maintaining osseointegrated implants. These methods include autogenous cancellous bone grafts and distraction histogenesis alone or with graft supplementation. A third approach alluded to above is the use of

rhBMP-2.[20] rhBMP-2 has been studied extensively in animal models, and human clinical trials in the areas of orthopedic surgery, spine surgery, and maxillofacial surgery have been ongoing during the past decade. rhBMP-2/ACS, which is the clinical combination of BMP with an absorbable collagen sponge carrier placed with a metal cage, received U.S. Food and Drug Administration (FDA) approval for spine fusion surgery in 2002. In March 2007, the FDA approved the use of rhBMP-2/ACS for localized alveolar ridge augmentation and two-staged maxillary sinus floor augmentation. Its approval was supported by data collected from feasibility studies, safety and efficacy studies, and dose-response studies involving either alveolar ridge buccal wall defects or posterior maxillary alveolar bone deficiency at sinus lift bone sites. Safety has been established, and a dose of 1.5 mg/mL, the same dose used for spine fusion, was chosen for maxillofacial applications. At this dose, the adequacy of new bone formation in response to rhBMP-2/ACS placed in buccal wall defects has been shown to be approximately twice as great as no treatment or placebo.[21] It has also been shown to induce denser bone formation when compared with 0.75 mg/mL.[22] More recently, Triplett and colleagues[23] completed a pivotal, randomized, 21-center study that demonstrated the effectiveness and safety of rhBMP-2/ACS compared with bone grafts for sinus floor augmentation. In this study, 160 patients with less than 6 mm of native alveolar bone height were randomized into a bone graft or a rhBMP-2/ACS treatment group. In each group, new bone formation was evaluated by CT scan 6 months postoperatively and at 6 months after dental restoration. In addition, core biopsies were performed at the time of implant placement. The mean change in bone height in the rhBMP-2/ACS group was 7.83 ± 3.52 mm compared with 9.46 ± 4.11mm. Significantly denser bone was found in the rhBMP-2/ACS group 6 months after functional loading. The authors concluded that rhBMP-2/ACS is a safe and effective material for maxillary sinus floor augmentation (Figure 6-11).

Similar to natural BMP, rhBMP-2/ACS has been shown to stimulate the cascade of bone-regeneration events, including chemotaxis, induction of pluripotent cells, and proliferation. To date, this technique has been shown to have the potential to significantly enhance patient care by providing an unlimited supply of nonimmunogenic sterile protein that can induce de novo bone formation. Bone regenerated by this process has also been shown to have characteristics of bone desirable for implant placement (Figure 6-12). In addition, the off-label use of rhBMP-2/ACS has also been demonstrated to be an effective alternative in osseous regeneration in areas of reconstruction where autogenous bone grafting has traditionally been utilized, such as alveolar cleft repair and mandibular continuity defects.[24,25]

Hopefully, the discussion of host properties and regenerative or graft techniques in this section will aid one in determining the best graft for sites to be reconstructed as part of a preprosthetic surgical treatment plan.

HARD TISSUE RECONTOURING

Current Trends in Alveolar Preservation

As dental implants continue to grow in popularity and play a major role in prosthetic reconstruction, the need for traditional bony recontouring at the time of extraction has been deemphasized. Current trends tend to lean toward preservation of alveolar bone and overlying periosteal blood supply, which enhances and preserves future bone volume. Alternatives to traditional alveoloplasty have emerged in an effort to maintain bone height and volume for the placement of implants to provide a stable platform for prosthetic reconstruction. Such alternatives include orthodontic guided tooth/root extraction, conservative extraction techniques using periosteotomes to maintain alveolar continuity, immediate grafting of extraction sites, relief of undercuts using bone grafts or hydroxyapatite (HA) augmentation, and guided

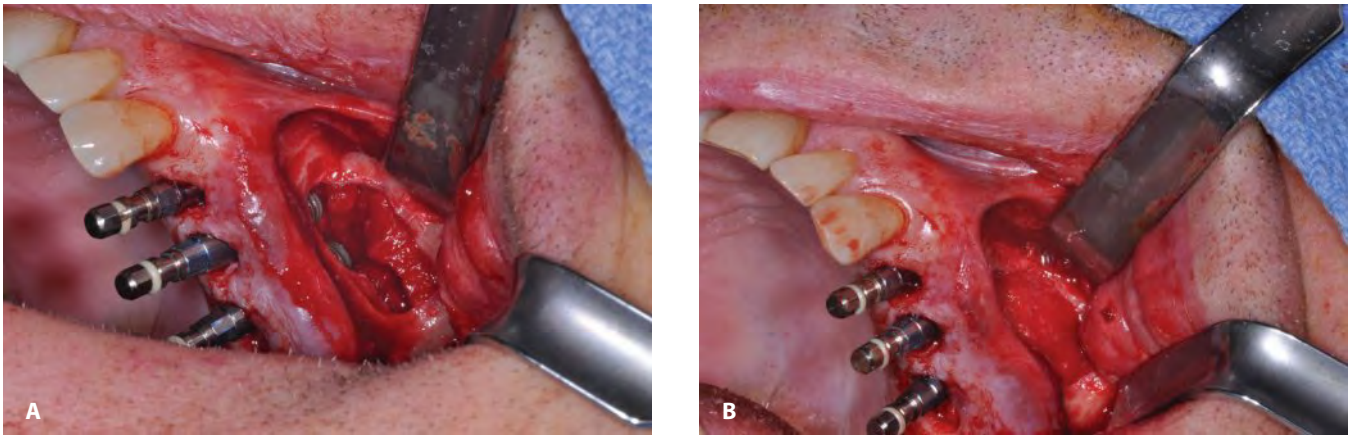


FIGURE 6-11. **A,** Simultaneous maxillary sinus lift and placement of implants into an alveolus measuring 5 mm in height. **B,** Maxillary ridge augmentation utilizing recombinant human bone morphogenetic protein-2 on an absorbable collagen sponge carrier (rhBMP-2/ACS).

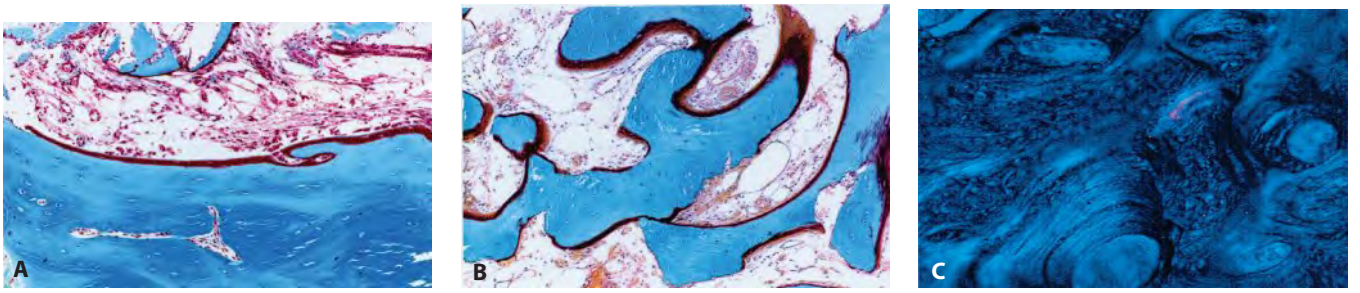


FIGURE 6-12. Stages of bone maturation are evident in these photomicrographs of autogenous bone grafts, autogenous grafts with bone morphogenetic protein (BMP), and distraction-regenerate. **A,** Autogenous tibial plateau with no filler was placed in this sinus lift site with less than 5 mm of native bone, procured by trephine, and sampled at 6 months after the graft. Viable osteocyte-rich bone trabeculae are evident with normal marrow spaces with a few residual foci of nonviable graft (original magnification ×100; hematoxylin and eosin stain). **B,** BMP was placed in an identical site to that shown in **A** (original magnification ×75, hematoxylin and eosin stain). This specimen reveals viable trabeculae with normal haversian canals, de novo bone growth, and no nonviable components. **C,** Regenerate was procured at the time of the distractor removal at this mandibular distraction site. The regenerated growth represents woven bone with some mature haversian systems (original magnification ×128, hematoxylin and eosin stain).

tissue regeneration. In cases in which bony abnormalities or undercuts require attention, selective alveolar recontouring is indicated.

Advances in implant technology have placed a greater emphasis on planning for alveolar ridge preservation. Beginning at the initial consultation, all extraction sites should be considered for implant reconstruction. Regardless of the reason for extraction (i.e., pulpal disease, periodontal disease, or trauma), every effort should be made to maintain alveolar bone, particularly buccal (labial) and lingual (palatal) walls. However, even with alveolar bone maintenance, there can be unpredictable resorption in a short period of time.[26] Multiple adjacent extractions may also contribute to extensive alveolar bone loss, precluding implant reconstruction.

Historically, techniques for alveolar ridge preservation were developed to facilitate conventional denture prostheses. HA materials were the first materials not plagued by host rejection and fibrous encapsulation. Previously, the use of polymethyl methacrylate, vitreous carbon, and aluminum oxide had led to poor results. Root form and particulate HA both were adapted and successful in preserving alveolar ridge form.[27] The obvious limitation with nonresorbable materials is that they preclude later implant reconstruction. Tricalcium phosphate is a resorbable ceramic that was originally thought would solve this problem, but it proved not to be truly osteoconductive because it promoted giant cell rather than osteoclastic resorption.[28] This resulted in limited osteogenic potential. Another alloplast that has been used for this purpose is bioactive glass, which consists of calcium, phosphorus, silicone, and sodium, but again, the biologic behavior of the replacement bone was never felt to be satisfactory for implant reconstruction.[29]

The gold standard for use for bony reconstruction anywhere has always been autogenous grafts. The dilemma with autogenous grafts involves donor site morbidity: whether from an intraoral or extraoral source, the additional surgery and inconvenience to the patient has precluded its general use. To avoid the use of a donor site, various allogeneic bone preparations have been advocated. Stringent tissue bank regulations have provided the public with greater confidence in the use of these materials. Anorganic bone has most recently been adapted for use in alveolar ridge preparation. Two products are currently available commercially. The first is a xenograft derived from a bovine source. The main advantage of this type of material is that it is available in an almost unlimited supply and is chemically and biologically almost identical to human bone. Minimal immune response is elicited because of the absence of protein; however, the resorption rate of bovine cortical bone is slow. In both animal and human studies, remnants of nonvital cortical bone have been shown to be present 18 months or longer in the grafted site.[30] A second product, derived from human bone, is processed by solvent extraction and dehydration. Animal studies have shown that there is near-complete remodeling with little or no remnant of the human anorganic bone left in the specimen.[31]

Both the deproteinized bovine bone and the solvent-dehydrated mineralized human bone appear to have great potential in alveolar ridge preservation. These materials take a long time to resorb, so a ridge form is maintained over an extended period of time, and are resorbed and remodeled via an osteoclastic process that results in bone ideally suited for implant placement.

The technique for alveolar ridge preservation at the time of extraction has been described by Sclar.[32] Atraumatic extraction is essential. Preservation of buccal or labial bone may be facilitated by the use of microosteotomes, and whenever possible, buccal or labial mucoperiosteal elevation is to be avoided or limited. The socket should be gently curetted and irrigated, and in the presence of periodontal infection, topical antibiotics may be helpful. Tetracycline powder mixed with the deproteinized bovine bone or the solvent-dehydrated mineralized human bone may allow for the use of

either of these types of bone in almost any clinical situation. It is not essential that the graft have complete watertight mucosal coverage. Collagen membrane is used to prevent spillage of the material from the socket, particularly in maxillary extractions. When temporary restorations are employed at the time of surgery, an ovate pontic provisional restoration helps to support the adjacent mucosa during soft tissue maturation. In selected instances, immediate placement of implants in the extraction site can be done in conjunction with the use of these deproteinized bone preparations. Because of the slow resorptive nature of both of these bone preparations, they may be ideally suited for buccal or labial defects that would otherwise be grafted with autogenous cortical bone.

Alveoloplasty

Often, hard and soft tissues of the oral region need to undergo recontouring to provide a healthy and stable environment for future prosthetic restorations. Simple alveolar recontouring after extractions consists of compression and in-fracture of the socket; however, one must avoid overcompression and overreduction of irregularities. Current trends endorse a selective stent-guided approach to site-specific bony recontouring, eliminating bony abnormalities that interfere with prosthetic reconstruction or insertion. Multiple irregularities produce undercuts that are obstructions to the path of insertion for conventional prosthetic appliances. These obstructions need a more complex alveoloplasty to achieve desired results. In many cases, the elevation of mucoperiosteal flaps using a crestal incision with vertical releases is necessary to prevent tears and to produce the best access to the alveolar ridge. During mucoperiosteal flap resection, periosteal and Woodson elevators are the most appropriate tools to prevent excess flap reflection, devitalization, and sequestrum formation. These conditions increase pain and discomfort for the patient and increase the duration needed before prosthetic restoration can proceed. The use of a rongeur or file for advanced recontouring is preferred to rotary instruments to prevent overreduction. For large bony defects, rotary instrument recontouring is preferred. Normal saline irrigation is used to keep bony temperatures below 47°C to maintain bone viability.

Owing to the physiology of bone and current restorative options available, interseptal alveoloplasty is rarely indicated. The main disadvantage of this procedure is the overall decrease in ridge thickness, which results in a ridge that may be too thin to accommodate future implant placement.[9] Removal of interseptal bone eliminates endosteal growth potential, which is necessary for ridge preservation. Therefore, if this technique is to be used, one must be cognizant of ridge thickness and reduce the labial dimension only enough to lessen or eliminate undercuts in areas where implants are not anticipated.

After hard tissue recontouring, excessive soft tissue is removed to relieve mobile tissue that decreases the fit and functional characteristics of the final prosthesis. Closure with a resorbable running or lockstitch suture is preferred because fewer knots are less irritating for the patient.

Treatment of Exostoses

Undercuts and exostoses are more common in the maxilla than in the mandible. In areas requiring bony reduction, local anesthetic should be infiltrated. This produces adequate anesthesia for the patient as well as an aid in hydrodissection of the overlying tissues, which facilitates flap elevation. In the mandible, an inferior alveolar neurovascular block may also be necessary. A crestal incision extending approximately 1.5 cm beyond each end of the area requiring contour should be completed. A full mucoperiosteal flap is reflected to expose all the areas of bony protuberance. Vertical releasing incisions may be necessary if adequate exposure cannot be obtained because trauma of the soft tissue flap may occur. Recontouring of exostoses may require the use of a rotary instrument in large areas or a hand rasp or file in minor areas. Once removal of the bony protuberance is complete and visualization confirms that no irregularities or undercuts exist, suturing may be performed to close the soft tissue incision. If nonresorbable sutures are used, they should be removed in approximately 7 days.

In areas likely to be restored with implants or implant-supported prostheses, irregularities and undercuts are best treated using corticocancellous grafts from an autogenous or alloplastic source. This can be done using a vertical incision only adjacent to the proposed area of grafting. A subperiosteal dissection is used to create a pocket for placement of the graft material. Visual inspection and palpation of the area should be done at the conclusion of the procedure to verify the relief of the defect. The incision can be closed with resorbable sutures. In areas that require a large amount of graft material, scoring of the periosteum can assist in closure of soft tissue defects. In addition, the use of a resorbable collagen membrane can be used to prevent tissue ingrowth into the surgical site.

Tuberosity Reduction

Excesses in the maxillary tuberosity may consist of soft tissue, bone, or both. Sounding, which is performed with a needle, can differentiate between the causes with a local anesthetic needle or by panoramic radiograph. Bony irregularities may be identified, and variations in anatomy as well as the level of the maxillary sinuses can be ascertained. Excesses in the area of the maxillary tuberosity may encroach on the interarch space and decrease the overall freeway space needed for proper prosthetic function. Access to the tuberosity area can be obtained easily using a crestal incision beginning in the area of the posterior tuberosity and progressing forward to the edge of the defect using a no. 12 scalpel blade. Periosteal dissection then ensues exposing the underlying bony anatomy. Excesses in bony anatomy are removed using a side-cutting rongeur. Careful evaluation of the level of the maxillary sinus must be done before bony recontouring is

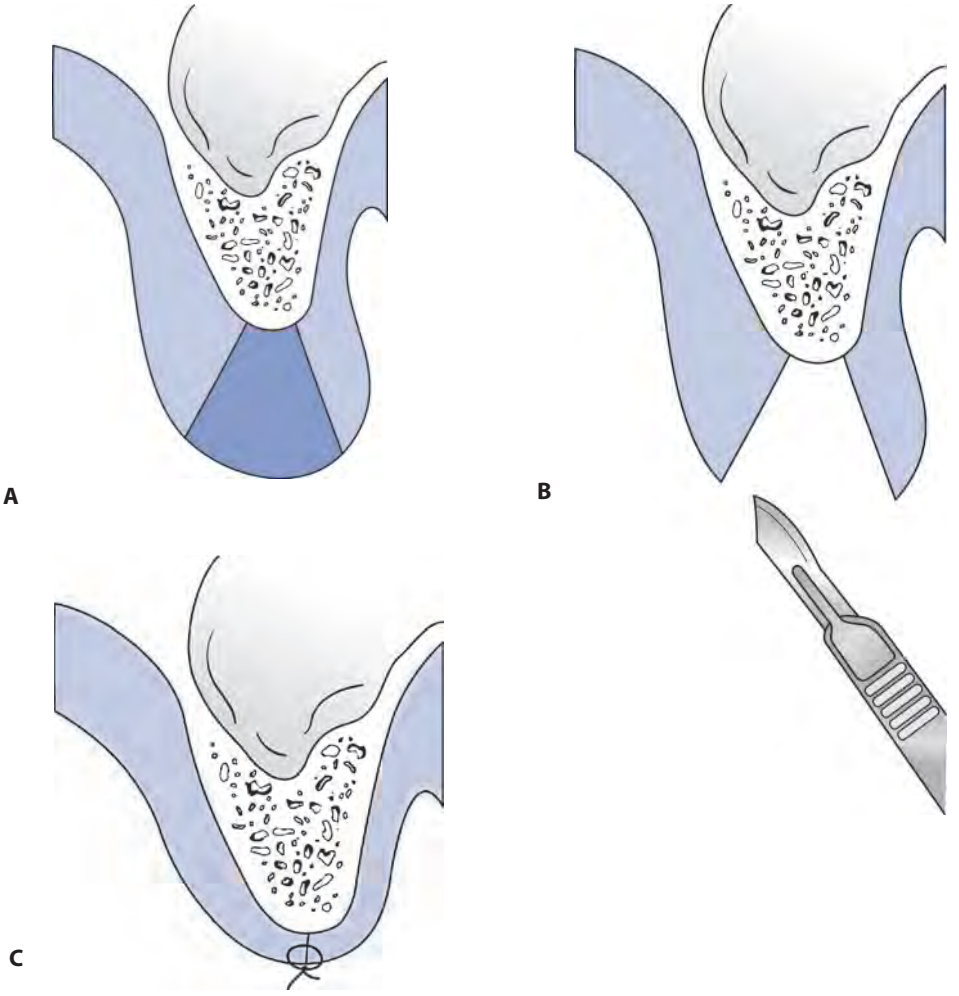


FIGURE 6-13. **A,** Area of soft tissue to be excised in an elliptical fashion over the tuberosity. **B,** Removal of tissue and undermining of buccal and palatal flaps completed. **C,** Final tissue closure.

attempted in the area of the tuberosity. Sharp undermining of the overlying soft tissue may be performed in a wedge-shaped fashion beginning at the edge of a crestal incision to thin the overall soft tissue bulk overlying the bony tuberosity. Excess overlying soft tissue may be trimmed in an elliptical fashion from edges of the crestal incision to allow a tension-free passive closure (Figure 6-13). Closure is performed using a nonresorbable suture in a running fashion. Small sinus perforations require no treatment as long as the membrane remains intact. Large perforations must be treated with a tension-free tight closure as well as antibiotics, preferably a penicillinase-resistant penicillin such as an amoxicillin/clavulanate potassium preparation or a second-generation cephalosporin. The patient is instructed to take sinus medications including antihistamines and decongestants for approximately 10 to 14 days and not to create excessive transmural pressure across the incision site by blowing her or his nose or sucking through straws.

Genial Tubercle Reduction

The genioglossus muscle attaches to the lingual aspect of the anterior mandible. As the edentulous mandible resorbs, this tubercle may become significantly pronounced. In cases in which anterior mandibular augmentation is indicated, leaving this bony projection as a base for subsequent grafting facilitates augmentation of mandibular height. During conventional mandibular denture fabrication, this bony tuberosity as well as its associated muscle attachments may create displacement issues with the overlying prostheses. In these cases, it should be relieved. Floor-of-mouth–lowering procedures should also be considered in cases in which genioglossus and mylohyoid muscle attachments interfere with stability and function of conventional mandibular prostheses.

Bilateral lingual nerve blocks in the floor of the mouth are necessary to achieve adequate anesthesia in this area. A crestal incision from the midbody of the mandible to the midline bilaterally is necessary for proper exposure. A subperiosteal dissection exposes the tubercle and its adjacent muscle attachment. Sharp excision of the muscle from its bony attachment may be performed with electrocautery, with careful attention to hemostasis. A subsequent hematoma in the floor of the mouth may lead to airway embarrassment and life-threatening consequences if left unchecked. Once the muscle is detached, the bony tubercle may then be relieved using rotary instrumentation or a rongeur. Closure is performed using a resorbable suture in a running fashion. The genioglossus muscle is left to reattach independently.

Tori Removal

The etiology of maxillary and mandibular tori is unknown; however, they have an incidence of 40% in males and 20% in females.[33] Tori may appear as a single or multiloculated bony mass in the palate or on the lingual aspect of the anterior mandible either unilaterally or bilaterally. In the dentate patient, they are rarely indicated for removal. Nevertheless, repeated overlying mucosal trauma and interference with normal speech and masticatory patterns may necessitate treatment. In the patient requiring complete or partial conventional prosthetic restoration, they may be a significant obstruction to insertion or interfere with the overall comfort, fit, and function of the planned prosthesis.

In the maxilla, bilateral greater palatine and incisive blocks are performed to achieve adequate anesthesia. Local infiltration of the overlying mucosa helps with hemostasis and hydrodissection that facilitates flap elevation. A linear midline incision with posterior and anterior vertical releases or a U-shaped incision in the palate followed by a subperiosteal dissection is used to expose the defect. Rotary instrumentation with a round acrylic bur may be used for small areas; however, for large tori, the treatment of choice is sectioning with a cross-cut fissure bur. Once sectioned into several pieces, the torus is easily removed with an osteotome. Care must be taken not to overreduce the palate and expose the floor of the nose. Final contouring may be done with an egg-shaped recontouring bur (Figure 6-14). Copious irrigation is necessary throughout the procedure. Closure is performed with a resorbable suture. Presurgical fabrication of a thermoplastic stent, made from dental models with the defect removed, in combination with a tissue conditioner helps to eliminate resulting dead space, increase patient comfort, and facilitate healing in cases in which communication occurs with the nasal floor. Soft tissue breakdown is not uncommon over a midline incision; however, meticulous hygiene, irrigation, and tissue conditioners help to minimize these complications.

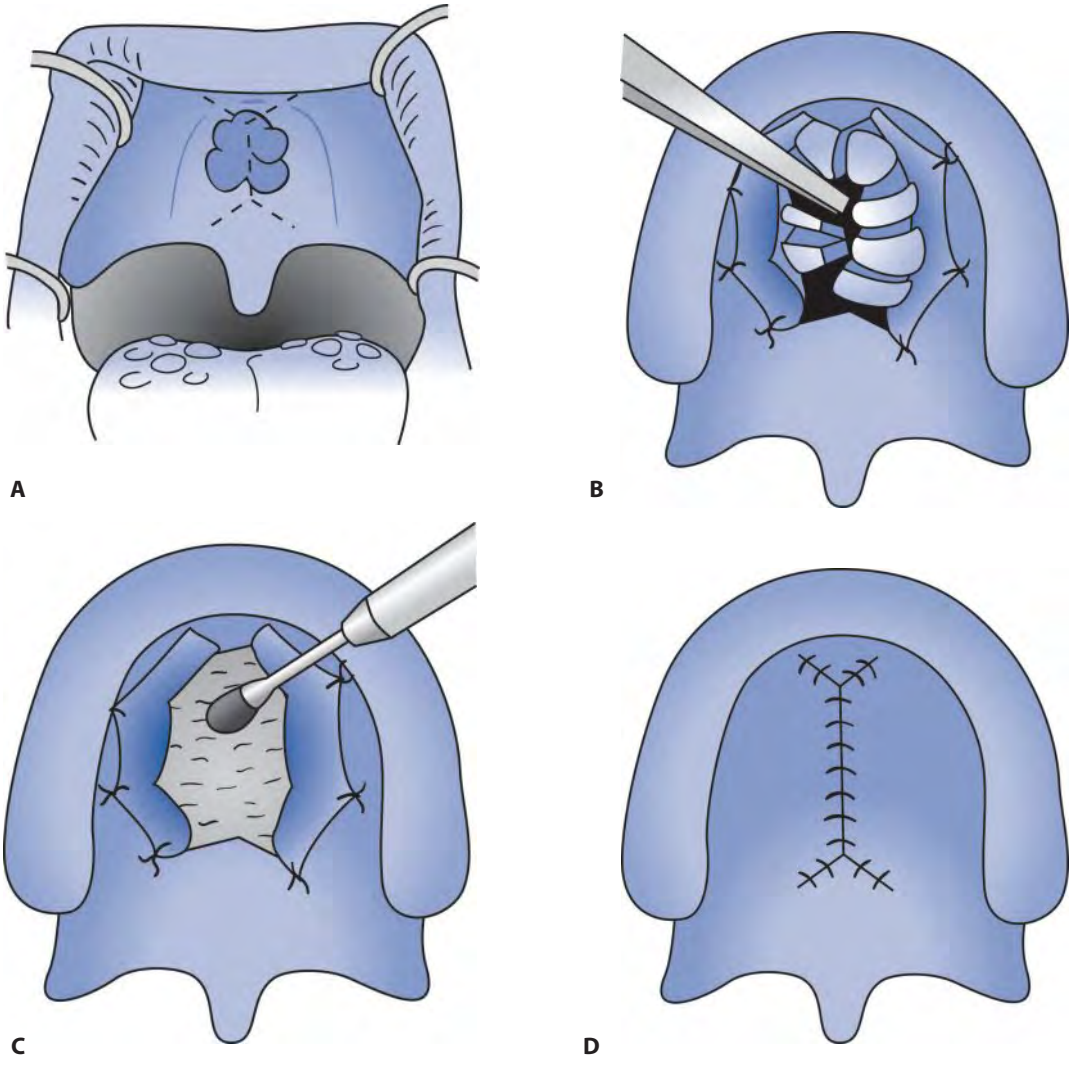


FIGURE 6-14. **A,** Preoperative view of a maxillary torus with the midline incision indicated *(dashed line)*. **B,** Removal of sectioned elements of the torus with an osteotome. **C,** Final smoothing of irregularities with a rotary bur. **D,** Final closure.

Mandibular tori are accessed using bilateral inferior alveolar and lingual nerve blocks as well as local infiltration to facilitate dissection. A generous crestal incision with subsequent mucoperiosteal flap elevation is performed. Maintenance of the periosteal attachment in the midline reduces hematoma formation and maintains vestibular depth. Nevertheless, when large tori encroach on the midline, maintenance of this midline periosteal attachment is impossible. Careful flap elevation with attention to the thin friable overlying mucosa is necessary because this tissue is easily damaged. Small protuberances can be sheared away with a mallet and osteotome. Large tori are divided superiorly from the adjacent bone with a fissure bur parallel to the medial axis of the mandible and are out-fractured away from the mandible by an osteotome, which provides leverage (Figure 6-15). The residual bony fragment inferiorly may then be relieved with a hand rasp or bone file. It is not imperative that the entire protuberance be removed as long as the goals of the procedure are achieved. Copious irrigation during this procedure is imperative, and closure is completed using a resorbable suture in a running fashion. Temporary denture delivery or gauze packing lingually may be used to prevent hematoma formation and should be maintained for approximately 1 day postoperatively. Wound dehiscence and breakdown with exposure of underlying bone is not uncommon and should be treated with local irrigation with normal saline.

Mylohyoid Ridge Reduction

In cases of mandibular atrophy, the mylohyoid muscle contributes significantly to the displacement of conventional dentures. With the availability of advanced grafting techniques and dental implants, there are fewer indications for the reduction of the mylohyoid ridge. In severe cases of mandibular atrophy, the external oblique and mylohyoid ridges may be the height of contour of the posterior mandible. In these cases, the bony ridge may be a significant source of discomfort because the overlying mucosa is thin and easily irritated by denture flanges extending into the posterior floor of the mouth. As a result, reduction of the mylohyoid ridge may accompany grafting techniques to provide greater relief and comfort for subsequent restorations. Historically, this procedure has been combined with lowering of the floor of the mouth; however, with the advanced armamentarium available today, there are few, if any, indications for these procedures alone or in combination.

Anesthesia is achieved with buccal, inferior alveolar, and lingual nerve blocks. A crestal incision over the height of contour is made, erring toward the buccal aspect to protect the lingual nerve. Subperiosteal dissection along the medial aspect of the mandible reveals the attachment of the mylohyoid muscle to the adjacent ridge. This can be sharply separated with electrocautery to minimize muscle bleeding. Once the overlying muscle is relieved, a reciprocating rasp or bone file can be used to smooth the remaining ridge. Copious irrigation and closure is completed, with particular attention to hemostasis. Placement of a stent or existing denture may also aid in hemostasis as well as inferiorly repositioning the attachment. Again, these procedures are rarely indicated and are included here essentially for historic reference, not for routine use.

SOFT TISSUE RECONTOURING

With the eventual bony remodeling that follows tooth loss, muscle and frenum attachments that initially were not in a problematic position begin to create complications in prosthetic reconstruction and to pose an increasing problem with regard to prosthetic comfort, stability, and fit. Often, these attachments must be altered before conventional restoration can be attempted. As dental implants become commonplace in the restoration of partially and totally edentulous patients,

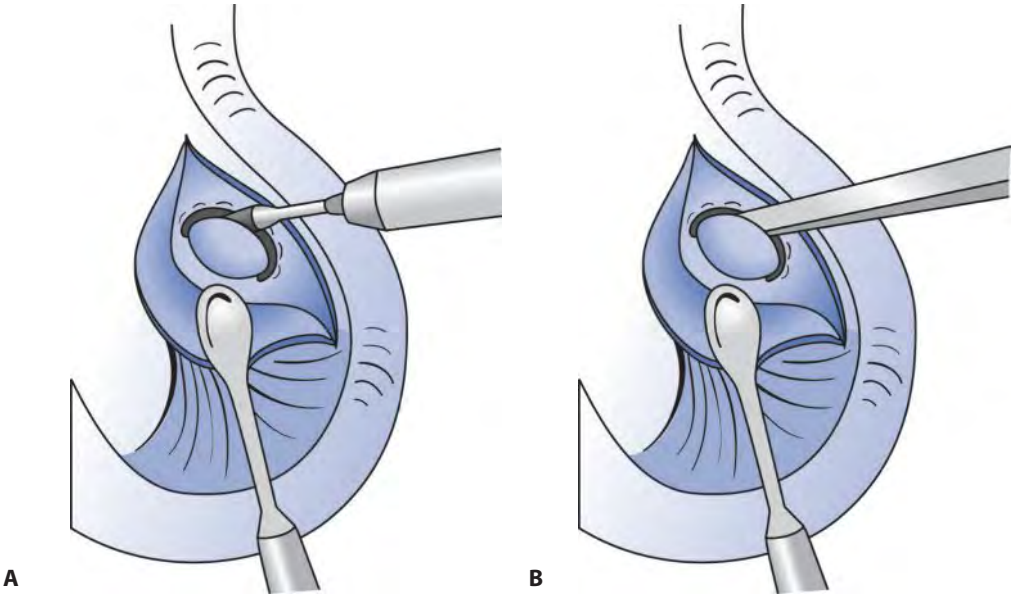


FIGURE 6-15. **A,** Rotary trough exposes a mandibular torus and creates a cleavage plane between the torus and the mandible. **B,** Osteotome shears the remaining attachment of the torus from the mandible.

surgical alteration of these attachments is indicated less often. Nevertheless, inflammatory conditions such as inflammatory fibrous hyperplasia of the vestibule or epulis and inflammatory hyperplasia of the palate must be addressed before any type of prosthetic reconstruction can proceed. Obviously, any lesion presenting pathologic consequences should undergo biopsy and be treated accordingly before reconstruction commences. In keeping with reconstructive surgery protocol, soft tissue excesses should be respected and should not be discarded until the final bony augmentation is complete. Excess tissue thought to be unnecessary may be valuable after grafting or augmentation procedures are performed to increase the overall bony volume.

Hypermobile Tissue

When excess mobile unsupported tissue remains after successful alveolar ridge restoration, or when mobile tissue exists in the presence of a preserved alveolar ridge, removal of this tissue is the treatment of choice. Usually, infiltrative local anesthesia can be performed in selected areas. Sharp excision parallel to the defect in a supraperiosteal fashion allows for removal of mobile tissue to an acceptable level. Beveled incisions may be needed to blend the excision with surrounding adjacent tissues and maintain continuity to the surrounding soft tissue. Closure with resorbable suture then approximates residual tissues. Impressions for prosthesis fabrication should proceed after a 3- to 4-week period to allow for adequate soft tissue remodeling. In cases in which denture flange extension is anticipated, the clinician must be careful to preserve the vestibule when undermining for soft tissue closure. Granulation is a better alternative if residual tissues cannot be approximated because it maintains the vestibule and increases the width of the attached keratinized mucosa.

Fibrous Inflammatory Hyperplasia

Fibrous inflammatory hyperplasia is often the result of an ill-fitting denture that produces underlying inflammation of the mucosa and eventual fibrous proliferation resulting in patient discomfort and a decreased fit of the overlying prosthesis. Early management consists mainly of adjustment of the offending denture flange with an associated soft reline of the prosthesis. When there is little chance of eliminating the fibrous component, surgical excision is necessary. In most cases, laser ablation with a carbon dioxide laser is the method of choice. When the treatment of large lesions would result in significant scarring and obliteration of the vestibule, sharp excision with undermining of the adjacent mucosa and reapproximation of the tissues is preferred. Again, maintenance of a supraperiosteal plane with repositioning of mucosal edges allowing for subsequent granulation is preferred over approximation of wound edges that results in the alteration of vestibular depth. This is accomplished with local anesthetic infiltrated into the proposed tissue bed, which is closed only if necessary with resorbable sutures.

Inflammatory Papillary Hyperplasia

Once thought to be a neoplastic process, inflammatory papillary hyperplasia occurs mainly in patients with existing prosthetic appliances.[34] An underlying fungal etiology most often is the source of the inflammatory process and appears to coincide with mechanical irritation and poor hygiene practices. The lesion appears as multiple proliferative nodules underlying a mandibular prosthesis likely colonized with *Candida*. Early stages are easily treated by an improvement of hygiene practices and by the use of antifungal therapy such as nystatin twice daily alternating with clotrimazole troches intermittently. Nocturnal soaking of the prosthesis in an antifungal solution or in an extremely dilute solution of sodium hypochlorite helps decrease the overall colonization of the prosthesis.

In proliferative cases necessitating surgical treatment, excision in a supraperiosteal plane is the method of choice. Many methods are acceptable, including sharp excision with a scalpel, rotary débridement, loop electrocautery as described by Guernsey,[35] and laser ablation with a carbon dioxide laser.[36,37] Because of the awkward access needed to remove the lesions, laser ablation is the method we employ. Treatment proceeds supraperiosteally to prevent exposure of underlying palatal bone. Subsequently, placement of a tissue conditioner and a denture reline is helpful to minimize patient discomfort.

TREATMENT OF THE LABIAL AND LINGUAL FRENUM

Labial Frenectomy

Labial frenum attachments consist of thin bands of fibrous tissue covered with mucosa extending from the lip and cheek to the alveolar periosteum. The height of this attachment varies from individual to individual; however, in dentate individuals, frenum attachments rarely cause a problem. In edentulous individuals, frenum attachments may interfere with fit and stability, produce discomfort, and dislodge the overlying prostheses.

Several surgical methods are effective in excising these attachments. Simple excision and Z-plasty are effective for narrow frenum attachments (Figures 6-16 and 6-17). Vestibuloplasty is often indicated for frenum attachments with a wide base.

Local anesthetic infiltration is performed in a regional fashion that avoids direct infiltration into the frenum itself; such an infiltration distorts the anatomy and leads to misidentification of the frenum. Eversion of the lip also helps one identify the anatomical frenum and assists with the excision. An elliptical incision around the proposed frenum is completed in a supraperiosteal fashion. Sharp dissection of the frenum using curved scissors removes mucosa and underlying connective tissue leading to a broad base of periosteum attached to the underlying bone. Once tissue margins are

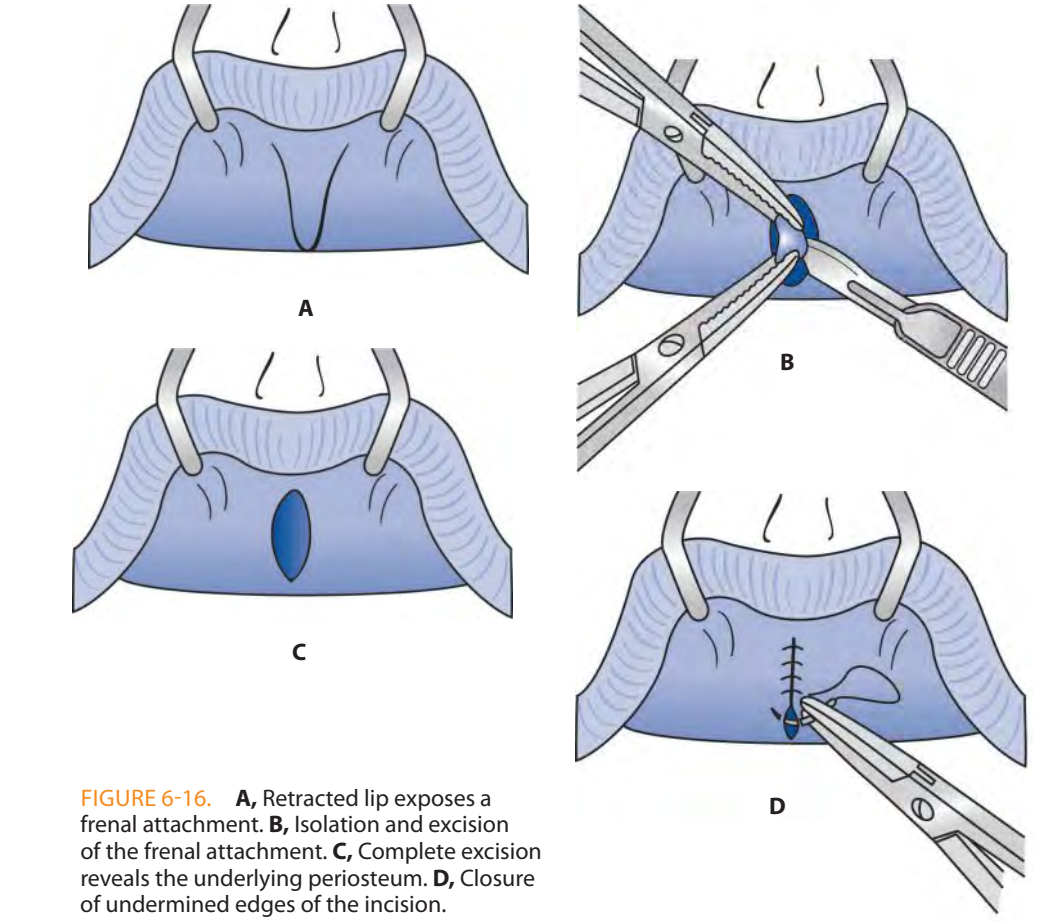


FIGURE 6-16. **A,** Retracted lip exposes a frenal attachment. **B,** Isolation and excision of the frenal attachment. **C,** Complete excision reveals the underlying periosteum. **D,** Closure of undermined edges of the incision.

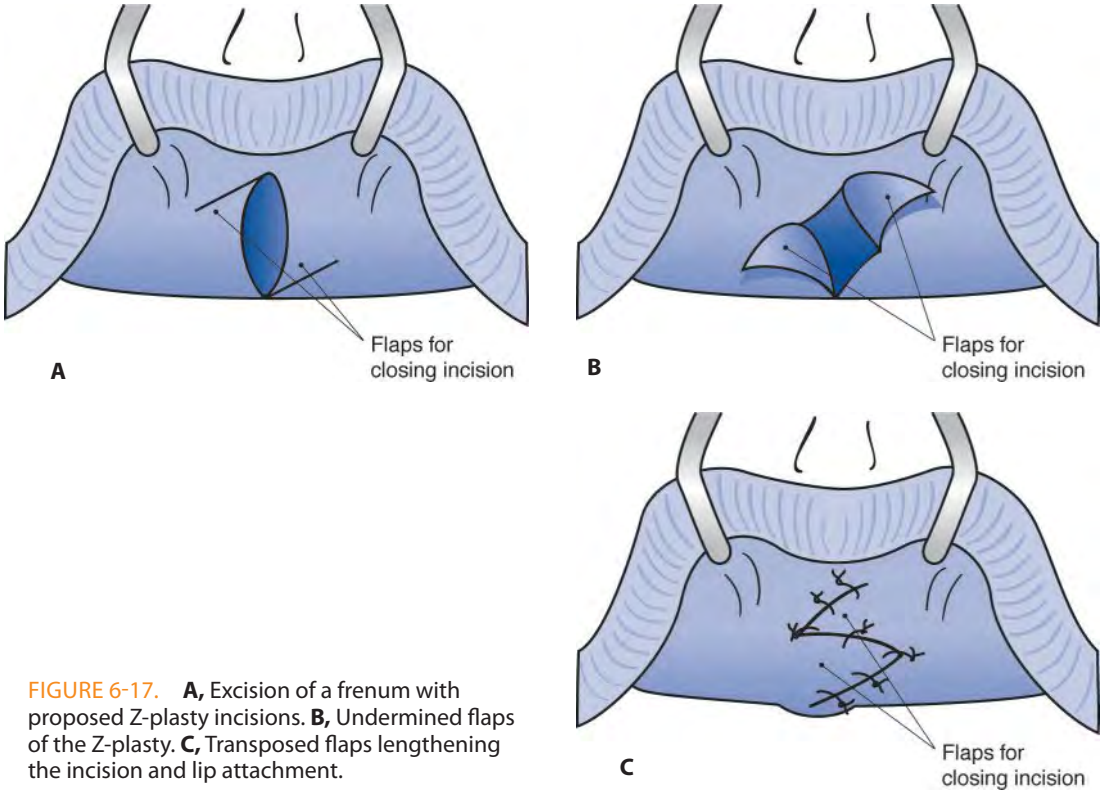


FIGURE 6-17. **A,** Excision of a frenum with proposed Z-plasty incisions. **B,** Undermined flaps of the Z-plasty. **C,** Transposed flaps lengthening the incision and lip attachment.

undermined and wound edges are approximated, closure can proceed with resorbable sutures in an interrupted fashion. Sutures should encounter the periosteum, especially at the depth of the vestibule, to maintain alveolar ridge height. This also reduces hematoma formation and allows for the preservation of alveolar anatomy.

In the Z-plasty technique, excision of the connective tissue is done similar to that described previously. Two releasing incisions creating a Z shape precede undermining of the flaps. The two flaps are eventually undermined and rotated to close the initial vertical incision horizontally. By using the transposition flaps, this technique virtually increases vestibular depth and should be used when alveolar height is in question.

Wide-based frenum attachments may best be treated with a localized vestibuloplasty technique. A supraperiosteal dissection is used to expose the underlying periosteum. Superior repositioning of the mucosa is completed, and the wound margin is sutured to the underlying periosteum at the depth of the vestibule. Healing proceeds by secondary intention. A preexisting denture or stent may be used for patient comfort in the initial postoperative period.

Lingual Frenectomy

High lingual frenum attachments may consist of different tissue types including mucosa, connective tissue, and superficial genioglossus muscle fibers. This attachment can interfere with denture stability, speech, and tongue range of motion. Bilateral lingual blocks and local infiltration in the anterior mandible provide adequate anesthesia for the lingual frenum excision. To provide adequate traction, a suture is placed through the tip of the tongue. Surgical release of the lingual frenum requires dividing the attachment of the fibrous connective tissue at the base of the tongue in a transverse fashion, followed by closure in a linear direction, which completely releases the ventral aspect of the tongue from the alveolar ridge (Figure 6-18). Electrocautery or a hemostat can be used to minimize blood loss and improve visibility. After removal of the hemostat, an incision is created through the area previously closed within the hemostat. Careful attention must be given to Wharton's ducts and superficial blood vessels in the floor of the mouth and ventral tongue. The edges of the incision are undermined, and the wound edges are approximated and closed with a running resorbable suture, burying the knots to minimize patient discomfort.

RIDGE EXTENSION PROCEDURES IN THE MAXILLA AND MANDIBLE

Submucous Vestibuloplasty

In 1959, Obwegeser[38] described the submucous vestibuloplasty to extend fixed alveolar ridge tissue in the maxilla. This procedure is particularly useful in patients who have undergone alveolar ridge resorption with an encroachment of attachments to the crest of the ridge. Submucous vestibuloplasty is ideal when the remainder of the maxilla is anatomically conducive to prosthetic reconstruction. Adequate mucosal length must be available for this procedure to be successful without disproportionate alteration of the upper lip. If a tongue blade or mouth mirror is placed to the height of the maxillary vestibule without distortion or inversion of the upper lip, adequate labiovestibular depth is present (Figure 6-19).[9] If distortion occurs, then maxillary vestibuloplasty using split-thickness skin grafts or laser vestibuloplasty is the appropriate procedure.

Submucous vestibuloplasty can be performed in the office setting under outpatient general anesthesia or deep sedation. A midline incision is placed through the mucosa in the maxilla, followed by mucosal undermining bilaterally. A supraperiosteal separation of the intermediate muscle and soft tissue attachments is completed. Sharp incision of this intermediate tissue plane is made at its attachment near the crest of the maxillary alveolus. This tissue layer may then be excised or superiorly repositioned (Figure 6-20). Closure of the incision

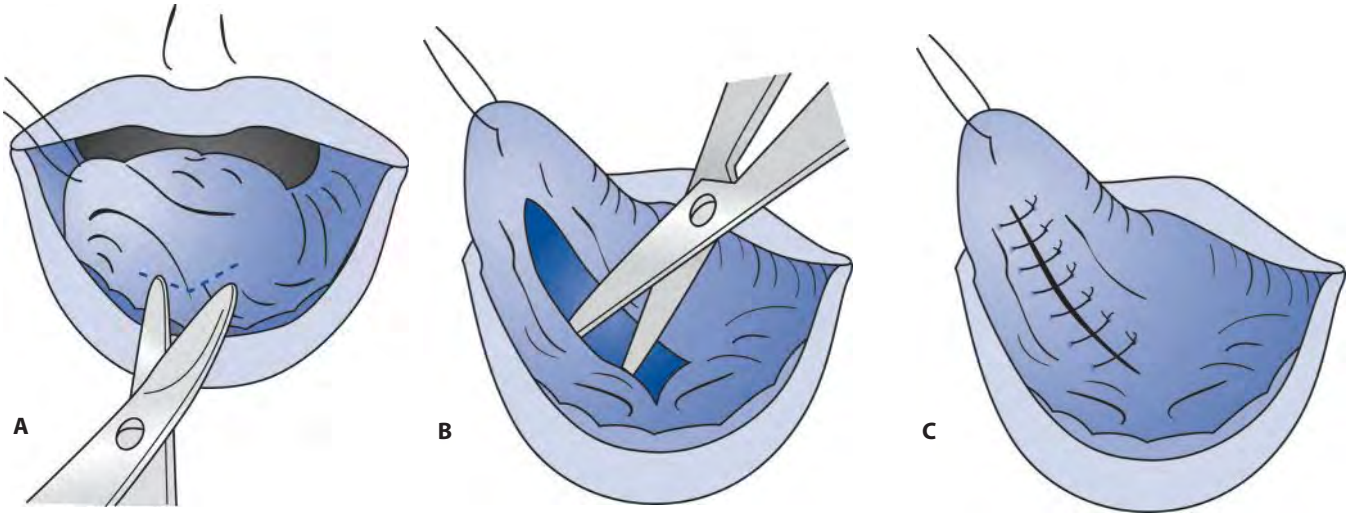


FIGURE 6-18. **A,** Lingual frenum attachment encroaches on an atrophic mandibular alveolus. **B,** Excision of the frenum with undermining of mucosal edges. *Note:* Care must be taken to avoid causing damage to Wharton's ducts. **C,** Final closure of mucosal edges.

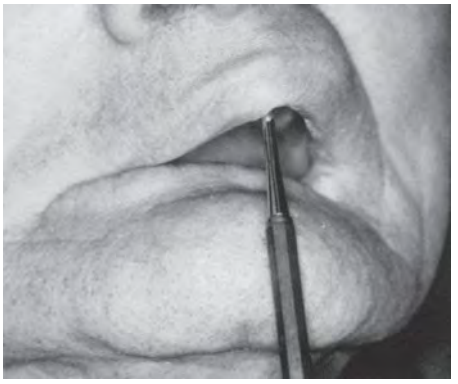


FIGURE 6-19. A mirror presses the vestibular mucosa to the desired height to evaluate the adequacy of lip mucosa. In this example, extension of the vestibular mucosa superiorly on the alveolar ridge does not result in thinning or intrusion of the lip. (Reproduced with permission from Tucker MR. Ambulatory preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; p. 1126.)

and placement of a postsurgical stent or denture rigidly screwed to the palate is necessary to maintain the new position of the soft tissue attachments. Removal of the denture or stent is performed 2 weeks postoperative. During the healing period, mucosal tissue adheres to the underlying periosteum, creating an extension of fixed tissue covering the maxillary alveolus. A final reline of the patient's denture may proceed at approximately 1 month postoperative.

Maxillary Vestibuloplasty

When a submucous vestibuloplasty is contraindicated, mucosa pedicled from the upper lip may be repositioned at the depth of the vestibule in a supraperiosteal fashion. The exposed periosteum can then be left to epithelialize secondarily. Split-thickness skin grafts may be used to help shorten the healing period. In addition, placement of a relined denture may minimize patient discomfort and help to mold and adapt underlying soft tissues and/or skin grafts.

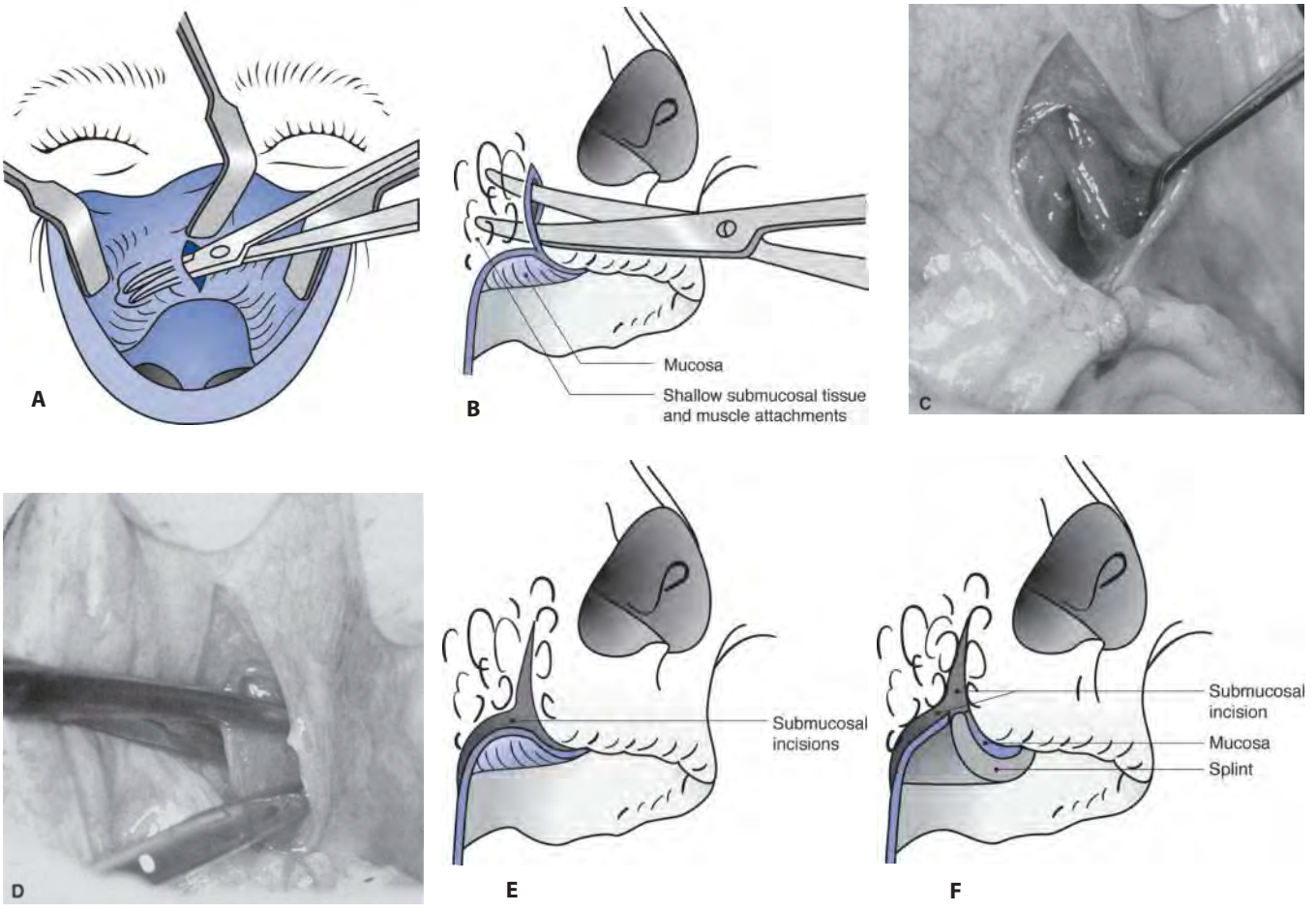


FIGURE 6-20. Maxillary submucosal vestibuloplasty. **A,** Following the creation of a vertical midline incision, scissors are used to bluntly dissect a thin mucosal layer. **B,** A second supraperiosteal dissection is created using blunt dissection. **C,** Interposing submucosal tissue layer created by submucosal and supraperiosteal dissections. **D,** Interposing tissue layer is divided with scissors. The mucosal attachment to the periosteum may be increased by removal of this tissue layer. **E,** Connected submucosal and supraperiosteal dissections. **F,** Splint extended in to the maximum height of the vestibule, placing the mucosa and periosteum in direct contact. **G,** Preoperative appearance of the maxilla with muscular attachments on the lateral aspects of the maxilla. **H,** Postoperative view. (**A, B, E,** and **F,** Adapted from Tucker MR. Ambulatory preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; pp. 1126–1127; **C, D,** *(Continued)*

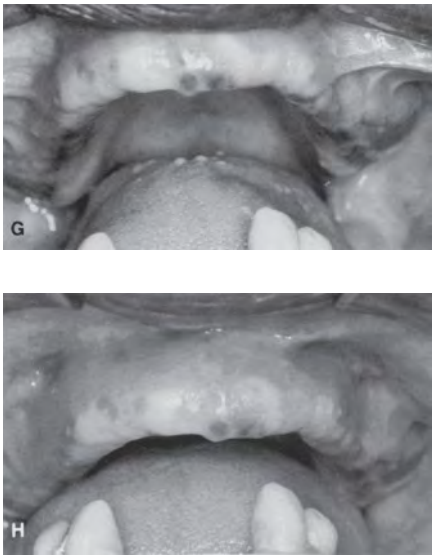


FIGURE 6-20. *(Continued)* **G,** and **H,** reproduced with permission from Tucker MR. Ambulatory preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; p. 1127.)

Another option in this situation is laser vestibuloplasty. A carbon dioxide laser is used to resect tissue in a supraperiosteal plane to the depth of the proposed vestibule. A denture with a soft reline is then placed to maintain vestibular depth. Removal of the denture in 2 to 3 weeks reveals a nicely epithelialized vestibule that extends to the desired depth (Figure 6-21).

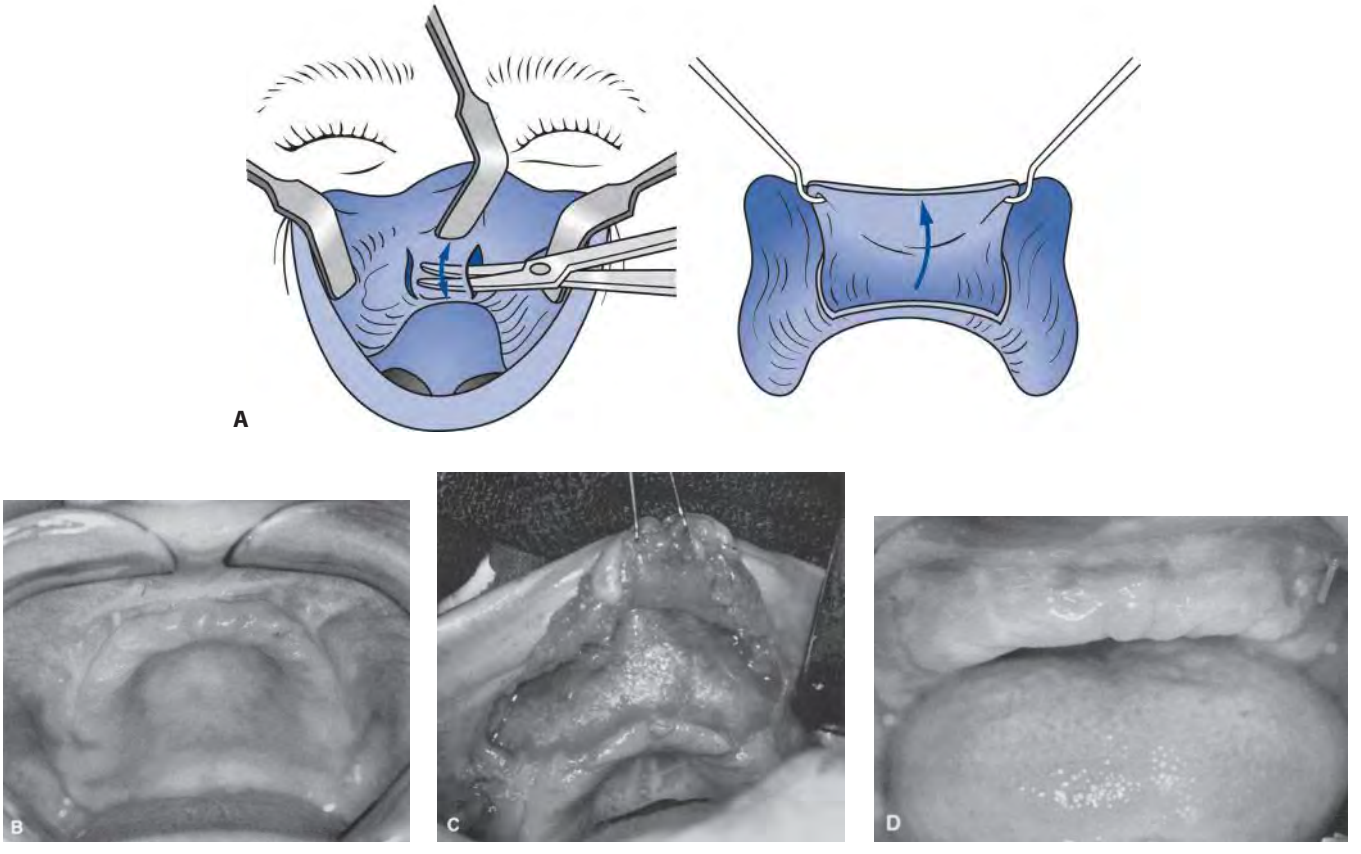


FIGURE 6-21. Open submucosal vestibuloplasty. **A,** Submucosal dissection between two anterior vertical incisions followed by an incision on the crest of the alveolar ridge. **B,** Preoperative appearance of the maxilla. Hydroxyapatite augmentation had been performed but resulted in inadequate vestibular depth. **C,** Intraoperative photograph after elevation of the mucosal flap and removal of submucosal tissue. **D,** Appearance at the time of splint removal. (**A,** Adapted from Tucker MR. Ambulatory preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; p. 1128; **B–D,** reproduced with permission from Tucker MR. Ambulatory preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; p. 1128.)

Lip-Switch Vestibuloplasty

Both lingually based and labially based vestibuloplasties have been described.[39,40] In the former, an incision in the lower lip and submucosa undermining to the alveolus is followed by a supraperiosteal dissection to the depth of the vestibule (Figure 6-22). The mucosal flap is then sutured to the depth of the vestibule and stabilized with a stent or denture. The labial denuded tissue is allowed to epithelialize secondarily.

In the transpositional vestibuloplasty, the periosteum is incised at the crest of the alveolus and transposed and sutured to the denuded labial submucosa. The elevated mucosal flap is then positioned over the exposed bone and sutured to the depth of the vestibule (Figure 6-23).

These procedures provide satisfactory results provided that adequate mandibular height exists preoperatively A minimum of 15 mm is acceptable for these procedures. Disadvantages include unpredictable results, scarring, and relapse.

Mandibular Vestibuloplasty and Floor-of-Mouth–Lowering Procedures

As with labial muscle attachments and soft tissue in the buccal vestibule, the mylohyoid and genioglossus attachments can preclude denture flange placement lingually. In a combination of the procedures described by Trauner[41] as well as Obwegeser and MacIntosh,[42] both labial and lingual extension procedures can be performed to effectively lower the floor of the mouth (Figure 6-24).[43] This procedure eliminates the components involved in the displacement of conventional dentures and provides a broad base of fixed tissue for prosthetic support. Again, adequate mandibular height of at least 15 mm is required. Split-thickness skin grafting is used to cover the denuded periosteum and facilitate healing.

As the popularity of implant-borne prostheses not requiring lingual and buccolabial flanges increases, the need for procedures providing a broad mandibular base in order to support a conventional denture has deminished. However, the vestibuloplasty and floor-of-mouth–lowering procedures still play an important role in mandibular reconstruction, especially in patients requiring mandibular resection owing to oral cancer or an intraosseous tumor or cyst. These patients typically lose keratinized mucosa during the initial resection, requiring the reconstructed mandible to be covered with movable mucosa. Unfortunately, this places muscle attachments near the planned prosthesis. A vestibuloplasty and floor-of-mouth lowering combined with a split-thickness skin graft is necessary to move muscle attachments away from the newly reconstructed mandibular ridge, while the skin graft helps minimize peri-implant trauma resulting from the prosthesis. This combination of a vestibuloplasty, floor-of-mouth lowering, and split-thickness skin graft therefore minimizes inflammation, peri-implantitis, vertical bone loss, and implant failure. If an implant-retained prosthesis in planned, these procedures can be performed before the time of implant placement, at the time of implant placement (Figure 6-25), or after implant placement. The advantage of placing the implants first is that they can then be used to support the surgical splint required for the skin grafting procedure.[44]

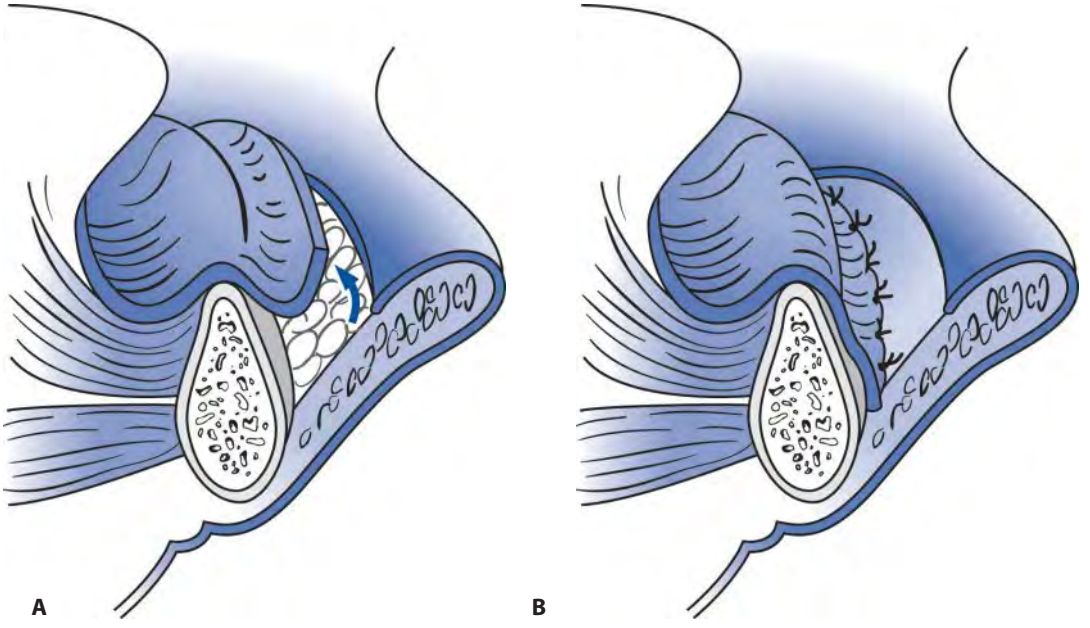


FIGURE 6-22. Kazanjian flap vestibuloplasty. **A,** An incision is made in the labial mucosa, and a thin mucosal flap is dissected from the underlying tissue. A supraperiosteal dissection is performed on the anterior aspect of the mandible. **B,** The labial mucosal flap is sutured to the depth of the vestibule. The anterior aspect of the labial vestibule heals by secondary intention. (**A** and **B,** Adapted from Tucker MR. Ambulatory preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; p. 1120.)

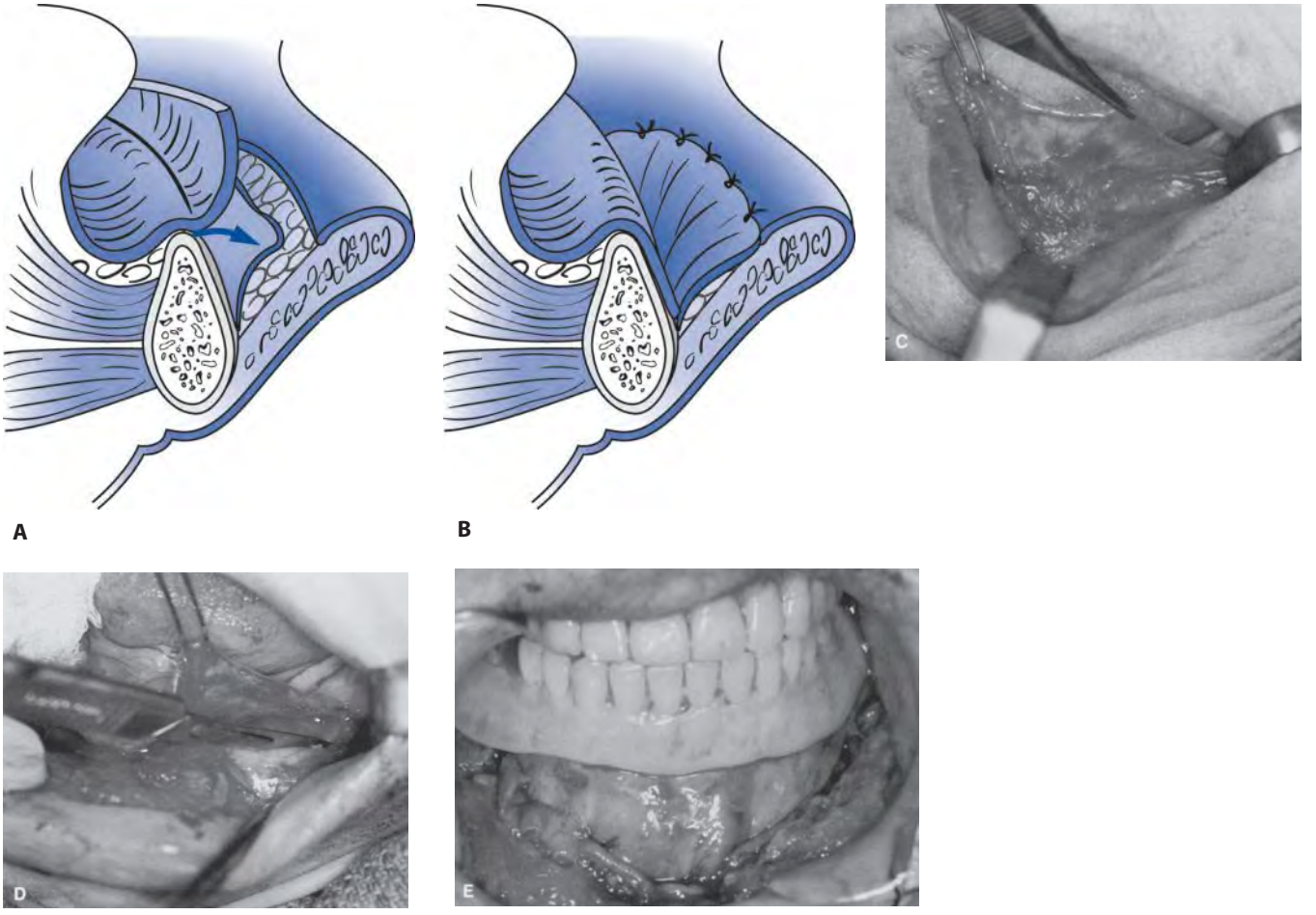


FIGURE 6-23. Transpositional flap (lip-switch) vestibuloplasty. **A,** After elevation of the mucosal flap, the periosteum is incised at the crest of the alveolar ridge and a subperiosteal dissection is completed on the anterior aspect of the mandible. **B,** The periosteum is then sutured to the anterior aspect of the labial vestibule, and the mucosal flap is sutured to the vestibular depth at the area of the periosteal attachment. **C,** Elevation of the mucosal flap. **D,** Periosteal incision along the crest of the alveolar ridge. **E,** Mucosa is sutured to the vestibular depth at the area of the periosteal attachment. Note the amount of vestibular depth extension compared with the old vestibular depth, which is marked by the previous denture flange. (**A** and **B,** Adapted from Tucker MR. Ambulatory preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; p. 1121; **C–E,** reproduced from Tucker MR. Ambulatory preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; p. 1121.)

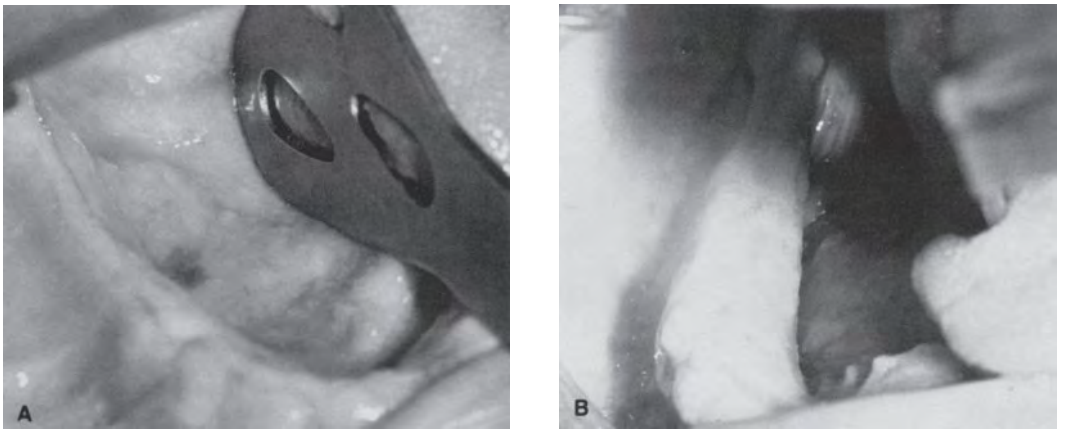


FIGURE 6-24. Floor-of-mouth lowering. **A,** Mucosal and muscular attachments near the crest of the alveolar ridge. **B,** Inferior repositioning of the mucosal flap after the mucosal incision and sectioning of mylohyoid muscle have been performed. *(Continued)*

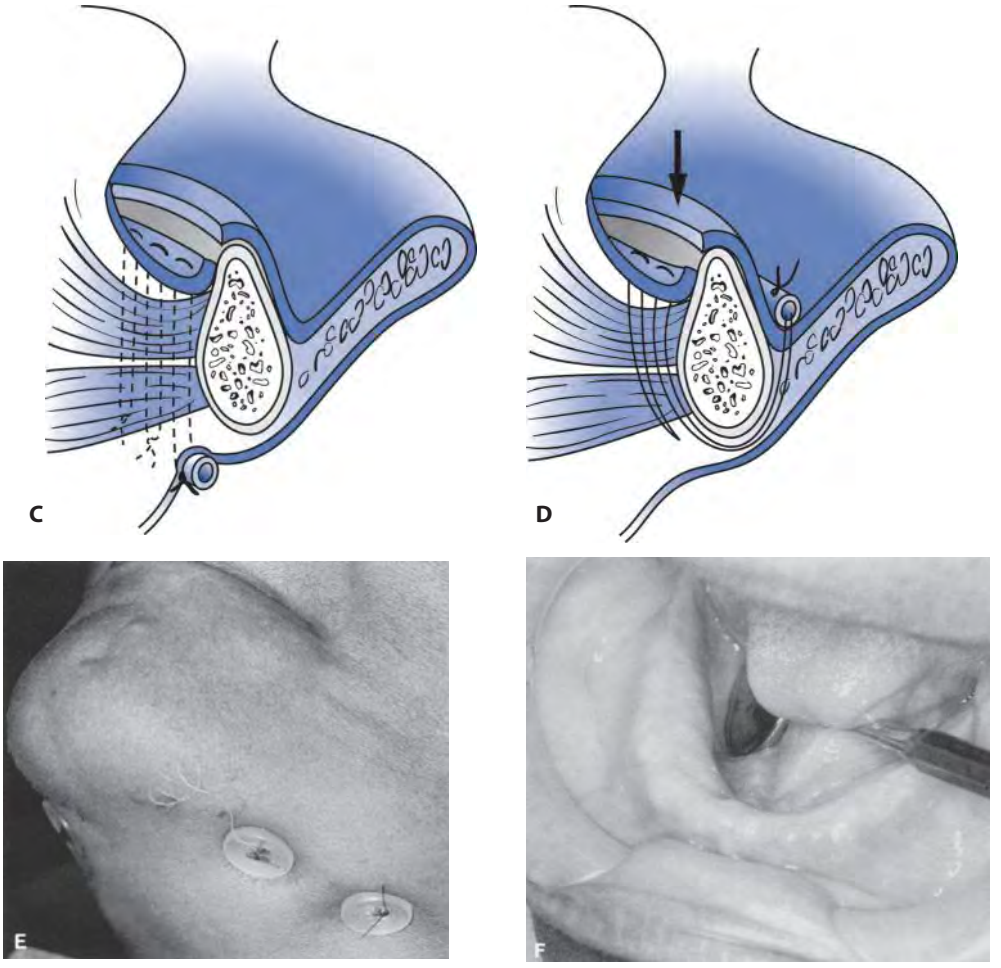


FIGURE 6-24. *(Continued)* **C,** Bolsters placed percutaneously to secure the flap inferiorly. **D,** Inferior mandibular "sling" sutures provide inferior traction on the mucosal flap. **E,** Buttons secure the mucosal flap sutures. **F,** Postoperative appearance of the floor-of-mouth extension. (**A, B, E,** and **F,** Reproduced with permission from Tucker MR. Ambulatory preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; pp. 1122–1123; **C** and **D,** adapted from Tucker MR. Ambulatory preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; p. 1122.)

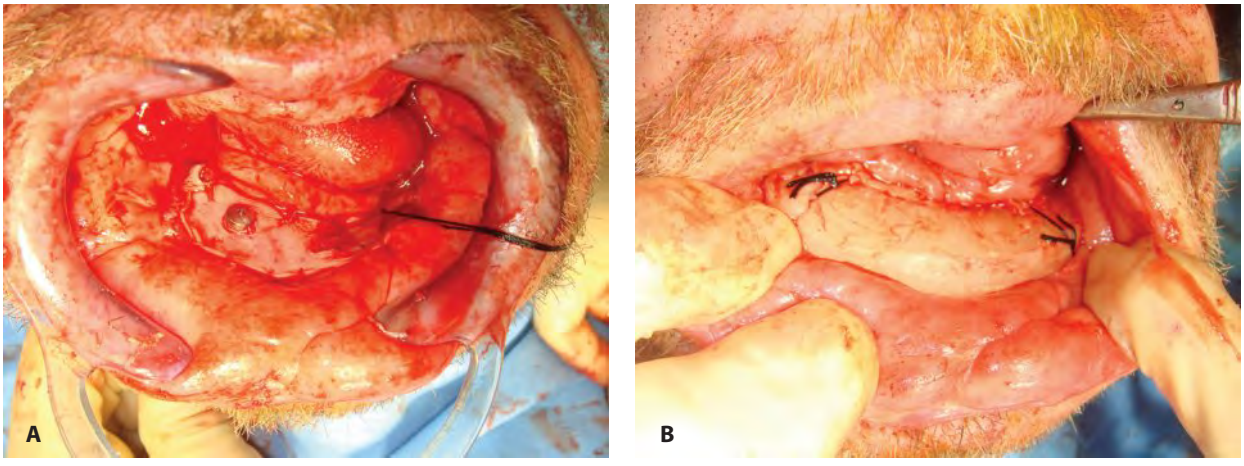


FIGURE 6-25. **A,** Floor-of-mouth–lowering procedure and mandibular vestibuloplasty were performed in conjunction with placement of dental implants. This was followed by a split-thickness skin graft to provide keratinized tissue adjacent to the implants. **B,** A splint secured with circummandibular sutures was used to prevent mobility of the skin graft.

HARD TISSUE AUGMENTATION

As stated previously, the overall goals of reconstructive preprosthetic surgery are to provide an environment for the prosthesis that will restore function, create stability and retention, and service associated structures as well as satisfy aesthetics and prevent minor sensory loss. There are many classification systems of rigid deficiencies associated with many treatment options; nevertheless, each patient must be evaluated individually. When atrophy of the alveolus necessitates bony augmentation, undercuts, exostoses, and inappropriate tissue attachments should be identified and included in the overall surgical plan before prosthetic fabrication.

Maxillary Augmentation

In the past, vestibuloplasties were the procedure of choice to accentuate the alveolus in the atrophic maxilla. Unfortunately, poor quality and quantity of bone combined with excessive occlusal loading by conventional prostheses continued to accelerate the resorptive process. Either augmentation or transantral implant cross-arch stabilization must be considered when anatomic encroachment of the palatal vault or zygomatic buttress and loss of tuberosity height affect overall fit and function of a conventional prosthesis. This section discusses conventional augmentation procedures of the maxilla to restore acceptable alveolar form and dimensions.

There is a fourfold increase in resorption in the mandible compared with that in the maxilla, combination syndromes notwithstanding. When severe resorption results in severely atrophic ridges (Cawood and Howell classes IV–VI[7]), some form of augmentation is indicated. Onlay, interpositional, and inlay grafting are the procedures of choice to reestablish acceptable maxillary dimensions.

Ridge-Split Osteoplasty

Ridge-splitting procedures geared toward expanding the knife-edged alveolus in a buccolingual direction help to restore the crucial endosteal component of the alveolus that is associated with preservation and response to transligamentary loading and maintains the alveolus during the dentate state. Replacement of this tissue allows for dental implant stimulation of the surrounding bone that can best mimic this situation and preserve the existing alveolus and possibly stimulate future bone growth. Adequate dimensions, however, should exist that allow for a midcrestal osteotomy to separate the buccal and lingual cortices (Figures 6-26 and 6-27). A labial

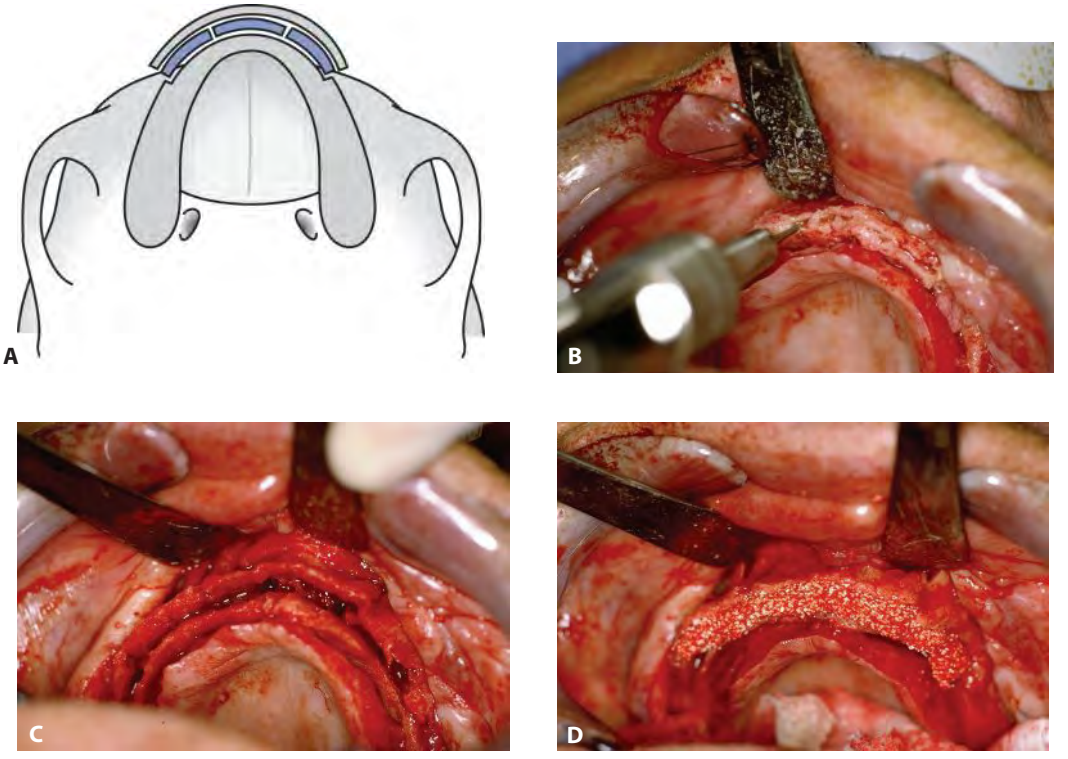


FIGURE 6-26. **A,** Diagram shows bone cuts and the position of the buccal fragment after the osteotomy. The bone grafts are already in position. **B,** Handpiece in position with small crosscut fissure bur performing osteotomy along the crest of the alveolar ridge. **C,** Osteotomy completed and buccal plate out-fractured to complete ridge split. The defect is now ready for interpositional graft to maintain the increased buccolingual width. **D,** Completed ridge split with interpositional corticocancellous and allogeneic bone used to fill the defect and maintain the increased buccolingual dimension. (**A,** Adapted from Stoelinga PJW. Preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Philadelphia: JB Lippincott; 1992; pp. 1169–1207.)

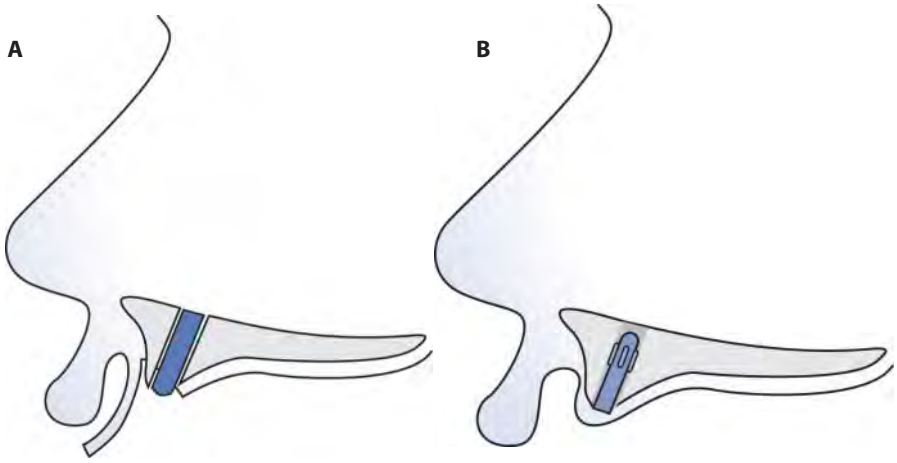


FIGURE 6-27. **A,** Schematic drawing of the bone graft position in relation to the nasal floor. Note the reflected buccal periosteum after palatal incisions. **B,** Position of endosteal implants after the bone graft has healed. (**A** and **B,** Adapted from Stoelinga PJW. Preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Philadelphia: JB Lippincott; 1992; pp. 1169–1207.)

incision originates just lateral to the vestibule and continues supraperiosteally to a few millimeters below the crest of the alveolus. A subperiosteal flap then originates exposing the underlying crest. Copious irrigation accompanies an osteotomy circumferentially anterior to the maxillary sinus from one side to the other. Mobilization of the labial segment can be achieved with careful manipulation with an osteotome, taking care to maintain the labial periosteal attachment. An interpositional cancellous graft can then be placed in the resulting defect, replacing the lost bony mass. Closure of the incision is away from the graft site and usually requires suturing of the flap edge to the periosteum with subsequent granulation of the remainder of the exposed tissue bed. Endosteal implants can be placed approximately 3 to 4 months later; waiting this length of time has been shown to increase overall long-term implant success.

Onlay Grafts

When clinical loss of the alveolar ridge and palatal vault occur (Cawood and Howell class V[7]), vertical onlay augmentation of the maxilla is indicated. Initial attempts at alveolar restoration involved the use of autogenous rib grafts; however, currently, corticocancellous blocks of iliac crest are the source of choice.[45,46] In a similar approach to that described previously, the crest of the alveolus is exposed and grafts are secured with 1.5- to 2.0-mm screws. Studies show increased success with implant placement in a second-stage procedure rather than using them as sources of retention and stabilization of the graft and alveolus at the time of augmentation. Implant success ranges from greater than 90% initially and falls to 75% and 50%, respectively, at 3 and 5 years postoperative.[47–51] Implant success may be directly proportional to the degree of graft maturation and incorporation at the time of implant placement. As a result, 4 to 6 months of healing is an acceptable waiting period when long-term implant success may be affected.

Interpositional Grafts

Interpositional grafts are indicated when adequate palatal vault height exists in the face of severe alveolar atrophy (Cawood and Howell class VI[7]) posteriorly, resulting in an increased interarch space. Because this method involves a Le Fort I osteotomy, true skeletal discrepancies between the maxilla and the mandible can be corrected at the time of surgery. The improvement of maxillary dimensions as a result of interpositional grafts may obviate the need for future soft tissue recontouring to provide adequate relief for prosthetic rehabilitation (Figure 6-28). Although early studies entertained the simultaneous placement of dental implants at the time of augmentation, more recently, several authors have demonstrated better success rates for implants placed in a second-stage procedure; this alleviates the need for excessive tissue reflection for implant placement and allows for a more accurate placement at a later date.[47] A relapse of 1 to 2 mm has been demonstrated in interpositional grafts using the Le Fort I technique with rigid fixation.[52–56] More data are needed to determine long-term overall success and relapse with these procedures.

Sinus Lifts and Inlay Bone Grafts

Sinus lift procedures and inlay bone grafting play a valuable role in the subsequent implant restoration of a maxilla that has atrophied posteriorly and is unable to accommodate implant placement owing to the proximity of the maxillary sinus to the alveolar crest. Incisions just palatal to the alveolar crest are created, followed by subperiosteal exposure of the anterior maxilla. A cortical window 2 to 3 mm above the sinus floor is created with the use of a round diamond bur down to the membrane of the sinus. Careful in-fracture of the window with dissection of the sinus membrane off the sinus floor creates the space necessary for graft placement; the lateral maxillary wall is the ceiling for the subsequent graft (Figure 6-29). Corticocancellous blocks or particulate bone may be placed in the resulting defect. Tears in the membrane may necessitate coverage with collagen tape to prevent extrusion and migration of particulate grafts through the perforations. Although implant placement can proceed simultaneously when 4 to 5 mm of native alveolus exists, we have found few cases in which the alveolus meets these requirements and, therefore, elect to place implants approximately 6 months



FIGURE 6-28. Graphic **(A)** and clinical **(B)** presentation of interpositional iliac crest grafts to the maxilla.

later. Block and Kent[57] have reported an 87% success rate with sinus-grafting procedures. They also have stated that in the literature, there is an overall success rate ranging from 75% to 100%.[57,58] As these procedures gain popularity and are routinely incorporated into mainstream preprosthetic surgery treatment plans, more accurate data and long-term follow-up will be available.

Treatment of Skeletal and Alveolar Ridge Discrepancies

Supraeruption of teeth and associated alveolar bone into opposing edentulous spaces in partially edentulous patients precludes prosthetic rehabilitation owing to functional loss of freeway space and the fact that the opposing arch cannot be restored without the extraction of the offending supraerupted dentition. With segmental alveolar surgery, these teeth can be repositioned to achieve a more appropriate relationship with the adjacent dentition and to increase the interarch space to allow for proper prosthetic restoration of the opposing dentition. A preoperative workup should include a thorough extraoral and intraoral examination. Cephalometric analysis and study models should be obtained. Close communication with the restorative dentist is necessary to determine expectations regarding the final position of the tooth-bearing segment postoperatively. Mounted models, model surgery to

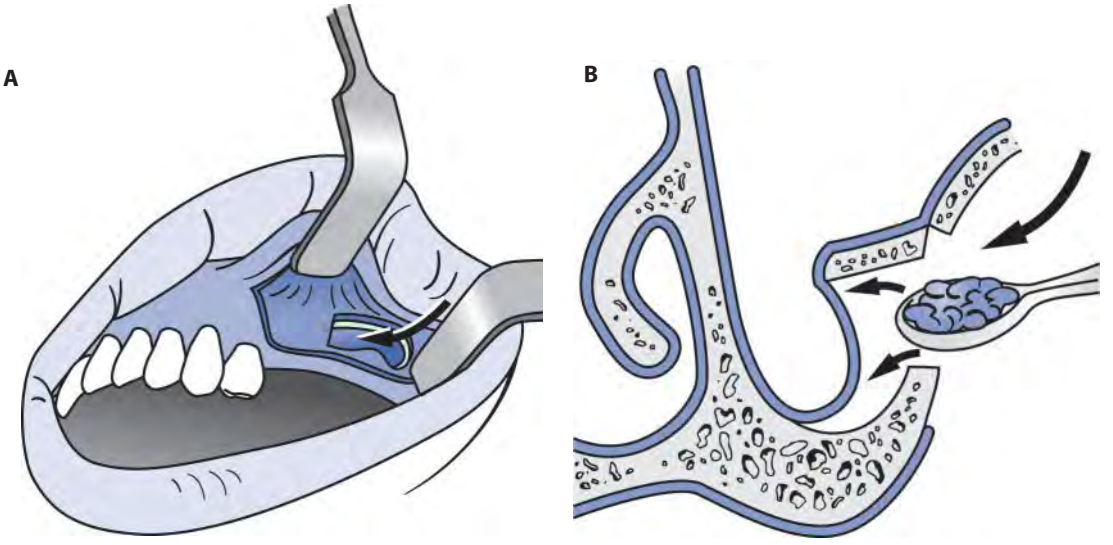


FIGURE 6-29. **A,** Sinus lift procedure with an inward trapdoor fracture of lateral sinus wall. **B,** Graft material is placed on the floor of the sinus. The sinus lining should not be perforated during the elevation of the bone. (**A** and **B,** Adapted from Beirne OR. Osseointegrated implant systems. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; p. 1146.)

reposition the segment, and diagnostic wax-ups of the proposed opposing dentition help one to verify the feasibility and success of the future prosthetic reconstruction. Surgical splint fabrication is necessary to support and stabilize the segment postoperatively. Increased stability is obtained if as many teeth as possible are included in the splint to help stabilize the teeth in the repositioned segment. The splint can be thickened to the opposing edentulous alveolar ridge to prevent relapse and to maintain the new vertical alignment of the repositioned segment. Techniques for segmental surgery are discussed in Chapter 58, and in other texts. An adequate healing period of approximately 6 to 8 weeks should precede prosthetic rehabilitation.

In totally edentulous patients with skeletal abnormalities that prevent successful prosthetic reconstruction owing to an incompatibility of the alveolar arches, orthognathic surgical procedures may create a more compatible skeletal and alveolar ridge relationship. This can aid the restorative dentist in the fabrication of functional and aesthetic restorations (Figure 6-30).[52] During the evaluation and treatment planning stage, the restorative dentist should play a major role in determining the final position of the maxillary and mandibular arches. Clinical examination, radiographic and cephalometric examinations, and articulated models should be attained to determine appropriate presurgical vertical and horizontal dimensions. This information should be combined with a cephalometric prediction analysis to determine the overall problem list and surgical treatment plan. Indexed surgical splints that can be rigidly fixed to the edentulous arches should be fabricated preoperatively at the time of model surgery; these splints aid in surgical repositioning of the maxilla, mandible, or both. Surgical procedures describing repositioning of the maxilla and mandible with rigid fixation are discussed in Chapters 58 and 59. Prosthetic reconstruction can usually proceed at 6 to 8 weeks postoperatively.

Mandibular Augmentation

One of the most challenging procedures in reconstructive surgery remains the reconstruction of the severely atrophic mandible (Cawood and Howell classes V and VI[7]). Patients exhibiting these deficits are characteristically overclosed, which creates an aged appearance, are usually severely debilitated from a functional perspective, and often present with significant risk for pathologic fracture of the mandible. Because the ideal graft should be vascularized and eventually incorporated into the host bone through a combination of osteoconduction and induction, autogenous bone grafts consistently meet these requirements and offer the most advantages to the reconstructive plan. Unfortunately, graft resorption and unpredictable remodeling have complicated grafting procedures; however, rigid fixation and later incorporation of dental implants have allowed for the needed stability postoperatively with regard to resorption and have promoted beneficial stimulation to preserve existing graft volume. Initially, mandibular augmentation with autogenous rib and ileum enjoyed little long-term success. However, recent incorporation of rigid-fixation, delayed implant placement 6 months after grafting (allowing for the initial stage of graft resorption), guided tissue regeneration, and BMP have all contributed to increased success rates in onlay augmentation of the mandible.[59–63]

Inferior Border Augmentation

Inferior border augmentation has been demonstrated using autogenous rib or composite cadaveric mandibles combined with autogenous cancellous bone (Figure 6-31).[64–66] The following describes our technique for inferior augmentation of the atrophic mandible using the latter method.

Incisions are placed as inconspicuously as possible from one mandibular angle to the other and proceed circumferentially



FIGURE 6-30. **A** and **B,** Preoperative views of a 52-year-old patient show severely collapsed circumoral tissues. **C** and **D,** The same patient 6 months postoperative. Note the improvement of the sagging chin and support for circumoral muscles. Better support for the lower lip also favorably affects the position of the upper lip. The patient is not wearing dentures in any of the photographs. (**A–D,** Reproduced with permission from Stoelinga PJW. Preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Philadelphia: JB Lippincott; 1992; pp. 1169–1207.)

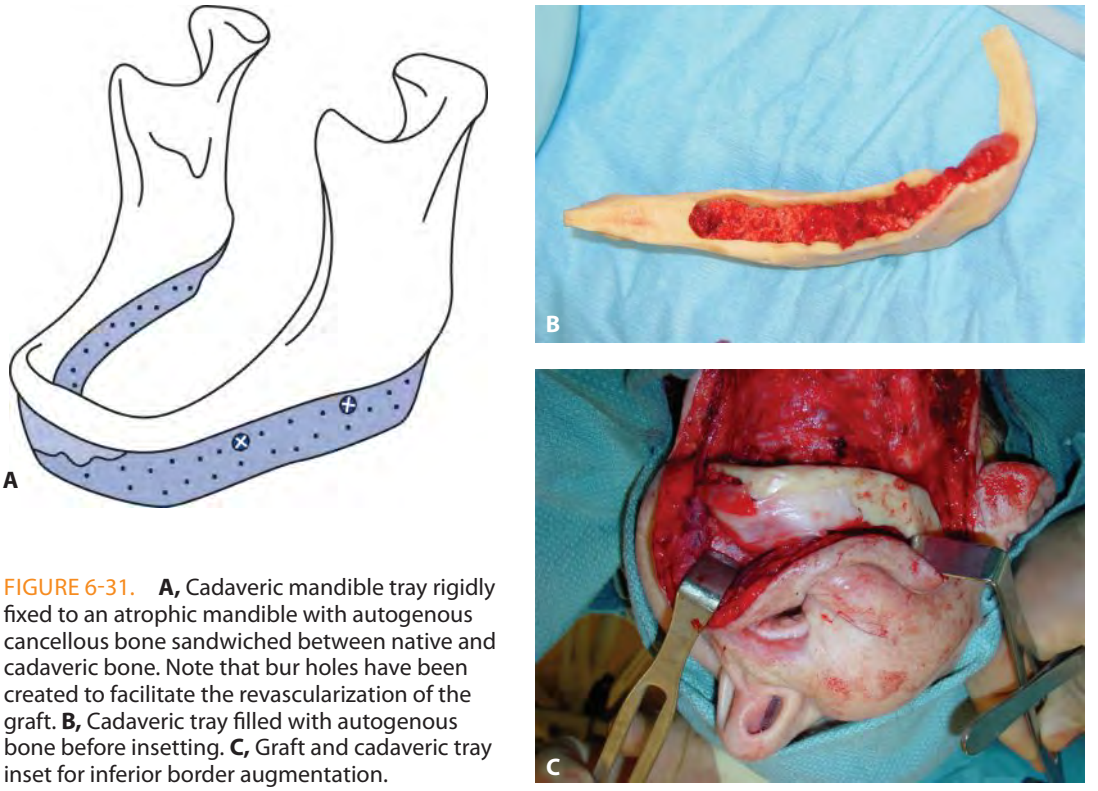


FIGURE 6-31. **A,** Cadaveric mandible tray rigidly fixed to an atrophic mandible with autogenous cancellous bone sandwiched between native and cadaveric bone. Note that bur holes have been created to facilitate the revascularization of the graft. **B,** Cadaveric tray filled with autogenous bone before insetting. **C,** Graft and cadaveric tray inset for inferior border augmentation.

3 to 4 mm below the inferior border of the mandible and anteriorly to the contralateral side. The superficial layer of the deep cervical fascia is sharply dissected. The fascia is then incorporated in the reflection; a nerve tester is used to perform a careful evaluation for the marginal mandibular branch of the facial nerve. Reflection superficial to the capsule of the submandibular gland allows dissection to the inferior border. Facial blood vessels are located and managed with surgical ties accordingly. The inferior border is exposed in a subperiosteal dissection with great care to avoid intraoral exposure. Cadaveric mandibular adjustment involves relieving the condyles and superior rami, thinning the bone to a uniform thickness of approximately 2 to 3 mm, and creating a scalloped tray to incorporate the autogenous bone. Repeated try-ins are necessary to evaluate the overall adaptation to the native mandible. Osseous interfaces as well as form and symmetry as they relate to the overall maxillomandibular relationship are evaluated. Once appropriate dimensions have been reached, the atrophic mandible fits securely inside the cadaveric specimen without creating a class III appearance, and flap closure is attainable, bur holes are drilled throughout the specimen to facilitate vascularization. Autogenous bone is then obtained from the ileum, morselized, and placed in the cadaveric specimen. BMP soaked in collagen is placed in the recipient bed as well as in a layered fashion over the autogenous graft. The entire specimen is fixed rigidly to the native mandible using screw fixation posterior to the area of future implant placement and in the mandibular midline, where implants are usually not placed. Postoperatively, patients can function with their preexisting prosthesis and enjoy increased stabilization of the mandible. When combined with implant placement at 4 to 6 months, this procedure results in an overall resorption rate of less than 5% and is associated with low rates of infection and dehiscence intraorally owing to the maintenance of mucosal barriers during reconstruction.

Pedicled and Interpositional Grafts

Placement of pedicled or interpositional grafts in the mandible is based on the maintenance of the lingual periosteum. The lingual periosteum maintains ridge form and its presence results in minimal resorption of the transpositioned basalar bone, as described by Stoelinga.[67] Peterson and Slade[68] as well as Harle[69] described the visor osteotomy in the late 1970s (Figure 6-32). Unfortunately, labial bone grafting of the superiorly repositioned lingual segment was necessary to reproduce alveolar dimensions that were compatible with prosthesis use. Schettler and Holtermann[70] and then Stoelinga and Tideman[71] described a horizontal osteotomy with interpositional grafts to augment mandibular height, with repositioning of the inferior alveolar neurovascular bundle (Figures 6-33 and 6-34). Unfortunately, neurosensory complications and collapse of the lingual segment became significant disadvantages to this technique. With the incorporation of mandibular implants and the success of full mandibular prostheses that are supported by four or five anterior implants between mental foramina, many of these pedicled and interpositional procedures are in decline today.

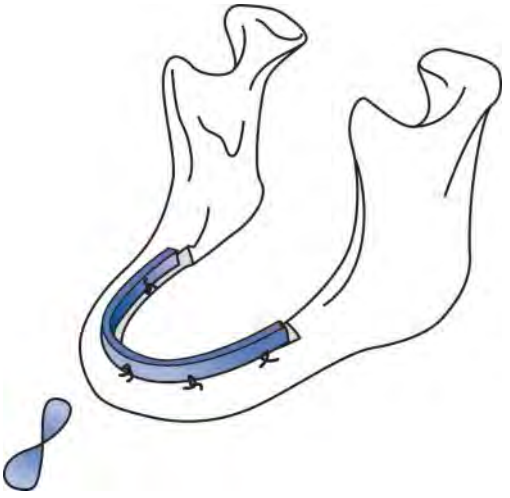


FIGURE 6-32. Visor osteotomy devised by Harle. (Adapted from Stoelinga PJW. Preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Philadelphia: JB Lippincott; 1992; pp. 1169–1207; and Harle F. A follow up investigation of surgical correction of the atrophied alveolar ridge with visor osteotomy. J Maxillofac Surg 1979;7:283–293.)

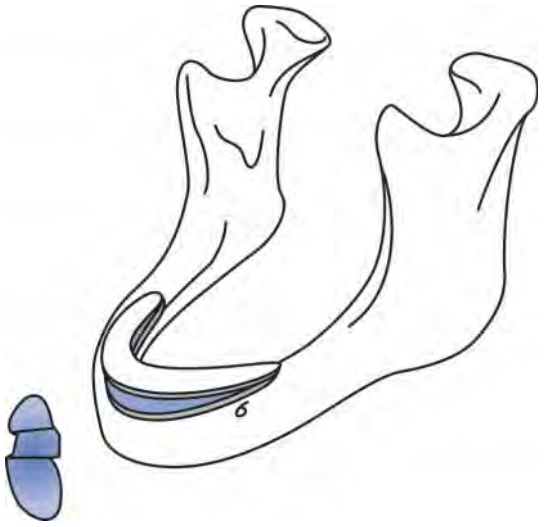


FIGURE 6-33. Sandwich osteotomy designed by Schettler and Holtermann. (Adapted from Stoelinga PJW. Preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Philadelphia: JB Lippincott; 1992; pp. 1169–1207; and Schettler D, Holtermann W. Clinical and experimental results of a sandwich-technique for mandibular alveolar ridge augmentation. J Maxillofac Surg 1977;5:199–202.)

Alveolar Distraction Osteogenesis

As alluded to previously, growing bone via the application of tension or stress has been shown to be a viable solution to defects of the long bone, mandible, and midface. Application to alveolar bone has been limited only by technologic advancement in appliances—the principles are still the same. Alveolar distraction offers some distinct advantages over traditional bone-grafting techniques. No donor site morbidity is involved, and the actual distraction process from the latency period through active distraction and consolidation is actually shorter than phase I and phase II bone remodeling and maturation. The quality of the bone grown in response to this tension/stress application is ideal for implant placement. The vascularity and cellularity of the bone promote osseointegration of dental implants. The greatest successes are related to the achievement of vertical graft stability. One of the

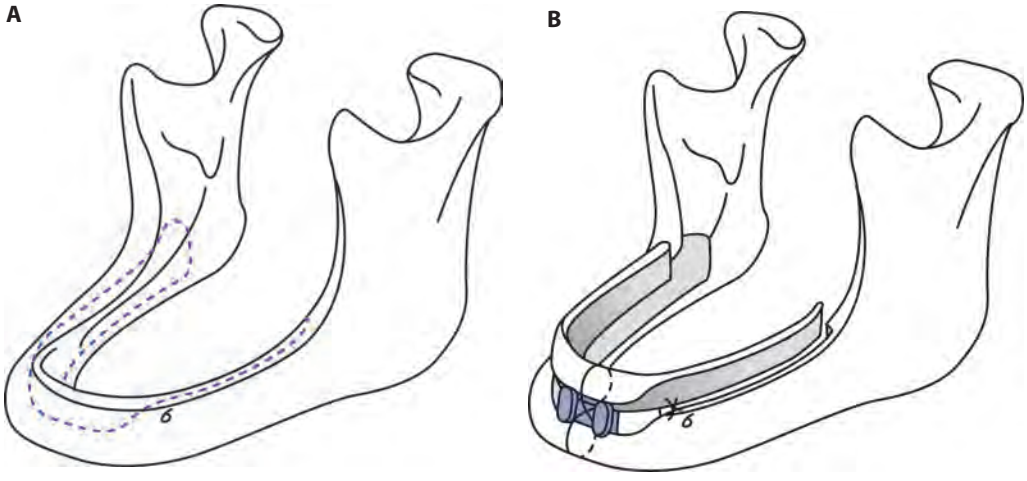


FIGURE 6-34. Sandwich-visor osteotomy according to Stoelinga and Tideman. **A,** Bone cut is outlined *(dotted line)*. **B,** Cranial fragment is lifted, supported by bone struts, and secured by a wire tied in a figure of eight. (**A** and **B,** Adapted from Stoelinga PJW. Preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Philadelphia: JB Lippincott; 1992; pp. 1169–1207; and Schettler D, Holtermann W. Clinical and experimental results of a sandwich-technique for mandibular alveolar ridge augmentation. J Maxillofac Surg 1977;5:199–202.)

biggest problems in alveolar bone grafting historically has been maintaining vertical augmentation of bone graft sites. When distraction is used, the transported alveolar segment does not undergo any significant resorptive process because it maintains its own viability through an intact periosteal blood supply. The intermediate regenerate quickly transforms into immature woven bone and matures through the normal processes of active bone remodeling. The sequencing of events is crucial to maintaining the newly augmented bone and is definitely applicable in cases in which the alternatives are limited.

Diagnosis and treatment planning of a typical case for alveolar distraction osteogenesis involve good clinical and radiographic examinations, primarily using panoramic radiographs. Anatomic structures such as adjacent teeth, the sinus floor, the nasal floor, and the inferior alveolar canal are all easily identifiable in these situations. It is rare that CT or other more sophisticated imaging studies are required. The prosthetic workup for these cases is also important. The ideal placement of the new alveolar crest both vertically and buccolingually determines the success of the distraction. The final position of the alveolus determines the exact alignment of the transport device and how it should be positioned in the bone.

The shape of the residual alveolar bone is also important to identify. Often, vertical bone defects are accompanied by a significant horizontal bone loss. This bone loss must be dealt with either by further reduction of the vertical height to achieve adequate horizontal width or by some type of pre- or postdistraction bone graft augmentation to achieve an adequate width.

Although the success rates with alveolar distraction are very high when cases are properly planned, there are surgical pitfalls to be avoided to ensure that alveolar distraction succeeds. First and foremost is maintenance of the blood supply of the distracted or transported segment. Many times, this is difficult when access to the osteotomies is limited. Although there is no minimum height or width for the transport segment, it should not exceed the distance across which the segment is being transported. Mistakes are often made related to the application of the distraction appliances. In the posterior mandible, the appliances are often inclined too far lingually for implant reconstruction. Similarly, in the anterior maxilla, an adequate labial projection of bone is difficult to achieve unless the appliance is proclined to transport the alveolus inferiorly and labially. In addition, care must be taken when handling soft tissues at alveolar ridge distraction sites. Mucosal flaps maintained with a substantial vascular supply are necessary to achieve predictable wound healing. In addition, we recommend both periosteal and mucosal closure to prevent segmental dehiscence during the distraction process.

Both intraosseous and extraosseous devices have been designed for alveolar distraction. The Lead R System device designed by Chin[72] is a simple one consisting of two bone plates and a distraction rod. A horizontal osteotomy is created, and the distraction rod is inserted from a crestal direction. The transport bone plate is then engaged and positioned on the transport section with a bone screw; the basal bone plate is engaged and likewise supported on the bone with a screw. There are some limitations with this device because the distraction rod may limit its use in areas in which the vertical dimension of occlusion is compromised. The rod is also visible anteriorly, which may be an aesthetic issue. Finally, the rod may interfere with future implant placement unless the implant can be placed directly into the site vacated by the rod.

The Robinson Inter-Oss alveolar device was designed to be used in a one-stage procedure in which the transport appliance actually becomes the implant when the regenerate has matured. Anatomic limitations require a fairly significant crestal bone height and width for use. A similar device, the ACE distraction dental implant system, allows for a distractor that can be placed and then replaced with a dental implant once the distraction has been completed. Again, this is a simple and easy implant to be placed, but anatomic constraints limit its use to certain situations. Both the ACE device and the Robinson Inter-Oss device have limitations in that they must be externally directed or the distraction may veer off course. Other devices available commercially that are similar to those previously discussed are the DISSIS distraction implant and the Veriplant. The Lead device mentioned previously provides relatively rigid stabilization of the transport segment, but these other devices may violate one of the prime requirements of successful alveolar distraction, namely, rigid fixation of the transport segments.

Extraosseous devices are much more successful and practical for distraction and rigid fixation of the segments. The Track Plus System manufactured by KLS Martin and the bone plate device manufactured by Walter Lorenz Surgical are two devices that adhere to the principles of distraction and rigid fixation (Figure 6-35).

After placement of a distraction device, a latency period must be observed, the duration of which is 4 to 7 days, depending on the age of the patient and the quality of tissue at the transport site. The latter is significant in patients who have previously undergone irradiation, multiple surgical procedures, or trauma, resulting in scar tissue and compromised blood supply. The active distraction period varies depending on the distance the segment is transported. Standard principles must be followed. The rate and rhythm of transport is 1 mm/day in divided segments—0.25 mm four times a day is the most practical for appliances as well as the patient. The consolidation phase commences when the distraction is complete. Generally, the consolidation period should be three times the length of the distraction period. The extraosseous appliances provide rigid fixation to promote faster maturation of the regenerated bone. At the conclusion of the consolidation phase, the appliance can be removed. Rather than waiting for full mineralization of the regenerate, one can place the implants, which then provide further rigidity to the transport segment and allow for healing of both the implant and the immature regenerate simultaneously. The total treatment time

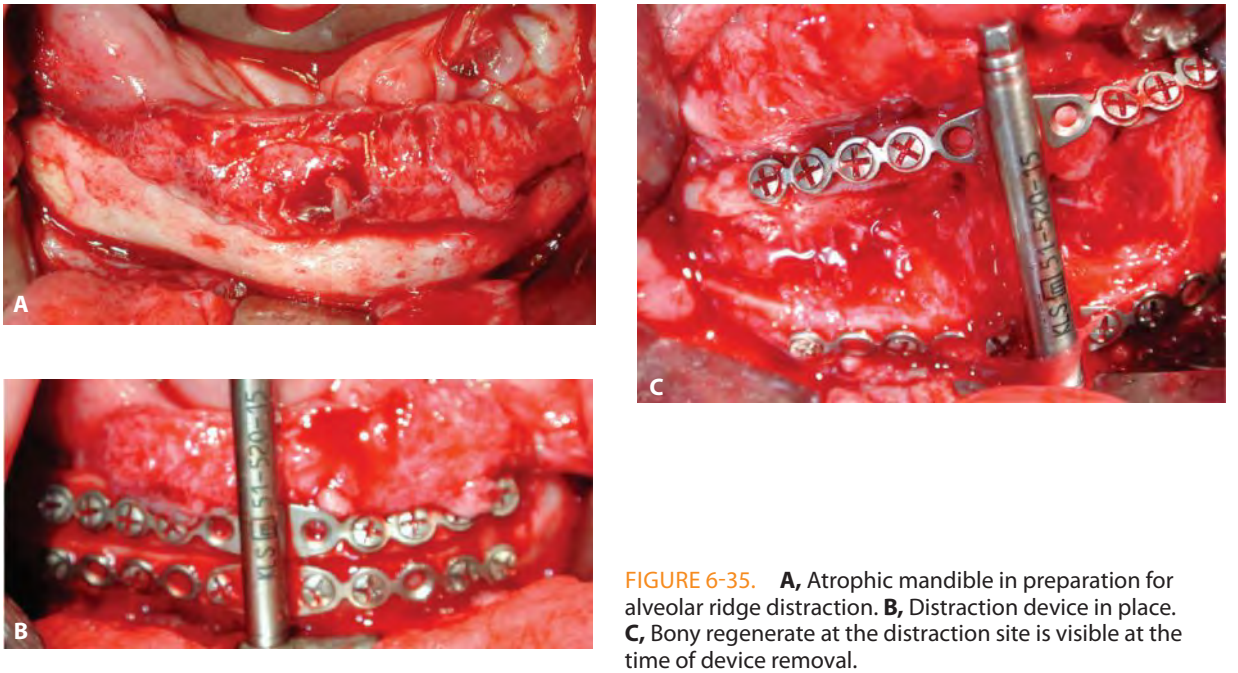


FIGURE 6-35. **A,** Atrophic mandible in preparation for alveolar ridge distraction. **B,** Distraction device in place. **C,** Bony regenerate at the distraction site is visible at the time of device removal.

is thus much shorter than with conventional bone grafting with either autogenous or allogeneic bone, and in most cases, the appliance does not interfere with day-to-day function. Other than the inability to wear a transitional prosthesis, there is minimal disruption of the normal activity and diet. Morbidity is generally minimal and is related strictly to management of soft tissue flaps, maintenance of adequate transport segment blood supply, and proper positioning of osteotomies.

CONCLUSION

With the evolution and success of dental implant technology, guided tissue regeneration, and genetically engineered growth factors such as BMP, current indications for grafting and augmentation are usually related to facilitation of implant placement. Time-honored reconstructive procedures including bone grafting and augmentation are also evolving to create the ideal environment for implant-supported and -stabilized prosthetic reconstruction.

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7

CHAPTER

Pediatric Dentoalveolar Surgery

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INTRODUCTION

Oral and maxillofacial surgeons commonly perform dentoalveolar procedures in adults. In children, the same procedures present different challenges. Behavioral management in pediatric patients is especially important. The surgeon should understand the child's stage of psychological development and must interact with these patients in an age-appropriate manner. To achieve a successful result, an attempt should be made to establish a supportive relationship with the child and parents before the operation. In some cases and for some procedures, conscious sedation or general anesthesia may be necessary if appropriate collaboration with the child is not possible. It is critical to avoid psychological trauma experience that could have an impact on future dental and medical treatment.

There are also age-related anatomic differences between adults and children. For example, in the mixed dentition, special attention must be paid to avoid damaging permanent tooth buds when removing supernumerary and impacted teeth. Sequence and timing of eruption of the permanent dentition must be understood so that interventions can be timed properly to facilitate normal eruption of teeth. Impacted canines, supernumerary teeth, and prominent frena are conditions more commonly seen in children than in adults. In this chapter, we review common dentoalveolar surgery procedures in pediatric patients.

IMPACTED TEETH

An *unerupted tooth* is defined as one that has normal eruption potential but has not yet progressed into the mouth.[1] An *embedded* or *impacted tooth* is one that is unerupted due to physical obstruction or a lack of normal eruptive force.[2] Normal eruption of teeth is essential for development of the maxilla and the mandible as well as the dentoalveolar bone. Missing or impacted teeth may affect the overall facial development.

A tooth may fail to erupt for many reasons, the most common being a lack of space due to underdevelopment of the maxilla and/or mandible.[2] Failure of eruption may also be associated with an overlying cyst or tumor (Figure 7-1), trauma to the tooth bud, mechanical obstruction by supernumerary teeth (Figure 7-2), scar tissue from previous surgery, ankylosis of primary or permanent teeth, habits (e.g., thumb sucking), ectopic position of the tooth bud, congenital deformities (cleft lip and palate, craniosynostosis), systemic diseases (e.g., hypothyroidism, hypopituitarism), or syndromes (e.g., cleidocranial dysostosis, Crouzon's, Apert's, Hurley's, or Down syndrome).[3–7] In many cases, no specific cause of eruption failure is identified.

Impacted deciduous teeth are uncommon. When they do occur, the most frequent tooth is the second primary molar.[2] Overall, mandibular and maxillary third molars are the most commonly impacted permanent teeth followed by maxillary canines, mandibular and maxillary premolars, and second molars.[8,9] Embedded first molars and central incisors are rarely seen in the absence of pathology.[10]

Appropriate treatment of an impacted tooth may include surgical extraction if there is dental caries, damage to adjacent teeth, or periodontal disease. When there is an associated cyst or tumor, extraction is indicated but treatment of the pathologic lesion takes precedence. Longitudinal observation of an impacted tooth is feasible but the patient should be educated on the risk of resorption of adjacent

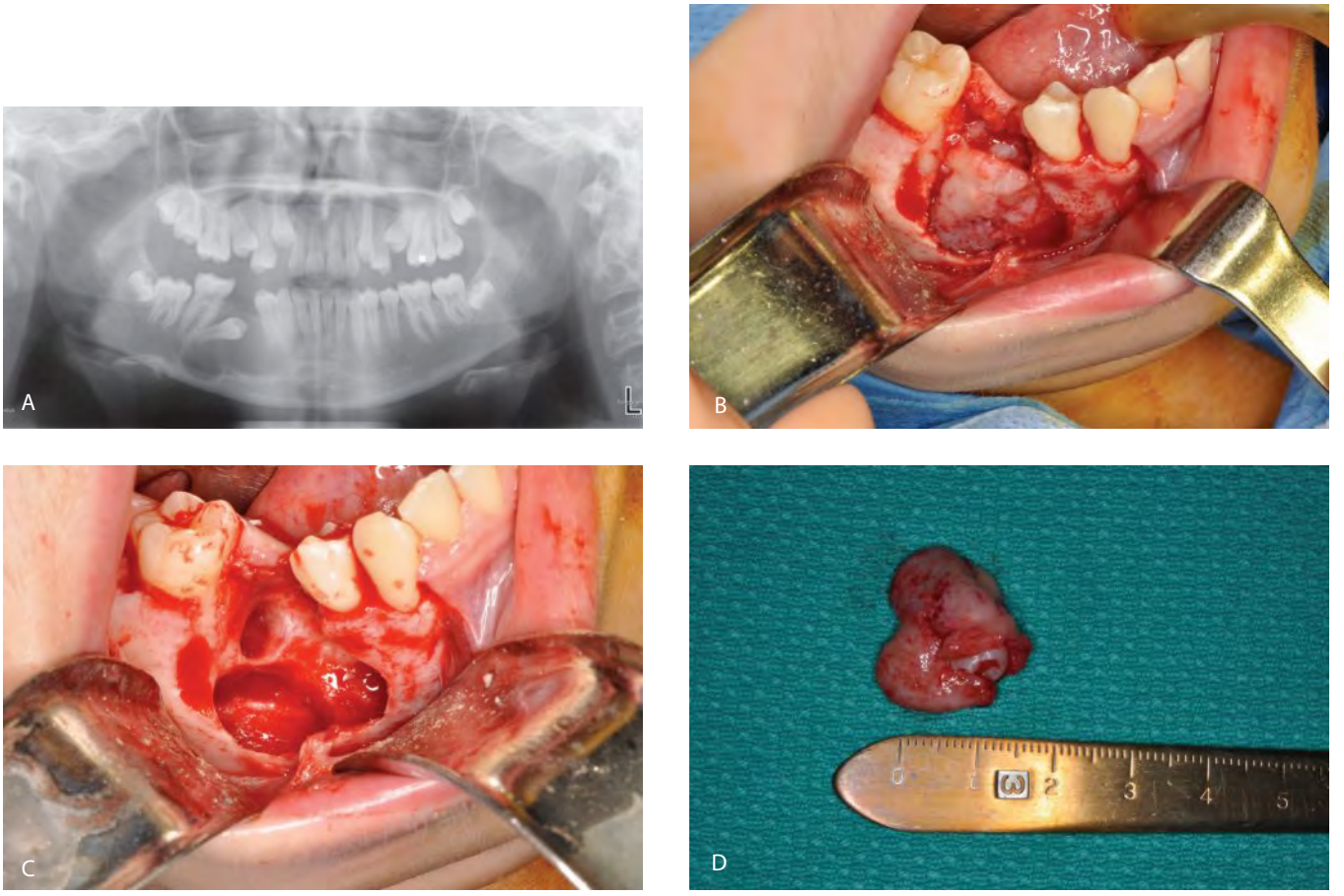


FIGURE 7-1. **A,** The second right mandibular premolar of this 13-year-old female patient failed to erupt due to a keratocystic odontogenic tumor. **B–D,** The cyst and the tooth had to be removed.

roots, development of cysts, tumors, or infection. When possible, and with the exception of wisdom teeth, every effort should be made to expose and orthodontically assist the eruption of impacted teeth.

Impacted Maxillary Canines

Maxillary canines are the most commonly impacted teeth, after third molars, with an incidence between 1.7% and 2.2%.[9] Females are affected twice as often as males. The tooth is located on the palate three times as often as on the labial aspect of the ridge.[11] Permanent canines should be erupted before the age of 14 years in girls and 15 in boys.[12] Crowding as a result of arch length discrepancy is the usual cause of failure of eruption of these teeth.

If recognized early and if the crown of the impacted tooth is not located too far anterior, extraction of the primary canine and creation of enough space orthodontically may allow spontaneous eruption of the impacted canine.[13–15]

When eruption is not anticipated, an impacted canine can be left in place, surgically extracted, exposed, and orthodontically moved to its normal location, or autotransplanted. Although it is usually preferable to preserve an impacted canine, in some situations, removing the unerupted tooth will shorten treatment time and provide an acceptable result (Figure 7-3). For example, if premolars have to be extracted for crowding, a deep, horizontally impacted canine can be removed instead and the premolar modified to look like a canine. Consultation and coordination with the patient's orthodontist is necessary to establish the appropriate treatment plan for each patient.

Maxillary Canine Exposure

The first step is to accurately localize the impacted tooth in three dimensions. Failure to do so will prolong the procedure and may result in inadequate or incorrect flap design and unnecessary soft tissue dissection. In addition, the young patient may be traumatized and his or her cooperation lost.

Visual inspection, manual palpation, and panoramic,[16] periapical, occlusal, and cephalometric radiographs are used alone or in combination to determine the exact position of the tooth. "Clark's rule" ("tube-shift" or "buccal-shift" technique) is also useful in cases in which the tooth cannot be palpated. Two periapical radiographs are taken, each with a different horizontal angulation of the x-ray beam. Impacted teeth on the palatal side of the ridge appear to move in the



FIGURE 7-2. This 25-year-old female patient with cleidocranial dysostosis had complete agenesis of both clavicles **(A)** and multiple impacted supernumerary teeth **(B).** Her permanent teeth failed to erupt due to lack of space and mechanical obstruction **(C).** All teeth were extracted **(D)** and she was reconstructed with dental implants **(E)**. (Courtesy of Dr. Dany Morais)

same direction as the x-ray beam, whereas teeth located labially appear to move in the opposite direction. A tooth located in the middle of the alveolar ridge will maintain its position relative to adjacent teeth on both x-rays.

More recently, cone beam computed tomography (CBCT) has been introduced and it provides three-dimensional visualization of the impacted tooth and adjacent hard tissue[17] (Figure 7-4). Radiation exposure is low when compared with standard multislice computed tomography (CT).[18] Although not indicated in every case, this imaging technology can provide valuable information on the position, inclination, amount of bone covering the tooth, three-dimensional proximity and resorption of adjacent roots, position of anatomic structures (maxillary sinus, nasal floor, inferior alveolar nerve), and dental development of the impacted tooth.[19] Conventional CT scans should be avoided in children for this indication because of the high radiation dose.

Complications encountered with surgical exposure of maxillary canines include infection, paresthesia of infraorbital and nasopalatine nerves, damage to adjacent structures

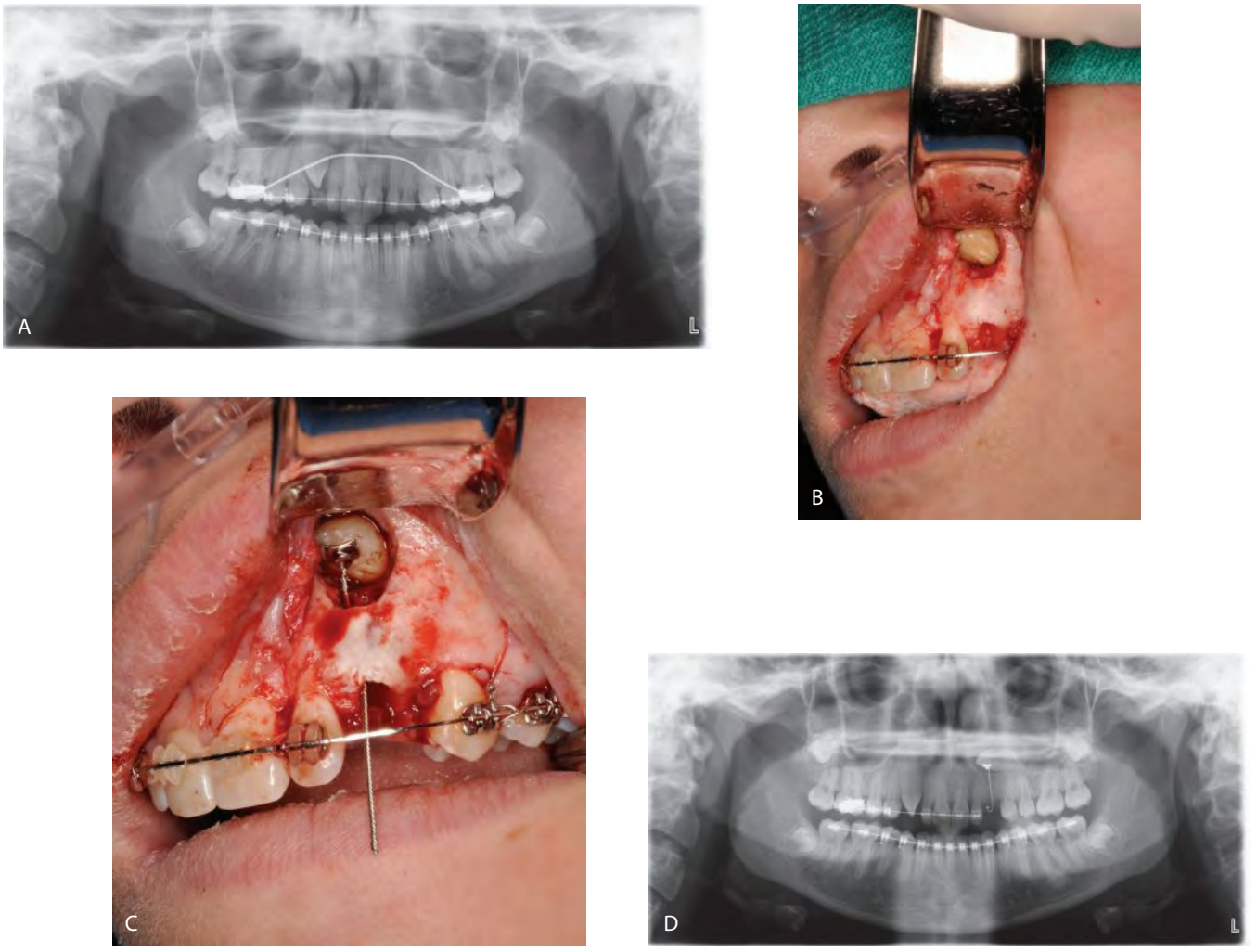


FIGURE 7-3. **A,** This 15-year-old female patient had a horizontally impacted left maxillary canine. **B** and **C,** The tooth was exposed and attempts were made to move this tooth in its normal position. **D,** After 1 year of traction, the position of the tooth did not change significantly. The orthodontic treatment of this patient was prolonged by an inappropriate treatment plan for this impacted canine.

(roots, maxillary sinus, nasal floor), failure of eruption, necrosis of the flap, lack of attached gingiva, or inadequate bone around the exposed tooth.[20]

Palatally Impacted Canines: Technique

A sulcular incision is made on the palatal side of the ridge and a full-thickness mucoperiosteal flap elevated. Releasing incisions are often not necessary and should be avoided. If bone is covering the crown of the impacted canine, it is easily removed with a periosteal elevator or a rongeur. It is sometimes necessary to use a bur if the tooth is covered with a thicker layer of bone. Care should be taken to gently shave the bone until the crown is definitively identified. Once the crown is exposed, the dental follicle is excised using curettes or electrocautery. The latter offers the advantage of keeping the operative field dry, making bonding of the orthodontic bracket, if necessary, easier. Removing bone to the cementoenamel junction is unnecessary and could lead to significant bone loss around the tooth.[21] Mobility of the embedded tooth should be verified with an elevator. If no movement can be elicited and there is a suspicion of ankylosis, the tooth should be luxated gently.

At this point, if the position of the tooth is favorable, excision of the overlying gingiva and bone may be sufficient for the canine to erupt.[20,22,23] If the inclination of the impacted tooth is unfavorable or if the tooth is deep, the crown is acid-etched and an orthodontic button or bracket is attached with a composite type adhesive. A bracket with gold chain is also an excellent choice in this situation. Often the bracket is best placed near the cusp tip. Self-cured or light-cured material can be used to fix the bracket to the tooth, depending on surgeon's preference. The chain is then attached with a wire or elastic power thread to the orthodontic arch wire. Other appliances have been used for traction on unerupted teeth, including a wire around the cervical margin, pins inserted in the enamel, and polycarbonate crowns. These devices are no longer recommended because of their potential complications and because of the efficacy of etched brackets and chains.

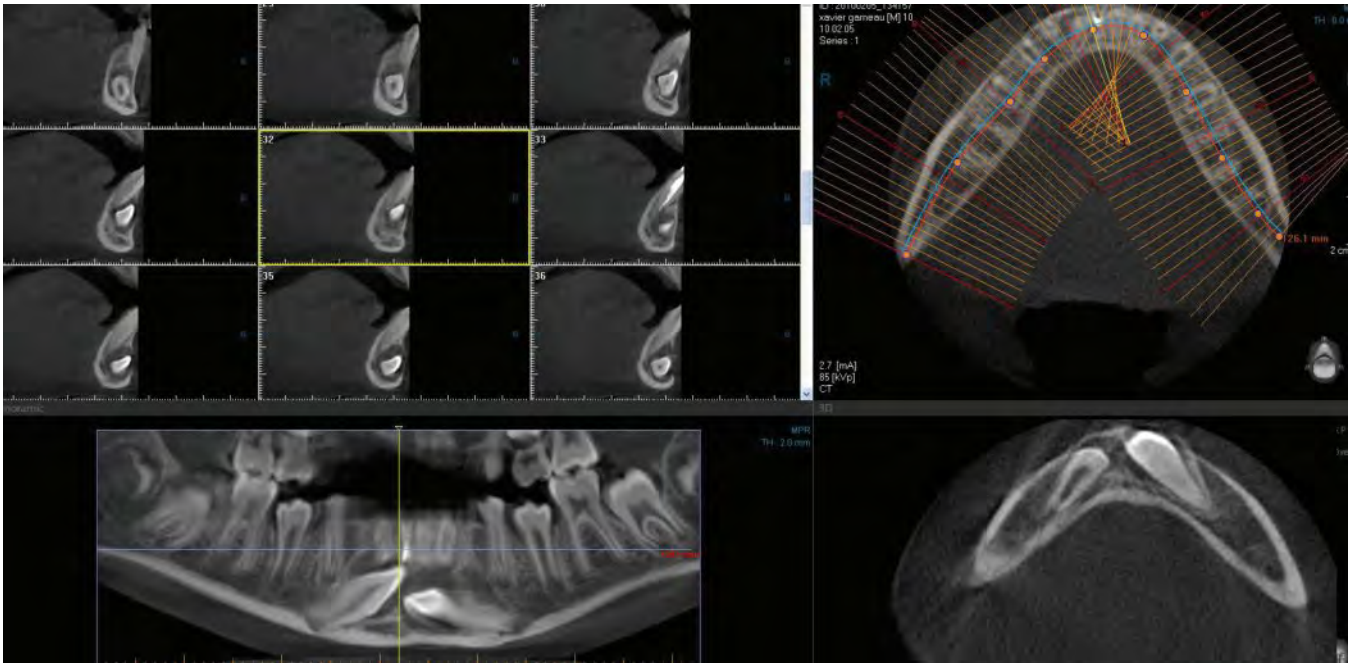


FIGURE 7-4. A cone-beam computed tomography (CBCT) was used to help localize precisely two impacted mandibular canines on this 10-year-old boy.

The flap is closed with resorbable sutures over the orthodontic bracket. The chain or wire is fed through the incision (Figure 7-5) or a window can be created through the palatal flap to expose the entire bracket and chain.[20] The latter technique gives access to the tooth if the bracket fails and needs to be rebonded and allows the orthodontist more freedom to adequately apply the correct vector to the tooth. The direction of eruption should be palatal and away from the root of the lateral incisor to prevent bone and root resorption. When the crown of the impacted tooth has erupted through the palatal gingiva, it then can be moved toward the alveolar ridge.[23] This technique has been shown to decrease periodontal problems between the lateral incisor and the canine.[24]

Labially Impacted Canines: Technique

The general principles for exposure of a labially impacted canine are similar to those described for palatally impacted teeth. However, specific attention must be paid to preserving the attached gingiva overlying the canine. Removal of keratinized tissue with electrocautery must be avoided. An apically repositioned flap including the keratinized attached gingival tissue is recommended.[3,25,26] This technique minimizes the risk of developing periodontal problems once the tooth is in position.[20] An alternative procedure is a closed eruption technique, in which the soft tissue is repositioned over the tooth and bracket. However, this could lead to increased treatment time, diminished control of orthodontic tooth movement, and need for further surgical procedures (uncovering, peridontal surgery).[27]

An incision is made on the alveolar crest with vertical releasing incisions on each side, leaving sufficient flap width to accommodate the canine in its erupted position. If a primary tooth is present, it should be extracted. The full-thickness flap is elevated and bone is removed to expose the crown of the impacted canine. Again, the dental follicle is removed and an orthodontic bracket bonded to the crown of the tooth. The inferior aspect of the flap is sutured apically at the cervical margin of the canine (Figure 7-6). A periodontal dressing can be applied for a few days for patient comfort.

Maxillary Canine Autotransplantation

Impacted canines that cannot be orthodontically moved into position after exposure, because of depth of impaction and orientation, can be autotransplanted. This is usually the case for canines with an angulation greater than 45 degrees to the vertical or that are close to the midline.[20]

The tooth is extracted atraumatically and moved to its desired position after a socket is prepared. The maxillary arch should be fully banded and the transplanted tooth stabilized to the arch wire for a period of 2 to 3 weeks. Both Moss[28] and Pogrel[29] have reported a success rate of approximately 70% with this procedure. Endodontic treatment with calcium hydroxide may be necessary if external root resorption develops. This will usually occur within the first 2 years.

Impacted Mandibular and Maxillary Premolars

Impacted premolars are more common in the mandible than in the maxilla. In the mandible, the second premolar is the second most common impaction after third molars and the tooth is usually located on the lingual side or in the middle of the alveolar ridge.[1,21] In the maxilla, premolars are most commonly found on the palate, similar to what is seen for impacted canines.[3]

Space should be created for eruption of an impacted premolar by extracting the second primary molar, if present,







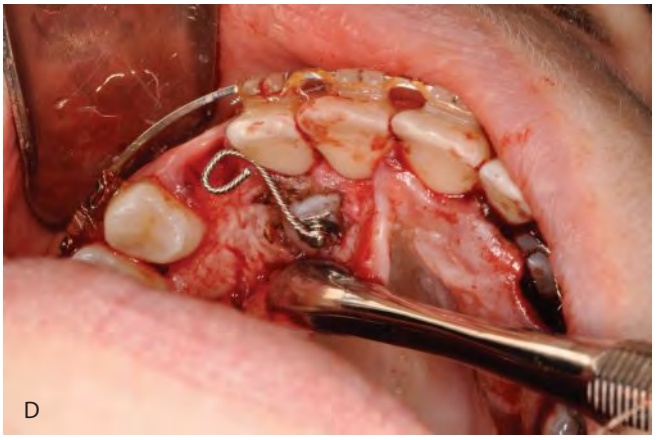






FIGURE 7-5. Preoperative intraoral picture **(A)** and panoramic x-ray **(B)** of a 16-year-old female patient with two retained maxillary deciduous canines and two palatally impacted permanent canines. The left **(C)** and right **(D)** canines were exposed by a palatal envelope flap and orthodontic buttons bonded to the enamel. Stainless steel wires were connected to the buttons to allow orthodontic traction of the impacted teeth. **E** and **F,** Postoperative intraoral pictures after eruption of the canines.

and/or by orthodontic tooth movement as necessary.[21] If the tooth does not spontaneously erupt, it should be exposed and the orthodontist should connect it to the arch wire and move it into position.

Impacted Premolars Exposure: Technique

Impacted mandibular premolars are located by inspection, manual palpation, and radiographic evaluation. Occlusal radiographs are very helpful to identify the position of the tooth. If the tooth is located on the lingual side, it is exposed by a sulcular incision on the lingual aspect of the ridge. If located on the buccal side or in the middle of the alveolar ridge, an apically repositioned flap may be necessary if the amount of keratinized tissue is an issue (Figure 7-7). Otherwise, a sulcular incision is performed and an envelope flap is elevated. The crown of the tooth is exposed, the dental folli-

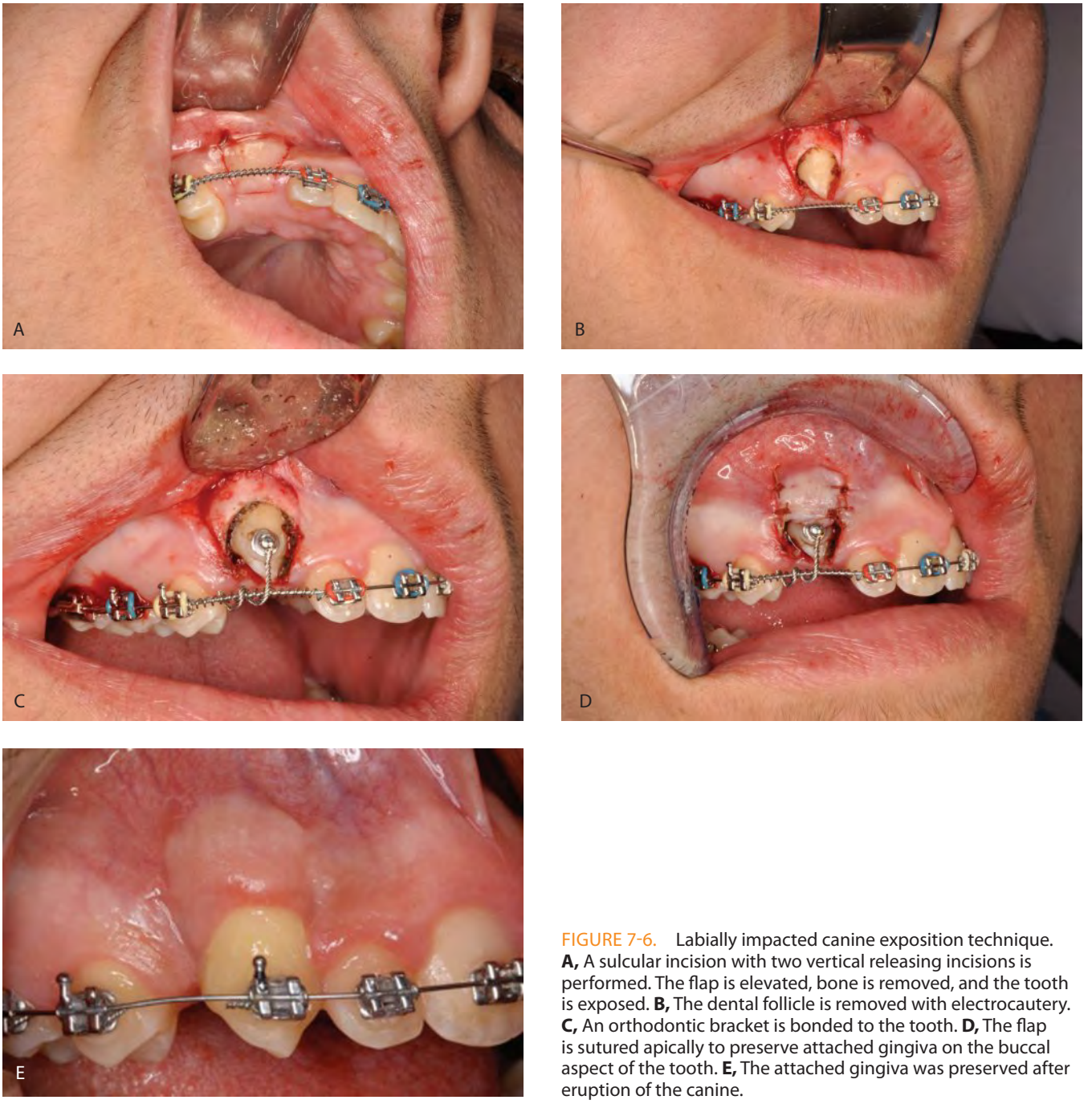


FIGURE 7-6. Labially impacted canine exposition technique. **A,** A sulcular incision with two vertical releasing incisions is performed. The flap is elevated, bone is removed, and the tooth is exposed. **B,** The dental follicle is removed with electrocautery. **C,** An orthodontic bracket is bonded to the tooth. **D,** The flap is sutured apically to preserve attached gingiva on the buccal aspect of the tooth. **E,** The attached gingiva was preserved after eruption of the canine.

cle is removed, and an orthodontic bracket is secured to the tooth with adhesive. Care must be taken to avoid damage to the mental nerve when elevating a flap in this area.

Impacted maxillary premolars are exposed by the same technique as described for impacted canines. Again, an apically repositioned flap should be performed if the tooth is positioned labially.

Impacted Maxillary Incisors

The incidence of impacted maxillary incisors is lower than that of impacted canines and premolars.[30] In tertiary hospital settings, where trauma to the anterior maxilla is frequently encountered, the incidence of impacted incisors may be higher than reported.[3] Primary incisors that are avulsed, intruded, loosened, or lost owing to infection will affect the alveolus and follicle of the permanent incisors, resulting in higher rate of impaction. Lack of eruption can also be a consequence of hypertrophic and fibrotic gingival tissue overlying the alveolus of the anterior maxilla. This is usually secondary to premature loss (before the age of 5) of primary central incisors. Mesiodens or impacted supernumerary incisors may also cause delayed or failure of eruption of maxillary incisors. These teeth should be removed

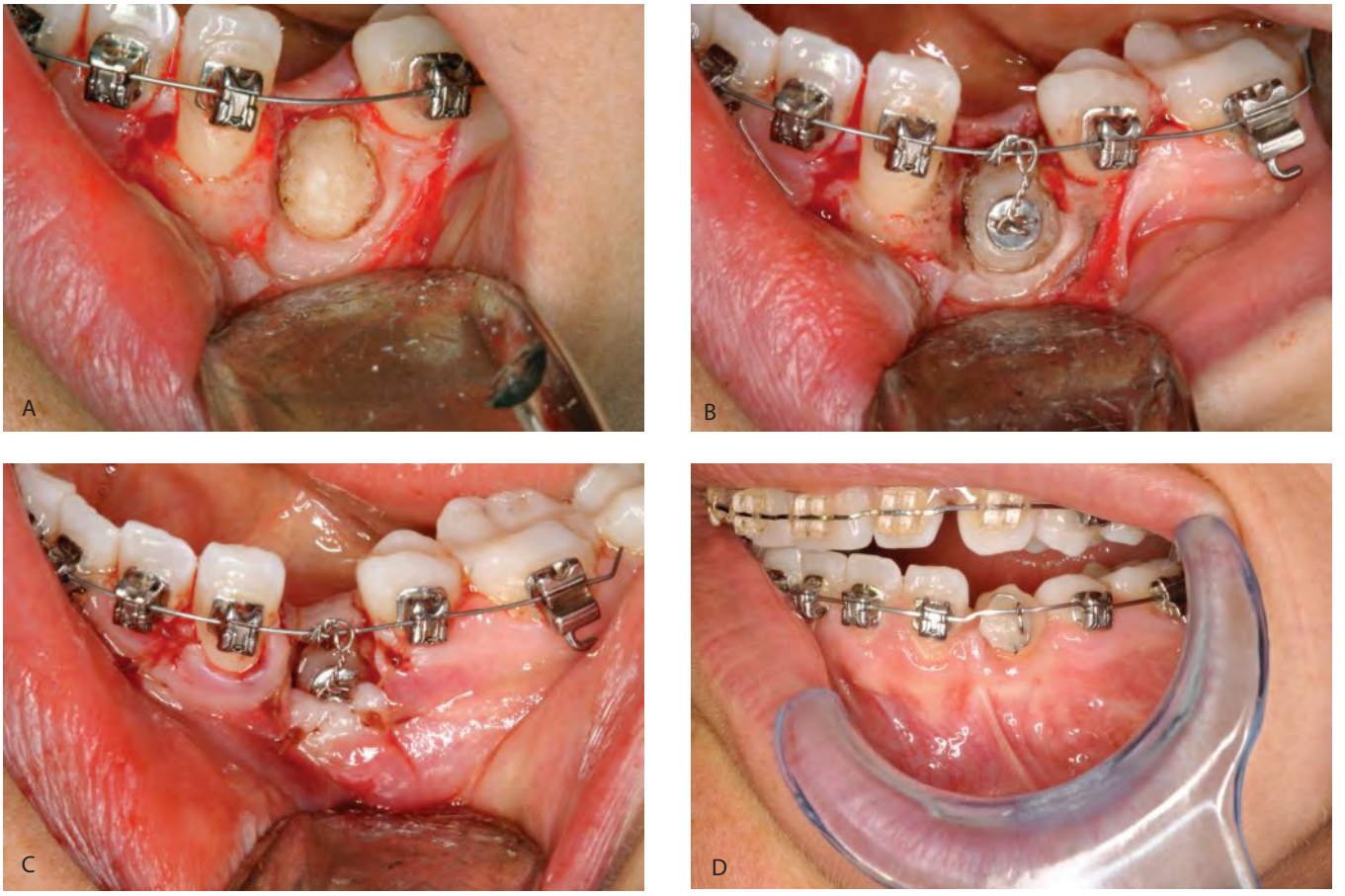


FIGURE 7-7 Labially impacted mandibular premolar technique. A sulcular incision with two vertical releasing incisions is performed, taking care to avoid the mental nerve. **A,** The flap is elevated, bone is removed and the tooth is exposed. An orthodontic bracket is bonded to the tooth **(B)** and the flap is sutured apically **(C). D,** Note the band of attached gingiva after eruption of the tooth.

when the roots of the permanent incisors are one half to two thirds developed to facilitate their identification and to prevent damage to developing permanent teeth during the procedure.

Maxillary Incisors Exposure: Technique

If the root of an impacted incisor is completely formed or if the apex is closed, there is little chance of spontaneous eruption. The tooth is first located by inspection, manual

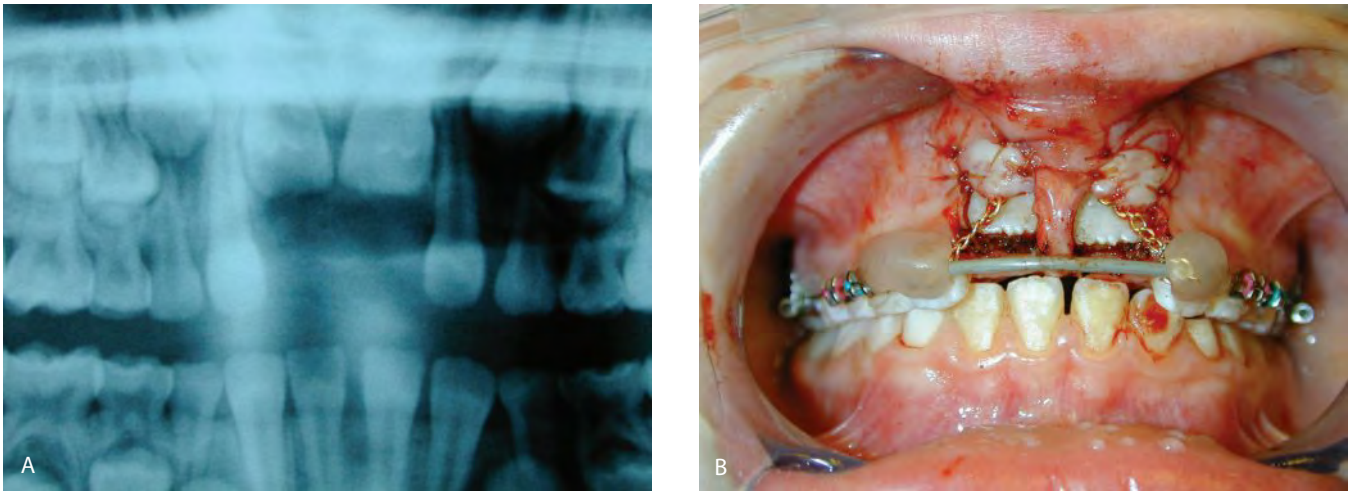


FIGURE 7-8. **A,** Preoperative radiograph of a 10-year-old female patient with impacted central incisors. **B,** Both teeth were exposed using an apically repositioned flap. *(Continued)*







FIGURE 7-8. *(Continued)* Intraoral pictures 1 month **(C),** 1 year **(D),** and 7 years **(E)** postoperatively.

palpation and radiographs as described previously. These teeth are commonly on the labial aspect of the ridge. A crestal incision, with vertical limbs at the mesial and distal margins of the incisor crown, is made. The full-thickness flap is elevated and the crown of the tooth uncovered. Remnants of the dental follicle are excised with electrocautery. A bracket is bonded to the tooth if no spontaneous eruption is anticipated. The flap is apically repositioned to ensure adequate attached gingiva as the tooth erupts (Figure 7-8). If the tooth is located in the middle of the ridge, a sulcular incision is used, a flap is elevated, and the follicle is removed by electrocautery. A button or bracket is placed on the tooth and connected to the orthodontic arch wire. The flap is reapproximated and sutured. The tooth is orthodontically moved into the arch.

If the incisor root is not completely formed, a path for eruption can be created by removing hypertrophic gingival tissue overlying the tooth, impacted supernumeraries, or other pathology. This will often allow spontaneous eruption. If the tooth does not erupt, it should be exposed, bracketed, and orthodontically moved into position as described previously.

Impacted First and Second Molars

Although the incidence of impacted first and second molars is low, this can result in significant periodontal and occlusal problems for adjacent and opposing teeth.[31] Mandibular first and second molars are more frequently impacted than those in the maxilla. As with any impacted teeth, it is better to make the diagnosis and treat the problem before the roots are fully developed.[32] This allows for spontaneous eruption.

As the roots develop, they may become deformed and delacerated along the inferior border of the mandible. If this occurs, it is almost impossible to get the impacted molar to erupt spontaneously.[25] Impacted second molars, in many cases, are a result of inadequate space and anterior migration of the third molar tooth bud. Treatment consists of removing the third molar prior to complete root development of the 12-year molar. This will provide enough space for the 12-year molar to upright into a normal position. For teeth with incomplete root development and before closure of root apices, Kaban and coworkers[31] obtained satisfactory result by surgical exposure and gentle luxation and extraction of the impacted third molar as appropriate.

Impacted Molars Exposure: Technique

A flap is elevated and bone is removed to expose the crown of the impacted molar. The dental follicle is excised using electrocautery. If there is a space problem, the impacted third molar is removed. The 12-year molar is luxated gently. If

necessary, an orthodontic bracket for traction is bonded to the tooth and connected to the arch wire with a chain or wire. If the second molar is mesially inclined and is locked below the first molar, it may need to be elevated and stabilized next to the marginal ridge.

SUPERNUMERARY TEETH

An increase in the number of teeth is a defined as *hyperdontia*. The additional teeth are termed *supernumerary*.[2] A possible explanation for an increased number of teeth is the development of excessive dental lamina, which in turn leads to the formation of additional tooth buds.[2,33] Syndromes associated with hyperdontia include cleidocranial dysplasia (see Figure 7-2) and Apert's, Crouzon's, Down, Gardner's, Ehlers-Danlos, and Sturge-Weber syndromes.[2]

In most cases, only one supernumerary tooth is present. The anterior maxilla is the site of predilection.[34] A supernumerary tooth in this area is called a *mesiodens* (Figure 7-9).[2] The next most common supernumerary tooth is a fourth maxillary molar (distomolar if located posterior to the last molar; paramolar if located medially or laterally of other molars), followed by a fourth mandibular molar and mandibular premolars.[34] Most supernumerary teeth fail to erupt and remain impacted.

A supernumerary tooth in the anterior maxilla should be addressed early because it can prevent normal eruption of permanent central incisors. They are usually extracted when one half to two thirds of the roots of the central incisors are formed. This makes it easier to identify the roots of the normal incisors and to identify the abnormal tooth.[3] The supernumerary is located by manual palpation and plain radiographs. A CBCT scan is also a valuable tool to locate the abnormal tooth if plain radiographs do not suffice.

Fourth molars are usually extracted at the same time as third molars, unless they are too deep. In these cases, they are left in place and extracted when they take the place of the extracted third molar.[3] Supernumerary premolars are removed when the permanent premolars exhibit one-half to two-thirds root development.

Teeth can also be present in the newborn (natal teeth) or appear shortly after birth (neonatal teeth).[35] Most of these teeth represent deciduous mandibular incisors. If they are supernumerary teeth, they are usually mobile and associated with eruption cysts and can be removed.

HYPODONTIA

The absence of one or multiple teeth is one of the most common developmental anomalies of the dentition.[2] Many









FIGURE 7-9. Intraoral **(A)** and panoramic **(B)** x-ray of a 5-year-old boy with two supernumerary teeth *(arrows)* in the anterior maxilla (mesiodens). **C** and **D,** Both teeth were extracted to allow normal eruption of permanent central incisors.

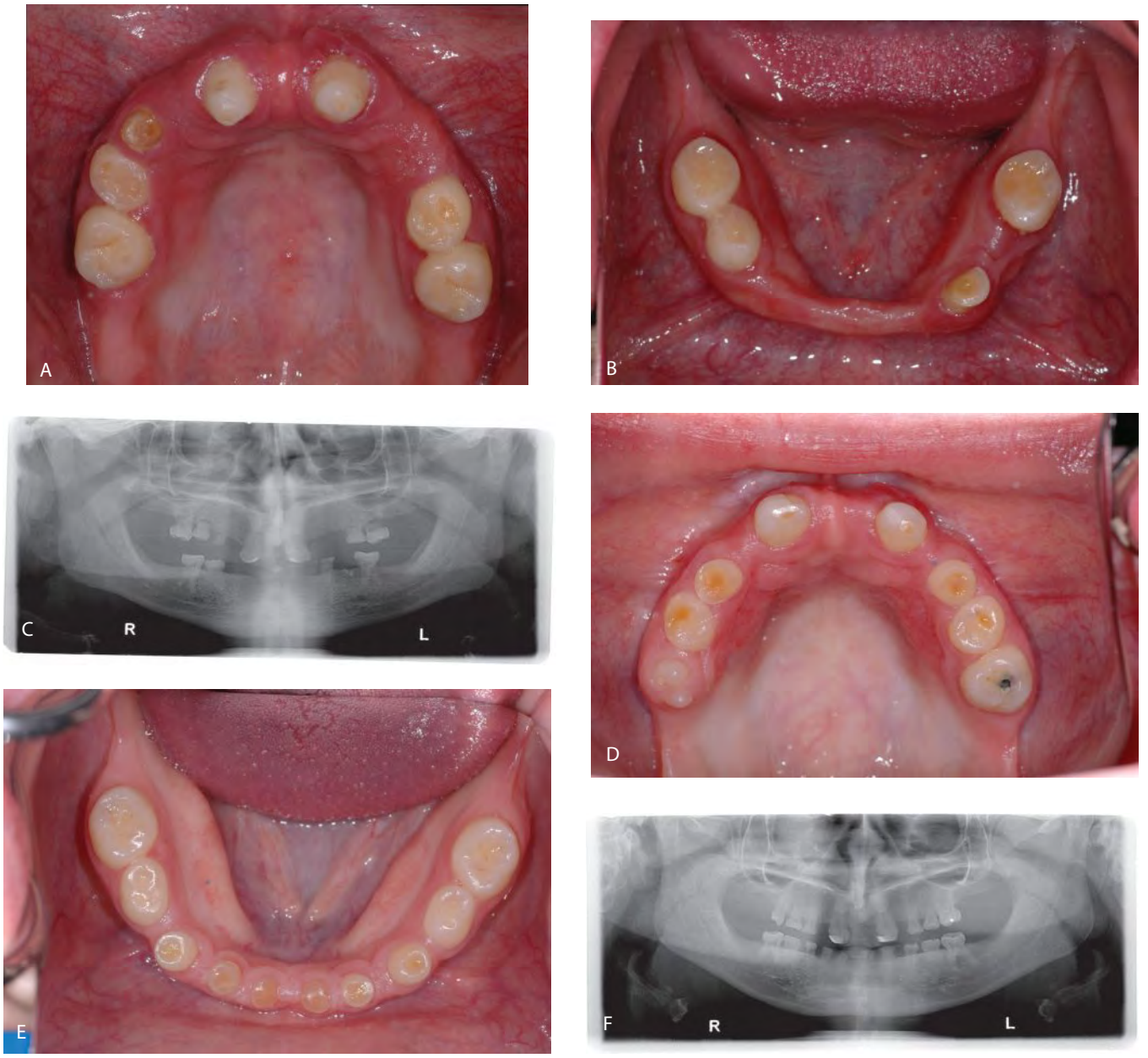


FIGURE 7-10. Intraoral and panoramic radiographs of two siblings aged 21 **(A–C)** and 19 **(D–F)** with ectodermal dysplasia. Both had agenesis of all permanent teeth.

syndromes have been associated with a decrease in the number of teeth, one of the most common being ectodermal dysplasia (Figure 7-10). These patients occasionally present with the absence of all teeth, a condition called *anodontia*.

The most common missing teeth are third molars, followed by first and second molars, second premolars, and maxillary lateral incisors.[2] Patients with complete cleft lip and palate have an increased incidence of a missing lateral incisor on the cleft side. The absence of teeth has an adverse effect on the development and growth of the alveolus. If a deciduous tooth is present, it should be retained until such time as the missing permanent tooth is replaced with a dental implant or the space is closed orthodontically.

TRANSPLANTATION OF TEETH

With the increased popularity and long-term success of dental implants, autologous tooth transplantation is rarely performed. However, it remains a useful technique for replacing a missing tooth at low cost in some situations. Allogenic tooth transplants, conversely, ultimately fail because the antigenic properties of the periodontal ligament cause a local immune response. Rejection is manifested by external and internal root resorption.

Autotransplantation of impacted canines and transplantation of third molars to replace a first or a second molar are the most commonly performed procedures. Canine transplantation was discussed earlier in this chapter.

A third molar can be transferred to replace a first or a second molar congenitally missing or lost due to dental caries. The transplanted tooth should be free of infection and the root should be at least 50%, ideally, two thirds developed.[36] If the donor tooth is extracted atraumatically and if the periodontal ligament is preserved, the overall success rate is high. Pogrel[29] obtained a success rate of 72% in a series of 400 transplanted teeth. The success of the procedure is inversely related to the amount of time between extraction of the donor tooth and transplantation and directly related to the fit of the transplant in the recipient socket. Initial stability of the transplant is also crucial. Some authors recommend prophylactic endodontic treatment with calcium hydroxide a few weeks after the procedure.[37] In most cases, endodontic treatment is performed only if external or internal root resorption is noted. Complications of autotransplantation include ankylosis, resorption, and development of periapical pathology.

Molar Tooth Transplantation: Technique

The recipient site should be free of pathology and infection. The donor third molar is extracted with care to prevent fracture of the roots and damage to the periodontal ligament. The tooth is inserted in the recipient socket and forced in place below the occlusal plane. Septal and periapical bone may have to be removed for a better fit. It is important to avoid occlusal interferences during the healing process.[38] Gingival tissues are sutured in place around the tooth. A periodontal dressing or an orthodontic wire can be used to maintain the tooth in place. Prophylactic antibiotics are recommended for 7 days. The tooth should erupt spontaneously and level with the occlusal plane.

DENTAL IMPLANTS IN THE GROWING CHILD

Dental implants are considered the standard of care to replace missing teeth in the adult. They are widely used and the success rate is high.[39–41] The use of dental implants in the growing child presents specific challenges related to the vertical growth of the alveolus. There is no periodontal membrane around an implant, and therefore, the implant does not stimulate bone formation by transmission of occlusal forces to alveolar bone.[42–45] Rather, similar to an ankylosed tooth, the implant becomes progressively submerged as the adjacent alveolus grows in height. (Figure 7-11).[46] In addition, because transverse growth of the maxilla occurs in part by bone apposition on the lateral aspect of the alveolus, an implant will take a more palatal position as the maxilla develops.[47] Fixed prosthesis crossing the midline should be avoided because this will have a negative impact on transverse growth at the midpalatine suture.[42]

Mandibular growth occurs both at the condyle and by apposition of bone on the posterior aspect of the ramus with resorption on the anterior surface.[48] Growth of the alveolar bone follows principles similar to what is observed in the maxilla, resulting in progressive lingual and inferior displacement of dental implants placed in growing children. Implants placed in the mandible should not be connected







FIGURE 7-11. Intraoral **(A** and **B)** and periapical **(C)** radiographs of a 19-year-old female who had tooth no. 9 replaced by a dental implant at the age of 13, before the end of growth of her maxilla. As a consequence, the implant appears too far apically in relation to adjacent teeth and the final restoration is not esthetically acceptable.

across the midline with a fixed prosthesis because this will affect the transverse growth of the mandible.[42]

Implants that are located too far apically and palatally/lingually are difficult to restore and the root-to-crown ratio may be affected negatively. The gingival margin around submerged implants will be located apically, resulting in poor esthetics of the final restoration.

Despite these concerns, there are some indications for implant placement in children. In ectodermal dysplasia, where the growth pattern is abnormal, dental implants offer several advantages over a removable prosthesis.[49] Replacement of teeth with dental implants in a reconstructed segment after tumor resection is another indication for implantation in children. No growth is expected in this area, so a prosthesis can be fabricated and modified over time as needed to prevent supraeruption of teeth.[3] Placement of implants and functional loading preserve the grafted bone.

PROMINENT MAXILLARY LABIAL FRENUM

It is common for pediatricians and parents to be concerned when there is a prominent labial frenum because it is sometimes accompanied by a diastema between the primary and the permanent central incisors. Labial frena are often more prominent in children and appear to decrease with dentoalveolar vertical growth. A labial frenum contributes to the persistence of a diastema if it crosses the alveolar ridge and is attached on the incisive papilla.[50] The papilla blanches when the lip is stretched (Figure 7-12). Other causes of diastema between maxillary incisors include thumb sucking, tongue thrusting, missing or impacted teeth, atypical swallowing pattern, bony pathology between incisors, midline bony cleft, and notching of alveolar bone between incisors.[50–53]

In infants, a prominent labial frenum inserted on the alveolar crest is not abnormal. With vertical growth of the maxilla, the frenum becomes positioned superiorly and eventually assumes a normal position in most children.[51] A frenectomy should not be performed before eruption of the permanent canines is under way, because as these teeth move inferiorly and anteriorly, the diastema will close in most cases.[50] Excising the frenum before the eruption of the six maxillary anterior teeth may create scar tissue between incisors, making closure of the diastema difficult, even with orthodontic forces.

A frenectomy may also be indicated to prevent periodontal problems, but in most cases, it is removed to aid in closure of a diastema or for esthetic and psychosocial reasons.[54]



FIGURE 7-12. This 24-year-old female patient had a prominent maxillary labial frenum attached to the incisive papilla. Note the blanching of the papilla when the lip is stretched. Because the frenum was not excised in time, she developed periodontal problems between the two maxillary incisors as demonstrated by the gingival recession.

Techniques

Standard Frenectomy

Histologically, the frenum is composed of a significant amount of elastic fibers. These fibers are thought to contribute to the development of the diastema.[51] Therefore, when performing a frenectomy, it is crucial to remove this fibroelastic band to prevent recurrence.

An incision is made on each side of the frenum, from its attachment on the incisive papilla to the labial mucosa. On the alveolus, the incision is extended down to the periosteum. The frenum is removed, resulting in an elliptical defect. The mucosa is undermined and releasing incisions can be made along the mucogingival junction on each side to facilitate closure. The labial flap is closed by medially transposing both edges to the midline. The resultant soft tissue defect below the mucogingival line heals by secondary intention (Figure 7-13). Prophylactic antibiotics are not necessary. Patients are instructed to maintain good hygiene in the area, and they are placed on a soft diet for a few days.

Laser Frenectomy

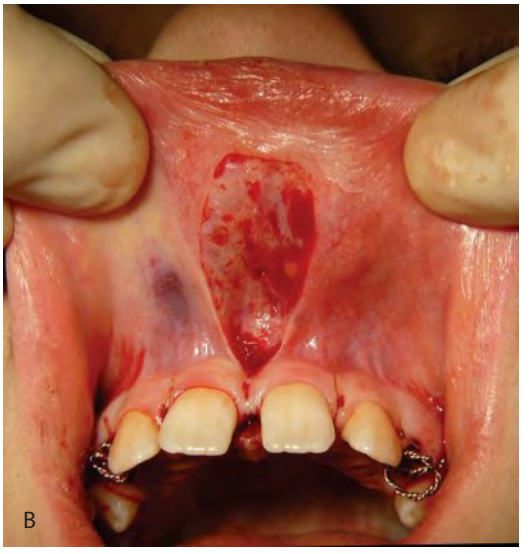
A laser frenectomy is a shorter operation than standard frenectomy and produces less swelling and postoperative discomfort. However, patient cooperation is necessary to prevent laser injury to surrounding structures. The loud suction and smoke may make this difficult in younger children and general anesthesia may be required.

The frenum is placed under tension by pulling on the upper lip. Then, a carbon dioxide laser is used to excise the frenum to the periosteum. No sutures are necessary and postoperative instructions are the same as with a standard open approach.

Mandibular Labial Frenum

A high labial frenum between lower central incisors can cause periodontal inflammation, gingival recession, and eventually bone loss if not treated. This is a result of the pulling action of the frenum on the interdental papilla. There is trapping of food, plaque accumulation, inflammation, and loss of attached gingiva. If periodontal problems persist even with good oral hygiene and intermittent scaling and root planning, a frenectomy is indicated.





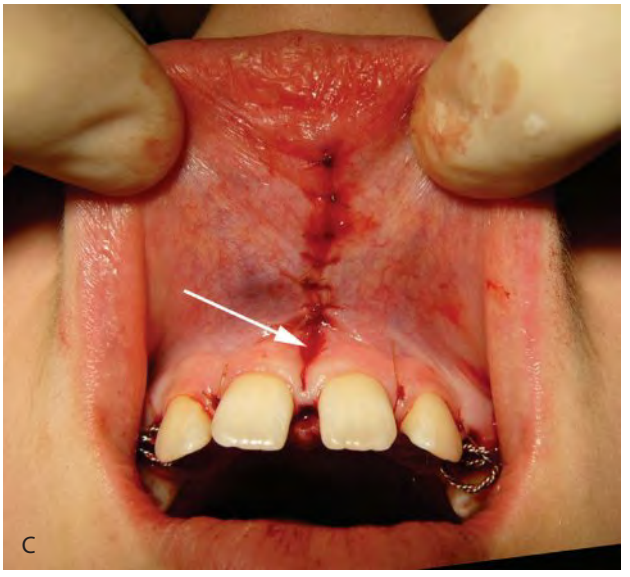


FIGURE 7-13. **A,** A frenectomy was performed on this 12-year-old male because the maxillary labial frenum was inserted on the incisive papilla. **B,** The frenum was completely excised with an elliptical incision. **C,** Soft tissue below the mucogingival junction was left to heal by secondary intention *(arrow)*. (Courtesy of Dr. Robert Paquin)

Techniques

Standard Technique

This technique is the same as the one described for a maxillary labial frenum. The frenum is excised elliptically and the labial mucosa reapproximated after undermining and advancing tissues on each side. The keratinized tissue on the superior aspect is allowed to heal by secondary intention, maintaining the width of the attached gingival.[55] A gingival graft is rarely necessary in pediatric patients.[50]

Z-Plasty Technique

The frenum is excised and the resultant defect is closed using a Z-plasty design (Figure 7-14). A Z-plasty is composed of three limbs of equal length. The vertical limb in this case is created by the excision of the frenum. Two limbs are drawn at 60 degrees from the vertical limb. The two triangles created are undermined, rotated, and translated toward each other. This changes the direction of the scar and lengthen the lip.[56] A gain in length of approximately 75% can be expected with 60-degree angles.[56]

Laser Frenectomy

This technique is identical as the one described for the laser maxillary labial frenectomy.

Lingual Frenum

A high lingual frenum, also called a tongue-tie, is attached on the tip of the tongue and on the lingual alveolar ridge.[50] If the frenum is short, tongue motion, particularly protrusion and elevation, is limited. Oral surgeons are often asked by parents and pediatricians to remove the frenum because of the fear of speech impairment. This often occurs if the frenum is very short and if the tongue exhibits decreased movement and particularly clefting when the child attempts to protrude or elevate it. In many cases, speech articulation develops



FIGURE 7-14. **A,** Preoperative intraoral picture of a high mandibular labial frenum. The Z-plasty incision is drawn **(B)** and the end result after closure is demonstrated **(C). D,** One year postoperative picture. **(A–D,** From Kaban LB. Dentoalveolar surgery. In Kaban LB, Troulis MJ, editors. Pediatric Oral and Maxillofacial Surgery. New York: Elsevier; 2004; pp. 150–151. With permission.)

normally and a frenectomy is not necessary. Consultation from a speech pathologist should be obtained.

A lingual frenum that extends high on the alveolar ridge may produce a diastema and periodontal problems related to the mandibular central incisors. Simply cutting through the frenum under these circumstances will not cure the problem and may produce a linear scar that may limit mobility of the tongue. Therefore, the frenum should be excised and a V-Y or Z-plasty closure should be carried out to increase the length of the ventral surface of the tongue. A laser frenectomy can also be performed, as described previously.

V-Y Closure Technique

Because this procedure requires more soft tissue manipulation, it should be performed under conscious sedation or general anesthesia. The frenum is excised in an elliptical fashion after it is released from the alveolar ridge. A relaxing incision is made on each side at the junction of the ventral aspect of the tongue and the floor of the mouth. The straight line created by the initial incision is converted into a V incision. The flaps are undermined and advanced medially, closing the incision as a Y, increasing the length of the ventral surface of the tongue (Figure 7-15).

Z-Plasty Technique

The Z-plasty technique will increase the length of the ventral surface of the tongue. The basic principles of this flap were described previously (Figure 7-16). The lower portion of the incision on the lingual aspect of the alveolar ridge is allowed to heal by secondary intention to prevent damage to the submandibular duct (Figure 7-17).

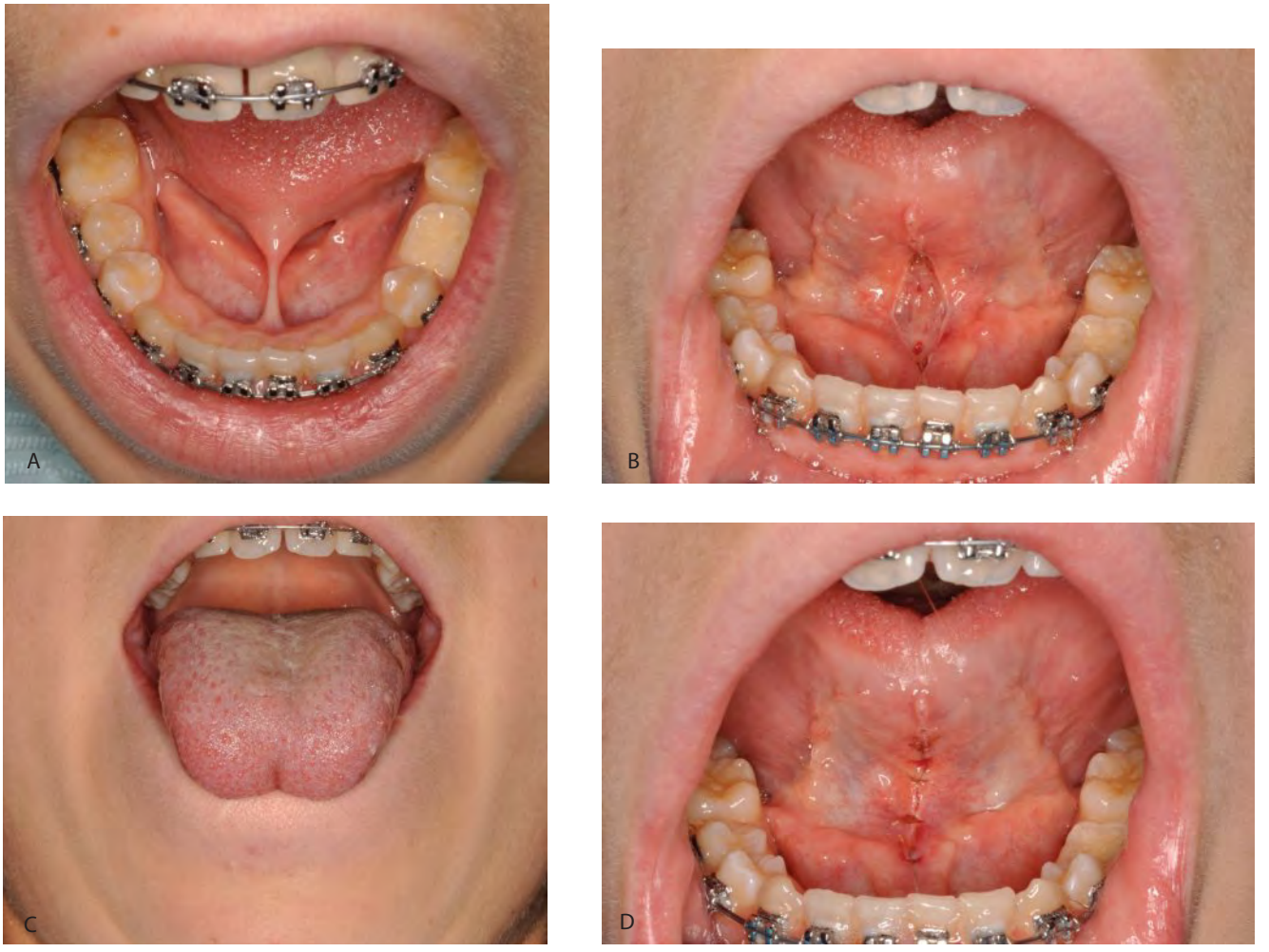


FIGURE 7-15. **A,** Intraoral view of a 13-year-old male patient with a tongue-tie. **B,** The lingual frenum is attached on the tip of the tongue, limiting its mobility. **C,** Wound resulting from the excision of the frenum and the two small relaxing excisions at the junction of the floor of the mouth and the ventral surface of the tongue. **D,** The tongue is free to move

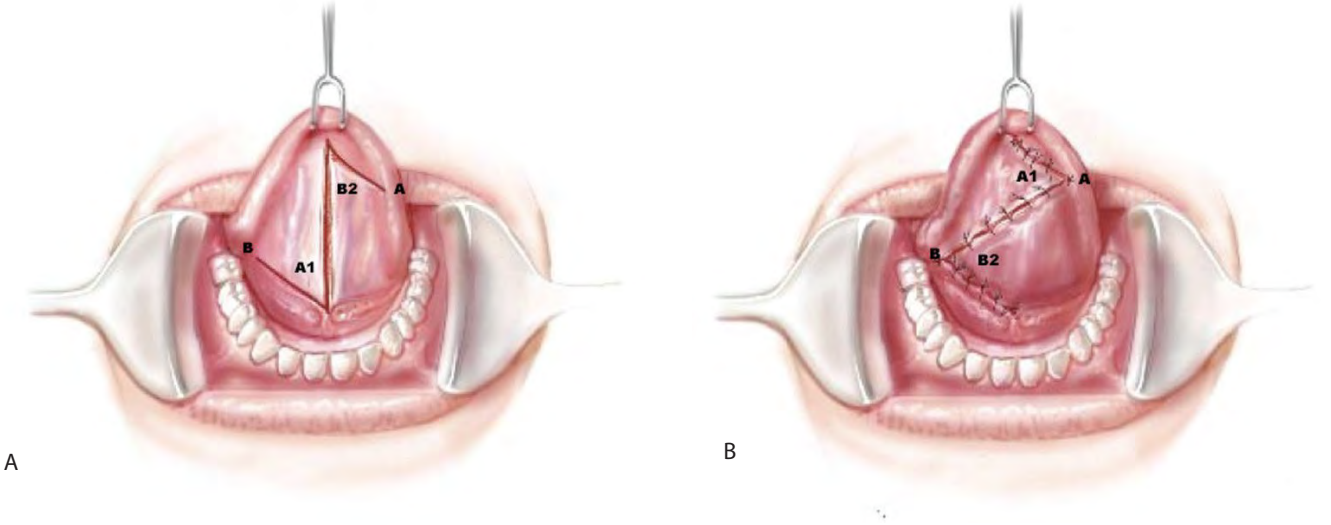


FIGURE 7-16. **A,** The basic principles of a Z-plasty triangular rotation flaps. A vertical limb is created by excising the lingual frenum. Two other limbs are then created at 60° from the first one. **B,** The two triangles created (A1, B2) are undermined, rotated, and translated toward each other. **(A** and **B,** From Kaban LB. Dentoalveolar surgery. In Kaban LB, Troulis MJ, editors. Pediatric Oral and Maxillofacial Surgery. New York: Elsevier; 2004; p. 154. With permission.)

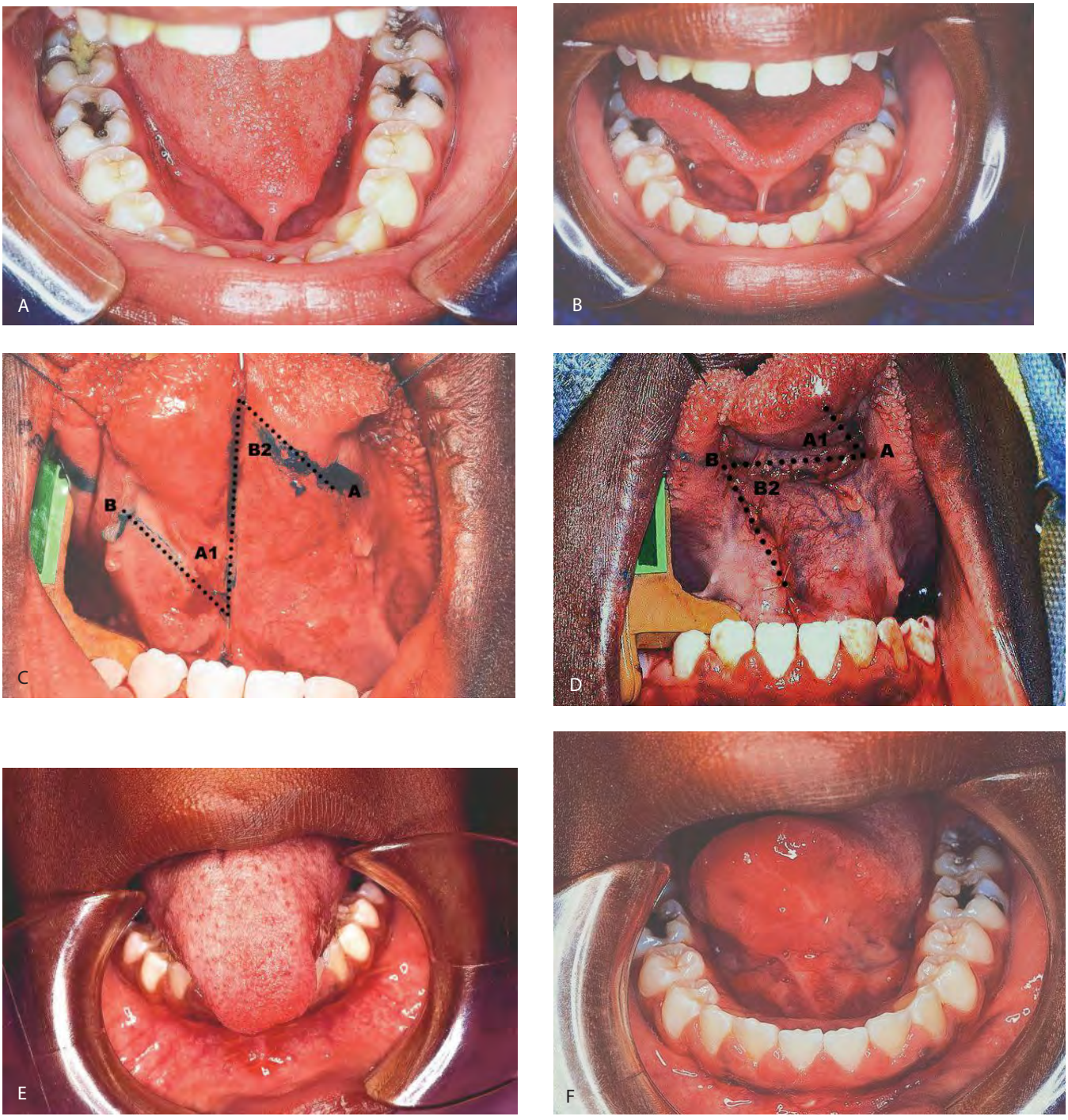


FIGURE 7-17. **A** and **B,** Intraoral pictures of a patient with a tongue-tie. The frenum is attached high on the lingual alveolus. **C,** The Z-plasty incisions are drawn, with the vertical limb on the frenum. **D,** Immediate result after rotation of the flaps and closure. **E** and **F,** Tongue protrusion and elevation 1 year postoperatively. **(A–F,** From Kaban LB. Dentoalveolar surgery. In Kaban LB, Troulis MJ, editors. Pediatric Oral and Maxillofacial Surgery. New York: Elsevier; 2004; pp. 154–155. With permission.)

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8

CHAPTER

Utilization of Three-dimensional Imaging Technology to Enhance Maxillofacial Surgical Applications

Scott D. Ganz, DMD

INTRODUCTION

The evolution in sophisticated diagnostic imaging modalities and associated software applications continues to impact the specialty of oral and maxillofacial surgery. The advent of computed tomography (CT) in dentistry began in the late 1980s with the use of medical-grade machines located in major hospitals or radiology centers. Patients were referred for CT scans to achieve a level of diagnosis beyond the scope of conventional two-dimensional panoramic or periodical radiography available in most dental offices. As computing power increased, the ability to move from two-dimensional to three-dimensional data marked the beginning of a new and exciting area in diagnostic imaging, paving the way for the development and adoption of interactive treatment planning software to help interpret the massive amount of anatomic data. The information gained from a CT scan allowed the clinician to appreciate the "reality of anatomy" for each patient presentation—yielding views of structures aided by advances in data processing power of the computer-driven technology. As dental implants increased in popularity as tooth replacements, the accurate assessment of patient anatomy and collaboration between restorative clinician and surgeon became more critical as determinants of successful outcomes. CT technology and interactive treatment planning software applications helped bridge the communication gap between all members of the implant team, while appreciably removing the guesswork from the process.

Despite all of the benefits of three-dimensional CT scan data, the actual use of the technology was quite limited due to several barriers including cost of the scan, proximity of the radiology center, and lack of proper training in interpretive three-dimensional diagnostics. Recently, a new family of imaging machines was introduced with cone beam computed tomography (CBCT) technology, which offered several immediate benefits for dental applications by helping to remove many of the barriers associated with the imaging modality. CBCT machines are far less costly than a medical-grade CT machine, can offer excellent image quality at a fraction of the radiation dose to the patient, and are small enough to fit into a dental office. Therefore, in the author's opinion, dental applications of three-dimensional imaging has become one of the most significant developments that will have the most impact on how surgical and restorative procedures are planned, communicated, and executed for years to come. As computing processing power, graphics emulations, and display screens continue to dramatically improve, the actual use of the techniques and concepts presented will be ultimately limited by the clinician's imagination. Regardless of the enormous benefits of this technology, there are risks

associated with even low dose radiation exposure. Therefore clinicians should be responsible when utilizing CBCT or CT in adhering to the ALARA principle (As Low As Reasonably Achievable) for each of our patients. The goal of this chapter is to illustrate how this technology can be applied for a variety of different clinical presentations.

Whether using standard CT or CBCT, the scan data requires further processing so that it can be visualized on the computer display. It is not the intent of this chapter to review the technical aspects of this process, as it is the application of the technology that is most rewarding for both clinician and patient. If the patient is referred to a radiology center, the data will be provided on a CD-type disk, with a self-contained viewing software. If an in-office CBCT machine is used, the data will be processed and viewed on a computer workstation. Regardless of how the data are acquired, the software will produce four basic views: the axial, the panoramic, the three-dimensional reconstruction, and the cross-sectional views (Figure 8-1). These four image types are the building blocks for analyzing data, formulating an accurate diagnosis, communicating with clinical teammates, educating the patient, formulating a definitive plan, and properly executing the plan. The key to the process is how each of these images can be utilized through direct interaction such as changing the gray scale, scrolling through the various slices, or simulating implant placement with sophisticated planning tools.

The image acquisition process can be limited by the type of machine used and the size of the data collection sensor. These differences can affect the field of view (FOV). A large FOV machine allows for inspection of the entire maxillofacial complex as displayed in a variety of different formats for diagnostic accuracy (Figure 8-2). An example of the diversity of how data can be displayed is found in the temporomandibular joint (TMJ) view, in which the software automatically generates a series of interactive images focusing on the anatomic landmarks for both the left and the right condyles (Figure 8-3). Various third-party interactive treatment planning software can manipulate the CT/CBCT data in an ever-expanding array of capabilities to facilitate the diagnosis, treatment planning phase, and surgical intervention. The use in maxillofacial surgical procedures includes, but is not limited to, impacted teeth, implant placement, block and particulate bone grafting, orthognathic procedures, distraction osteogenesis, TMJ dysfunction, sinus augmentation, and many others. Each of these procedures can be enhanced through the application of three-dimensional imaging technologies. The following clinical presentations demonstrate how new three-dimensional tools can be applied.

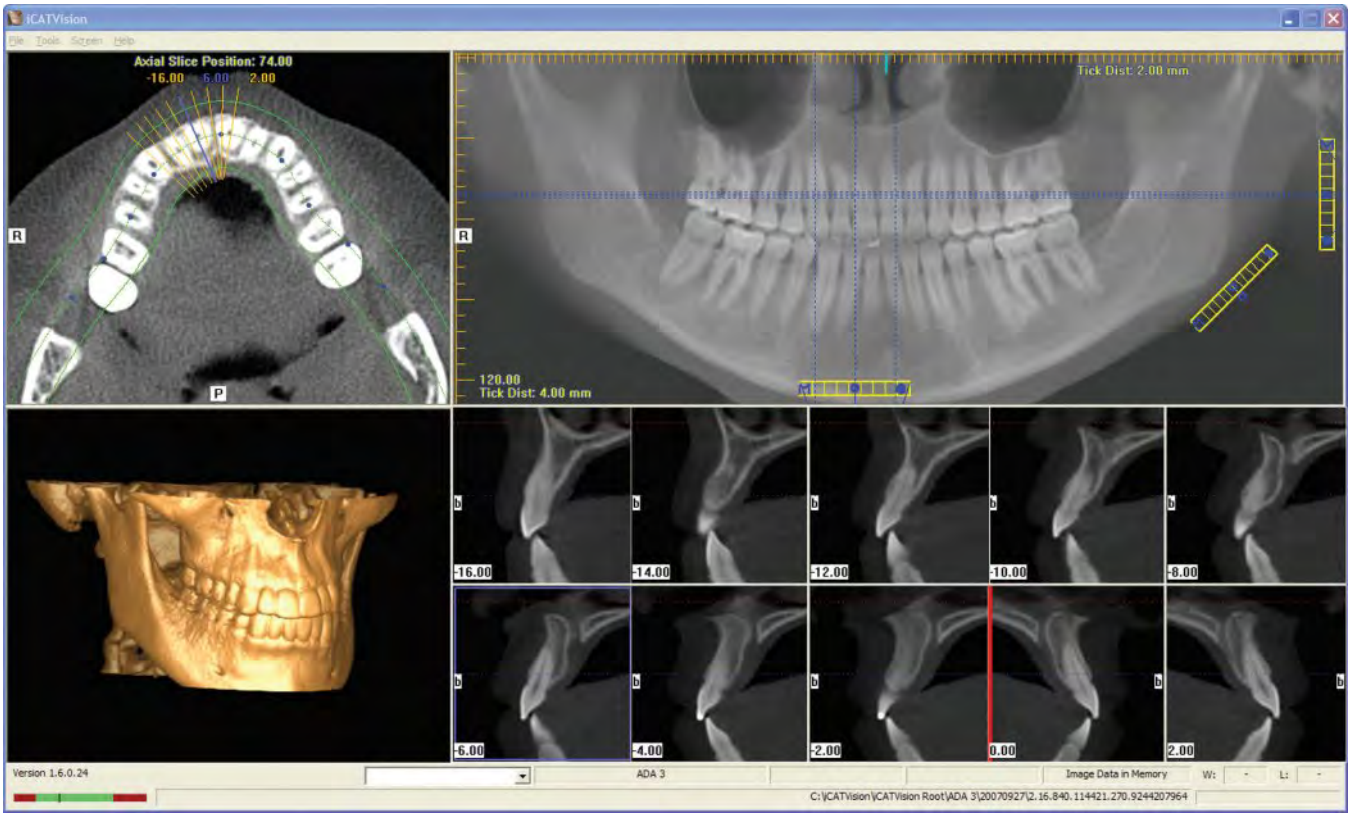


FIGURE 8-1. Cone beam computed tomography (CBCT) data display four primary views: the axial view **(A),** the panoramic view **(B),** the three-dimensional reconstructed view **(C),** and the cross-sectional views **(D).**

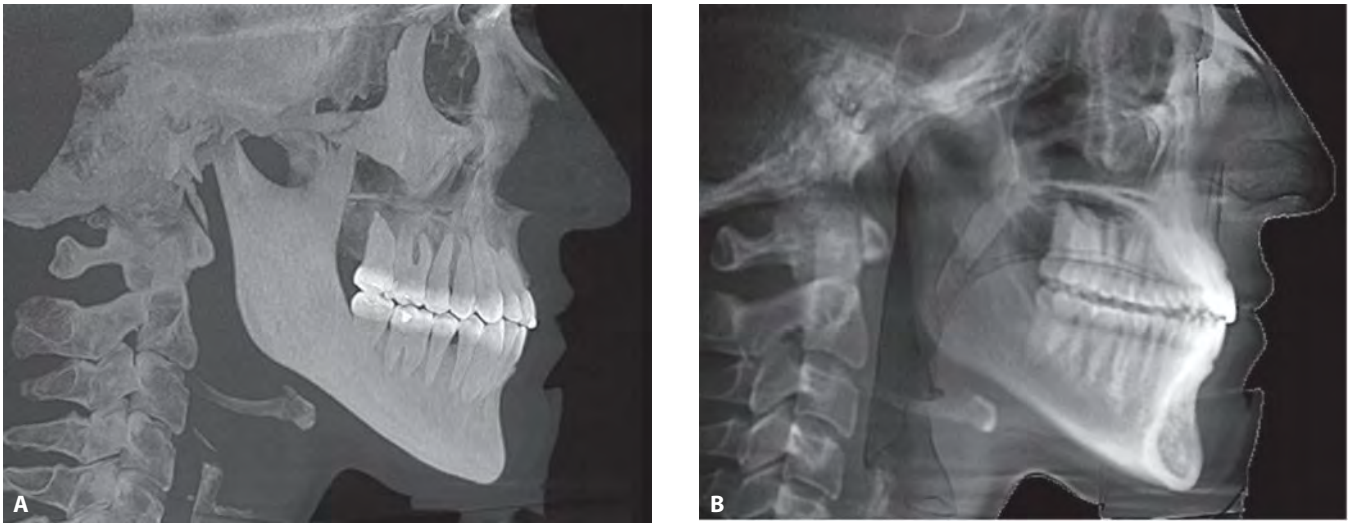


FIGURE 8-2. **A** and **B,** Large field of view (FOV) machines allow for inspection of the entire maxillofacial complex, which can be displayed in a variety of different formats for diagnostic accuracy.

CASE ONE: CBCT Diagnostics for a Horizontally Impacted Maxillary Canine and More . . .

A 10-year-old boy was referred for a three-dimensional CBCT scan by his orthodontist when it was determined that he had an overretained deciduous canine and a horizontally impacted permanent canine. Two-dimensional radiographs were not sufficient to diagnose the spatial position of the canine relative to adjacent vital structures. With the parent's approval, a scan was performed and the three-dimensional data were reviewed. The mixed dentition can be clearly visualized in the initial view of the maxilla (see Figure 8-4). The horizontally impacted canine can be seen in the upper right side of the image, with confirmation that the contralateral canine had erupted properly.

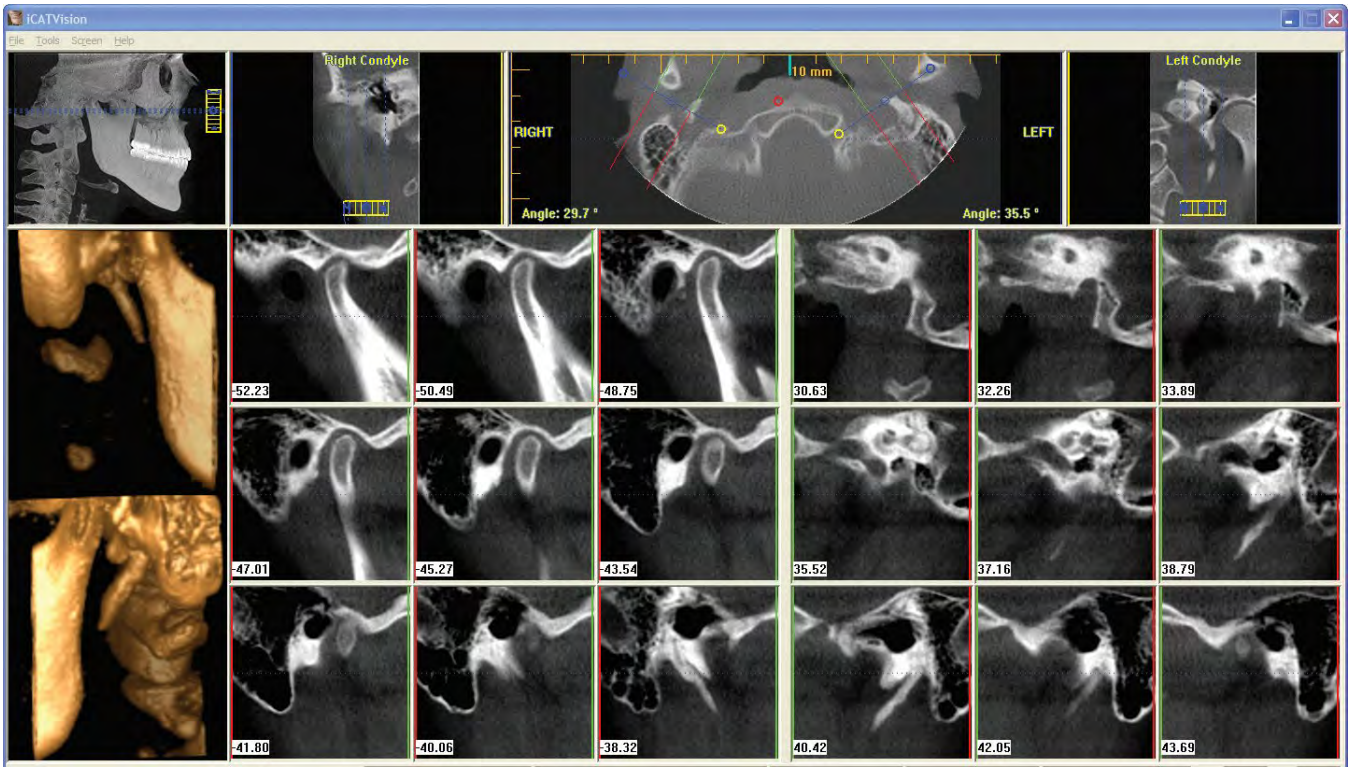


FIGURE 8-3. An example of the diversity displays can be found in the temporomandibular joint (TMJ) view, which automatically creates a series of interactive images focusing on the anatomic landmarks for both the left and the right condyles.

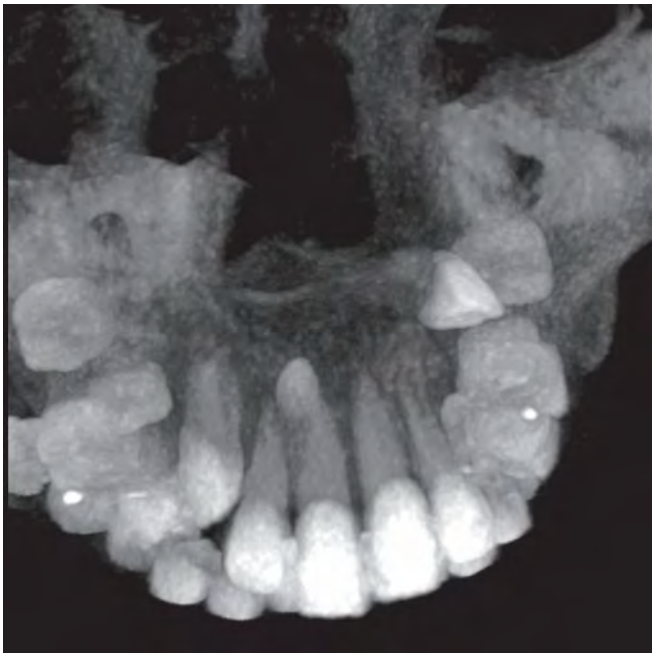
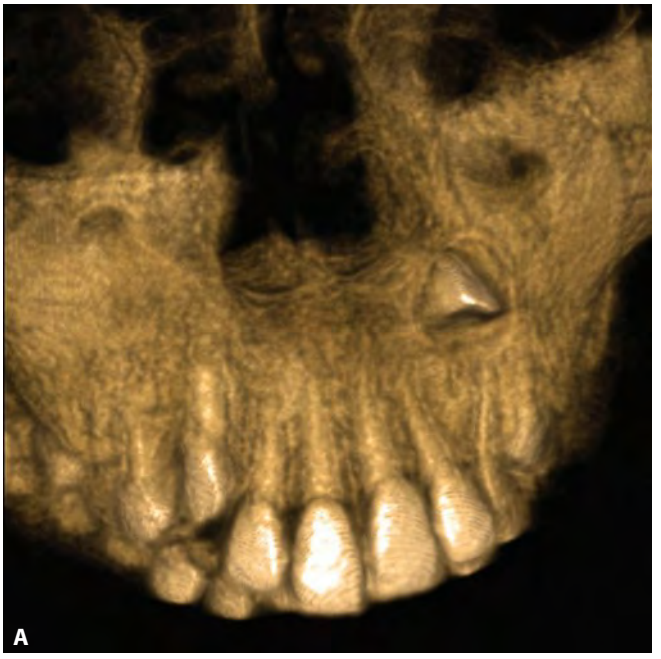


FIGURE 8-4. CBCT scan of mixed dentition in a 10-year-old boy with a horizontally impacted canine tooth.



FIGURE 8-6. The axial view allows inspection of the impacted tooth, the root apex, and proximity to other vital structures including the floor of the nose and maxillary sinus.

Utilizing the CBCT scan data, the maxillary arch was reconstructed as a solid interactive three-dimensional model. The three-dimensional reconstruction accurately revealed the maxillary bone, the properly erupted right canine, the left deciduous canine, and the left impacted canine (see Figure 8-5A). The lateral three-dimensional view exhibited the horizontal position of the permanent canine and the vertical position of the primary canine (see Figure 8-5B). Proximity to adjacent vital structures can also be appreciated in this view. However, the author recommends that all views be assimilated before a definitive diagnosis and treatment plan is determined. The axial view allows further inspection of the



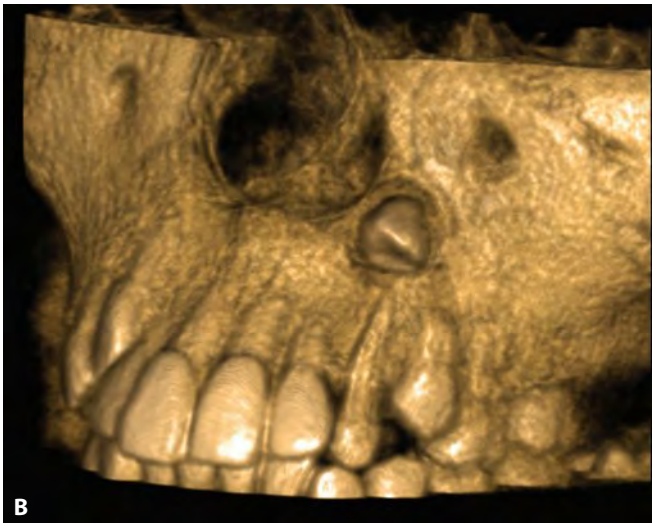


FIGURE 8-5. **A,** Three-dimensional reconstruction reveals the right canine, left deciduous canine, and left impacted canine. **B,** The lateral view reveals the horizontal position of the impacted canine and vertical position of the primary canine.

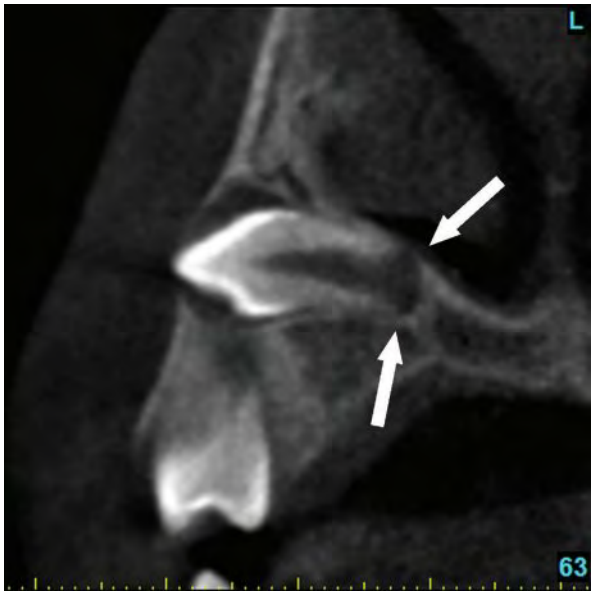


FIGURE 8-7. The cross-sectional view revealed the lack of closure of the root apex, and surrounding structures.

impacted tooth and root apex (see Figure 8-6). The root apex was not fully developed, a fact to be considered if it was determined that the canine tooth could be orthodontically moved into a proper position. The axial view also displayed adjacent vital structures such as the floor of the nose and the maxillary sinus (see *arrows*). Confirmation of the location of the tooth and its proximity to vital anatomy was found in the cross-sectional slice (Figure 8-7). The status of the root apex was also confirmed (see *arrows*).

Using the maximum-intensity projection (MIP), one method of mapping three-dimensional points to a two-dimensional plane, the mixed dentition was clearly visualized but was difficult to interpret, which can sometimes lead to an unexpected incidental finding (Figure 8-8A). Further inspection of the three-dimensional data revealed a supernumerary tooth (mesiodens) near the right maxillary central incisor (see Figure 8-8B). The axial image (Figure 8-9) confirmed the palatal position *(yellow arrow)* and close proximity to the incisal canal *(white arrow)*. A series of cross-sectional image slices (44–49) revealed the 180-degree vertically rotated position of the second impacted tooth and radiolucent area surrounding the clinical crown (Figure 8-10).

The valuable information acquired through three-dimensional imaging technology was essential in accurately diagnosing two impacted teeth within the maxillary scan. Once this information was assimilated, a proper plan of treatment was developed and discussed by all members of the treatment team: the oral surgeon, the orthodontist, and the prosthodontist. The three-dimensional images allowed for improved communication between members of the team, providing the necessary information for a proper course of treatment resulting in a successful outcome.

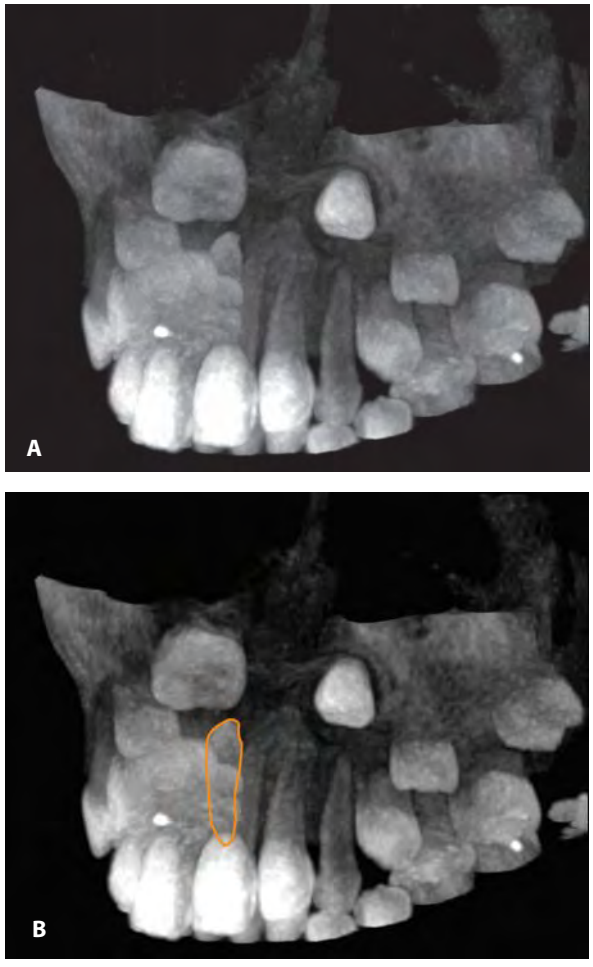


FIGURE 8-8. **A,** Another view of the mixed dentition was helpful to identify other important structures which might have been missed. **B,** A second impacted tooth was found on further inspection of the 3-D images.

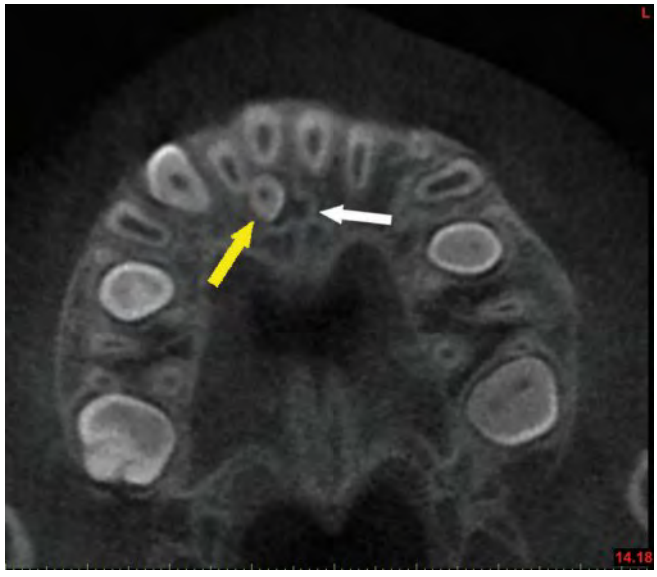


FIGURE 8-9. The axial reveals a mesiodens located palataly to the right central incisor tooth (yellow arrow) with close proximity to the incisal canal (white arrow).

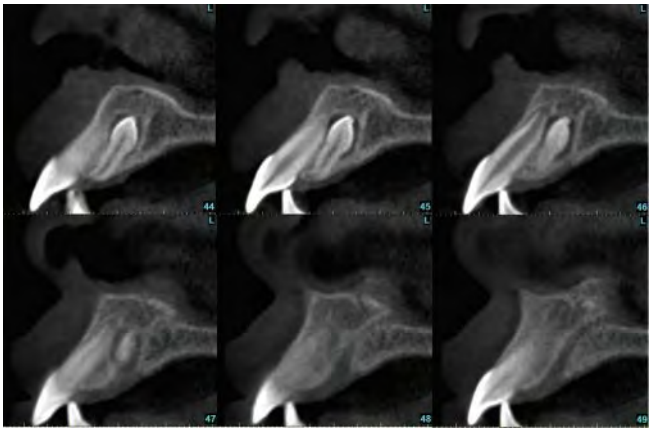


FIGURE 8-10. The cross-sectional slices (44-49 clearly illustrate the position and shape of the mesiodens and radiolucent area surrounding the clinical crown.

CASE TWO: CBCT Diagnostics for a Mandibular Implant Evaluation and More . . .

A 60-year old-man requested an evaluation and treatment recommendation for two missing mandibular first molar teeth. Clinical examination was unremarkable except for the volumetric change in the bone as a consequence of tooth loss. His chief complaint was that he wanted to restore the spaces in his lower arch; he had no other symptoms. A periapical radiograph revealed a suspicious radiolucent area anterior to the potential implant receptor site (see Figure 8-11A). The *arrows* indicate the outline of the area. The intraoral view showed that the mandibular right lateral incisor, canine, and two bicuspid teeth were all within normal limits (see Figure 8-11B). Due to the loss of bone in the first molar area, the proximity to the inferior alveolar nerve, and an irregular appearance on the two-dimensional radiograph, a CBCT scan was recommended.

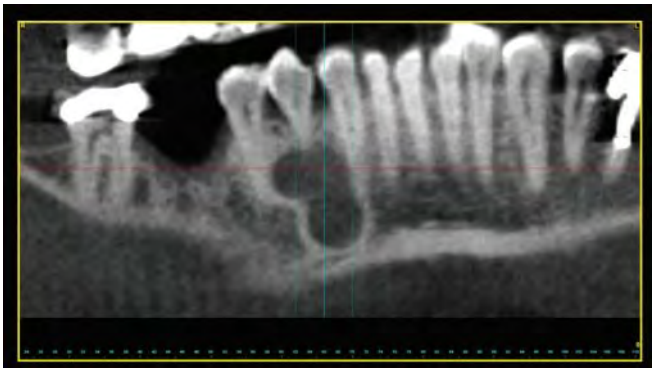


FIGURE 8-12. Panoramic reconstructed view reveals a well-defined mandibular right lesion.

The panoramic reconstructed view from the CBCT data revealed the potential implant receptor sites in the areas of the mandibular first molar teeth. An unexpected incidental finding further reinforced the need for three-dimensional assessment of patient anatomy. A well-circumscribed lesion was detected in the areas of the mandibular canine and premolar teeth (see Figure 8-12). Further inspection of the cross-sectional images revealed the size and extent of the lesion within the mandible and in relation to the adjacent teeth (see Figure 8-13). The lesion was radiolucent within the confines of the dense facial and lingual mandibular cortical plates (see Figure 8-14). The lesion was accurately measured in all dimensions. The three-dimensional reconstructed image showed the area of the missing right mandibular molar and its proximity to the mental foramen (see Figure 8-15).

The axial slices illustrated the well-circumscribed lesion from a different view, revealing a solid anterior border within the symphysis (see Figure 8-16). The path of the inferior alveolar nerves had been previously traced in *orange*. The scope of the lesion seemed to extend to the right mental

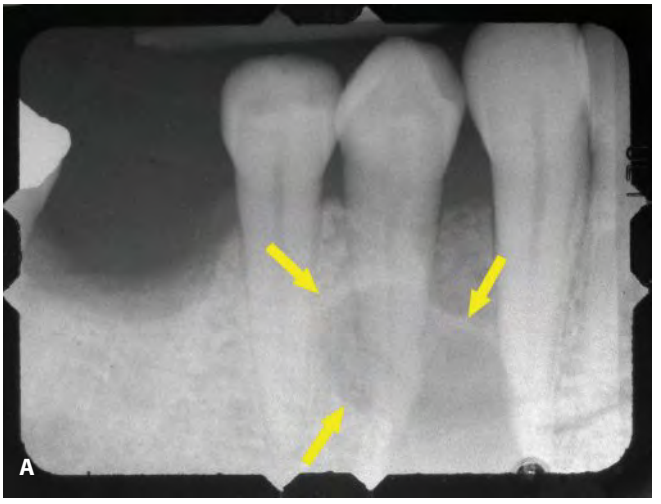




FIGURE 8-11. **A,** A periapical radiograph revealed a suspicious radiolucent area. **B,** Intraoral view.

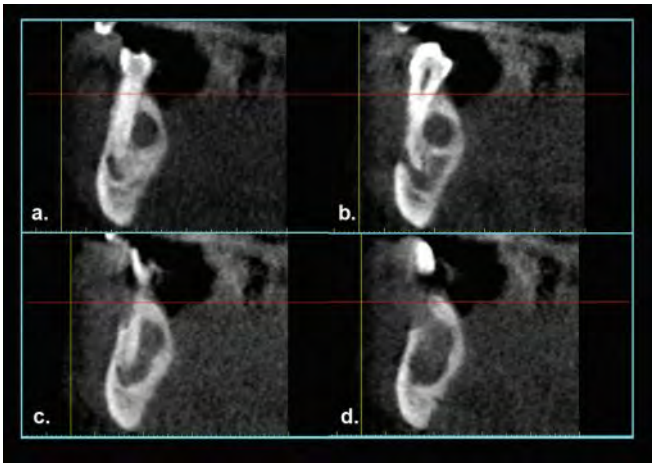


FIGURE 8-13. Cross-sectional images reveal the extent of the lesion within the mandible and in relation to the adjacent teeth.

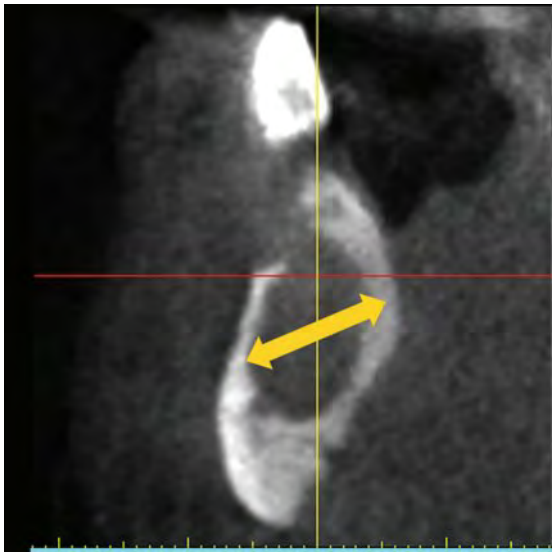


FIGURE 8-14. The radiolucent lesion and the buccal and lingual cortical plates can be visualized and measured *(arrow)* in all dimensions.



FIGURE 8-15. Native three-dimensional reconstructed view of the right side mandible with a missing first molar tooth.

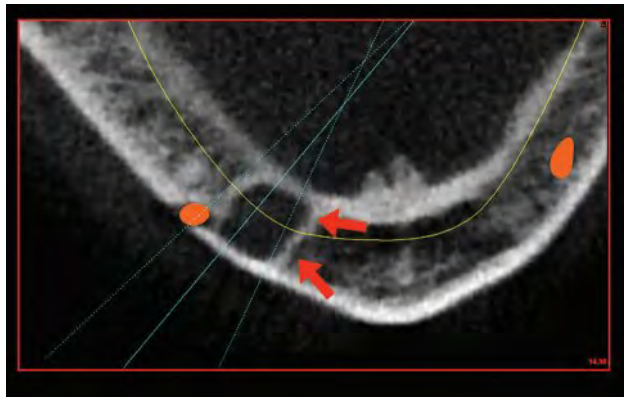


FIGURE 8-16. The axial view reveals a well-circumscribed lesion *(arrows)* near the mental nerve.

foramen as seen in the axial view (see Figure 8-17A). To determine whether the lesion was expansive, accurate measurements were made from the facial to the lingual cortical plates over the lesion and in the area of the contralateral side of the mandible (see Figure 8-17B). The

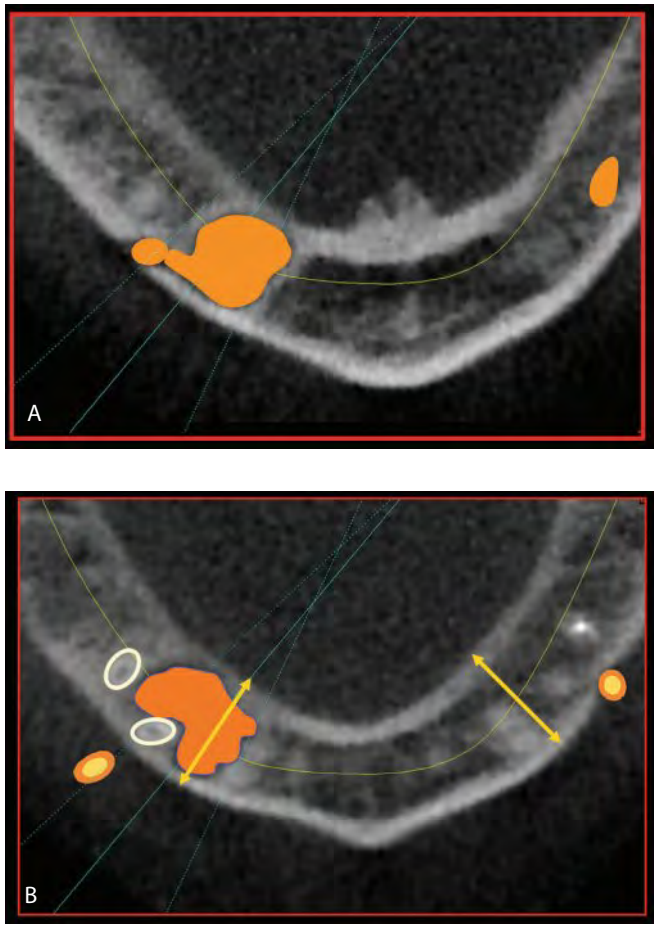


FIGURE 8-17. **A,** The extent of the lesion is marked in *orange.* It appears to extend to the right mental foramen. **B,** Measurements of the right and left side areas indicate that the lesion is nonexpansive in nature.

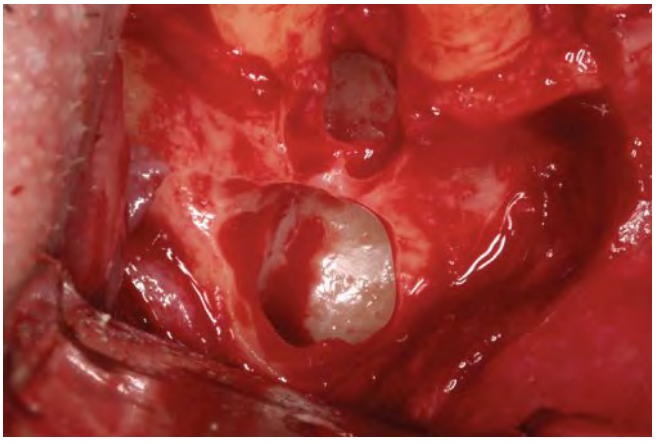


FIGURE 8-18. After excision of the lesion, the shiny lingual cortical plate can be seen. Photo Courtesy of Dr. Richard Kraut

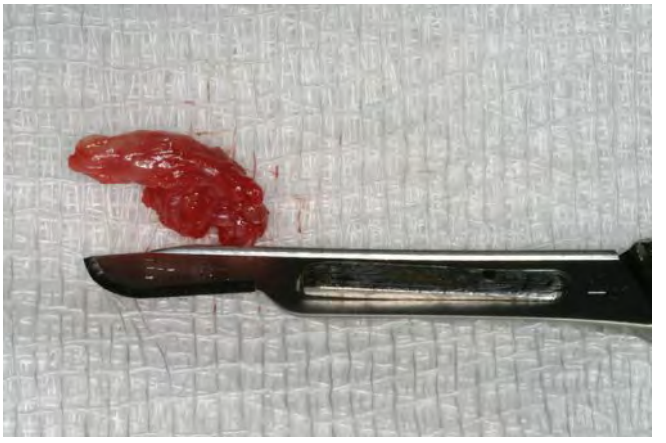


FIGURE 8-20. Part of the excised lesion, sized near the scalpel, was sent for biopsy. Photo Courtesy of Dr. Richard Kraut

measurements were the same, indicating a nonexpansive lesion. Due to proximity to the adjacent teeth, pulp tests were performed and both teeth were found to be vital. The patient was advised of the findings and referred to an oral surgeon. The nature of digital data workflow and CBCT data provides clinicians with one of the best communication tools for diagnosis and treatment planning. The data set was sent via email to the local surgeon for review and discussion. The surgeon was able to view the interactive data and offer a plan for treatment after consultation with the referring dentist. The information was reviewed with the patient and, based upon a thorough review of the findings, it was agreed that a surgical intervention was recommended.

After local anesthesia was administered utilizing a full-thickness mucoperiosteal flap, the area was exposed, and the lesion was excised leaving a shiny appearance to the lingual cortical plate (Figure 8-18). It was elected to fill the lesion with a sterile, biocompatible natural porous bovine bone mineral matrix (Figure 8-19). The excised lesion was sent for biopsy (Figure 8-20), and the area healed uneventfully. The histologic evaluation indicated that the lesion was an odontogenic keratocyst. This type of lesion has a high recurrence rate, which means that it should be followed carefully after initial treatment. The low dosage of CBCT makes it an ideal modality for postoperative follow-up. The 5-month postoperative CBCT cross-sectional image reveals complete fill of the defect site (Figure 8-21). The postoperative axial view and the resultant density of the grafted site can been seen at 5 months in Figure 8-22. The lesion has been followed for over 3 years without any sign of recurrence (Figure 8-23).

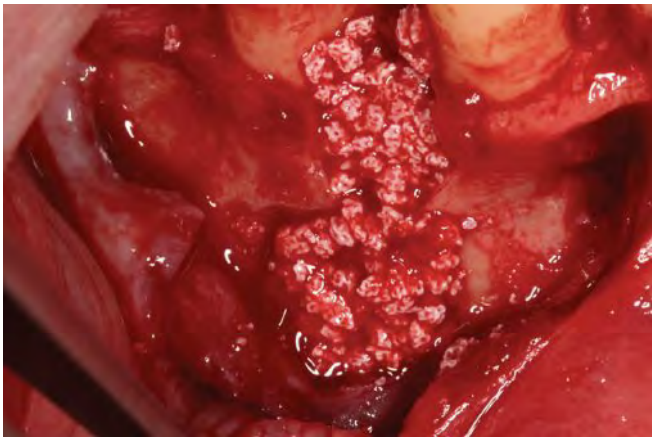


FIGURE 8-19. The lesion was filled with graft material and allowed to heal. Photo Courtesy of Dr. Richard Kraut

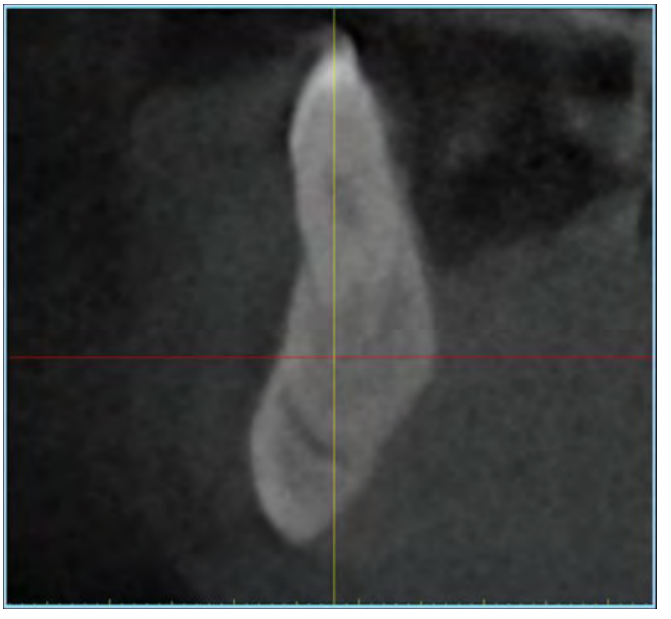


FIGURE 8-21. The 5-month CBCT postoperative cross-sectional image reveals complete fill of the defect site.

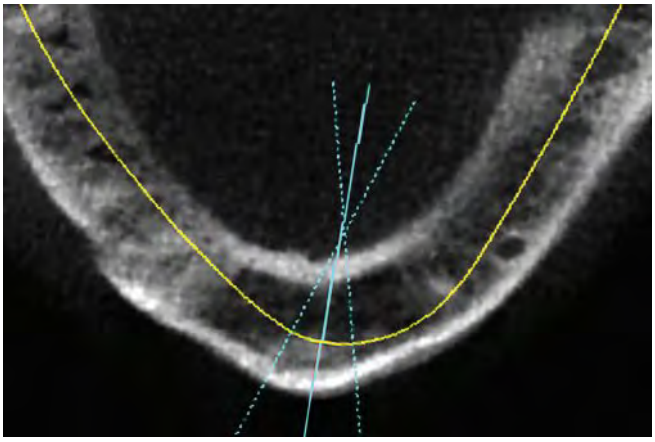


FIGURE 8-22. The postoperative axial view and the resulting density of the grafted site at 5 months.

This case represented a CBCT scan that resulted in an incidental finding of an intra-osseous lesion. Due to the location of the lesion and its proximity to the mental nerve and adjacent teeth, the subsequent treatment could have been debilitating to the patient. The diagnostic accuracy afforded by the three dimensional views allowed for a complete understanding of the size and scope of the lesion, so that the oral surgeon could make an educated decision on the recommended course of treatment. Therefore, the ability to visualize the lesion and its properties are essential for successful outcomes.

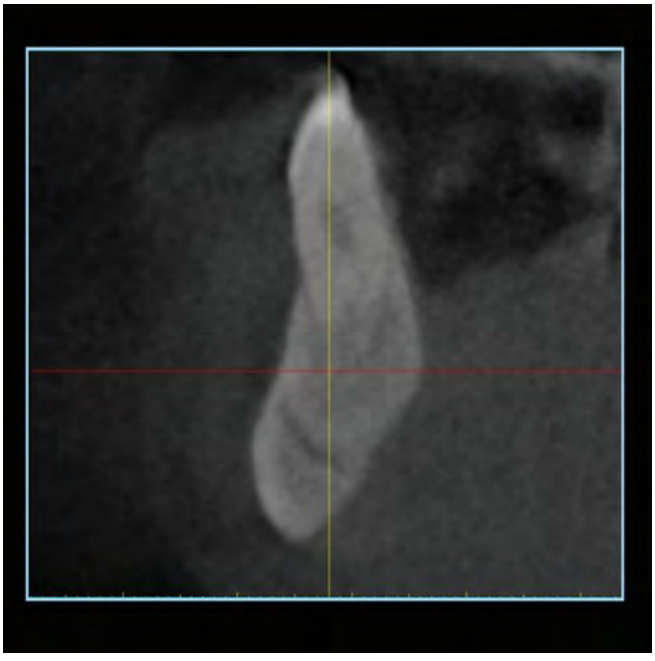


FIGURE 8-23. The CBCT cross-sectional image at the 3-year recall. No evidence of recurrence.

CASE THREE: CBCT Diagnostics to Assess Potential Graft Augmentation

A 17-year-old female was referred for routine dental implant evaluation of a congenitally missing maxillary left lateral incisor. Clinical examination revealed a facial concavity in the area of the missing tooth, leaving a narrow facial-palatal dimension of bone for an implant. Conventional periapical radiographs were not helpful in determining the quality of the bone, the width of bone, or the thickness of the facial or palatal cortical plates. Further radiographic studies were suggested and a CBCT scan was performed.

The panoramic reconstructed view based upon the CBCT data revealed blunted root apexes for the maxillary anterior teeth and allowed inspection of the potential lateral incisor implant receptor site (see Figure 8-24). To gain an understanding of the space required to place and restore a single implant in the esthetic zone, all three-dimensional views were considered. The axial view sliced near the crest of the bone displays the root morphology of the entire arch and the facial-palatal width of the residual alveolar crestal bone (see Figure 8-25). The shape of the individual teeth as seen in the axial view demonstrates what the author has termed as the "restorative dilemma". The shape of our natural teeth as they emerge from the bone are consistent with normal tooth morphology. The dental implants we may use to insert into the bone are "round" in shape. Going from the round shape of the implant to a morphologically correct tooth form is not easy. It is a restorative dilemma.

Utilizing nondistorted measuring tools in the axial view *(red)*, the facial-palatal dimension can be determined for the right existing lateral incisor tooth, as transposed to the left lateral space (see Figure 8-26). A realistic implant known to be 3.8 mm in diameter can be virtually placed to assess the potential volumetric space requirements of this receptor site *(yellow arrow)*. The narrow ridge can then be assessed in the cross-sectional views (see Figure 8-27A). Utilizing specialized three-dimensional planning software, a simulated bone graft can be positioned on the facial aspect of the alveolar ridge (see Figure 8-27B). If this were to be a block graft taken from mandibular ramus or the anterior symphysis fixation would be an issue to ensure success—and this aspect can also be virtually planned (see Figure 8-28B and C). The

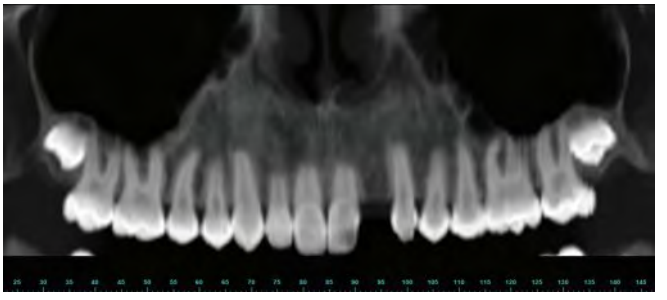


FIGURE 8-24. Panoramic reconstructed view reveals a congenitally missing maxillary left lateral incisor.



FIGURE 8-25. Axial view shows the facial-lingual dimension of the left lateral incisor area.

length and diameter of the fixation screw should be commensurate with the graft thickness and density of the underlying host bone. Once an appropriate ridge width and volume has been achieved, the implant can be planned.

In research developed in 1992 and first published in 1995, the author proposed a method for receptor-site evaluation within the cross-sectional slice depicting the available bone. This concept is called the *Triangle of Bone® (TOB).* The base of the triangle is created by drawing a line from the widest portion of the facial bone to the widest portion of the palatal bone. The apex of the triangle is positioned above the alveolar crestal bone (or proposed position of the crest when considering grafting), and the lines are connected. The most ideal position to maximize bone volume surrounding an implant would be to bisect the triangle. A realistic 3.8-mm-diameter tapered-design implant placed within the TOB is surrounded by bone in the apical two thirds, leaving the

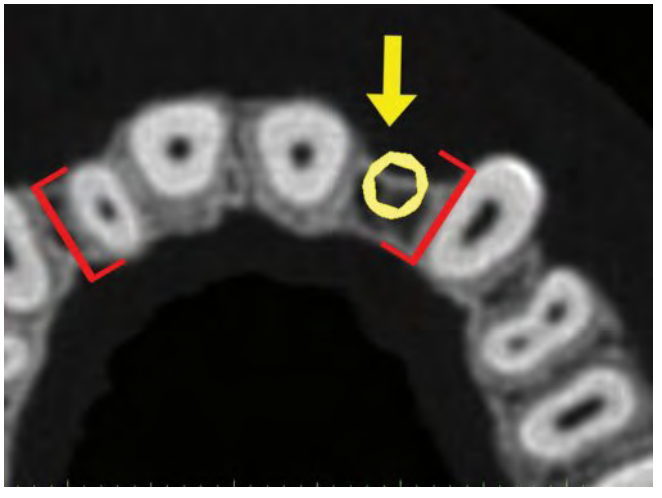


FIGURE 8-26. The axial view allows for precise measurements (in *red*) to compare the right side existing lateral incisor with the potential space requirements for the implant receptor site *(arrow).*

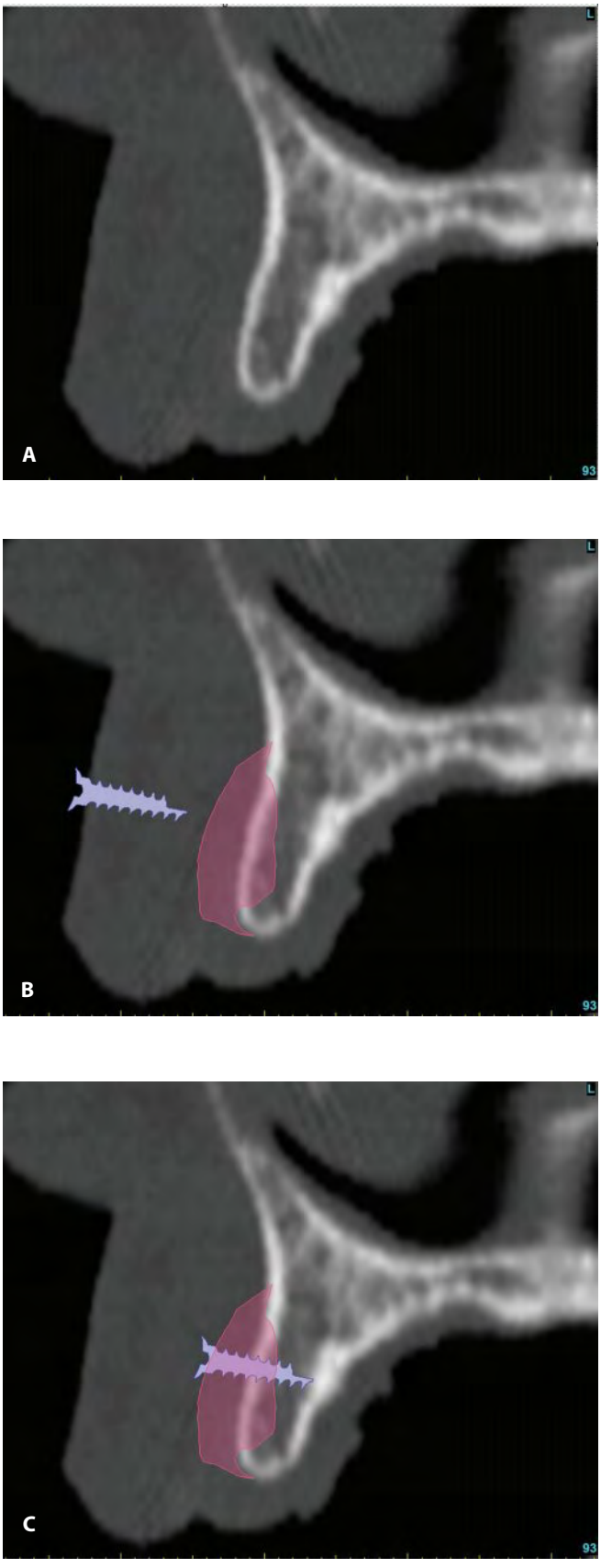
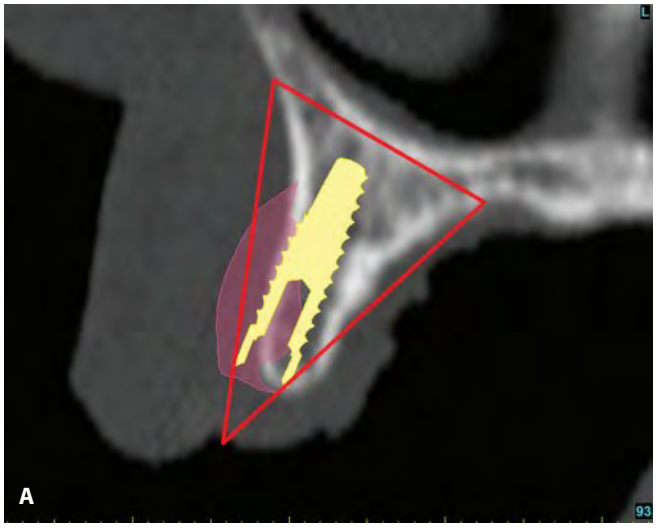


FIGURE 8-27. The cross-sectional view shows a narrow ridge **(A),** a simulated bone graft **(B),** and the plan for fixating the donor graft with a screw **(C).**



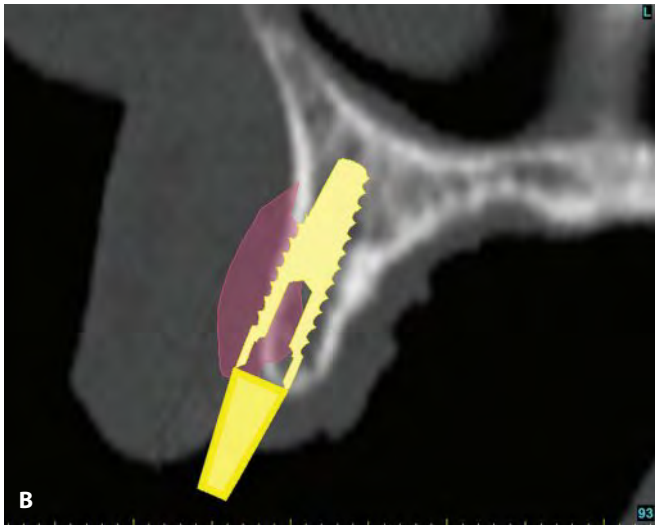


FIGURE 8-28. A simulated 3.8-mm diameter implant positioned within the "triangle of bone" **(A)** and a simulated abutment to help visualize the emergence profile **(B).**

facial aspect exposed to the simulated bone graft (see Figure 8-29A). However, the goal of implant dentistry is not the implant, it is the tooth. Therefore, the restorative aspect must be considered before finalizing the placement of the implant. The abutment simulation helps to visualize the path of emergence of the implant within the envelope of the restoration (see Figure 8-29B).

The three-dimensional planning concepts and software tools have empowered clinicians with new paradigms for assessing implant and/or grafting sites. Closer inspection of the positioning of the simulated bone graft, implant, abutment, and virtual tooth reveals that at the facial-coronal aspect of the implant, there is 1.65 mm of new simulated bone, 2.93 mm at the middle of the implant, and 1.65 mm on the palatal aspect near the first thread (see Figure 8-30). A virtual tooth has been properly positioned within the space requirements for a left lateral incisor tooth. A 12-degree angulated abutment has been simulated to fall within the envelope of the virtual tooth for

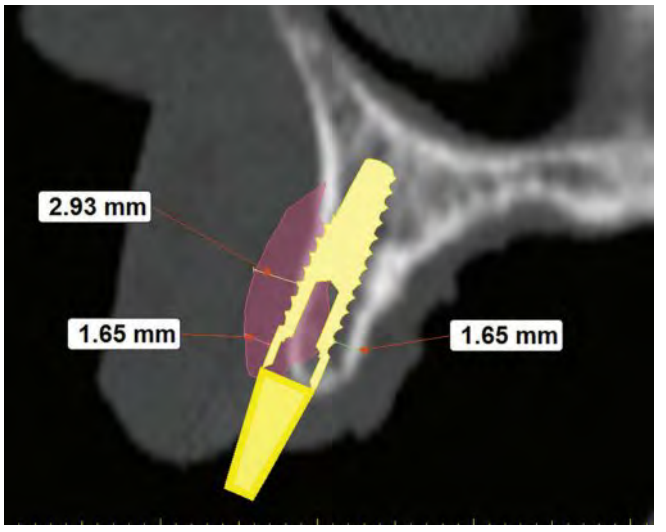
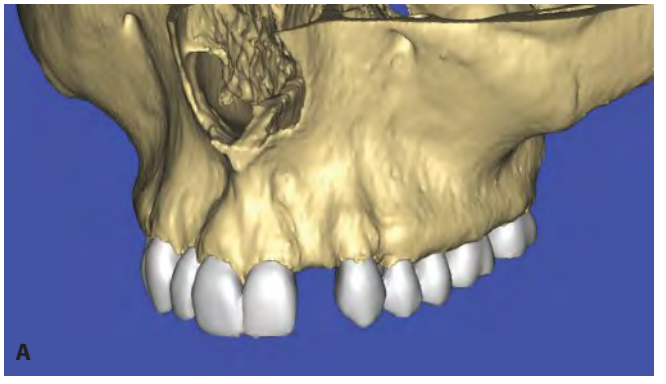


FIGURE 8-29. Closer inspection of the positioning of the simulated bone graft, implant, abutment, and virtual tooth reveals that at the facial-coronal aspect of the implant, there is 1.65 mm of new simulated bone, 2.93 mm at the middle of the implant, and 1.65 mm on the palatal aspect near the first thread.

assessment of emergence profile for the planned cemented-type restoration. However, final verification of bone graft and implant placement should be completed utilizing the three-dimensional reconstructed model.



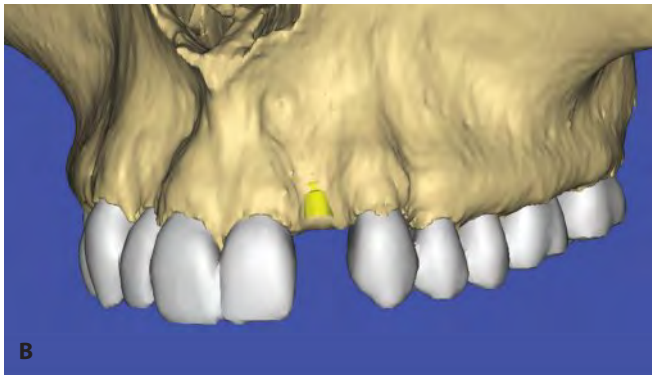
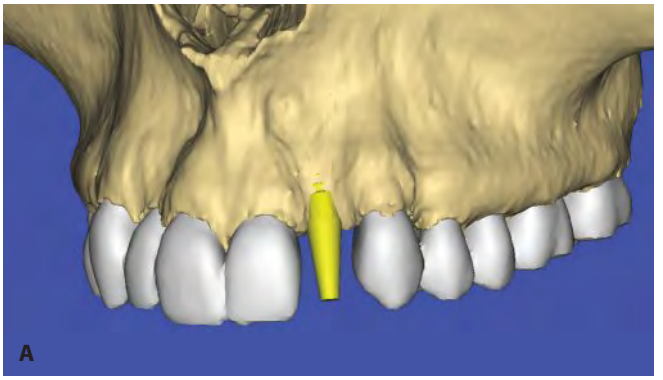


FIGURE 8-30. The three-dimensional reconstruction reveals the severe concavity of the lateral incisor area **(A),** with a simulated implant with the coronal aspect exposed at the alveolar crest **(B).**



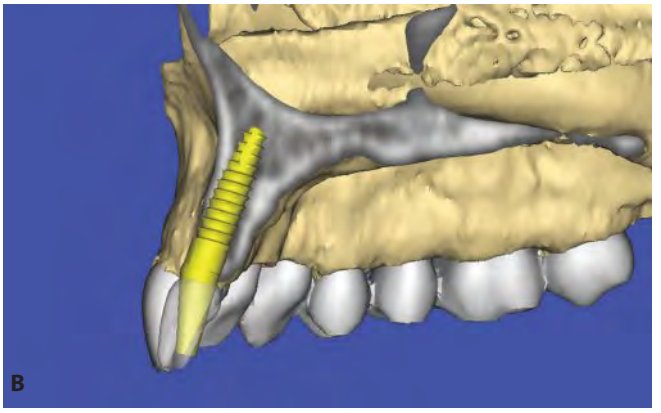


FIGURE 8-31. The abutment projection extends from the implant indicating direction **(A),** which is further demonstrated in the three-dimensional cross-sectional slice through the virtual tooth **(B).**

Examination of the three-dimensional reconstructed maxilla reveals the dentition, the surrounding root eminences, and the resulting concavity in the area of the congenitally missing left lateral incisor tooth (see Figure 8-31A). The placement of a simulated implant helps to visualize the depth of the concavity and assess the facial exposure of the coronal aspect of the implant (see Figure 8-31B). An abutment projection that extends from the implant is helpful in determining implant positioning and angulation (see Figure 8-32).

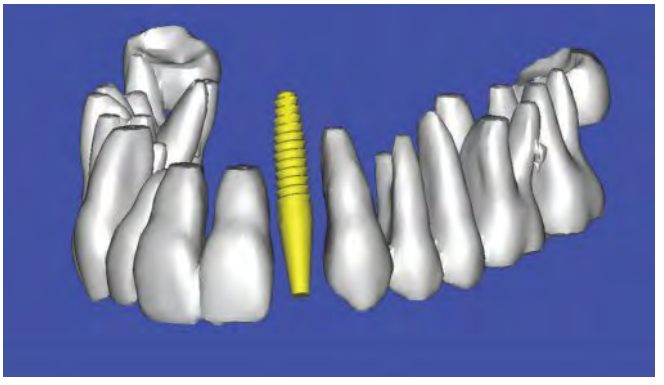


FIGURE 8-32. Removing the bone allows access to the root morphology of the adjacent teeth.

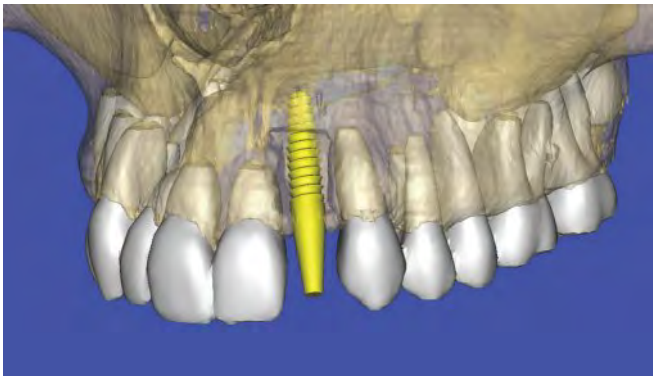


FIGURE 8-33. Using "selective transparency" reveals the implant and abutment projection within the planned receptor site.

Using advanced "clipping" features of the planning software, the three-dimensional maxilla can be sliced to provide additional vital information regarding the implant position and abutment within the envelope of the virtual tooth (see Figure 8-32). The proximity of the implant to the adjacent teeth can be confirmed by removing the maxillary bone and leaving only the clinical crowns and tooth roots (see Figure 8-32). Interactive treatment planning software can provide a variety of methods to reveal the three-dimensional data, and perhaps, the most striking is the ability to selectively control the opacity of different anatomy, a concept that the author has termed *selective transparency*. Using selective transparency, the outer maxillary bone remains translucent, showing the underlying implant and adjacent teeth and roots (see Figure 8-33).

For illustrative purposes, the bone graft site can be virtually prepared to receive a block bone graft as demonstrated utilizing innovative software modules (Figure 8-34A). The facial bone has been modified, and the simulated receptor site de-corticated for better vascularity to the graft (see Figure 8-34B). Using the a special "graft volume tool" the prepared defect site can be virtually filled with an adequate volume of bone to support an implant based upon the paradigms addressed previously (see Figure 8-35). The fixation of the graft can also be visualized in advance of the procedure for proper positioning within the graft and the receptor site bone (see Figure 8-36A and B). Through three-dimensional "clipping," the entire surgical and restorative complex can be visualized including the implant, the bone graft, the abutment, and the virtual tooth (Figure 8-37). The final simulation on the three-dimensional reconstructed maxilla is useful as an educational tool to explain the individual process to the patient, and a communication tool to the referring clinician regarding all of the components of the procedure (Figure 8-38). The implant can be seen emerging from the proposed bone graft (see Figure 8-38A), the virtual translucent tooth (see Figure 8-38B), the opaque tooth helpful in evaluating contact area in relation to the interproximal height of bone (see Figure 8-38C), and a simulation of the healed graft site with the restoration in place (see Figure 8-38D). This case highlighted the wide

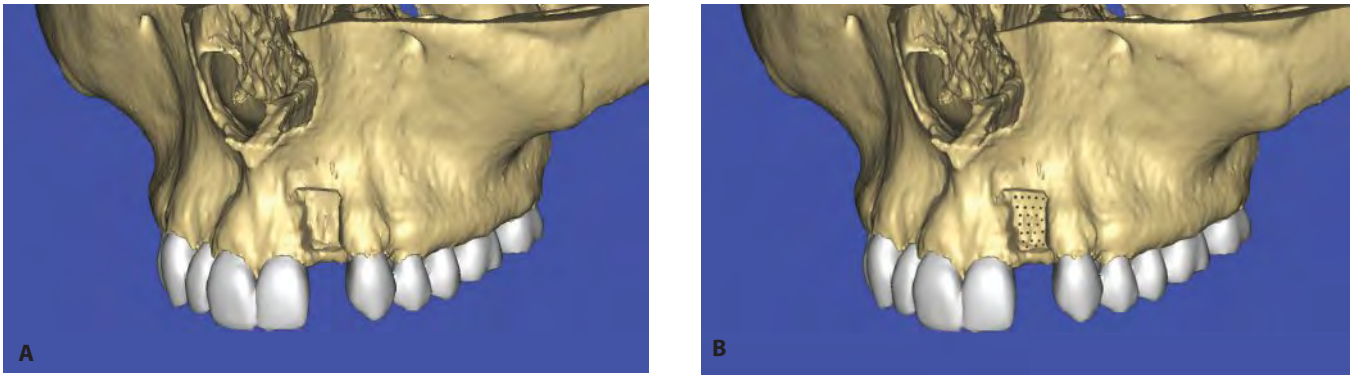


FIGURE 8-34. Using three-dimensional planning tools, the simulated graft receptor site can be virtually "prepared" **(A)** and the bone decorticated **(B).**

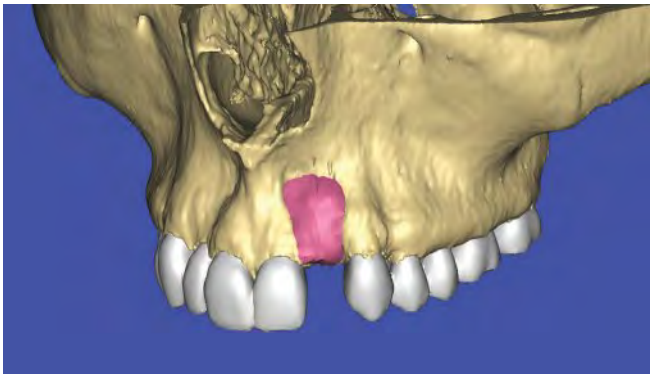


FIGURE 8-35. The graft site can be simulated to fill the concavity to allow for adequate volume of bone to support an implant.

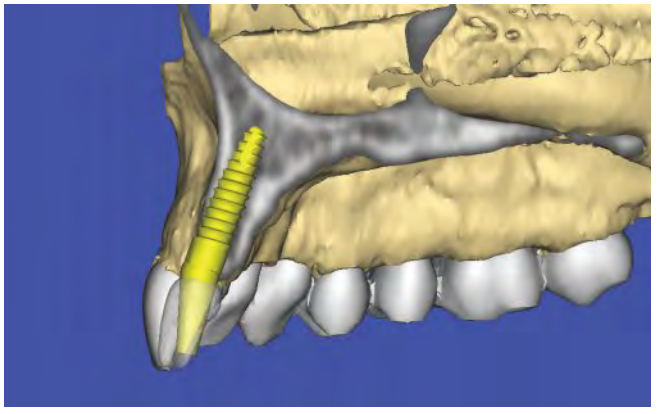


FIGURE 8-37. The implant, the graft, the abutment, and virtual tooth can be visualized.

variety of tools that can be utilized to enhance the diagnostic and treatment capabilities for a single tooth and bone augmentation application.

CASE FOUR: CBCT Diagnostics in the Mandible—Implants and More . . .

A 62-year-old man presented with complete maxillary and mandibular removable dentures. He wished to review treatment options for implant-supported restorations, both fixed and removable. As part of the presurgical prosthetic workup, maxillary and mandibular study casts were fabricated to facilitate capturing vertical dimension records. A complete denture setup was achieved with the appropriate tooth mould to fit the arch form. Although it appeared that the patient had adequate bone for implant placement, in order to plan with greater accuracy, a barium sulfate scanning template was fabricated by duplicating the tooth setup (BariOpaque®, Salvin Dental, Charlotte, NC). The scanning template was placed intraorally, with a putty bite registration to stabilize

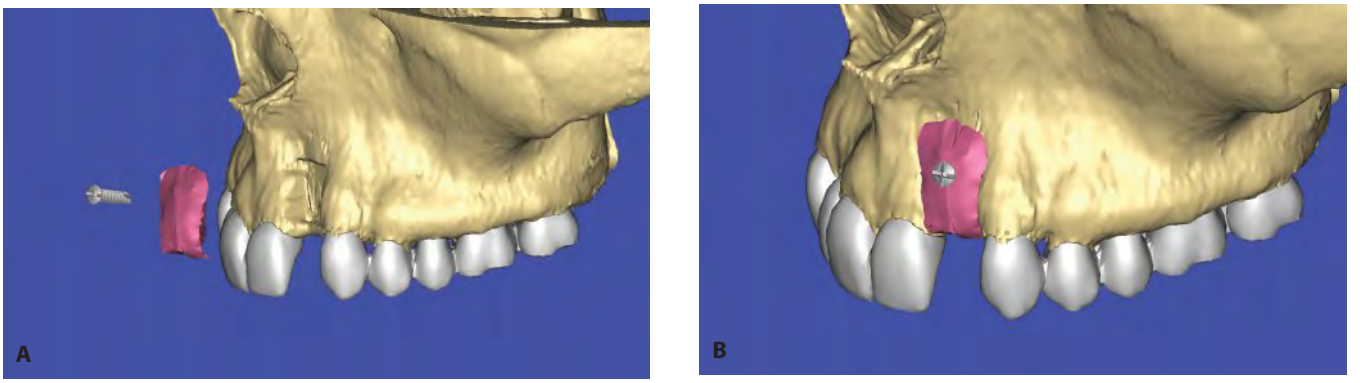


FIGURE 8-36. **A,** The fixation screw can also be visualized for proper positioning within the graft. **B,** The fixation screw in place.

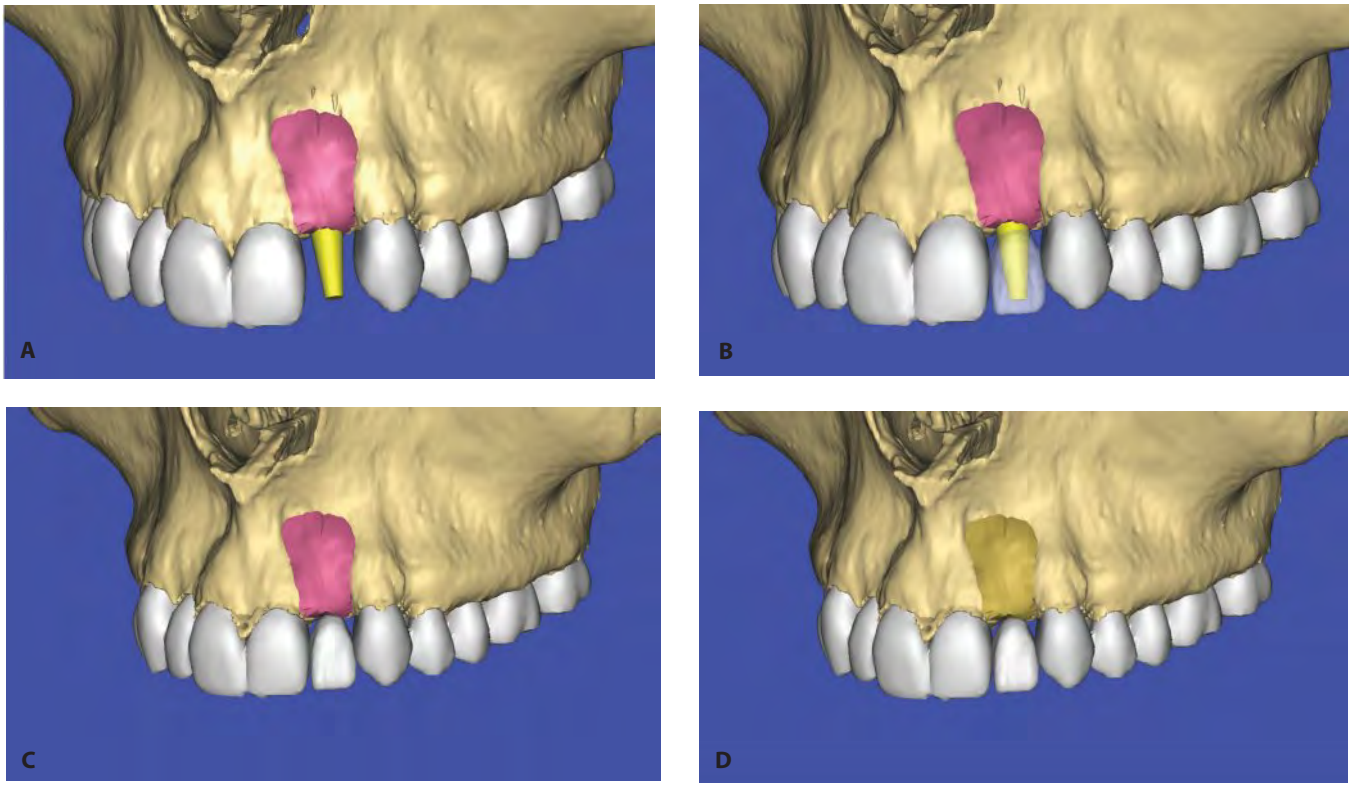


FIGURE 8-38. The implant abutment emerging from the graft **(A),** with the virtual translucent tooth **(B),** the virtual tooth (opaque) **(C),** and a simulation of the healed graft and restoration in place **(D).**

the maxillary and mandibular position and to minimize movement before the CBCT scan. The panoramic radiograph with the mandibular barium sulfate scanning template can be seen in Figure 8-39. For the purposes of this chapter, only the information from the mandible is presented.

The three-dimensional reconstructed view allows for inspection of the mandibular morphology and an appreciation of the mental foramina (see Figure 8-40). However, it does not reflect the relationship between the bone and the desired occlusion. A properly fabricated radiopaque scanographic template affects the treatment plan by linking the tooth position to the underlying bone, establishing important landmarks that help to direct implant positioning (see Figure 8-41 A and B). Whereas it is possible to simulate the placement of implants in the anterior symphysis without a template, there is no assurance that the implants can be restored properly within the boundaries of the restorative envelope (see Figure 8-42). Noting the location of the mental foramen and the path of the inferior alveolar nerve is essential to avoid potential serious complications. Most interactive treatment planning software applications will provide the necessary tools to trace out the nerve from the lingula to the mental foramen. Using the cross-sectional images, the location of the mental foramen can be located and the nerve traced (see Figure 8-43). Once the nerves have been located, the implants can be placed at the desired position within the confines of the scanning template. Ideally, the implant should be positioned within the bone, with an abutment pro-

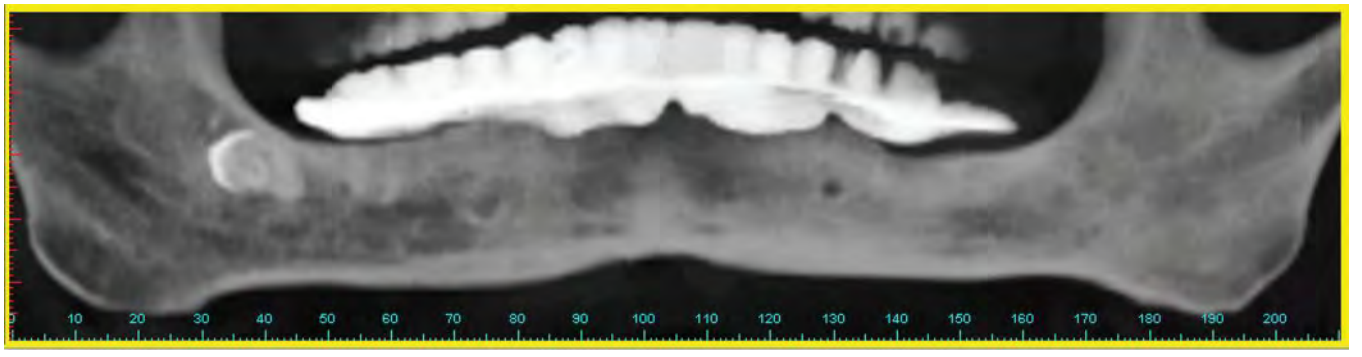
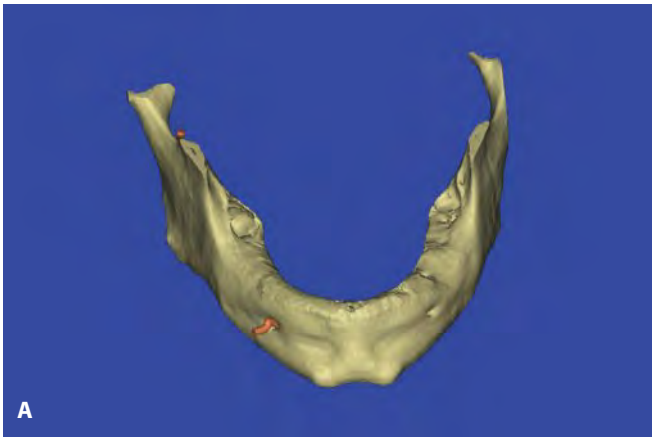


FIGURE 8-39. Panoramic radiograph with mandibular barium sulfate template.



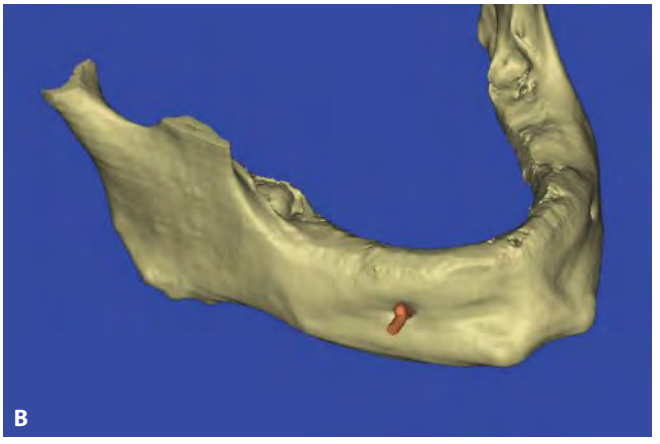


FIGURE 8-40. **A** and **B,** Mandibular reconstructed three-dimensional view shows the simulated nerve at the right mental foramen.

jection extending above the occlusal plane and adjusted after taking into consideration the other views (see Figure 8-44A). Note the difference in bone quality, density, and morphology from one implant receptor site to the next (see Figure 8-44B). Implant length and diameter can also be evaluated in the cross-sectional view.

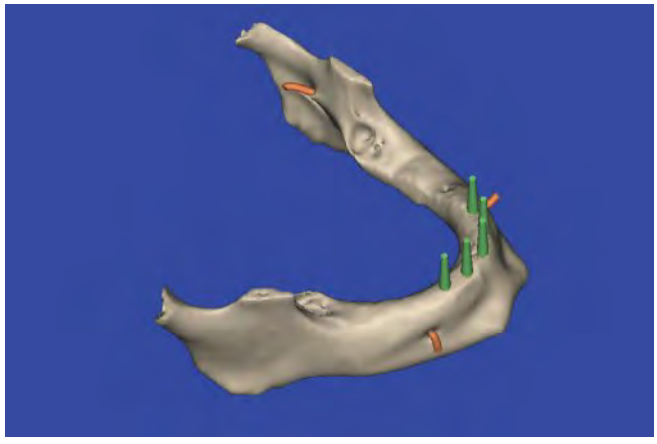


FIGURE 8-42. Five simulated implants placed in the anterior mandible.

Returning to the three-dimensional reconstructed model and the occlusal view, the inter-implant distances and angulation can be evaluated to fit the needs of the restorative scheme. If desired, the software can help to parallel the implants (see Figure 8-45A). Using the concept of selective transparency, the path of the mental nerves and proximity to the implants can be clearly visualized. The initial positioning of the implants can then be verified by superimposition of the scanning template (see Figure 8-46). Adjustments can be accomplished if necessary. The left distal-most implant exits in the embrasure between the left canine and the first bicuspid tooth. If this were to be a screw-retained hybrid-type restoration, the implant could be repositioned to achieve a better location for the screw access hole. Once the implant positions have been verified, the oral surgeon can confer with the referring clinician, and prepare for the surgical intervention. Based upon the accepted virtual plan, a CT-derived bone-borne or soft-tissue borne template could be fabricated - referred to by the author as “template-assisted” surgery. Or the surgeon can place the implants free-hand, using the CT data as provided by the plan to guide the implant placement



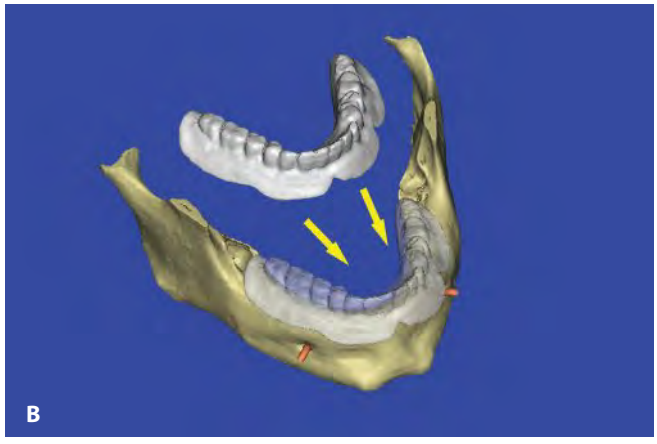


FIGURE 8-41. The radiopaque scanographic template helps visualize desired tooth position in relation to the underlying bone.

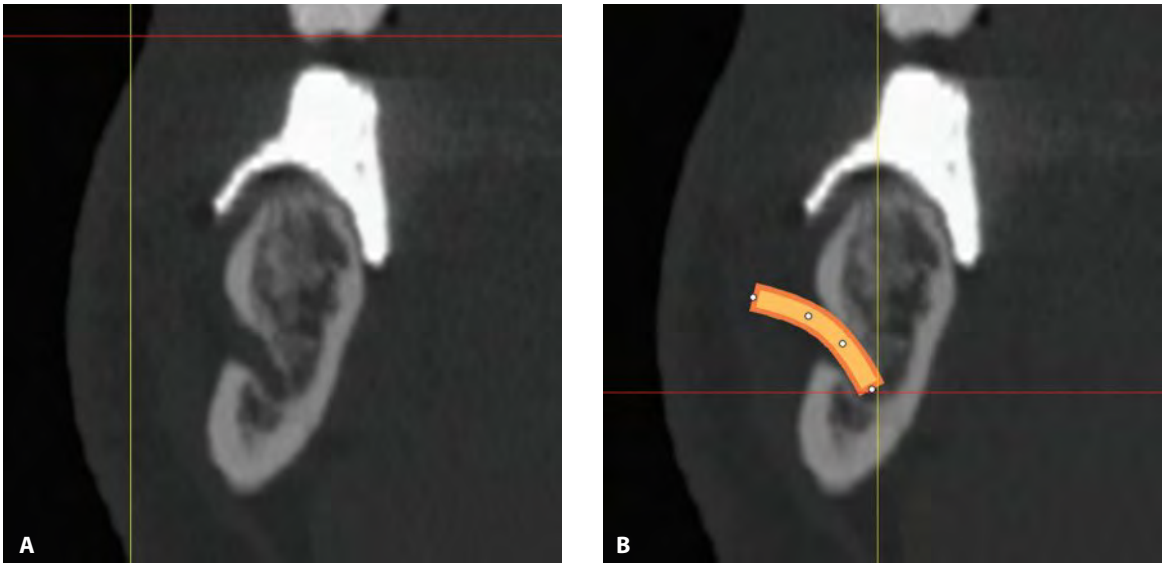


FIGURE 8-43. The mental foramen is noted **(A)** and the path of the mental nerve traced **(B).**

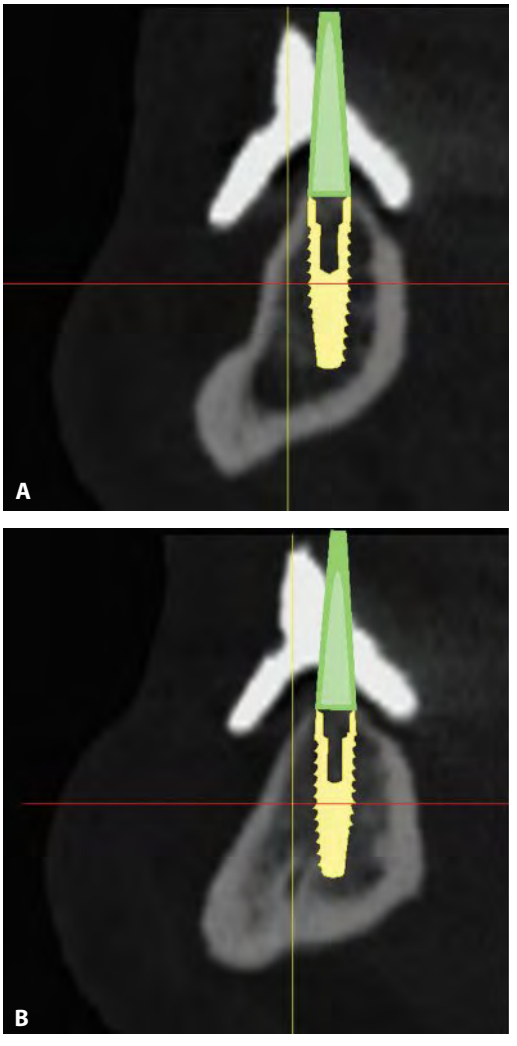


FIGURE 8-44. **A** and **B,** Using the radiopaque template as a guide, implants are positioned within the bone and restorative envelope.

based upon computer print-outs of the various slices - referred to as "CT-assisted" surgery.

Upon inspection of the entire mandible in either the panoramic or axial view, additional significant anatomy

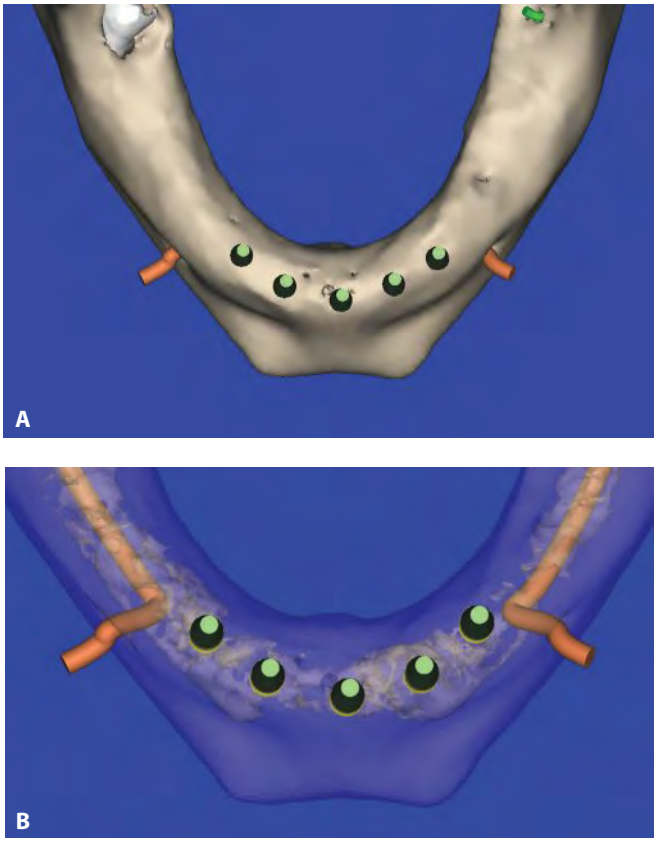


FIGURE 8-45. **A,** If desired, the implants can be placed in a parallel orientation with appropriate implant-to-implant spacing. **B,** Selective transparency allows visualization of the mental nerves and proximity to implant placement.

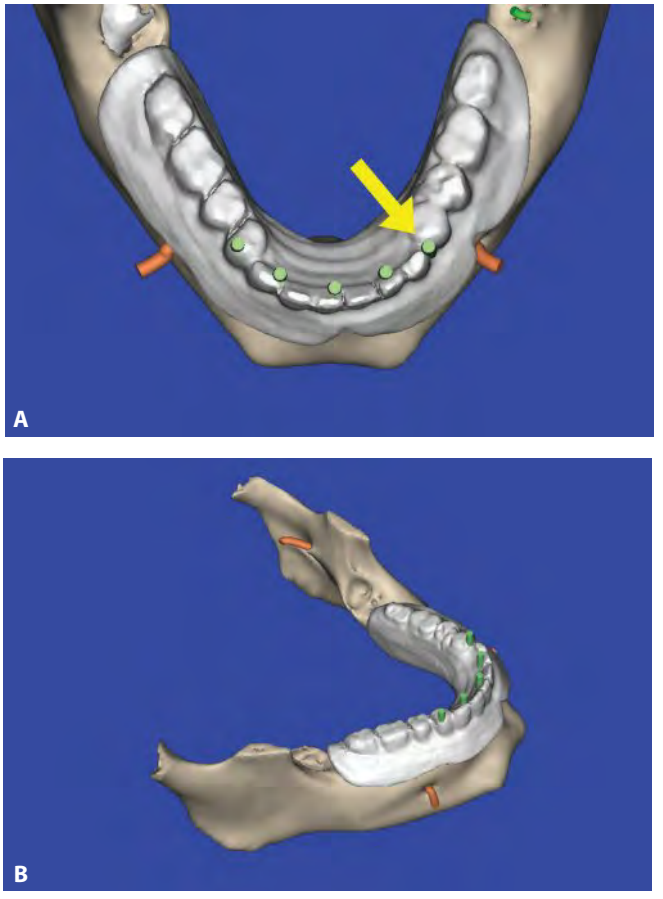


FIGURE 8-46. **A** and **B,** The initial positioning of the implants can be verified by superimposition of the scanning template. The final implant placement can then be finalized based upon the restorative envelope.

was discovered. In the posterior right side mandible, a horizontally impacted molar was identified (see Figure 8-47). The ability to zoom in and focus on a small area of interest is an important feature of three-dimensional imaging technology. The close-up axial view reveals the horizontal tooth position with the clinical crown facing to the lingual (see Figure 8-48A). Scrolling through the next axial slice revealed the dilaceration of roots embedded in the thick facial cortical bone (see Figure 8-48B). The proximity to the inferior alveolar nerve can be confirmed in the cross-sectional view if the nerve had been previously traced to the posterior aspect of the mandible. (see Figure 8-49). If the software allows for the tooth to be virtually separated from the bone, the coronal aspect of the impacted tooth would be visible in the three-dimensional reconstructed view (see Figure 8-50). The area of interest can be expanded to clearly visualize the molar, the extent of the bone surrounding the coronal portion, and its proximity to the path of the inferior alveolar nerve using transparency features of the software (see Figure 8-51). Rotation of the three-dimensional mandible allows for a lingual view of the impacted molar and the relationship to the nerve from another perspective (see Figure 8-52). The bone can be totally removed

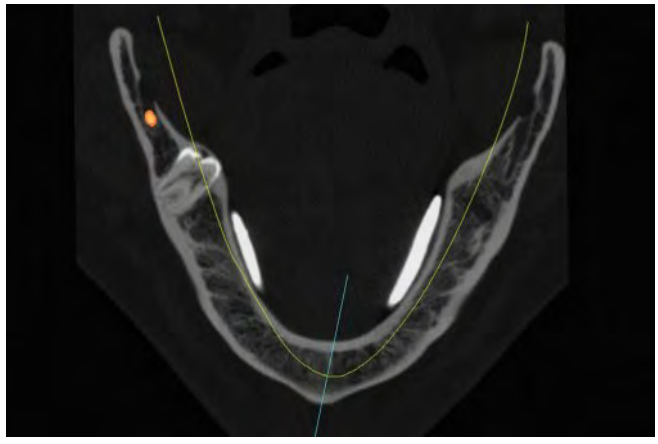


FIGURE 8-47. The axial view revealed a horizontally impacted third molar.

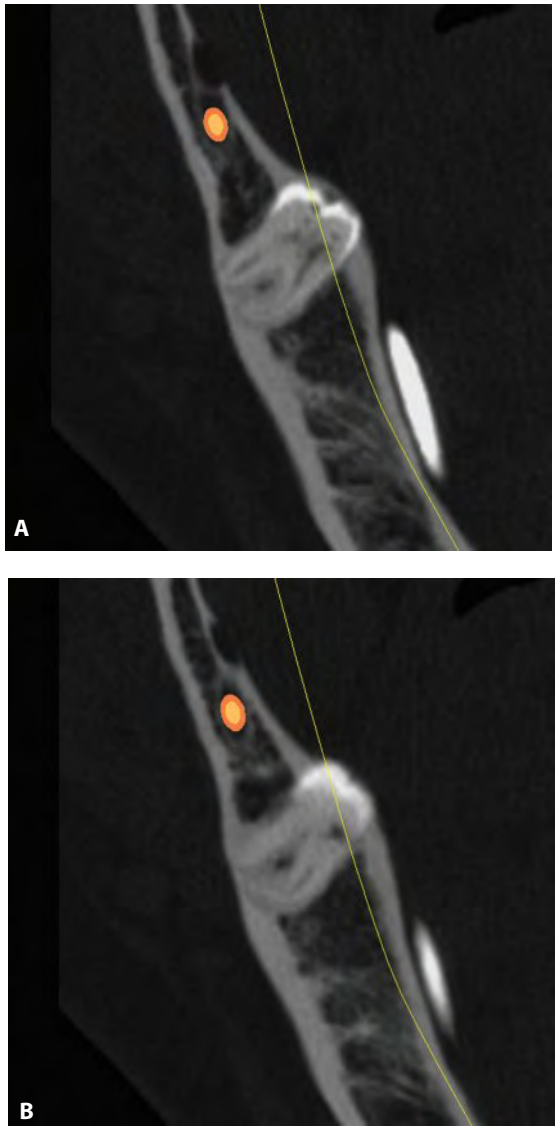


FIGURE 8-48. A close-up axial view reveals the tooth position **(A)** and the dilaceration of the roots embedded in the facial cortical bone **(B).**

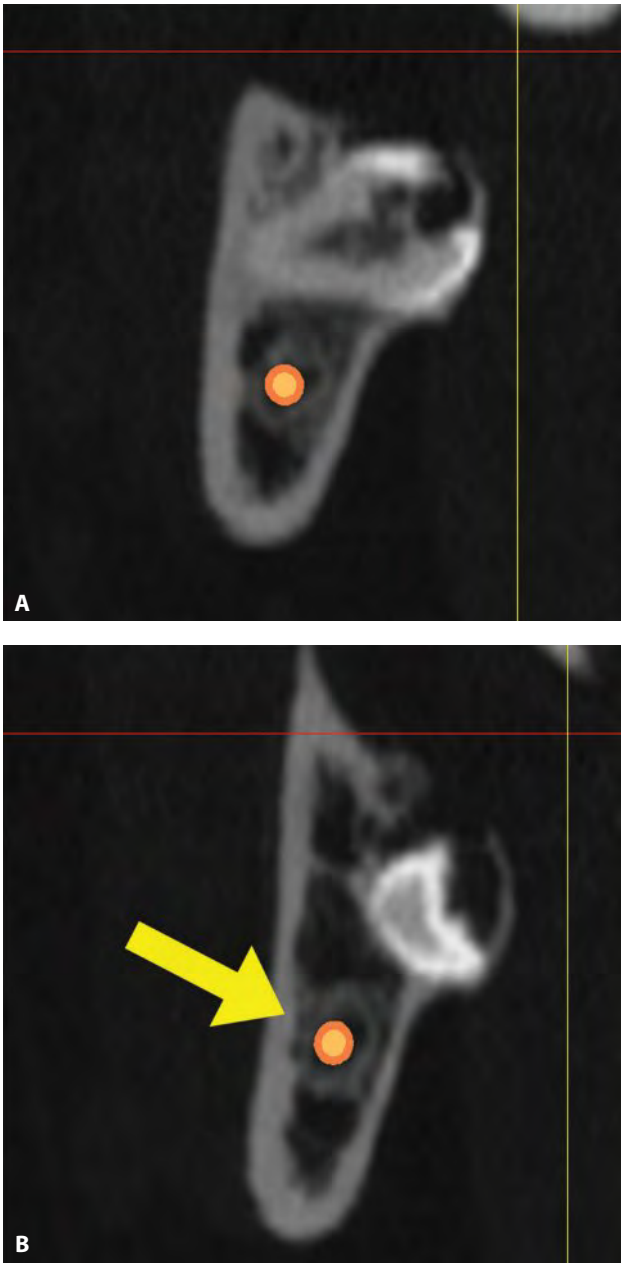


FIGURE 8-49. **A** and **B,** The cross-sectional view illustrates the relationship between the impacted molar and the inferior alveolar nerve.

to provide an even closer inspection of the vital anatomy and root morphology (see Figure 8-53).

Fortunately, in this example, there was adequate spacing between the molar tooth and the nerve to avoid potential damaging complications. If the molar required removal, these views would prove to help guide the oral surgeon in planning the surgical intervention with the least amount of patient morbidity.

If the posterior ramus were to be utilized as a potential donor graft site, the three-dimensional reconstructed view would provide ample information to determine whether the procedure could be successfully accomplished (Figure 8-54).

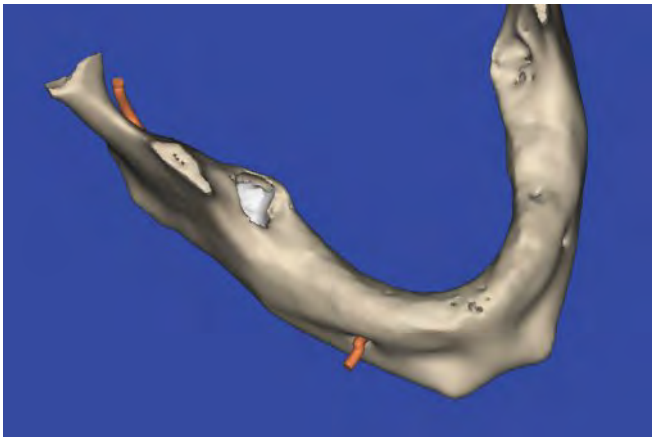


FIGURE 8-50. The three-dimensional reconstructed view reveals the coronal aspect of the impacted molar.

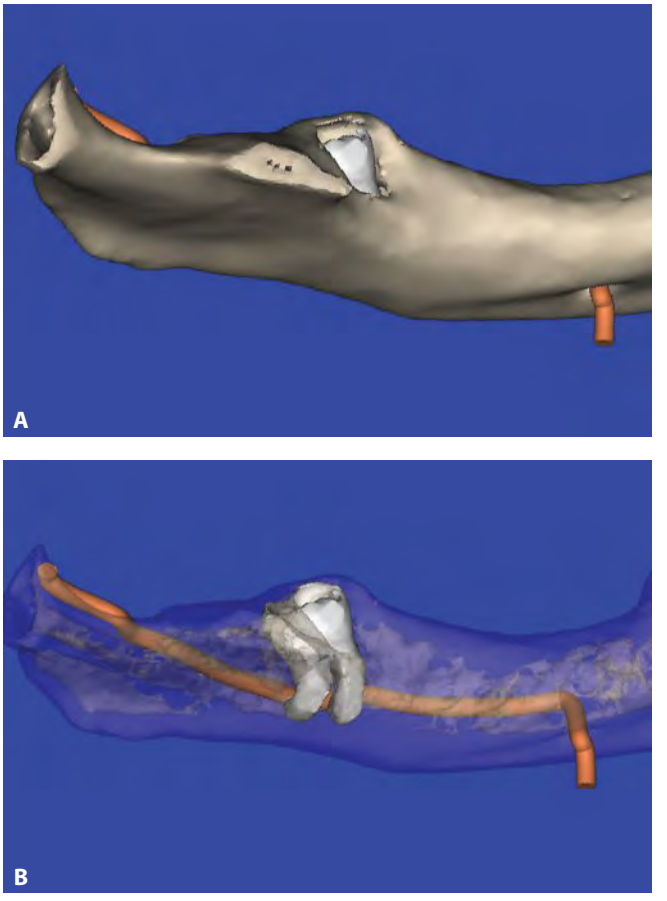
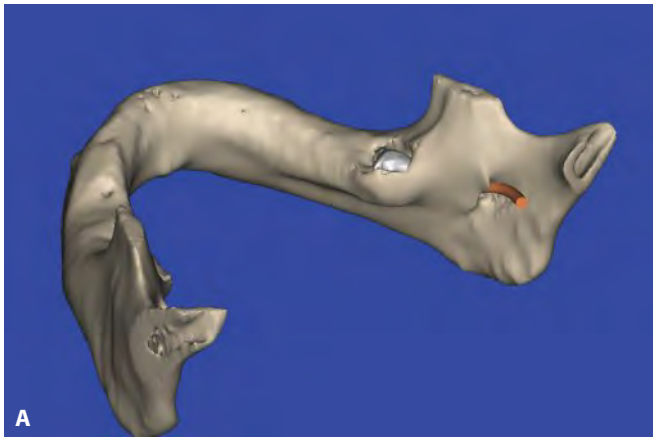


FIGURE 8-51. The close-up view of the impacted molar **(A)** more clearly visualized near the inferior alveolar nerve by modifying the transparency of the mandibular bone **(B)**.

Whereas the bone might look excellent during the harvesting procedure, without a complete understanding of the underlying vital anatomy, complications could occur. Therefore, it is imperative to locate the path of the inferior alveolar nerve in relation to the donor site. The cross-sectional view reveals the thickness of the facial cortical plate and the proximity of



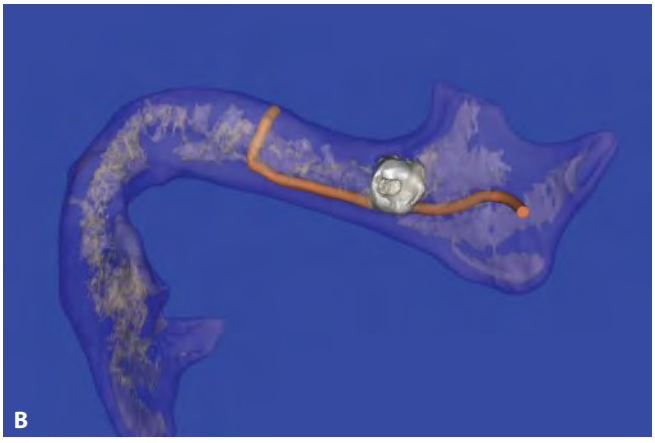


FIGURE 8-52. Rotating the three-dimensional mandible allows for a lingual view of the impacted molar **(A)** and proximity to the inferior alveolar nerve **(B).**

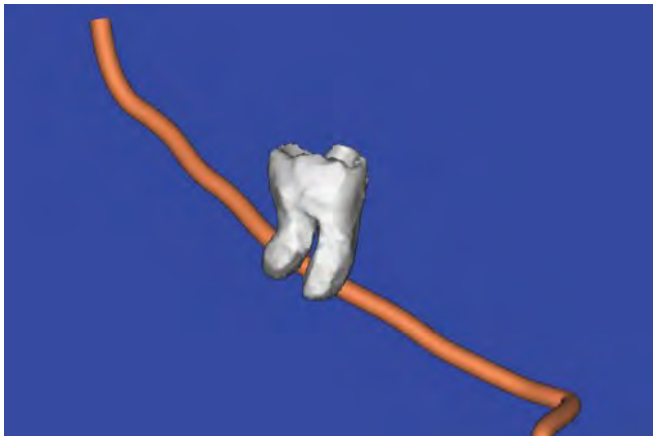


FIGURE 8-53. The bone can be removed totally to allow for a close inspection of the vital anatomy.

the inferior alveolar nerve (Figure 8-55). If there was adequate clearance, and the quality of bone sufficient for the bone to be utilized as a donor, a plan can be formulated that would allow for proper harvesting while limiting potential

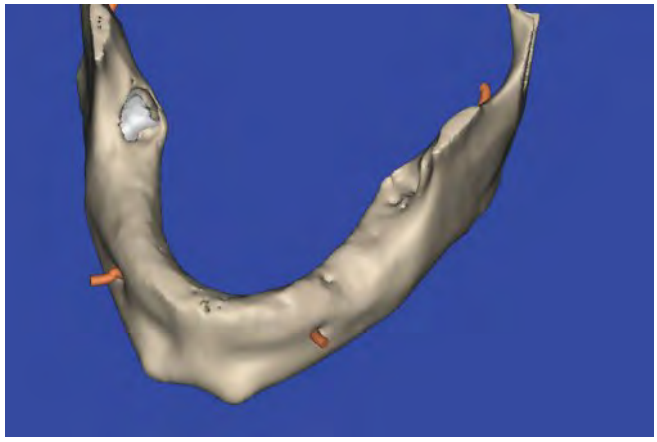


FIGURE 8-54. Assessing the ramus area as a potential donor graft site.

complications. Using advanced software specifically designed for oral surgery applications, different procedures can be simulated including segmental osteotomies and more. Once the dimensions of the donor site have been established, the surgery can be simulated on the three-dimensional model. The donor graft can be virtually removed from the mandible and positioned where it is needed to augment the distant receptor site (Figure 8-56). If the receptor site was part of the three-dimensional reconstructed maxilla, it could be moved to the site to see if it fit properly. This can all be accomplished before touching the scalpel to the patient.



FIGURE 8-55. The cross-sectional view reveals the thickness of the facial cortical plate and the proximity of the inferior alveolar nerve.

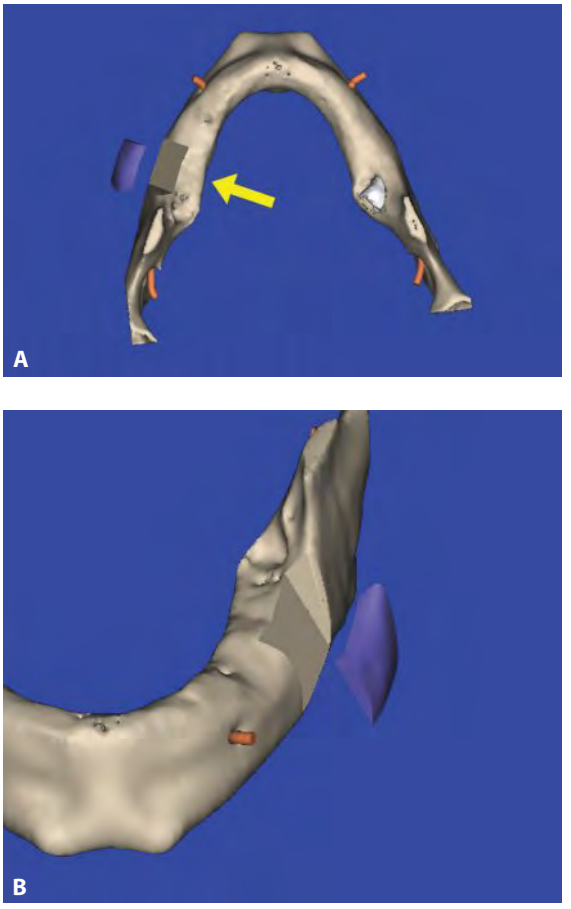


FIGURE 8-56. **A** and **B,** The bone graft of correct dimensions can be virtually "removed" from the donor site and positioned where it is needed to augment the receptor site.

CONCLUSION

It has been well documented that panoramic and periapical radiography are limited in diagnostic accuracy due to the inherent distortion factor, overlap of adjacent structures, the inability to realistically evaluate spatial relationships, assess bone width, or quality. The use of CT/CBCT resolves these issues by accurately supplying undistorted three dimensional assessment of bone width, density, location of nerves, vessels, volume of the maxillary sinus, location of septum, root morphology, the temporomandibular joint complex, spatial position of impacted or supernumerary teeth, and magnitude of potential pathological lesions. The improved diagnostic acuity affords the clinician with the vital information required to formulate and present one or more treatment options to the patient.

The ability to interactively assess patient anatomy with three dimensional imaging has empowered clinicians with an expanded set of tools which can greatly improve diagnostic accuracy. Applications for the use of this technology apply to all specialities of dentistry, but are particularly suited for oral surgery procedures as illustrated in this chapter. The examples are almost limitless as each patient presents with an individual set of anatomical realities which need to be appreciated prior to the development of a definitive treatment plant. Virtual simulation of "routine" extractions can be found to be anything but routine after reviewing the axial or cross-sectional slices of a CT/CBCT scan of the region in question. The shape and dimension of a defect requiring bone grafting can be pre-operatively assessed and visualized in great detail so as to understand the volume required to manage the site, helping to decide between particulate or block grafting techniques. Implant or bone grafting receptor sites can be evaluated for thickness of the buccal or lingual cortex, density of the intermedulary bone, and proximity to vital structures. Manipulation of the 3-D interactive data with new and innovative software tools can yield unprecedented views which allow for inspection of the patient's unique anatomical presentation and provide the means to remove guesswork from the process.

The case examples contained in this chapter were selected to illustrate how three-dimensional imaging and interactive software applications can significantly enhance the diagnosis and treatment planning process through the use of a variety of different applications for the technology. It is important to note that the concepts presented are not limited to specific case types, but provide the potential for expansion into every area of dentaloalveolar surgical, implant, and prosthetic reconstruction. It is the author's opinion that the utilization of CT/CBCT diagnostic imaging by the oral surgeon will significantly impact on the everyday practice workflow including; case presentation, reduction of potential complications, supplemental medical-legal documentation, improved treatment planning capabilities, enhanced communication with referring clinicians, and reduction in patient morbidity during and after the ultimate surgical intervention.

ACKNOWLEDGEMENTS

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CHAPTER 9

Implant Prosthodontics

Thomas J. Salinas, DDS

BIOMECHANICAL CONSIDERATIONS

Peri-Implant Biology

Considerations for tooth replacement with osseointegrated dental implants include the biologic principles of soft and hard tissues of adjacent teeth to the implant site. The placement of an implant between two periodontally healthy teeth is a unique situation whereby the bone and soft tissue are maintained in part by the teeth. Original studies by Waerhaug[1] and Gargiulo and colleagues[2] showed the width of the dentogingival complex surrounding natural teeth approaching 3 mm. Although the peri-implant tissues are fundamentally different, a study by Cochran and coworkers[3] assimilated the peri-implant tissues to a similar dimension. Based on these principles, the suggested depth of placement of an implant below the free margin of soft tissue is approximately 3 to 4 mm (Figure 9-1). This distance provides room for biologic width, proper emergence of restoration, and aesthetics and also should allow for remodeling of the soft tissue and bone, which occurs between 6 months and 1 year.[4] It has been postulated by some that the type of periodontium influences how extensive this remodeling process is. In other words, thin, scalloped gingiva recedes more extensively than does thick, nonscalloped gingiva.[5,6] Restorative interfaces with metal should be kept below the free margin of tissues in anticipation of this remodeling. Tarnow and associates[7] have shown that there is a relationship of the underlying bone to soft tissue in the interdental spaces between natural teeth. A relationship from both implant to natural tooth and implant to implant has been demonstrated.[8] Therefore, the distance suggested from the side of the implant to the adjacent tooth should be about 2 mm to avoid horizontal bone loss affecting the adjacent tooth. Similarly, Tarnow and colleagues[8] showed the critical distance between implant surfaces approached about 3 mm before the mutually destructive process of lateral bone resorption accelerated each other's processes (Figure 9-2). Typically, each implant loses peri-implant bone within the first year and then stabilizes—one criterion of success as outlined by Albrektsson and coworkers.[9] Whereas this pattern of bone loss is typically followed by Branemark-type implants, other types of implant systems demonstrate a different pattern of bone loss that can emulate one of four different patterns.[10]

Patient Factors

Soft tissue evaluation before implant placement is critical for long-term success and maintenance. A sufficient volume of keratinized and fixed tissue is needed to properly maintain hygiene around an implant, just as it is needed around a natural tooth. Occasionally, it may be necessary to incorporate subepithelial connective tissue or full-thickness soft tissue grafts to prospective implant sites. When restoring single missing teeth, the interproximal bone between the remaining

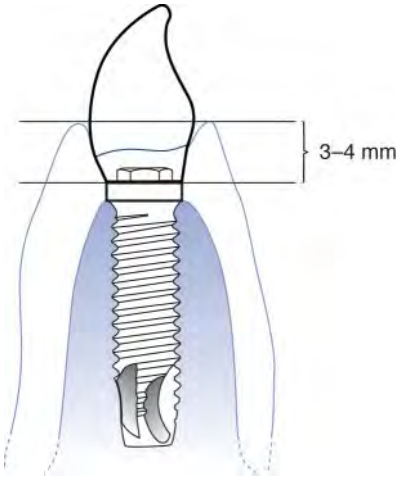


FIGURE 9-1. Osseointegrated implant placed at a depth of 3 to 4 mm for biologic width and emergence profile.

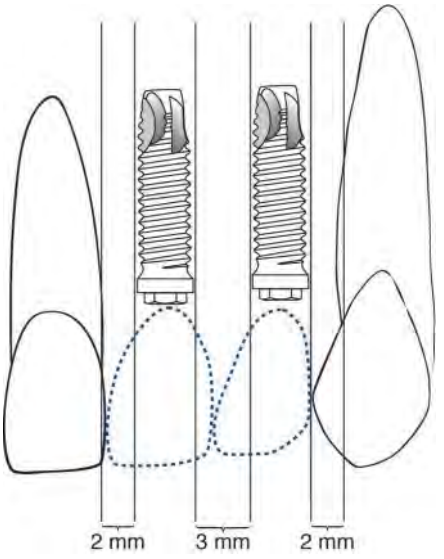


FIGURE 9-2. Suggested minimum distances of implant to natural tooth and implant to implant.

teeth is a good prognostic indicator of the likelihood of creating and preserving interdental papilla. Generally, the distance from the residual alveolar bone to the contact area of the restoration can be assessed on a periapical film. The likelihood of having a papilla is depicted in Table 9-1.

Bone volume is best assessed by radiographic techniques, although a rudimentary estimate can be made clinically by palpation and inspection. Assessing a patient for mandibular implant reconstruction may include intraoral/extraoral palpation as well as panoramic, occlusal, and lateral cephalometric radiographs. Single-tooth replacement in the aesthetic zone also can be assessed by comparison of the bony topography of the adjacent teeth as well as periapical/panoramic radiographs. Bone is a scaffold for soft tissue, and it is typical for bone loss to occur on a scale of 0.2 mm/yr after stabilization of initial bone loss pattern. Therefore, it is not unusual that soft tissue recession occurs in this period of time. This recession should be anticipated, especially when considering placing implants in the aesthetic zone and elsewhere.

It is well documented that local and systemic factors such as cigarette smoking have a deleterious effect on the long-term success of dental implants.[11–13] In one study, failure rates of implants placed in type 4 bone approached 35% in smokers; placement of implants into types 1, 2, and 3 bone of smokers resulted in a failure rate approaching 3%.[14] A recent publication on surveying the success of moderately roughened surfaces discloses a more favorable outcome in smokers and also in type 4 bone.[15]

TABLE 9-1. **Potential of Creating/Preserving Papilla**

Distance from Bone to Contact Area (mm)	Chance of Creating Papilla (%)
4.0	100
5.0	100
6.0	56
7.0	27

Adapted from Tarnow DP, Magner AW, Fletcher P. The effect of the distance from the contact point to the crest of bone on the presence or absence of the interproximal dental papilla. J Periodontol 1992;63:995–996.

Although osteoporosis can be a negating factor to bone density, this disease seems to affect the hip and spine of those afflicted. No clear correlation can be demonstrated that osteoporosis is a contraindication to the placement of dental implants.[16]

Periodontal disease is a local factor that should be under control to avoid adverse effects of a unique population of microbiota affecting these diseased sites.

Bruxism is another local factor that can compromise long-term success. Generally, bruxism promotes micromovement of the implant bone interface. In bone types 3 and 4, bruxism may have a more pronounced effect on the long-term osseointegration. Off-axis and lateral loading of dental implants by bruxism or other parafunctional forces can be deleterious in the long term with respect to accelerated bone loss and prosthetic failure. Self-awareness and occlusal splint therapy may provide appropriate protection. If these factors cannot be controlled preoperatively, alternative treatment should be considered.

Radiation to the head and neck in excess of 50 Gy is considered a contraindication to dental implant placement in most cases. There are instances in which the radiation has created a significant degree of xerostomia, which is incompatible with retaining natural teeth or stabilizing prostheses. Given the risks of osteoradionecrosis, hyperbaric oxygen should be considered if placement of implants would significantly improve the oral health and quality of life in these individuals.[17–19] However, several studies refute the benefit of hyperbaric oxygen to the long-term survival of dental implants.[20,21] Standard protocol suggested by Marx and Ames[22] is 20 preoperative dives and 10 postoperative dives.

Systemic factors such as diabetes, connective tissue diseases, autoimmune diseases, and human immunoceficiency virus (HIV) are considered relative contraindications to treatment with osseointegrated implants. If these disease processes are well controlled, it may be advisable to treat the patient to improve the overall quality of life. Chemotherapy given to patients during osseointegration has not been shown to be subtractive in success.[23–25]

Radiographic Evaluation

Periapical radiographs are an excellent way to evaluate single missing teeth because they depict a minimally magnified amount of bone and root topography. Adjacent root angulation, pulp chamber size, periodontal defects, interproximal bone, and residual pathology are some of the factors critical to the treatment planning of single-tooth implant restorations (Figure 9-3).

Occlusal radiographs for mandibular arch assessment also can give an appreciation of the size of the buccal and lingual cortices as well as the position of the mental foramina

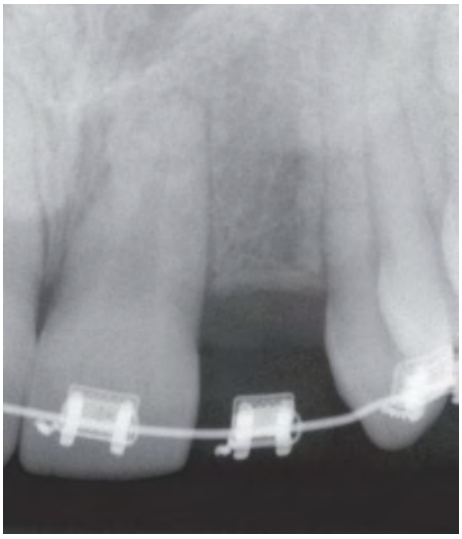


FIGURE 9-3. Presurgical planning for placement of an implant into site no. 10. Minimal magnification is noted from the periapical radiograph.

(Figure 9-4). It may be also feasible to incorporate a radiographic marker on the patient's denture to give a perspective of the relationship of the mental foramina to the overlying prosthesis. This can be done with either lead foil from a film packet adhered to the underside of the patient's denture or a stainless steel wire attached to the buccal or occlusal portion of the mandibular denture.

Panoramic radiographs are excellent screening examinations that give a broad perspective on the inferior alveolar canal, maxillary sinuses, mental foramina, and nasal floor; they are used for treatment planning of single and multiple missing teeth. The panoramic film generally has a magnification factor of about 25%, which should be anticipated on the workup to gain a better appreciation of the actual position of vital structures and the size of implant to be selected. Methods of standardizing the magnification factor include the use of known-diameter stainless steel shots incorporated in a vacuum-formed stent worn at the time of radiography (Figure 9-5). This varies from patient to patient, by location,

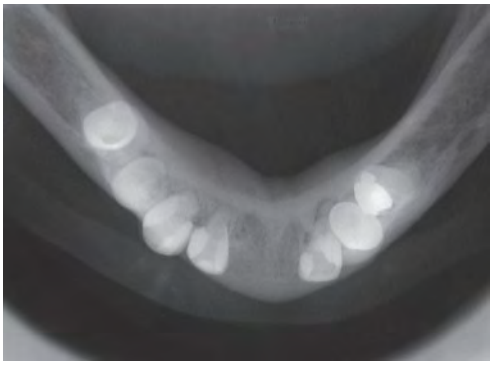


FIGURE 9-4. Occlusal radiograph gives the relative position of mental foramina and the taper of mandible.

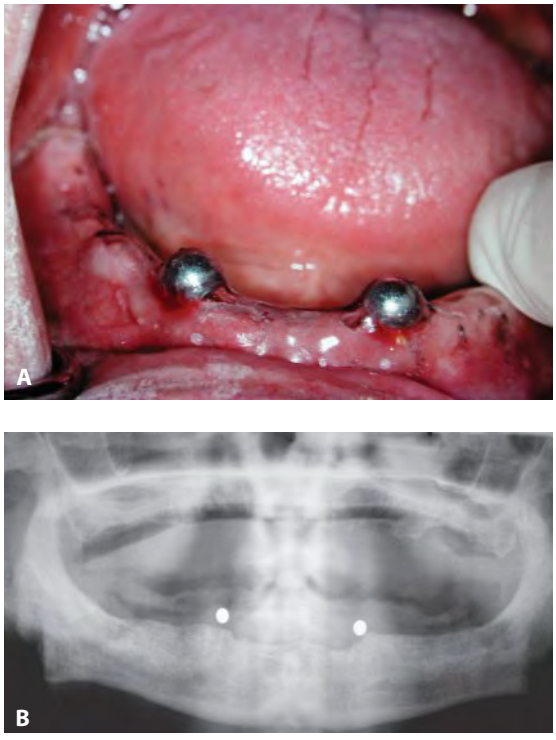


FIGURE 9-5. **A** and **B,** Five-millimeter stainless steel shots in vacuum-formed stent to calculate the magnification factor.

and also with the machine used. Panoramic radiographs are also useful for verifying complete seating of impression and restorative components. Use of this film over a standard periapical radiograph is preferable because the incident beam of the tube is more likely to be perpendicular to the long axis of the implant. Also, many edentulous patients have a shallow floor of mouth and flat palatal vault owing to resorption. It is far easier to obtain a perpendicular view of the implant platform in these circumstances, which is critical to the accurate performance in the treatment stages.

Lateral cephalometric radiographs assess the maxillomandibular relationship as well as that of the maxilla and mandible to the cranial base. A lateral cephalogram may give an appreciation of the concavity of the lingual surface of the anterior mandible vitally important to surgical consideration of implants in the anterior mandibular area. Development of anticipated implant occlusion is well assessed with lateral cephalography, which becomes especially useful when recreating anterior guidance and posterior occlusal schemes (Figure 9-6).

Cone beam computed tomography (CT) is a more contemporary technique that gives a reasonable three- dimensional perspective to the interpretation of the adjacent anatomy of a proposed implant site. This technique may be indicated in an implant placement scenario in which the volume of bone is compromised or diminished, such as an extensively resorbed jaw or prominent maxillary sinus or inferior alveolar canal (Figure 9-7). The advantage of using the cone beam technique is a markedly reduced radiation dose,



FIGURE 9-6. Lateral cephalograms may assist in the workup for determining maxillomandibular relationships and occlusal schemes.

increased survey volume, and a reduced scan time in comparison with conventional single-plane CT.

CT can be helpful when considering maxillary rehabilitation with a full complement of implants or when other craniofacial landmarks are planned for use. CT may be used in conjunction with computerized technology to aid implant placement. These images may be reformatted to construct a three-dimensional image of the selected part of the craniofacial skeleton. CT scans are useful in assessing the health of the maxillary sinus before augmentive procedures (Figure 9-8).

A radiographic or imaging stent can be used when there is a need to join the prosthetic information to the bony topographic information. In creating these stents, acrylic resin can be mixed with 30% or less barium sulfate as a radiographic marker to create the contour of the intended restoration. Some denture teeth are true to anatomic form and create a radiopaque appearance when included in the stent. As an alternative, access channels can be filled with gutta-percha as a radiographic marker. If verified radiographically, this imaging stent may double as a surgical stent.

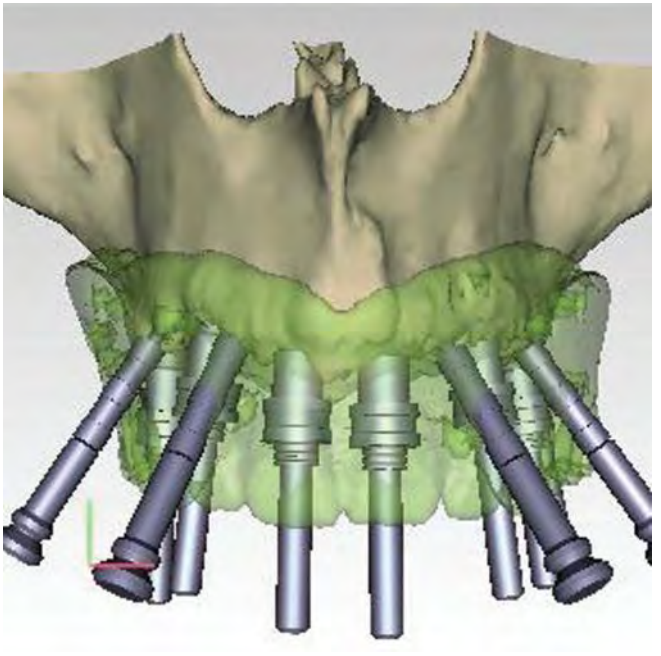


FIGURE 9-7. Cone beam imaging technique to show hard tissue deficit related to proposed positions of teeth.



FIGURE 9-8. Three-dimensional reconstruction with computer software manipulation of computed tomographic data of a patient with a maxillectomy.

Surgical Stents

Fabrication of surgical stents for implant placement should be part of every case because the placement is permanent and irrevocable after integration. Planning of each case includes the collection of all diagnostic data, as previously mentioned. Once this data has helped create a thorough treatment plan, fabrication of a surgical stent can begin from the diagnostic models and other information from the workup.

Construction of prostheses begins with a confirmation of occlusal relationships and the need to direct occlusal forces over the long axes of the implants. This becomes exceptionally critical when a fixed restoration is to be used. On this basis, a site is selected and a stent made to guide the surgeon at placement (Figure 9-9). This information may also be translated from radiographic findings to a surgical stent in the position of the mental foramina (previously described).



FIGURE 9-9. Stent used to place the implants within the confines of the denture base.

This information can be used to place implants far enough away from the foramina and each other to be mechanically advantageous. Again, parallelism is of paramount importance if a stud-retained overdenture is used. This stent can be as simple as a vacuum-adapted thermoplastic sheet over an edentulous cast or a clear processed duplicated denture.

Implant-supported overdenture construction may incorporate the use of the surgical stent to keep the implant fixtures away from the peripheral confines of the prosthesis. This may be beneficial to avoid encroaching on the peripheral seal in either aesthetic or functional areas. Also, occlusal forces may be better directed over the long axes of the implants. The stent can be either a duplicate of a diagnostic wax-up in clear resin or simply a duplicate of the patient's denture, if acceptable. A stent may be critical in this situation because it will be supported with a splinted structure in which cantilevering may be used. More sophisticated stent design can incorporate the use of the cone beam CT planning to positionally coordinate implant placement within the anatomic confines of the jaw. These stents contain a series of placement guides that telescope to the final placement drill guide, making precision placement attainable in compromised host sites.

Fixed-implant–supported complete dentures mandate the use of a surgical stent because the occlusal access channels are desired to be through the posterior teeth and the lingual aspects of the anterior teeth. In these situations, a slot can be created through these areas to provide the surgeon with latitude in site selection. A clear processed duplicate of the patient's denture may be the best technique in surgical stent design. If immediate loading protocols are to be used, the patient's complete denture can be used for this purpose and modified to fit to the placed implants.

Surgical stent design for fixed prostheses is mandated in that selection of a specific prosthetic design may be entirely dependent on implant position and orientation. In the aesthetic zone, the cemented design may be the preferred method of prosthesis, and placement of an implant in an orientation just palatal through the incisal edge is optimal. Also, the implant platform should be approximately 3 to 4 mm below the free edge of the gingival margin. Two vital pieces of information contained on a surgical stent are the occlusal/incisal plane and gingival margin of the proposed restoration (Figure 9-10). To obtain this information, a wax-up is performed in the desired occlusal position. Once completed, this model should be duplicated into another cast. A vacuum-adapted stent can be made on this duplicate cast. The matrix can be trimmed with a hot knife and rotary instrument. Guide channels can be created with previously surgical drills or laboratory burs. The constant access diameter of these stents is based on the concentric enlargement of each succeeding drill diameter. These stents are usually easily made, are cost effective, are self-retaining, and do not require prefitting. because these stents fit well, it is only necessary to extend the stent two to three teeth on either side of the edentulous spaces for partially dentate cases.

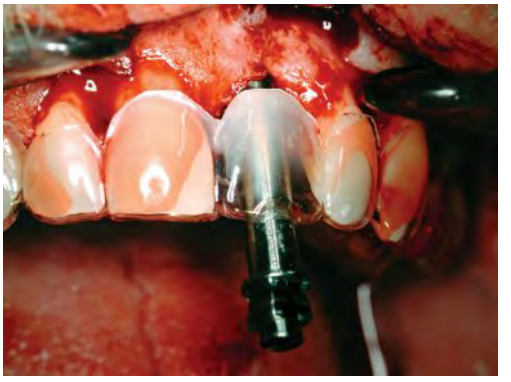


FIGURE 9-10. Surgical stent showing proposed gingival margin and incisal/occlusal plane. (Surgery performed by Michael S. Block, DMD.)

Crown-to-Implant Ratio

Ideally, a crown-to-implant ratio of 1:1 or less is desired (Figure 9-11). For this reason, the minimum length needed approaches 10 to 12 mm because the clinical crown length frequently approaches this measurement. Often, replacement of teeth in a compromised site gives rise to single or multiunit restorations that have poor or unfavorable crown-to-implant ratios. If the restoration participates in anterior guidance, it should be splinted to other implants. If the restoration participates in posterior occlusion, it should be protected by natural canine teeth to limit lateral loads in excursions. If it is placed in conjunction with other implants in the posterior, it may be splinted for mutual support.

Occlusion

Several axioms in implant dentistry relate to occlusion:

- Avoid lateral component forces whenever possible.
- When restoring occlusion of an entire arch, favor the weaker of the two arches. (In other words, an implant-borne restoration opposing a complete denture should be restored with bilateral balanced occlusion.)

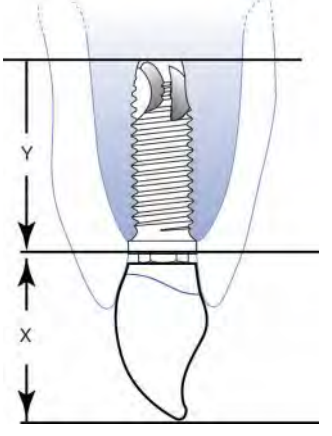


FIGURE 9-11. Ideal crown-to-implant ratio occurs when X ≤ Y.

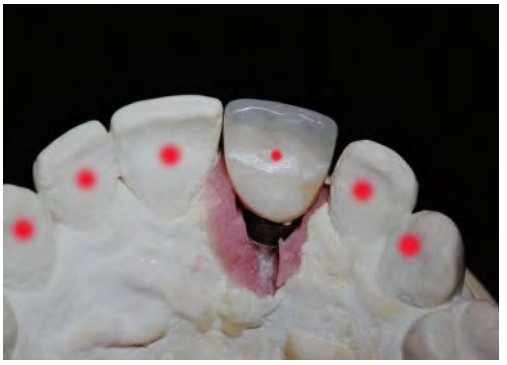


FIGURE 9-12. Contact of the implant occlusion should be over the platform of the implant and slightly less intense than that of natural teeth.

One additional consideration is that, unlike natural teeth, implants have no proprioception. In fact, many patients restored with dental implants have a significantly increased bite force within the first year.[26–28] In partially dentate cases, the implant restoration should have equal or slightly less occlusal loading than the natural tooth (Figure 9-12). Also, the occlusal contacts should preferably be placed over the platform of the implant to minimize the possibility of screw loosening. Although this often may not be possible, it should be striven for to minimize complications.

Full-Arch Restorations

Although generally recommended, complete arch reconstructions of the maxilla has been based on placement of 8 to 10 implants splinted for cross-arch stability.[29–32] Reasonable length implants (>10 mm) should be considered, especially in the posterior maxilla because shorter implants into this relatively soft bone have been shown to do poorly in the long term.[14] The maxillary sinuses may preclude placement of a full complement of implants, and sinus augmentation or perhaps the use of extended-length implants into the zygomatic bones bilaterally may allow an optimum force distribution for full-arch prostheses (Figure 9-13). Additional publications have explored the used of using four implants placed anterior to the maxillary sinuses by incorporating a tilt to the posterior implants, thereby increasing the anteroposterior spread.[33,34] This technique has been further augmented by the use of immediate load and splinting with a provisional prosthesis. The use of cortical stabilization of all implants, the use of implants greater than 13 mm in length, and risk profiling to avoid smokers, bruxers, and those with poor bone quality hasve seemed to be favorable with initial success.



FIGURE 9-13. Full-arch reconstruction using two zygomatic implants and three endosseous implants.

Full-arch reconstruction of the mandible can involve different considerations because the mandible is a dynamic bone that flexes and rebounds as it opens and closes. In addition, treating edentulous patients by the use of the approach of four to six implants between the mental foramina with a minimal cantilever to the posterior was used in the pilot studies of osseointegration.[35,36] The greater the anteroposterior spread, the greater the amount of cantilever possible. On average, a 16-mm distal cantilever is permitted (Figure 9-14). Although successful for edentulous patients, this model is not universally applicable to all configurations of an edentulous mandible and may be limited by the amount of anteroposterior spread of the implants. To avoid using a cantilever, it may be necessary to place implants distal to the mental foramen. In such a case, division of the prosthesis into two components prevents unfavorable stress transfer. Another option is to use the distal fixtures for vertical support and not engage the abutment-implant junction with an abutment-coping screw. This allows some flexure of the mandible without transferring stress to the prosthesis and/or implants.

Implant Selection

Historically, osseointegrated dental implants were introduced in their original configuration as a machined parallel-walled screw. The implant possessed a platform with a 4.1-mm diameter, an external hex implant platform (originally used to drive the implant into position), and a 3.75-mm-diameter body; this has been the most common implant type placed worldwide (Figure 9-15). The original applications were piloted for the edentulous patient, and limited restorative options were available in the first years of its introduction. In later years, the use of surface-textured press-fit type implants also became popular because their surgical installation was simplistic and achieved earlier integration into softer types of bone (Figure 9-16). At this time, the connection of abutments or prostheses to the surface of the implant was characterized as a butt-joint connection. Abutment stability with single- and multiple-tooth replacement using standard externally hexed implants has a history of cyclic fatigue with abutment screw loosening. As extended applications developed for the use of replacements for single and multiple teeth and with immediate loading, an increased need for secure abutment connections, aesthetic versatility, and improved surgical stability in trabecular bone became more apparent. Significant mechanical improvement in abutment and screw-retained components occurred in the early 1990s and markedly

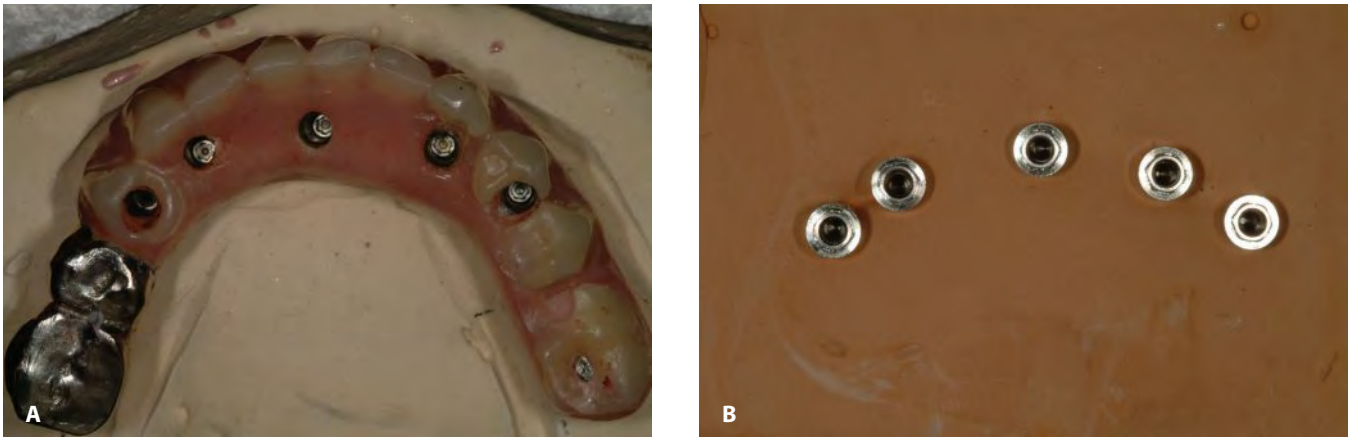


FIGURE 9-14. **A** and **B,** Mandibular edentulous arch with limited anterior posterior spread limiting the amount of posterior cantilever extensions.

decreased complications.[37] Since the early 2000s, industrial trends are toward the use of tapered macroretentive implant configurations, based on the fact that tapered screw-type implants have increased surgical stability in soft bone. An example of these types of implants is shown in Figure 9-17. Implant surfaces have also changed in the early 2000s to incorporate the use of moderately rough implant surfaces. The roughness values are often denoted by two variables: the first is a three-dimensional measurement of the peak-to-valley arithmetic mean (Sa value); the second is a measure of the randomness of surface roughness called "isotropism" In animal studies, it has been demonstrated that isotropic (regularly irregular) surfaces with Sa values of 1 to 2 µm elicit the more robust bone response.[38]

The Morse taper, a cone within a cone attachment mechanism, is a feature of some implant systems that allow the abutment-prosthetic connection to facilitate installation and to maintain stability (Figure 9-18). This taper creates a seating effect of the connection to the internal aspects of the implant; therefore, fewer lateral stresses are transferred to the abutment screw, resulting in a less frequent incidence of screw loosening and fracture. Morse tapers are measured in percentage units that reflect the shaft length relative to the radius of the shaft. Thus, if for every centimeter of shaft the radius increases 0.01 cm, this would by definition be a 1% Morse taper. Most Morse tapers are anywhere from 1% to 7%, and dentistry most commonly employs the 4% to 7% series. Use of specific implants resistant to the problems of abutment screw loosening and immediate stability is probably more critical in cases of single missing teeth or in which a cemented implant crown and bridge are planned. The traditional parallel-walled screw continues to enjoy success in the general population of edentulous patients restored with implants. The vast majority of prospective and retrospective studies have concluded that this specific implant is highly successful for restorations in edentulous patients. Long-term development has resulted in an increased number of components for edentulous and partially dentate applications. It is advisable for the surgeon to become familiar with the restorative components available when treatment planning for implants cases.

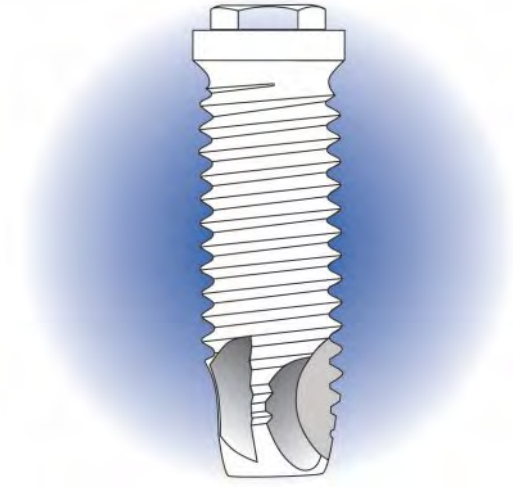


FIGURE 9-15. Standard externally hexed implant.



FIGURE 9-16. Press-fit cylinder-type implant.

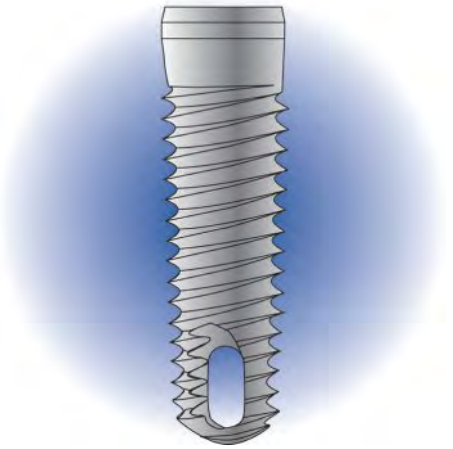


FIGURE 9-17. Tapered-wall screw implant.

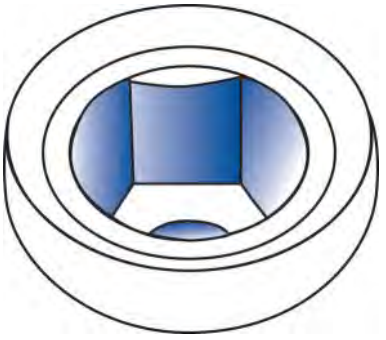


FIGURE 9-18. Morse taper internal connection.

Implant Components

A wide array of dental implant components are available for impression procedures, laboratory fabrication, and direct restorative dentistry. The various types of osseointegrated implants are discussed previously.

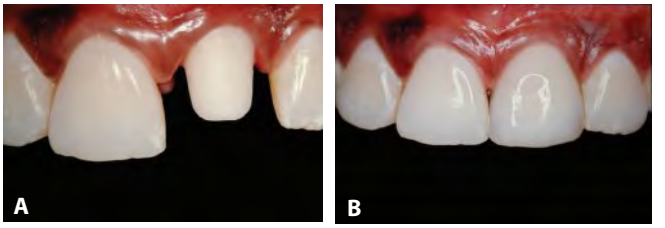


FIGURE 9-20. **A** and **B,** Aluminum oxide cemented abutment with an all-ceramic crown. (**A** and **B,** Prostheses prepared in collaboration with Avishai Sadan, DMD.)

Abutments are simply transmucosal extensions for the attachment of prostheses. Abutments can be used to provide a restorative connection above soft tissues and to provide for the biologic width. Abutments can be used for attachment of screw-retained or cemented connections and can be made of metal or ceramic. The most commonly used abutment material is machined titanium, which has been shown to be strong and resistant to plaque retention and to react favorably to soft tissues. Titanium abutments have been used historically for the attachment of screw-retained connections. Two of these types of abutments are shown in Figure 9-19. Titanium abutments are also used in many cases in which a cemented prosthetic connection is desired. With thin gingiva, the gray hue of these abutments can be problematic in aesthetic areas. Cast gold has been used for abutment connections owing to its blend with translucent gingival tissues. Although no hemidesmosomal attachment is found with cast alloys or dental porcelain,[39] yellow gold creates a warm appearance in aesthetically critical areas. In aesthetic areas, ceramic abutments have also been used in cemented designs for single- and multiple-unit crowns (Figure 9-20). Similar to titanium,

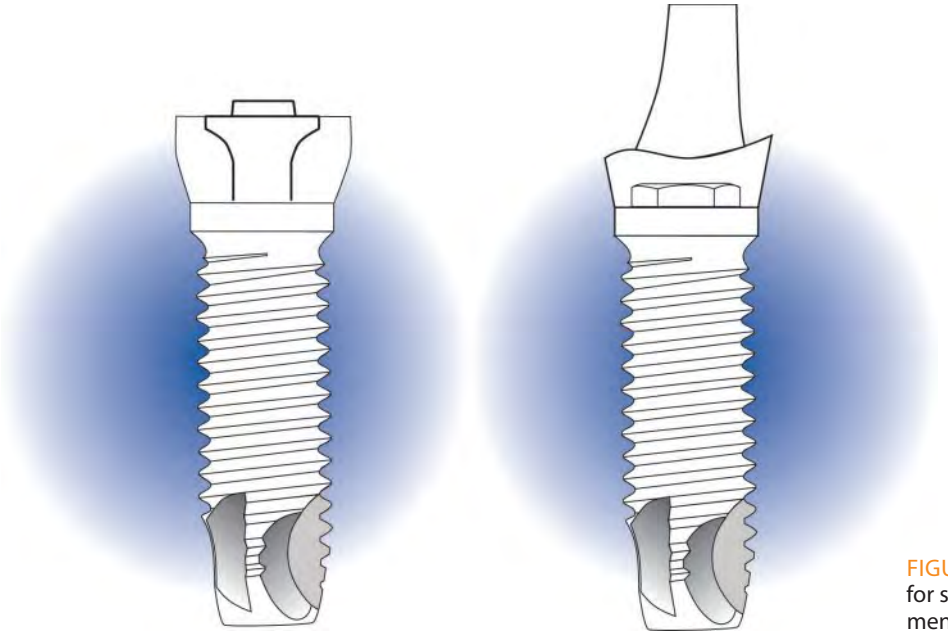


FIGURE 9-19. **A,** Premachined abutment for screw-retained restorations. **B,** Abutment for cement-retained restorations.

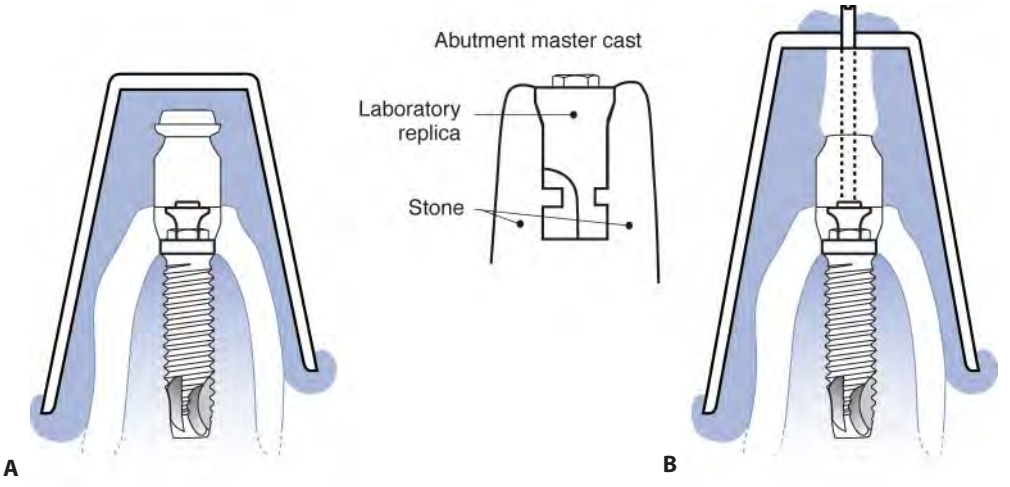


FIGURE 9-21. **A,** Abutment transfer impression using closed-tray technique. **B,** Abutment transfer impression using open-tray technique.

these abutments manifest a biologic attachment. The material used in these products has been mainly aluminum oxide and zirconium oxide. Generally, when soft tissues are less than 2 mm in thickness in the aesthetic zone, the use of ceramic abutments are indicated.[40]

The decision to use an abutment for screw-retained restorations can be made based on the depth of tissue. Generally, 3 mm or more of tissue depth necessitates the use of an abutment. As with any restorative procedure, biologic width is the driving force between the alveolar bone and the prosthetic margin. If the tissue depth is less than 3 mm, biologic width is probably created from a portion of the implant; therefore, the prosthesis may be connected directly to the implant, bypassing the need for an abutment. If the restorative dentist is unsure of which abutment to use, a fixture level impression can be recorded and the selection process completed in the laboratory.

Impression procedures used for dental implants are based on transferring either the abutment position or the implant position to the laboratory. If abutments are to be used for a screw-retained restoration, an impression coping is placed on the abutment and either a closed- or an open-tray technique can be used (Figure 9-21). The open-tray technique is considerably more accurate and is indicated for multiple splinted units. At this point, an abutment analogue or replica is attached in the impression and a cast is poured in the laboratory to simulate the oral situation.

If no abutment is to be used or if a cemented design is to be employed, a fixture level impression with an impression post can be made in a similar open- or closed-tray technique. Subsequently, an implant analogue or replica is attached to the impression post in the impression and simulated gingival material is placed; then a cast is poured to create a soft tissue master model (Figure 9-22). The simulated gingival material allows the dentist or technician to select an appropriate abutment and/or design the prosthesis while preserving the actual position of the gingiva.

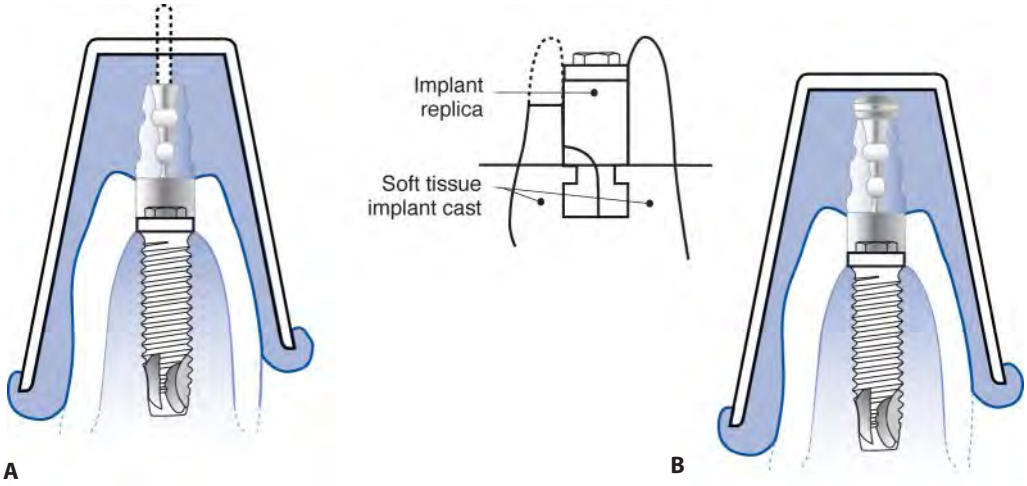


FIGURE 9-22. **A,** Implant level transfer impression using open-tray technique. **B,** Implant level transfer impression using closed-tray technique.

SINGLE-TOOTH REPLACEMENT

The Nonrestorable Tooth

Replacement of a single missing tooth should start with an evaluation of the periodontium and structural support. Periodontal defects, periapical pathology, bone loss, mobility, and pain are indications for periodontal/endodontic treatment or extraction. Other factors that require assessment before consideration for either restoration or extraction are the remaining coronal tooth structure, root fracture, and restorative space. The decimated tooth may have only one wall of the coronal structure remaining. Structural deficits of this type can be restored by using intracoronal anchorage methods (i.e., elective endodontics or post and core). However, vertical deficits that encroach upon the biologic width may necessitate crown elongation to provide enough tooth structure necessary for a ferrule or external bevel, which provides encasement of remaining tooth structure. A 2-mm amount of coronal tooth structure has been shown to improve long-term structural resistance to failure[41–43]; in total, biologic width plus a 2-mm ferruled tooth structure necessitates about 4 to 5 mm of suprabony tooth structure. If this is not available, it may be created by either orthodontic extrusion or crown elongation, which may sometimes create unfavorable crown-to-root ratios or furcation exposure. In this scenario, it may be prudent to consider extraction and either replacement with a fixed partial denture (FPD) or a single-tooth implant-supported restoration. Much of the literature indicates standard FPD survival to be in the high 80th percentile at 10 years and the 60th percentile at 15 years.[31] However, typical complications occurring are related to endodontics, recurrent caries, periodontal factors, and failures in retention. Single-tooth implant studies reveal complications as well.[32] However, in comparison with other implant restorations, the implant single crown is the most successful. If sufficient bone, soft tissue, and restorative dimension exist, replacement with an implant-supported single-tooth restoration is considered the standard of care and should be offered to the patient.[44,45]

The Edentulous Arch

The success of removable prostheses relies on the combination of retention, support, and stability, which can be deficient. A conventional mandibular prosthesis should be evaluated for retention, support, and stability. Difficulty with speech, swallowing, and mastication should be considered when evaluating prostheses. Patient acceptance of conventional prostheses may be contingent on stability and comfort when masticating. A patient's chief complaint should be closely scrutinized and correlated with the clinical examination to help formulate the proper treatment; the complaint is the foundation for a wide array of considerations that determine avenues possible for a candidate considering treatment with osseointegrated implants. Patient factors of prosthetic satisfaction can be quantified and evaluated before proposing initiation of further treatment.[46] Many of these considerations help to determine which imaging studies, preparatory treatment, and number of ancillary procedures are needed; if the treatment goals are feasible; and what time and cost commitment is involved. Treatment should be targeted at specific goals to achieve a predictable outcome that addresses the patient's functional and/or aesthetic problem. The treatment may encompass several different routes paying attention to time, cost, longevity, and levels of invasiveness.

The amount of keratinized/fixed tissue, vestibular depth, available bone, and opposing occlusion are all important factors to consider before implant treatment (i.e., natural dentition, edentulous arch, and implant-borne occlusion). It may be appropriate to recommend only an implant-retained overdenture for a favorable mandibular arch. However, mandibular arches with limited support, vestibular extension, and extensive bone resorption may require an implant-borne prosthesis.

The Aesthetic Zone

Aesthetic considerations encompass additional complex concerns such as gingival display, proportion of teeth in the aesthetic zone, and bone density support. The aesthetic zone is generally considered to be the maxillary anterior area. When considering replacement of a single tooth in the aesthetic zone, the adjacent dentition should also be evaluated for proportionality and position. From a frontal plane, the lateral incisor should be about two-thirds the width of the central incisor. Likewise, the width of the canine when viewed from the same vantage point should be about two-thirds the width of the lateral incisor, and so on. The width-to-length ratio of aesthetically pleasing central incisors should be approximately 66% to 80%.[47] The axioms are ranges found in nature and are considered pleasing to the human eye. If these proportions are not present, they may be created by surgical periodontics, restorative dentistry, orthodontics, and if appropriate, osseointegrated implants.

Occasionally, replacement of maxillary or mandibular canines may present a compromise in either occlusion or aesthetics for the functional goal of eliminating lateral forces on the restoration/implant. Aesthetic and/or functional correction may dictate the need for pretreatment orthodontics, endodontics, periodontics, and concurrent restorative dentistry. A complete examination that includes diagnostic models, radiographs, and clinical photographs can be invaluable.

Aesthetic considerations for removable prosthodontics may be a concern for lower edentulous arches when restoring the facial contours typically lost in mandibular resorption. This is especially true when restoring the skeletal class II patient. The use of a flange may be necessary to eliminate the labiomental fold usually apparent in these cases. Likewise, the use of a flange in the edentulous maxillary arch may be beneficial to restore upper lip support as well as the aesthetic integrity so critical to this area. A functional lingual maxillary alveolar seal is essential for correct labiodental consonant

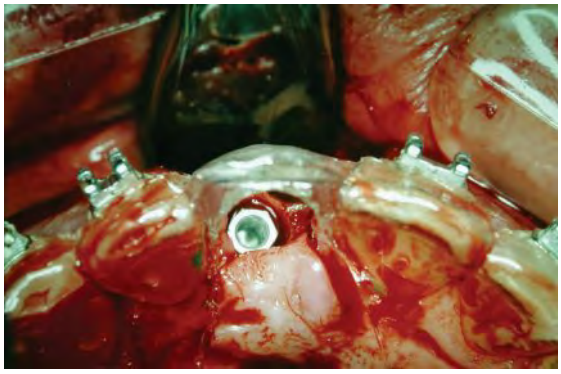


FIGURE 9-23. Long axis of implant placement through the incisal edge of the stent for cement-retained prostheses. (Surgery performed by Michael S. Block, DMD.)

production; in cases of advanced resorption of the maxilla, an overdenture may be the appropriate treatment.

Cemented Single Units

Cemented prostheses may be preferable to screw-retained designs for single-unit crowns in the anterior areas. They tend to provide minimized bulk of the restoration. Overcontoured bulky restorations are not hygienic and are detrimental to the maintenance of peri-implant tissues. The axis of implant placement should be aimed through the incisal edge for standard-diameter implants (Figure 9-23). This results in predictable aesthetics and manageable soft tissues. If a comparably wider implant is placed (4.3, 5.0, or 6.0 mm) in an aesthetic site, the long axis should traverse just palatal through the incisal edge. Errors in placement to the facial of the incisal edge produce not only difficulties with angulation correction but also a soft tissue problem because the bone support in this area is lost owing to the osteotomy (Figure 9-24). Errors in placement too far palatally create ridge-lapping and hygiene difficulties. The superior/inferior placement of the implant platform should be 3 to 4 mm below the anticipated free gingival margin. The use of a surgical stent in placement aids in creating an optimal site for implant restoration. The choice of cemented restorations for a posterior tooth is plausible and becomes especially useful when angulation in placement is less than ideal. However, the resistance and retention form of the abutment should be sufficient to resist dislodgment. The choice of specific abutments can be

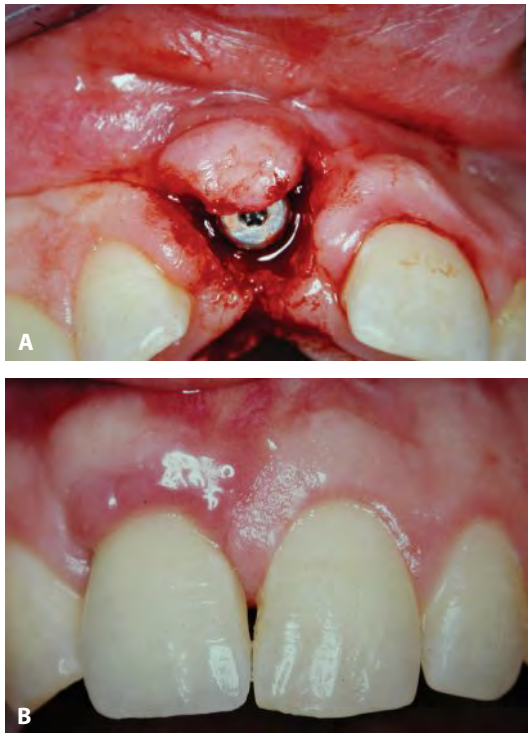


FIGURE 9-24. **A** and **B,** Implant placed too far facially resulting in compromised peri-implant soft tissues.

planned in advance if placement is based on an ideal scenario. Anatomy should not dictate placement of the implant position, but rather, the placement should be based on restorative parameters. This information can be obtained by the use of surgical stents, which may provide critical information about where to develop the occlusion and where to re-create the emergent path as the restoration exits the gingival sulcus. It is critical to ensure removal of all luting cement by the use of retraction cord, vent holes, and radiographic verification because this can lead to soft tissue recession/irritation and bone loss.

Screw-Retained Single Units

The treatment plan for replacement of a single tooth with screw retention is the professional preference of the restorative dentist. There are advantages and disadvantages to using this design for single and multiple missing teeth (Table 9-2).

TABLE 9-2. **Screw Retention versus Cement Retention**

Advantages		Disadvantages	
Screw Retention	*Cement Retention*	*Screw Retention*	*Cement Retention*
Retrievability	Aesthetic	Implant placement critical	Cement removal subgingivally
Porcelain emergence	Angle correction possible	Screw access channel visible	Abutment selection critical in anterior
Cost effective	Less bulk of restoration in anterior areas	Deep channels should be sealed	Provisional restoration needed in anterior
Elimination of cement retrieval	Built-in load indicator by two interfaces		Cost factor with abutment/restoration
			Problematic retrievability







FIGURE 9-25. **A,** Incisal view of screw-retained central incisors. **B,** Facial representation of restorations placed. **C,** Occlusal view of a screw-retained restoration for a posterior single unit.

Screw-retained prostheses are simplistic to retrieve, are easy to trial fit, and can be shaped to the desired emergence with either porcelain or metal. This design also eliminates the uncertainties of loosening and incomplete debris removal associated with cemented prostheses. However, using screw-retained prostheses requires strict attention to placement and confines the axis of the implant through the desired area of emergence within the restoration (Figure 9-25). Screw-retained prostheses are especially useful in the posterior dentition because retrievability is much easier than with the cemented prosthetic design and a controlled degree of retention is afforded as well.

RESTORATIONS FOR THE PARTIALLY EDENTULOUS PATIENT: FPDS

FPDs require the first assessment of site planning as with other types of restorations. It is of prime importance to understand that the implant bridge should be supported entirely by dental implants. Combining the support with natural teeth has been shown to involve prosthetic complications and intrusion of the abutment teeth for a number of reasons. Although these studies may use the specific scenario of a three-unit FPD supported by a natural tooth and implant, other studies have advocated strategic teeth in combination with implants for full-arch prostheses. It is prudent to keep the restoration supported entirely by dental implants to avoid problems concerning abutment fracture, screw loosening, tooth intrusion, malocclusion, and other complications. Designing the FPD to be screw-retained as opposed to cement-retained is largely based on personal preference but may be tailored to what can be serviced and maintained most easily.

FPDs in the Aesthetic Zone

Placement of multiunit restorations in the anterior maxilla should bring to mind several anatomic considerations for surgical planning:

- Adequate depth of the residual alveolar ridge to the nasal floor.
- Buccolingual width of the bony ridge to provide for implant placement.
- Assessment of the lip when relaxed and upon activation of a smile.
- Participation of the restoration in anterior guidance in conjunction with the adjacent teeth.

Anterior FPDs or any restoration in the aesthetic zone should first begin with a diagnostic wax-up or template (Figure 9-26).

This will give an idea as to the incisal edge position as well as the available restorative dimension and should be

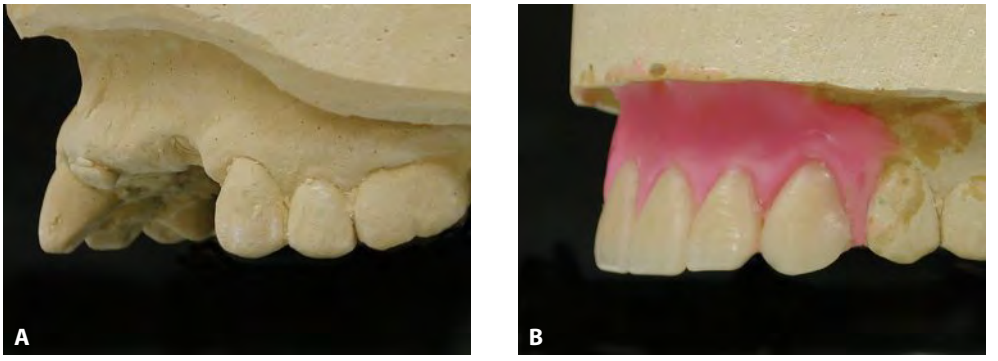


FIGURE 9-26. **A** and **B,** Diagnostic cast and wax-up of missing maxillary anterior teeth.

verified in the patient's mouth to correspond with facial landmarks such as the center of the face and interpupillary line. Also, a proportional relationship should exist from the central incisor to the canine from an anterior perspective. This proportionality becomes critical in aesthetically prominent areas. The wax-up may also indicate how much tissue has been lost as a result of the missing teeth, soft tissue, and associated alveolar process. In these cases, it may be necessary to consider horizontal or vertical bone augmentive procedures as a first phase followed by placement of implants in a second phase. In some cases, it may not be feasible to perform bone grafting owing to local or systemic factors. Making precision detachable bridgework that replaces teeth, soft tissues, and alveolar bone may be more predictable in these circumstances. If the surgical workup determines implant placement will be done concomitantly with or without a bone graft, the diagnostic wax-up should be used to fabricate a surgical guide or stent for implant placement. If a bone graft is necessary, the surgical guide references the incisal edge and gingival aspect of the future restoration to aid in establishing the proper amount and positioning of the bone graft (Figure 9-27). Superior/inferior positioning of implants is virtually the same as for single units, described previously. However, the mesiodistal assessment of restorative space should be done first to determine the appropriate implant number and dimension to be placed. Using a 2-mm rule from each adjacent tooth and a 3-mm rule from implant to implant, the appropriate implant number and dimension can be calculated (see Figure 9-2). If the available space does not allow an appropriate number of implants or encroachment upon the implant-implant proximity, either restorative dentistry and/or orthodontics may be indicated. Occasionally, use of a cantilevered bridge design can be advantageous where space constraints or insufficient bone prohibits placement. If it becomes necessary to cantilever the FPD either mesially or distally, a screw-retained design permits a framework that better withstands the cyclic loading of occlusion and subsequent problems with porcelain fracture or other material failure. Screw-retained prostheses require an entirely passive fit. It is considerably more difficult to create a passive-fitting screw-retained framework than a cemented framework that has intimate fit with the supporting abutments. Conversely, it occasionally becomes necessary to perform angle correction because there is frequent disparity between the long axes of tooth and the long axis of bone available in the anterior maxilla. An intimate fit of FPDs is far easier to achieve with a

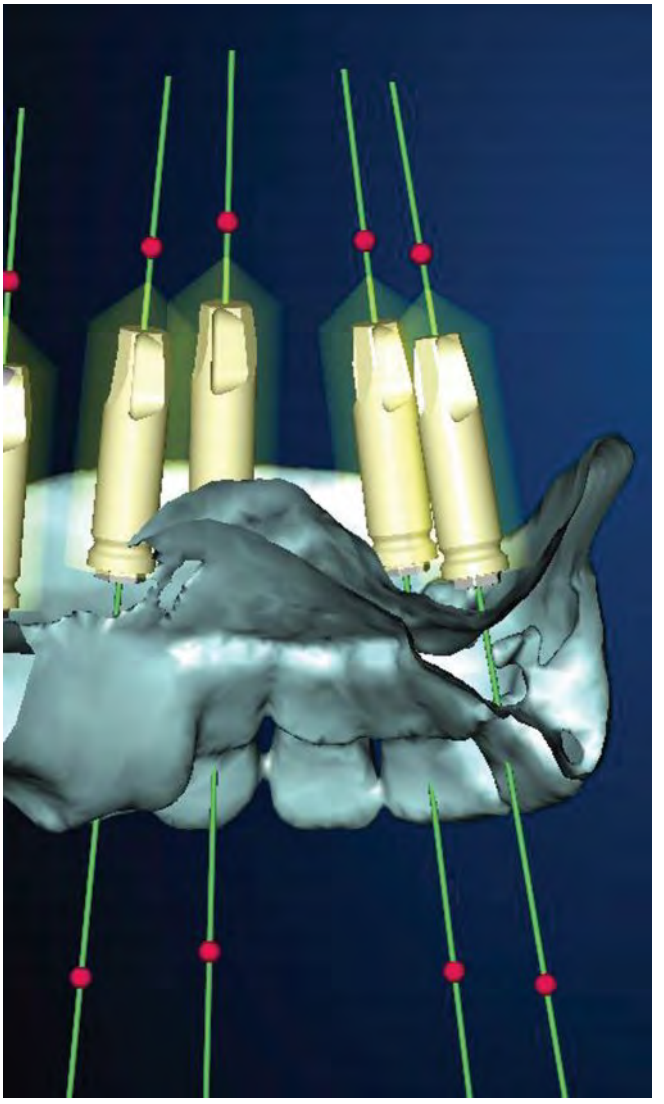


FIGURE 9-27. Computerized navigation reveals amount of soft/hard tissue loss.

cemented prosthetic design than with a screw-retained restoration. The subtle inaccuracies of impression making, alloy casting, and porcelain application make the simultaneous and coincident fit of screw-retained FPDs difficult; thus, a cemented prosthetic design is a more appropriate choice. With a cemented design, the creation of a surgical stent is critical for accurate placement and aesthetic success of the implant restoration. After placeing and uncovering the implants, it is prudent to create provisional restorations to develop soft tissues. Only in this way can an acceptable aesthetic outcome become predictable in the aesthetic zone.

FPDs in the Anterior Mandible

Placement of multiple-unit restorations in the anterior mandible requires similar forethought as with the anterior maxilla. Placement of multiple implants in the anterior mandibular area presents a unique challenge in that one-to-one replacement of teeth with implants can create proximity concerns (Figure 9-28). Tarnow and colleagues[8] have outlined the pattern of bone loss to be about 3 mm from the edge of the implant to an adjacent implant. Therefore, placement of implants closer than 3 mm to each other creates accelerated bone loss patterns in these areas. This pattern seems to be somewhat less (~2 mm) when the implant abuts a natural tooth. Because the anterior mandible is mostly composed of dense compact bone, an implant-to-tooth replacement ratio of 1:2 may be acceptable as long as the crown-to-implant length ratio is 1:1. Gingival adaptation in the anterior mandible is not as critical as it is in the anterior maxilla because phonetics are primarily made in relation to the maxilla. Screw-retained designs for FPDs in the anterior mandible seem to work well (Figure 9-29). Implant proximity should also be assessed before placement for hygiene procedures because the placement of even an appropriate number of small-diameter implants in this area can create hygiene difficulties.

FPDs in the Posterior Maxilla

Placement of implants in the posterior maxilla requires sufficient bone buccally and lingually as well as inferior to the maxillary sinus. Although short implant lengths have been used, 12 mm of bone in actual height is the minimum required for a macroretentive screw-type implant to adequately support occlusal forces. After the loss of a tooth in the posterior maxilla, this required dimension might not be available (Figure 9-30). Progressive enlargement of the maxillary sinus as well as residual ridge resorption is often seen after tooth loss. Diagnosis of either of these problems helps one determine the appropriate treatment. If pneumatization has taken place, sinus augmentation procedures can be indicated with either concomitant or delayed implant placement. Residual ridge resorption or traumatic destruction of alveolar bone by trauma or periodontal disease may also have taken



FIGURE 9-29. **A** and **B,** Screw-retained prosthesis permits hygiene access.



FIGURE 9-28. Placement of two implants in strategic locations to permit hygiene access and force distribution.



FIGURE 9-30. Alveolar bone loss resulting in the need for an onlay bone graft before implant placement.



FIGURE 9-31. Cranial onlay bone graft in the posterior maxilla. (Image courtesy of Leon F. Davis, DMD, MD.)

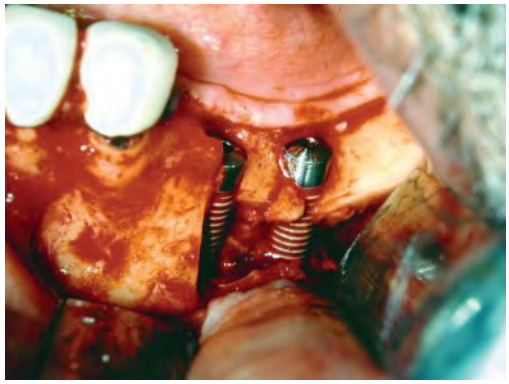


FIGURE 9-33. Placement of two implants in the posterior mandible after inferior alveolar nerve transpositioning.

place. In these cases, onlay bone grafting may be a more appropriate treatment (Figure 9-31). The decision to replace a posterior maxillary quadrant with individual crowns versus fewer splinted implants acting as an FPD may be related to the length of implant or the presence of natural canine teeth with cuspid-protected occlusion (Figure 9-32). Other strategies place the implants in a slightly staggered configuration from buccal to lingual and then splint them together. Screw-retained designs seem to allow retrievability and offer advantages for modifying hygiene and performing reparative ceramometal procedures.

FPDs in the Posterior Mandible

As with the posterior maxilla, tooth loss for an extended time can result in residual ridge resorption. In such cases, onlay bone grafting may provide an appropriate bone volume for implant installation. A limiting factor for implant placement in the posterior mandible is not only residual ridge resorption but also relative position of the inferior alveolar canal. Panoramic radiographs may give a full appreciation of the position of the inferior alveolar canal. In some patients, this may assume a relatively high position making placement of implants of reasonable length impossible. In these cases, lateral positioning of the inferior alveolar nerve with implant placement may be the only option for treatment other than a removable partial denture. Nerve repositioning is an effective adjunct in implant placement, but the technique can have significant adverse nerve injury (Figure 9-33). In some cases, it may be preferable to sacrifice teeth anterior to the edentulous area to permit placement of sufficient length implants anterior to the mental foramen with extension to the posterior.

Cantilevered FPDs

Cantilevered FPDs may be used in implant dentistry provided there is adequate length to the supporting implants and limited distance to the cantilever. This may be especially useful when there is an insufficient amount of bone or when significant site morbidity may result. Posterior cantilevering probably is a more common scenario, typically owing to a greater availability of bone in the anterior area of the jaws. Anterior cantilevering may be used in areas where posterior anchorage is superior to anterior anchorage (Figure 9-34). Cantilevering requires that a framework be connected at a maximum clamp force; such stability is best achieved with screw-retained frameworks. A further strategy is to use direct



FIGURE 9-32. Individual fixed units protected from canine rise in lateral excursions.

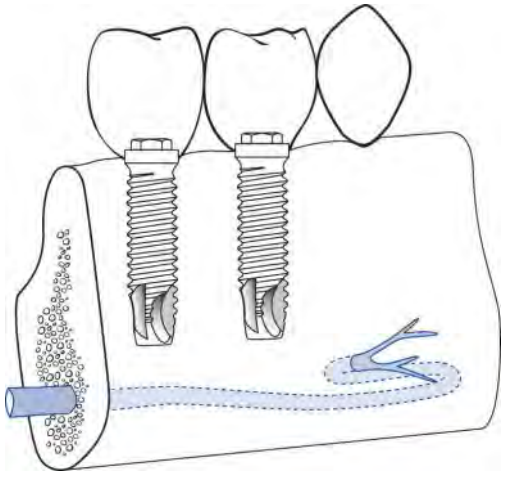


FIGURE 9-34. Anterior cantilever fixed partial denture.

to implant connections owing to a comparably robust screw joint connection. Occlusal contact created on the pontic should be very light to coincident.

RESTORATIONS FOR THE EDENTULOUS PATIENT

Implant-Retained Overdentures

Those older than 65 years are said to represent a significant proportion of the U.S. population, and the average life expectancy has risen by 30 years since 1900. A sizable portion of this group is edentulous or partially dentate in at least one arch.[48] Many in this age group have difficulty wearing mandibular complete dentures owing to poor support and retention precipitated by advanced bone resorption, xerostomia, loss of attached keratinized tissue, and neuromuscular degeneration. The use of implants for these edentulous patients has been shown to actually preserve existing bone as opposed to results with conventional dentures.[49] Increased support and anchorage can be improved with the use of at least two osseointegrated implants in the anterior mandible. Although the use of stud attachments connected to the implants can be a cost-effective measure to improve retention, stability, and support (Figure 9-35), there is an increasing need for aftercare and servicing for these patients. If a stud-retained denture is planned, the implants should be as parallel as possible to avoid premature wear of the attachment mechanism. Alternative cylindrical attachment mechanisms have been introduced to allow for an easier rate of servicing in the aftercare period.(Figure 9-36). The vertical height of the attachment should be considered because some edentulous mandibular arches do not provide more than 4 mm of restorative dimension for the mandibular denture. Preoperative planning calls for the evaluation of the patient's present difficulty. Reasonable aesthetics, occlusion, and extension should be evaluated first. If these factors seem to be appropriate, panoramic radiographs and possibly an occlusal radiograph are helpful in determining the position of the mental foramina. A prime objective is to place at least two implants as far apart as possible within this area. The anterior loop of the inferior alveolar nerve can extend as far forward as 7 mm before exiting the mental foramen; thus, consideration should be given to proper site selection.[50] A radiographic marker such as a standardized stainless steel shot can be secured to the patient's denture and placed in the mouth before panoramic and/or occlusal radiography. This will give an indication of the correct site selection for implants in the anterior mandible. After the site has been selected, an open channel can be created in the stent to allow surgical latitude. Either duplication of the patient's denture or a wax trial tooth arrangement subsequently processed in clear acrylic resin can be helpful in determining the position. In general, tapered arch forms with extensive resorption may direct placement of implants in close proximity to each other. In other words, implants placed less than 20 mm apart may not be mechanically advantageous for use independently as stud attachments. In these cases, it may be desirable to connect the implants with a bar attachment to create a wider base of anchorage. There are several reasons to plan the implant-retained denture for a bar attachment. First, short (≤10 mm)



FIGURE 9-35. Stud-retained overdenture using O-ring attachments.





FIGURE 9-36. **A** and **B,** Cylindrical attachments intraorally with corresponding placement of matrix in overdenture base.

implants or implants placed in cancellous bone or types 3 and 4 bone, not typically seen in the anterior mandibular area, may be better supported by the splinting effect of a bar attachment. Second, nonparallel implants create different paths of insertion, which subsequently serve to wear and disable the stud attachment prematurely. In these cases, the bar attachment can correct this problem by providing a single path of insertion. Third, implants placed in close proximity to each other may provide better anchorage to the overdenture if a bar attachment is incorporated that places the attachment mechanism at a wider base than the interimplant distance.

There are some spatial considerations of using a bar attachment that should be evaluated before treatment planning. The vertical height needed for a bar attachment can approach 11 mm. This measurement is taken from the occlusal plane to the highest point of the alveolar process. This distance will provide for the height of the bar (2–4 mm), 1 to 2 mm under the bar for maintenance of hygiene, and at least 7 to 8 mm of restorative material in the overdenture (usually acrylic resin) (Figure 9-37). Some early evidence points to the added benefit of using metal reinforcing frameworks in mandibular overdentures to decrease the risk of fracture of these restorations.[51]

Implant-retained overdentures for the maxilla can incorporate the use of bar attachments. A minimum of four implants in the anterior maxilla splinted with a bar seems to be appropriate treatment. Whenever possible, cross-arch stabilization is preferred for maxillary implant-retained or -supported overdentures. In these cases, it may be prudent to also incorporate full palatal coverage to assist with some residual load transfer to the hard palate. The prosthetic treatment of these implant cases is assimilated to the Kennedy class I partially edentulous arch in that stress-breaking attachments and stress distribution to the soft tissue support posteriorly are important considerations. Also shown to be successful is the use of abutment retention for maxillary overdentures. Discretion should be made for distribution of the abutment relative to rotational force requirements as many abutment systems are resilient.

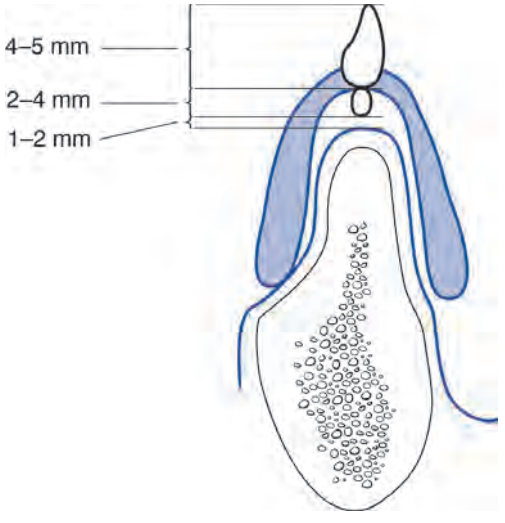


FIGURE 9-37. Minimum clearances needed for a bar-attached overdenture.

Implant-Supported Overdentures

Implant-supported overdentures may be indicated when a patient has significant difficulty in all factors of support, retention, and stability. Anatomically, there may be cause to suspect that extensive resorption has taken place that has resulted in the loss of alveolar structure. Consequently, implant anchorage can be used to aid in the support and retention of overdenture prostheses.

Historically, most of the literature available on implant-supported restorations in the mandible has been planned for four to six implants intraforaminally. More contemporary literature suggests the use of four widely spaced implants in this region opposing an edentulous arch with equally successful rates.[52–54] The strategy for using implants in the anterior mandibular area allows segments to be cantilevered posteriorly in accordance with the anteroposterior spread of the implants. On average, this equates to 10 to 20 mm or the area of the lower first molar. The decision to extend the cantilever can be based on the arch form of the fixtures, fixture length, anterior cantilevering, natural maxillary dentition, and parafunctional habits. Favorable factors for extension of the cantilever are a tapered arch with long fixtures, no anterior cantilevering, edentulous maxillary arch, and no parafunctional activity. The most posterior implant supports a load typically of compression in comparison with the anterior implants, which are placed under tension. Also, the mandible may be viewed as a dynamic bony structure undergoing flexure. This can approximate 2 mm at the mandibular angle upon maximum opening. For this reason, implants placed distal to the foramen should not be rigidly connected to the contralateral side.

Planning for implant support of a prosthesis in the edentulous maxilla has traditionally involved at least eight fixtures. This may require the use of sinus augmentation, or for consideration of fewer implants, extended-length implants into the zygomatic process have been successfully used. The use of zygoma implants have been favorable, approaching a success rate that surpasses those implants to which they are attached.[55,56]

Attachment mechanisms for implant-supported overdentures can range from the simple to the sophisticated. Bar-clip attachments are a cost-effective and predictable means of connecting implants. More sophisticated milled-bar and plunger attachments can be precision methods in telescopic placement of a removable prosthesis. The milled bar can be machined to a 2-degree taper, allowing a precise path of placement (Figure 9-38). The underside of this overdenture has a cast metallic housing that acts as a guide over the milled-bar attachment (Figure 9-39). Usually, this restoration contains either plunger or swivel attachments that lock the overdenture as it comes to complete placement over the bar attachment. This technique is very effective but can allow a small degree of micromovement.



FIGURE 9-38. Bar attachment milled to a 2-degree taper for implant-supported overdenture.



FIGURE 9-40. Mandibular hybrid denture.

An additional method of electrical discharge machining, also known as "spark erosion," can be used in these cases; it results in a precise fit between the superstructure and the bar. This technology, which results in an essentially detachable fixed bridgework, may be significant in costs.

Fixed Detachable Prostheses

One alternative treatment method for an edentulous mandible is the use of an implant-supported fixed complete denture also known as a *fixed/removable restoration (hybrid denture)*. This restoration contains a screw-retained metal framework with a veneer of acrylic resin and denture teeth, thus coining the term *hybrid*. Such restorations are fixed and are not removable by the patient; however, they do allow adequate room for oral hygiene procedures (Figure 9-40). As might be expected, no denture flange is present and a minimum vertical restorative space of 15 mm is necessary for structural integrity and hygiene access. Placement of implants for a hybrid denture should incorporate the use of a surgical stent because the exit sites for the access channels are critical. The surgeon may be cautioned on careful implant placement in those patients with a skeletal class III or severe class II relationship as revealed by cephalometric radiography. Recently, application of this immediate-load technique has become popular.

Of course, a full-arch ceramometal restoration could also be used in these circumstances in which a minimal restorative dimension exists. In this circumstance, screw-retained prostheses offer stable occlusal support while allowing some degree of posterior cantilevering.

Treating patients with an edentulous maxilla is dependent upon a number of factors. The primary determining factor is one of available space. Generally, the more space available (13+ mm vertically), the more indication there is for an overdenture prosthesis. Incipient resorption or minimal space availability (9–12 mm vertically) may indicate the use of a ceramometal design (Figure 9-41). Implant-supported maxillary overdentures are frequently used in cases of moderate to severe resorption because they replace not only missing mastication and aesthetics but also phonetic physiology as well. Speech production may rely heavily on adaptation of the prosthesis to the palatal gingiva. If there is ample space, this is best accomplished with an overdenture prosthesis to seal this linguoalveolar area phonetically. Attachment mechanisms for the maxillary implant-supported overdenture are the same for the mandibular overdenture with the exception of plunger or locking attachments placed palatally (Figure 9-42).



FIGURE 9-39. Precision detachable overdenture with attachments for engaging the bar. (Prostheses courtesy of Northshore Dental Laboratory, Lynn, MA.)

CONTEMPORARY TECHNIQUES

Immediate Placement

Immediate placement of implants into extraction sockets has been considered for some time. Although it has been performed successfully, inflammation and infection should be eradicated for predictable osseointegration to occur. Considerations for using immediate placement capitalize on the osteogenic potential of a recent extraction site and the chance to preserve what bone remains. The use of tapered implants in these sites has become popular to obliterate the socket defect while being firmly anchored in the majority of the

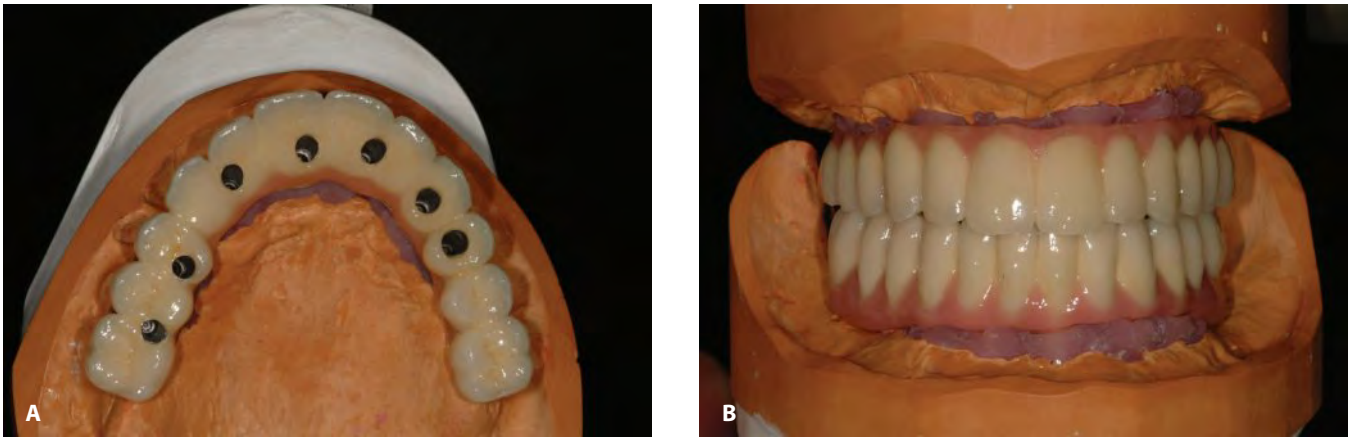


FIGURE 9-41. **A** and **B,** Screw-retained ceramometal prosthesis, occlusal and frontal views.

bony walls. A word of caution is advised for those teeth that have drifted or are not in an ideal location because tooth position influences implant position. Indications for placement into a recent extraction socket are freedom from infection and intact nature of the remaining socket. Ways of facilitating this technique may incorporate orthodontic extrusion to create a smaller socket in the bone, facilitating extraction, and overcorrecting bone apposition to re-create missing architecture (Figure 9-43). The extrusion should take place slowly, usually over 3 to 6 months.

Early Placement

As an alternative, early placement (4–8 wk after extraction) may afford an advantage in offering some bone formation into the socket, a surplus of soft tissue for closure, and more predictable soft tissue profiles long term.[57] This will often result in improved primary stability of the implant as well as an opportunity to place the implant platform where the prosthetic and biologic indication is strongest.

Surgical Installation Stability

Installation of implants into bone usually is characterized by minimizing the inherent gap between the implant and the bone surface. If placed in perfect adaptation of the osteotomy, this results in primary implant stability that is often appreciated by a resistance to further torque applied. Depending on the type of bone that the implant is placed in, further assessment of the implant's secondary stability can be recorded by a radiofrequency device. However, during the period of osseointegration, the secondary stability changes in proportion to some bone remodelling. Although similarly high osseointegration rates have not been surpassed, some implant surfaces have been shown to reduce the time taken for this secondary stability to take place.

Although achieving this stability can be accomplished with both screw-type and press-fit implants, parallel- and tapered-walled screws are uniquely suited to providing firm stability at surgical placement. This becomes an important consideration when achieving osseointegration under placement either in an extraction site, where a provisional restoration will also be inserted, or where other implants will be joined for an immediate-load prosthesis. For immediate placement after extraction, the socket should be obliterated by the implant and/or grafting materials. Micromovement in excess of 50 to 75 μm has been shown to inhibit osseointegration to a fibrous tissue deposition instead of bone



FIGURE 9-42. Swivel latches placed to the palatal aspect for a maxillary spark erosion overdenture prosthesis.



FIGURE 9-43. Orthodontic extrusion of a nonrestorable tooth to aid with migration of the soft/hard tissue as well as atraumatic root removal.

apposition; therefore, occlusion placed on a provisional restoration during the critical period of osseointegration must be carefully controlled to reduce this scenario. If this modality is desired, a more controlled technique of protecting the occlusion with a centric relation orthotic may be appropriate. Immediate loading for single teeth mandates more data before it can be recommended for routine use. However, controlled immediate loading of multiple connected implants in the anterior mandible has been favorably surveyed and can be recommended as long as there is careful control of occlusion.

Immediate Restoration

Immediate restoration of a single-tooth implant may be incorporated in the aesthetic zone (Figure 9-44). The indications are freedom from occlusal overload and lateral forces. Sometimes, it is difficult to control occlusion, and the creation of an occlusal splint may be a prudent way to protect the implant while osseointegration takes place (Figure 9-45). The advantages of immediate restoration are the establishment and preservation of the peri-implant tissues. It is easier

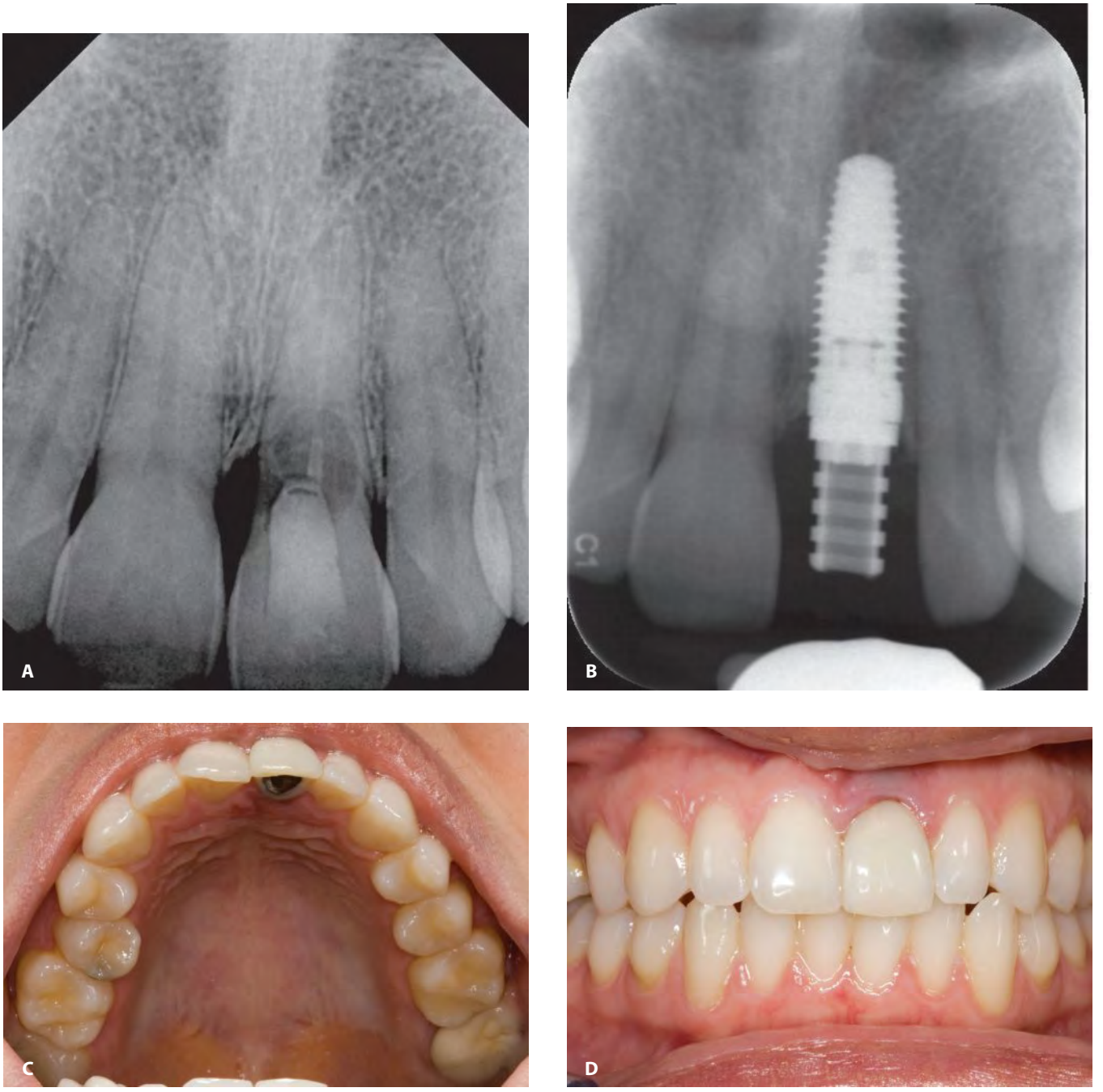


FIGURE 9-44. **A,** Initial presentation of external resorption of tooth no. 9. **B,** Post implant placement fit with screw retained abutment. Incisal **(C)** and frontal **(D)** views of modified natural tooth crown.



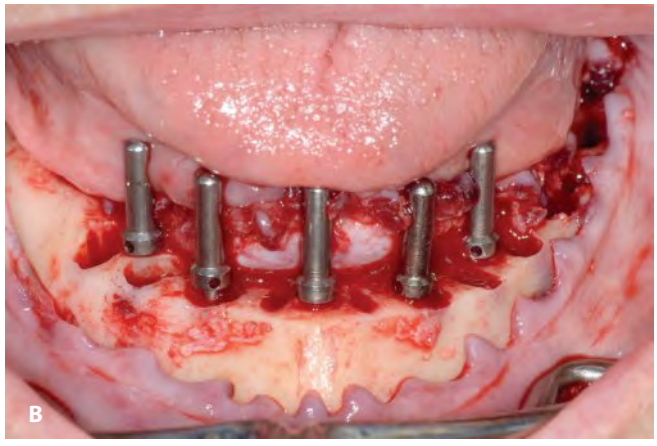
FIGURE 9-45. Use of occlusal splint to protect an immediately placed implant/restoration in site no. 9. Note that the splint is relieved from contacting tooth no. 9.

to preserve this tissue than to re-create it by using a staged approach. However, higher incidences of failures are associated with immediately loading of single-tooth restorations.[58]

Fixed or Overdenture Prostheses

The use of splinted implants immediately loaded in the mandibular anterior region has been discussed by Schnitman and associates[59] and others.[60] The edentulous interforaminal mandible appears to be favorable for immediate loading of multiple endosseous root form implants (Figure 9-46). Clinical experience and literature reviews indicate that the results obtained are favorable for either fixed or overdenture





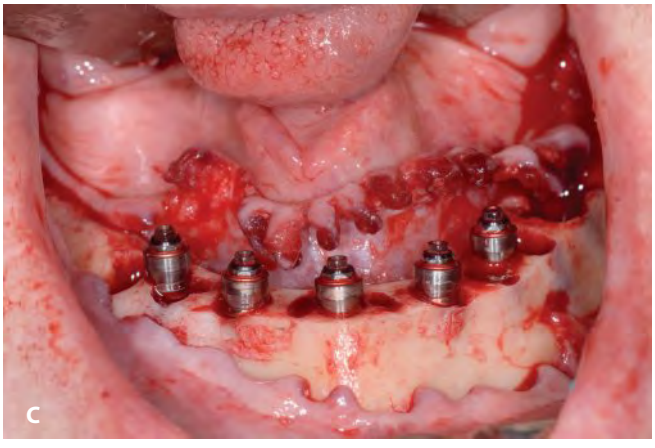






FIGURE 9-46. **A,** Nonrestorable dentition indicated for removal. **B,** Ostectomy with pilot osteotomy parallel pins. **C,** Implant placement interforaminally with abutments secured. **D,** Denture used as guide in preparation for conversion prosthesis. **E,** Final fixed conversion prosthesis secured.

alternatives.[61] Requisites of primary implant stability are suggested at or above 30 N/cm.

MAXILLOFACIAL PROSTHESES

Patients treated for tumor ablative surgery of the oropharyngeal area may have a significant deficit of anatomic structures necessary for oral function. The incidence of oral cancer approaches about 5% of all new cancers diagnosed in the U.S. general population.[62] A significant number of these patients are treated for malignant neoplasms of the lip, tongue, oropharynx, mandible, maxilla, soft palate, larynx, external ear, orbit, and external nose. To successfully eradicate disease, these tumors are treated with multimodal therapy of tumor ablative surgery, radiotherapy, and chemotherapy. The highest incidence of this disease afflicts those individuals with significant risk factors of excessive use of alcohol and tobacco and other factors such as ultraviolet light exposure and infection with human papillomavirus. A common site of development of squamous cell carcinoma is seen in the lower lip and ventrolateral tongue. Occasionally, this disease expands by direct extension to involve structures of the mandible and maxilla.

Mandible Defects

Resection of a portion of the mandible may be necessary to control disease and may create a discontinuity defect. Because the mandible is so integral to oral physiology, it is desirable to preserve function as much as possible.

If a marginal mandibulectomy is performed, the remaining mandible may be reconstructed with osseointegrated dental implants. Preservation of the inferior alveolar nerve may preclude placement if there is minimal bone available above the canal position to stabilize implants (Figure 9-47). In these cases, either nerve transposition or onlay bone grafting may serve to provide osseointegrated rehabilitation. If mandibular continuity is not preserved with resection, it may be desirable to reconstruct the area with an autologous or alloplastic graft. Autologous grafts offer a greater volume of viable bone with progenitor cells capable of creating a more favorable environment for osseointegration. Nonvascularized or vascularized osteomyocutaneous flaps can be used for reconstruction. In previously operated fields, it may be preferable to use a vascularized flap that may offer a secure opportunity for the graft to remain viable because the blood supply is preserved. The iliac crest has been used with some degree of success for mandibular defects and some maxillary defects as well. Introduced by Hidalgo[63], the use of fibular grafts has also shown a promising degree of success in reconstruction of these complex mandibular defects. Being a non–weight-bearing bone, the fibula is of reasonable dimension to functionally and cosmetically reconstruct the mandible. Bicortical stability for concomitant or delayed implant placement can also be well obtained at surgical installation, and long-term success has been observed (Figure 9-48). The

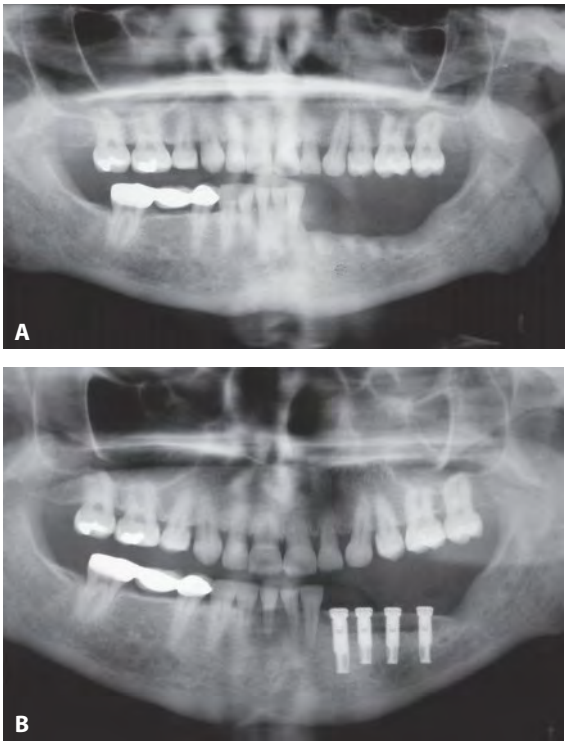


FIGURE 9-47. **A,** Mandible with insufficient supracanal height for implant installation. **B,** Iliac crest graft to mandible stabilized by placement of osseointegrated implants. (**A** and **B,** Surgery performed by Michael Miloro, DMD, MD.)

choice of whether to use either a sectional overdenture design or a screw-retained fixed prosthesis may be based on the amount of tissue missing, the function of the tongue, perioral scarring, and adjacent/opposing occlusion. Frequently, the crown-to-implant ratio is seen to be greater than 1:1 (Figure 9-49). Passive splinting of these implants is crucial to their long-term success, and close attention must be paid to development of the occlusal scheme. Occasionally, it may be necessary to perform soft tissue revision procedures if the skin pedicle is thick or if a greater vestibular depth is needed.



FIGURE 9-48. Implants placed into a vascularized fibula graft to the mandible. (Surgery performed by Perry Johnson, MD, and Michael Miloro, DMD, MD.)



FIGURE 9-49. Mandibular fixed partial denture supported by a vascularized fibular graft in the patient viewed in Figure 9-48.

This ensures soft tissue health and visibility for hygiene procedures.

Maxillary Defects

The maxilla may require resection for tumor control, which creates a host of problems related to speech and aesthetics. Traditional resection of the maxilla involves an infrastructure procedure or may involve the medial portion or a total removal of the maxilla. Infrastructure maxillectomies are used to control incipient disease of the oral cavity and have been classified by Aramany based on frequency of occurrence.[64] Obviously, the more teeth, bone, and soft tissue available, the easier prosthetic rehabilitation can be employed. However, edentulous patients requiring this operation may have significant difficulty in obtaining stability with their prosthesis, and in these cases, a consideration for the use of implants is warranted.

The use of sinus augmentation has been well documented and deemed to be successful with the incorporated use of implants. This technique may be used on a nondefect side where a unilateral or posterolateral defect of the opposite side is present. Splinting of approximately four or five implants with a stress-breaking bar is generally suggested and provides the patient with a retentive stable prosthesis that may offer improved support as well (Figure 9-50). The use of zygomatic implants has been suggested as an alternative to sinus lifting in the early 1990s.[65] The implant protocol for zygomatic implants mandates bilateral placement, and preservation of the defect side of the infraorbital rim may improve surgical stability. This has proved successful in the use of treating oral defects and compromised maxillae (Figure 9-51).

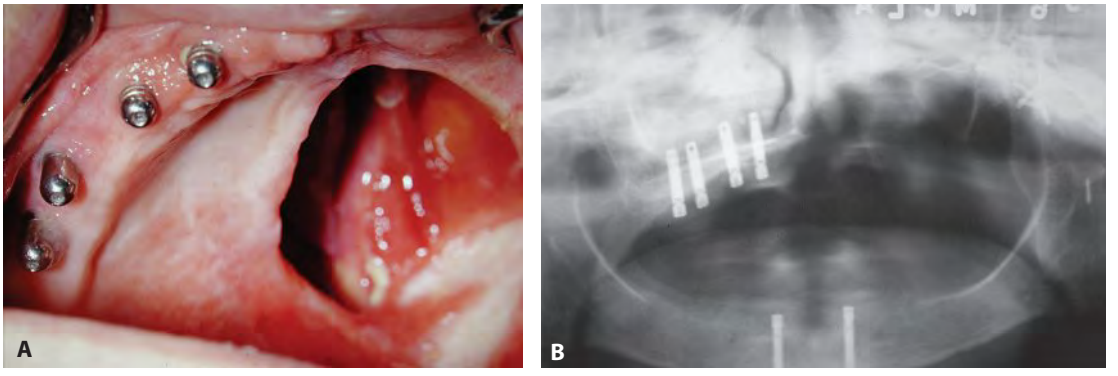


FIGURE 9-50. **A** and **B,** Implants placed in the nondefect side of a sinus-lifted maxilla. (**A** and **B,** Reproduced with permission from Salinas TJ, Guerra LR, Rogers WA. Aesthetic considerations for maxillary obturators retained by implants. Pract Proced Aesthet Dent 1997;9:265–276.)

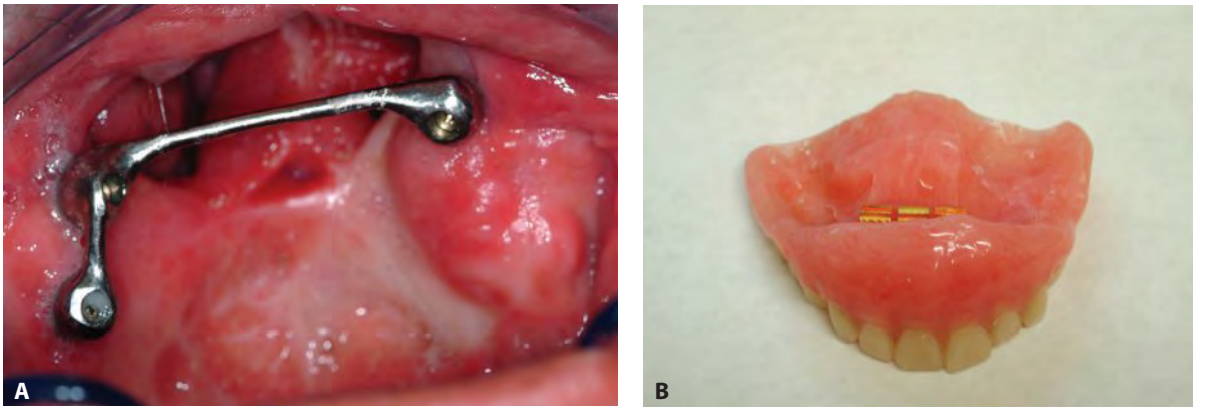


FIGURE 9-51. **A** and **B,** Implant-retained obturator using two zygomatic and one pterygoid implants.

Craniofacial Defects

Resection of portions of the craniofacial skeleton for disease control can result in both functional and aesthetic defects. These defects may not be suited to plastic surgical reconstruction owing to local or regional factors. Traditional roles for prostheses are to replace architecture with alloplastic materials that mimic the color and textures of adjacent skin. A method of retaining these prostheses can be attachment by medical-grade adhesives, which may be unpredictable in holding and irritate underlying soft tissues. In such instances, the use of osseointegrated technology can provide similar anchorage used intraorally. The rates of success in the craniofacial skeleton of implants are also well documented and should be planned out with specialized imaging. Three-dimensional reconstruction techniques may provide valuable information to maximize success of placement exclusively in the confines of intended site selection. The temporal bone is the most predictable site for the placement of implants in comparison with frontal nasal areas.[66] This is true even if radiation has been used to treat malignant tumors in this area. The choice of a minimum of two splinted implants in the temporal bone can serve well to provide a bar-retained prosthesis. Workup should include CT images with 2-mm axial cuts while a radiographic stent is worn (Figure 9-52).[67] This should affirm site selection as well as placement into sound bone. Bone-anchored hearing aids (BAHAs) can be used as well in treating patients with Treacher Collins syndrome or other forms of auditory agenesis.

Placement of implants into frontal nasal bone is possible with the use of specialized computer software to delineate the frontal sinus, anterior cranial fossa, orbit, and other vital structures adjacent to proposed site selection. Extraoral anchorage can, in some cases, assist with anchorage of an intraoral prosthesis as well (Figure 9-53).

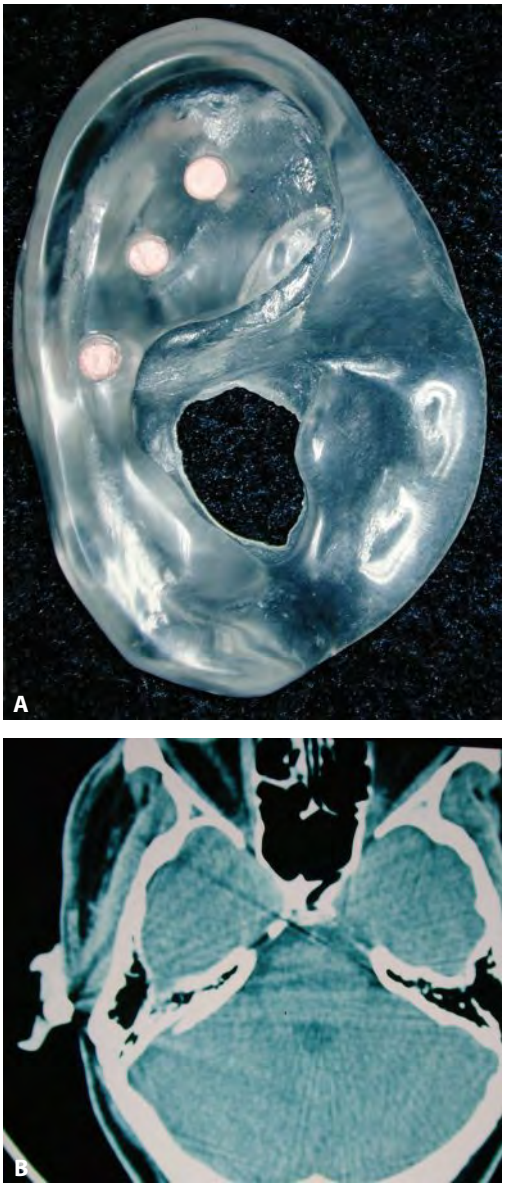


FIGURE 9-52. Stent **(A)** and computed tomography scan shows site selection **(B)** for implant placement into temporal bone.

Radiotherapy Concerns

Unlike elective implant placement, there are particular concerns when providing a patient with osseointegrated anchorage in cases in which optimal oral function is essential after tumor ablative surgery. Judicious use of interdisciplinary preoperative planning helps in deciding which cases may be appropriate for osseointegrated implants. This becomes critical when consideration is given to the relative risks of complications after radiotherapy to the head and neck. As with any oncologic case, radiation therapy may be incorporated to improve long-term survival. Because of absorptive changes in the osseous tissues, osteoblast populations are typically affected by dosages exceeding 50 Gy. The possibility of creating osteoradionecrotic wounds increases with bone manipulation above this dosage. However, osseointegrated implants have been successfully employed in previously radiated fields without undue complications.[68] Hyperbaric oxygen therapy has been objectively shown to reduce the risk of osteoradionecrotic complications in both the craniofacial skeleton and the intraoral regions.[69] As with any hypoxic wound, increasing oxygen tension above 40 P_{O_2} (partial pressure of oxygen) in comparison with a nonradiated control site increases the likelihood of healing. With this increase of O_2 concentration comes angioneogenesis and the subsequent effect of pleuripotential cell differentiation into osteoblasts.

COMPLICATIONS

Soft Tissue Complications

Soft tissue complications with dental implants can be seen in areas where the quantity of keratinized soft tissue is minimal. As with natural teeth, implant restorations rely on attached and keratinized tissue for long-term maintenance. Soft tissues may also be compromised in sites where implant angulation is not ideal in an aesthetic area. Finally, soft tissue depths



FIGURE 9-53. Facial and intraoral prosthesis anchored with two zygoma and three endosseous implants.

surrounding implants exceeding 5 to 6 mm may present problems with long-term maintenance. This can be especially true for areas grafted with soft tissues or in osteomyocutaneous flaps where dermis is quite thick. In these cases, it may be wise to reduce the soft tissue thickness surgically before making a restoration or even placing the implants.

Radiographic Bone Loss

Bone loss is expected with the placement of any implant; however, this loss should not exceed 1.5 mm in the first 12 to 18 months. Bone loss in excess of this value exposes a significant portion of the implant surface, making hygiene procedures difficult. If the choice of implant is a machined titanium screw, this problem is less than with implants having a textured surface, but in either case, it is desirable to see bone loss of no more than 0.2 mm/yr after the initial period of osseointegration and abutment/prosthetic connection. Evaluation of implants in edentulous patients by panoramic radiography may be more formidable than when using periapical examinations. However, partially dentate patients may benefit from periapical radiographs made with a silicone putty standardized bite block. In this way, radiographs would be standardized at each exposure, allowing interpretation at a consistent dose and incident beam angle.

Screw Loosening

Abutment and prosthetic screw loosening can be a recurrent problem seen often with single-tooth restorations. The incidence of screw loosening was significant before modern developments in screw mechanics.s. A further method of reducing screw loosening is to use a new abutment or prosthetic screw, torque once to the recommended torque application, wait 5 minutes, and then torque again.[70] In these circumstances, screw loosening is minimized. Repeated loosening of screws should bring to mind occlusal overload, excessive contact in lateral excursions.

Abutment Fracture

Abutment fracture is a relatively uncommon occurrence but can be problematic, particularly for cemented restorations. Material choices for implants subjected to heavy occlusion or unavoidable lateral loads should be carefully selected. Although strong, ceramic materials are used with caution in areas of high stress application. Premachined abutments used for screw-retained restorations can usually be replaced if they fracture.

Implant Fracture

Although relatively uncommon, implant fractures have been reported in only a few studies which account to less than 0.6%. These seem to be associated with commercially pure titanium implants of 3.75 mm diameter.[71,72] Further, the incidence appears to be higher in partially dentate patients than edentulous patients, perhaps owing to a rectilinear arrangement more so than a curvilinear arrangement. The trend toward use in internally connected implants continues, although no studies to date have surveyed the incidence of fracture. Pathognomic precursors are repeated screw loosening and peri-implant bone loss.

MAINTENANCE

Patients restored with osseointegrated implants should receive regular and frequent follow-ups in the first year after implant placement. Factors to evaluate include bone loss, mobility, and pain. Clinical examination should include light percussion and gentle evaluation of soft tissue. Radiographic evaluation includes both periapical and panoramic radiographs. If the restoration is screw-retained, it can be removed every 2 years, cleaned, and resecured or cleaned in position. Cleaning of implant and titanium abutment surfaces should be done with either gold or polyethylene (Teflon) instruments so as not to scratch these biologically critical surfaces and make them prone to plaque accumulation (Figure 9-54).[73]

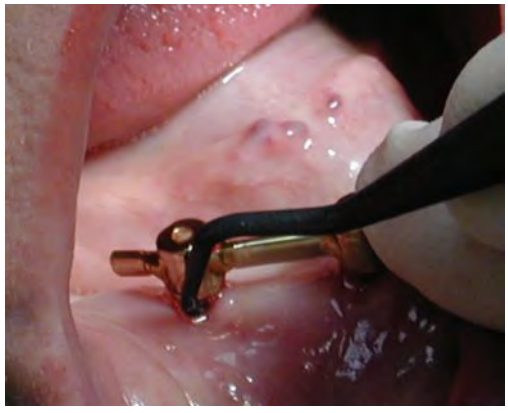


FIGURE 9-54. The use of polyethylene-tipped instruments facilitates plaque removal from titanium implant surfaces.

Any scratches or crevices created by this or other processes impose a nidus for plaque and calculus accumulation. After cleaning, polishing with either toothpaste or a light prophylaxis paste is recommended. Because a perimucosal seal exists between the implant and the abutment and tissue, it is not suggested that cemented restorations be removed routinely because this may jeopardize the integrity of the restoration and surrounding tissues. If abutment or coping screws have been torqued previously, it is generally suggested that they be replaced to avoid future fatigue fracture.

SUCCESS CRITERIA

Historically, the criteria of success have involved one of quantification of pain, mobility, and peri-implant radiolucency. These criteria were established by Albrektsson and coworkers[9] and remain one of the standards in long-term evaluation of Branemark-style dental implants. More recently, attaching a functionally aesthetic restoration to the implants were added as a criteria and removal of the original success rate of 85% at 5 and 80% at 10 years were updated at a 1998 Toronto Conference.[74] Even though the Albrektsson criteria have been established and validated in these types of implants in multiple populations in different areas of the mouth, they do not lend themselves well as guidelines for other types of implants. It will be necesssary to create success criteria for these other implant systems as they develop.

Removing the prosthesis (especially if it is splinted with other implants) and gently percussing with either a blunt instrument or a standardized torque instrument will give an indication of mobility. Other methods involved the use of Periotest instruments or nanodevices that promote radiofrequency response from the osseointegrated implant to give an indication of mobility.

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10

CHAPTER

The Science of Osseointegration

Craig M. Misch, DDS, MDS, and Carl E. Misch, DDS, MDS

THE BIOLOGY OF OSSEOINTEGRATION

Introduction

The science of osseointegration was introduced over 40 years ago by Professor Brånemark. *Osseointegration* has been defined as a direct structural and functional connection between ordered living bone and the surface of a load-carrying implant.[1] Although there have been several advancements in the field of implant dentistry, this basic concept remains the foundation of this treatment modality. This chapter focuses on the biologic development and maintenance of the bone-implant interface as well as the biomechanical effects of implant loading.

Implant Materials

Several biomaterials for dental implants have been evaluated over the years including surgical stainless steel, gold alloy, cobalt-chromium alloy, aluminum oxide, carbon, and titanium. The superior biocompatibility of titanium has rendered this material the preferred choice in modern dental implant therapy. The proven high biocompatibility of titanium as an implant material is connected with the properties of its surface oxide.[2] When exposed to air, titanium forms a dense 4-nm layer of oxide. Titanium dioxide (TiO_2) is the most common surface oxide. TiO_2 is chemically stable and very corrosion-resistant.[3]

Titanium

There are four grades of commercially pure titanium. The difference has to do with the small percentage of trace impurities in the metal.[4] The greater percentages of these contaminants, the harder the metal becomes, giving it higher strength. Grade 5, also known as Ti6Al4V, is the most commonly used titanium alloy. Titanium alloy offers equal biocompatibility but better tensile strength and fracture resistance than commercially pure titanium.[4]

Although the incidence of dental implant fracture is very low (<1.0%), it can have drastic consequences for the patient (Figures 10-1 and 10-2). Implant fracture can be attributed to design and/or material selection.[5] Grade 1 titanium implants with reduced diameter (3.75 mm) have been noted to have higher fracture rates. Thinner walls at the implant neck have also been found to be subject to fracture during insertion in dense bone and fatigue failure with loading[6,7] (Figure 10-3). The reduction of implant diameter is ultimately limited by material strength, especially with two-piece implants. Approaching a 3.0-mm diameter appears to be the limit unless a solid one-piece design is utilized[8] (Figure 10-4). Smaller-diameter one-piece implants are useful in limited spaces between adjacent teeth such as lower incisors and maxillary lateral incisors (Figures 10-5 to 10-7).

Zirconia

Zirconia, a ceramic material, has also been evaluated in the manufacture of dental implants and abutments. Zirconia

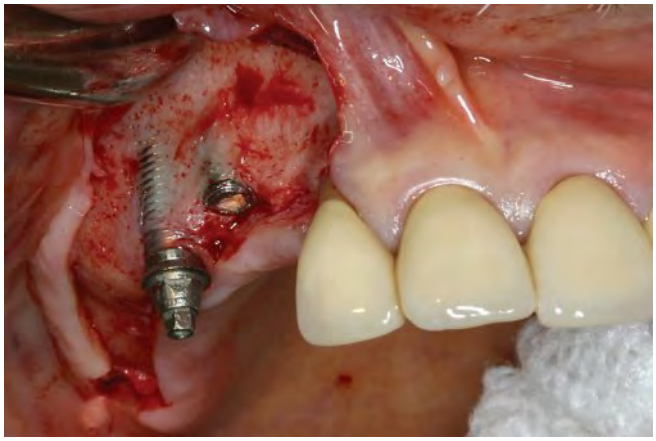


FIGURE 10-1. Fractured 3.75 mm Brånemark titanium implant.

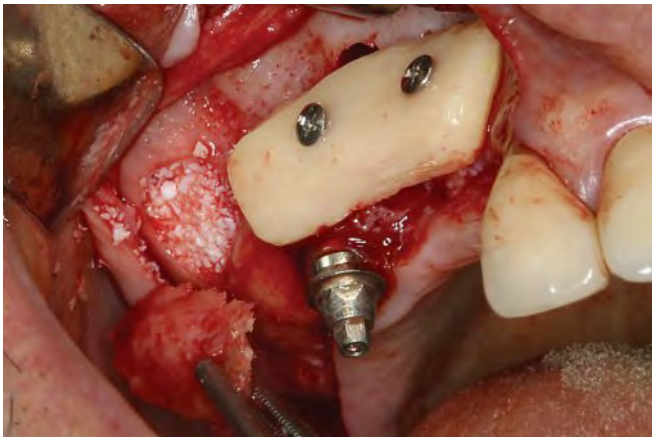


FIGURE 10-2. Reconstruction of the maxillary defect following removal of the fractured implant.

(ZrO_2), a dioxide of zirconium, has biocompatibility properties similar to those of titanium.[9] The obvious advantage of a ceramic is the potential for improved cosmetic appearance of the tooth replacement because there is no dark metal transgingival component (Figures 10-8 and 10-9). The strength of zirconia is much higher than the first-generation aluminum oxide abutments.[10] However, fracture resistance of zirconia is still a concern (Figure 10-10). Abutment preparation and

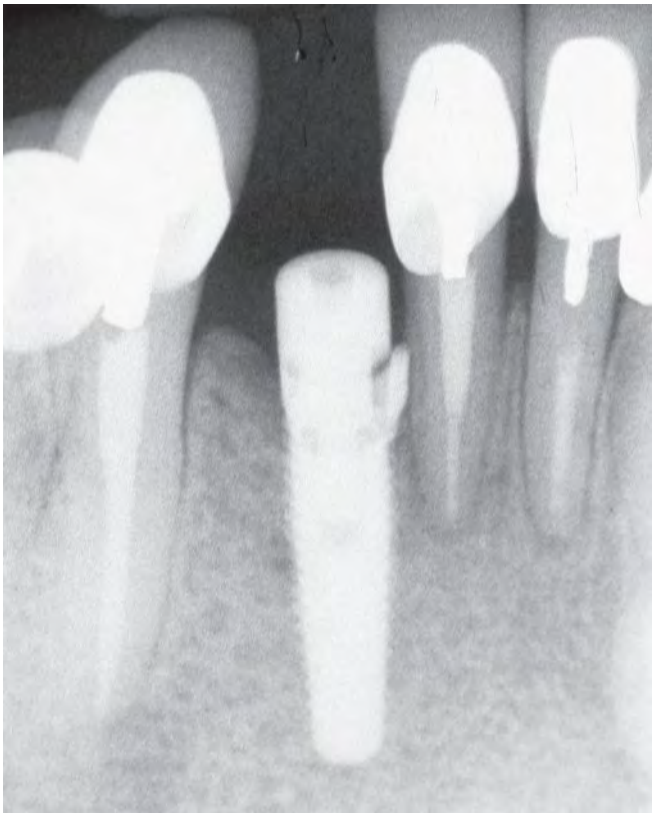


FIGURE 10-3. Fractured neck of a small-diameter (3.5-mm) titanium implant.

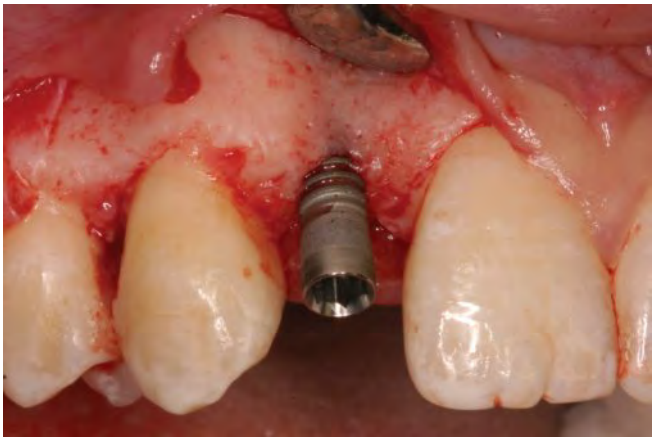


FIGURE 10-4. Two-piece, small-diameter (3.0-mm) titanium alloy implant.



FIGURE 10-5. One-piece, small-diameter (3.0-mm) titanium implant used to replace a lateral incisor.



FIGURE 10-6. Periapical radiograph of a small-diameter titanium implant (note limited space between the roots).



FIGURE 10-7. Final cement-retained implant crown.



FIGURE 10-8. Custom zirconia implant abutment.



FIGURE 10-9. Ceramic crown cemented onto the ceramic abutment.

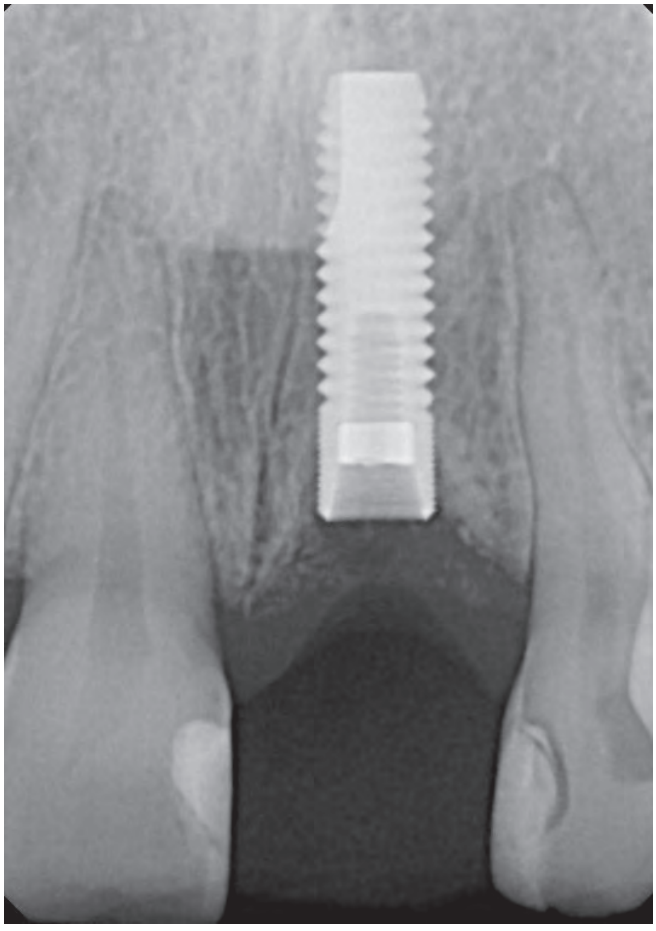


FIGURE 10-10. Periapical radiograph of the fractured zirconia abutment (see Figures 10-7 and 10-8).

improper seating can lead to fracture.[11] An inherent disadvantage of ZrO_2 dental implants is that they are one piece, requiring permucosal healing and an inability to correct angulation[12] (Figure 10-11). Long-term clinical data are necessary before one-piece ZrO_2 dental implants can be recommended for routine use.[13] Another recent development is a combination of titanium and zirconium alloy (TiZr1317) under the brand name Roxolid (Straumann USA, Andover, MA). This alloy is 50% stronger than titanium and shows significantly better mechanical attributes with respect to elongation and fatigue strength.[14] This material may be suitable for two-piece smaller implants and components exposed to high mechanical constraints.

Implant Surfaces

The surface properties of dental implants play an important role in molecular interactions, cellular responses, and bone formation. With the inception of osseointegrated implants, the titanium Brånemark fixture was consider the gold standard for implant surfaces. The implant was machined from a titanium rod by a turning process.[15] The resulting surface was rather uniform with ridges and valleys along the turning

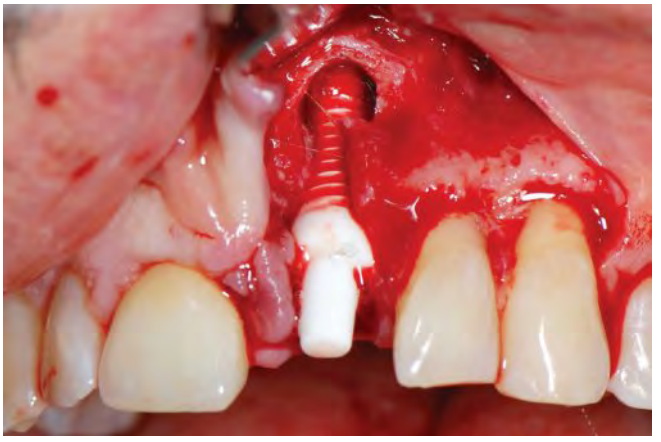


FIGURE 10-11. One-piece zirconia implant (CeraRoot, Oral Iceberg S.L., Barcelona, Spain).

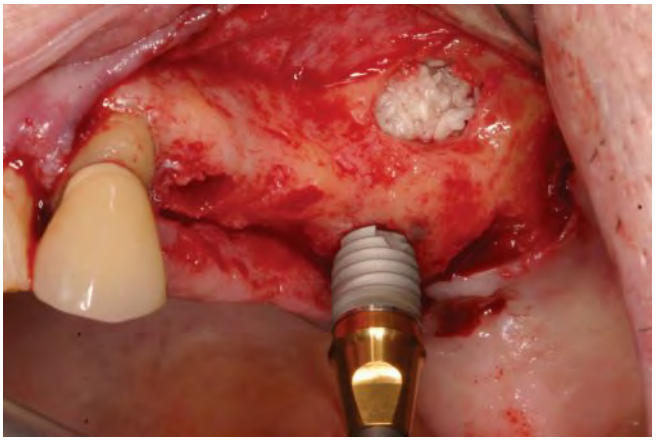


FIGURE 10-13. Microtextured implant placed into a sinus bone graft.

direction (Figure 10-12). Although many scientific publications describe this implant as a "machined" surface, there are several other types of machined surfaces including milled, ground, sandblasted, electrodischarged, and polished.[16]

Second-generation implant surfaces have been developed with chemical and topographic modifications to produce an increased surface topography. There is a general consensus that roughening the implant surface above the level of a turned or machined surface leads to a stronger bone response.[17,18] Experimental evidence from in vitro and in vivo studies strongly suggests that some types of surface modifications promote a more rapid bone formation than do machined surfaces.[19] However, this response could depend on an altered surface chemistry and/or an increased texture on the micrometer scale.[20] An implant with a rough surface may well perform better in sites with poor-quality or -quantity host bone. For example, improved implant survival is found in sinus bone grafts when microtextured implants are used instead of machined fixtures[21] (Figure 10-13). In these less favorable clinical situations, early and higher bone-to-implant contact would obviously be advantageous. Shorter implant healing times and earlier loading are other potential clinical benefits. Although implant treatments that provide a surface microtexture may improve the bone response, there is no clinical evidence demonstrating the superiority of any particular implant surface.[22]

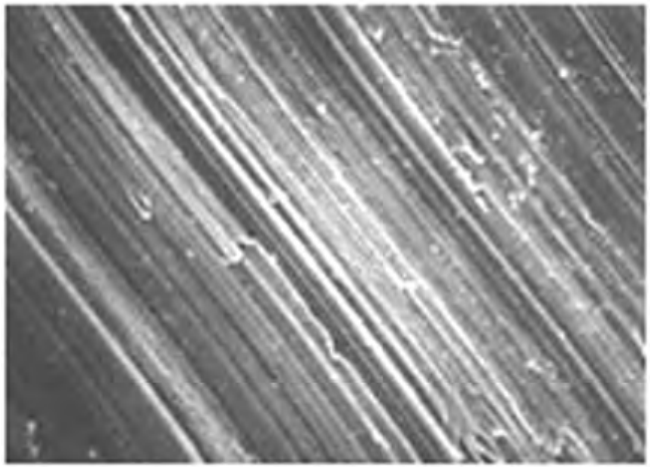


FIGURE 10-12. SEM photomicrograph of a machined implant surface.

A complex cascade of biologic events occurs in association with insertion of a dental implant. These events begin with the injury to bone from preparation of the implant osteotomy. Immediately after implant placement, there is an ionic exchange between the blood plasma and the implant surface.[23] Plasma proteins absorb onto the implant surface and are followed by initial cellular contact with erythrocytes, platelets, and leukocytes. The activated platelets release growth factors into the surrounding tissues. These cytokines are chemotactic for fibroblasts, osteoblasts, and mesenchymal stem cells. As tissue healing progresses, the surrounding blood clot serves as a reservoir of growth factors and acts as a provisional scaffold into which osteogenic mesenchymal cells can migrate.[23] In this regard, the implant surface topography is of extreme importance for retention of the blood clot. The microtextured implant surface increases fibrin adhesion and resists clot detachment.[23] This stabilization of the fibrin clot helps ensure that osteogenic cells will migrate directly to the implant surface and begin bone deposition.

Implant Surface Alterations

Unfortunately, there is no consensus in the literature on what surface topography constitutes smooth or rough. The height-descriptive two-dimensional profiles R_a, R_q, R_z, and R_t are the most commonly used parameters to describe the texture of dental implant surfaces.[16] Surface roughness, also known as surface profile R_a, is a quantitative calculation of the relative roughness of a linear profile or area. Three-dimensional surface area roughness is expressed as S_a. In the comparison of implant surfaces, average roughness over a surface (Sa) is more important than average roughness (Ra), a two-dimensional measurement.[19] Albrektsson and Wennerberg[20] suggested the following parameters: smooth surfaces have an Sa

value of less than 0.5 µm; minimally rough surfaces have an Sa of 0.5 to 1.0 µm; moderately rough surfaces have a, Sa of 1 to 2 µm; and rough surfaces have am Sa of greater than 2 µm. In animal experiments, it seems as though a moderately rough surface with an S_a of about 1.5 µm appears optimal to promote the strongest bone response.[19] Both smoother and rougher surfaces present less robust bone responses than this hypothetical ideal.[19] The long-term influence of a moderately rough implant surface on soft tissue implant health and the risk of developing peri-implantitis is less certain.[24,25]

The surface topography of dental implants may be altered by subtractive or additive processes. Subtractive processes include electropolishing, mechanical polishing, blasting, and etching. Additive processes include oxidation, hydroxyapatite (HA), and other forms of calcium phosphate coating, titanium plasma spray, and ion deposition. Blasting, etching, plasma spray coatings, and oxidation create increased surface roughness and enlarged surface area.

Subtractive Implant Surfaces

Blasting

Blasting may be done by aluminum oxide, HA, or TiO_2 powder with various grit particle sizes (small, medium, large). This process produces a moderately rough surface. Aluminum oxide is insoluble in acid and is thus hard to remove from the titanium surface. There has been some concern with sand blasting using aluminum oxide as residual particles may have a negative effect on bone formation.[26] The use of calcium phosphate, a resorbable blast media, has been offered as an alternative more biocompatible material[27] (Figures 10-14 and 10-15). Titanium oxide is also used for blasting titanium dental implants (Figure 10-16). Surface roughness depends upon particle size, time of blasting, pressure, and distance from the source of particle to the implant surface. Blasted titanium implants demonstrate improved bone integration

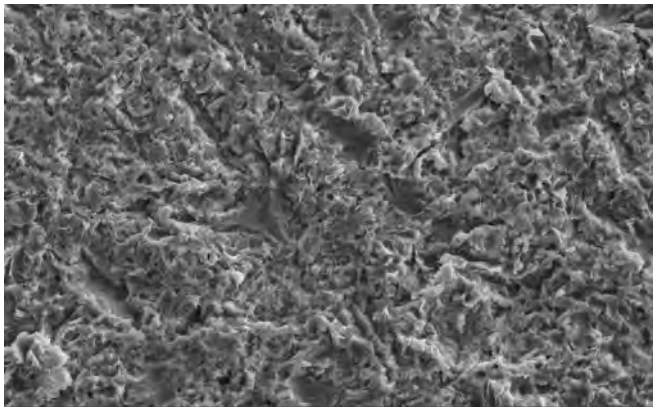


FIGURE 10-15. SEM photomicrograph of an implant surface blasted with HA particles (MTX, Zimmer Dental, Carlsbad, CA).

over turned/machined implants.[28–30] Comparative clinical studies gave higher marginal bone levels and survival rates for TiO_2 grit-blasted implants than for machined-turned implants.[31,32]

Acid Etching

Although titanium is corrosion-resistant, some acids can be used to etch the surface. Sulfuric, hydrochloric, and hydrofluoric acids have been used for the etching process. The acid erodes the implant surface, creating micropits of specific diameter and shape.[33] This process may increase surface area but not necessarily the average height deviation. The result is a clean, highly detailed surface texture, devoid of impurities and minimal to moderate roughness[16] (Figure 10-17). In animal and human studies, etched surfaces demonstrate better bone-to-implant contact at earlier time periods than machined/turned implants.[16,34] With this enhanced bone response, a clinically shortened implant healing time has been proposed.[35] Comparative clinical studies with machined/turned

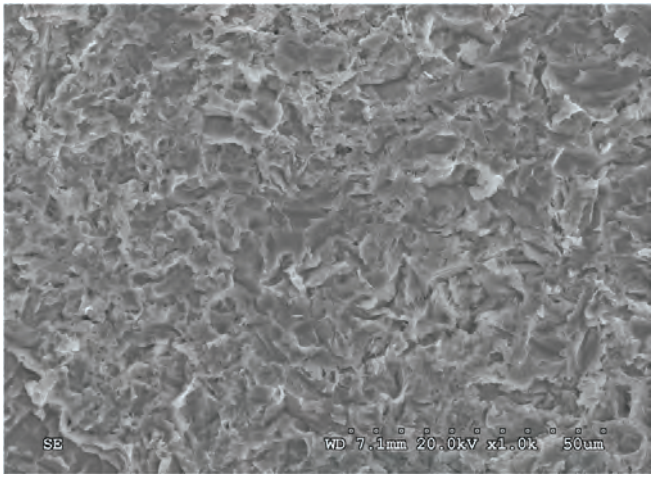


FIGURE 10-14. SEM photomicrograph (1000×) of an implant surface blasted with hydroxyapatite (HA) particles (RBT, BioHorizons, Birmingham, AL).

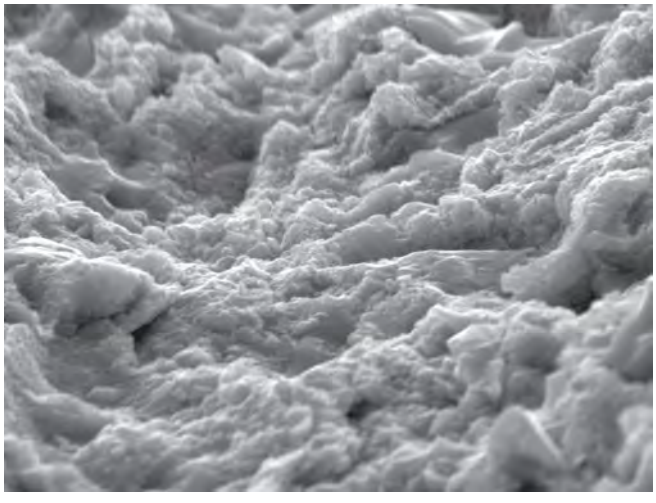


FIGURE 10-16. SEM photomicrograph of an implant surface blasted with titanium oxide particles (Osseospeed, Astra Tech, Molndal, Sweden).

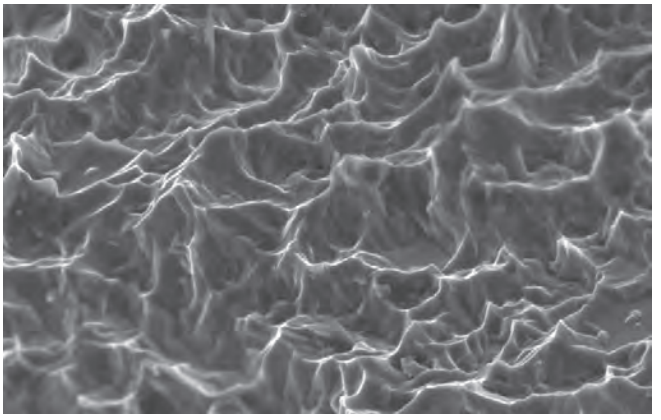


FIGURE 10-17. SEM photomicrograph (2000×) of an acid-etched implant surface (Osseotite, Biomet 3i, Palm Beach Gardens, FL).

implants suggest higher implant survival in poorer quality bone, compromised sites, and smoking patients.[36–38] A hybrid design, with only partial acid etching, was originally proposed to avoid bacterial adherence onto the rougher surface (Figure 10-18). However, this minimally rough implant surface does not appear to be at risk of peri-implant tissue breakdown.[39]

Blasting and Etching

A combination of blasting and acid etching has become another commonly utilized surface modification method for titanium dental implants. The surface is produced by sand-blasting with a large-grit aluminum oxide followed by etching with hydrochloric/sulfuric acid. Sand-blasting results in a moderately rough surface and acid etching creates a more complex microtexture while cleansing the implant[40] (Figure 10-19). The abbreviation SLA was introduced by Buser and coworkers[17] to describe the unique surface and stands for sand-blasted, large grit, and acid-etched. Compared with machined/turned implant surfaces, blasted and etched implants are definitely found to be more strongly integrated in bone.[16] The comparison of blasted and etched implants with other surfaces is less certain.



FIGURE 10-18. A hybrid design of the implant surface with the acid-etched portion placed into the bone and the machined surface near the ridge crest.

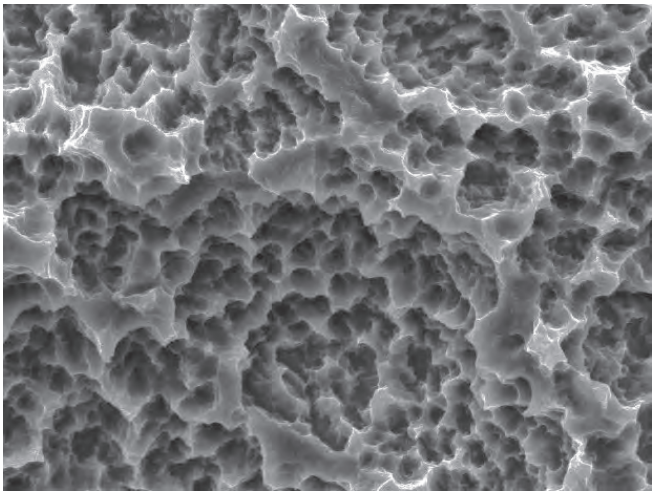


FIGURE 10-19. SEM photomicrograph of an implant surface sand-blasted with large-grit aluminum oxide followed by acid etching (SLA, Straumann, Basel, Switzerland).

Additive Surfaces

Titanium Plasma Spray

The titanium plasma-sprayed surface (TPS) is created by applying titanium particles to the implant surface under high velocity and extreme heat. This yields a bumpy, irregular surface configuration. It has been shown that this rough three-dimensional topography increases the tensile strength at the bone/implant interface.[17] TPS surfaces appear to elicit a better bone response than turned/machined–surfaced implants.[41,42] Because a cylinder implant lacks a macrogeometry for load transfer to the bone, many press-fit implant systems used the TPS surface to enhance bone integration. A patient study comparing SLA and TPS implant surfaces found no clinical difference was observed between these two surfaces.[43] However, clinical reports have suggested that dental implants with TPS surfaces often have more marginal bone resorption and peri-implantitis than other minimally to moderately rough surfaces[44–46] (Figure 10-20). As such, their production and clinical use has significantly diminished.

HA Coating

Implant coatings with calcium phosphate and HA are another type of additive surface treatment. Although several different methods have been developed to coat metal implants with HA, the plasma-spraying technique has been used most often in clinical applications (Figure 10-21). To obtain mechanical retention of the coating, the surface of the metallic implant is usually roughened by grit blasting. Under a plasma flame, HA particles are propelled onto the titanium implant surface in an inert environment, such as argon. The HA particles condense and fuse together, resulting in a surface thickness between 50 and 100 μm. Thinner surface coatings can be produced by

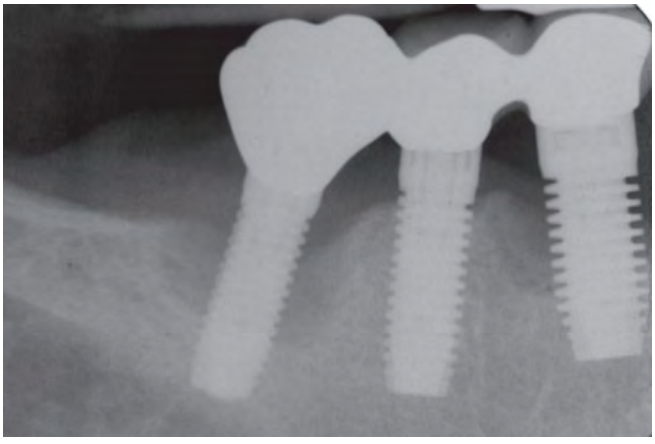


FIGURE 10-20. Periapical radiograph of severe marginal bone loss from peri-implantitis around titanium plasma-sprayed (TPS) implants.

an ion sputtering technique. This is done by directing a beam of ions onto an HA block, which vaporizes to create a plasma, and then recondensing this plasma on to implant.

HA coating of the implant offers some unique features. Animal studies have found bone bonding with greater interfacial implant to bone strengths compared with uncoated implants.[47] In addition, HA coatings have been shown to accelerate bone formation and produce a greater surface area of bone apposition around implants.[48,49] The earlier bone formation is attributed to contact osteogenesis between the prepared host bone and the osteoconductive implant surface.[50,51] The enhanced bone response around HA-coated implants makes them an attractive choice for compromised sites.[52] Clinical comparison has found greater implant survival of HA-coated implants in poor bone quality.[53]

Some reports suggest that the HA coating may separate from the substructure, undergo dissolution in tissue fluids, and contribute to bone loss around the implants.[54] The chemical properties of the HA coating influence the long-term stability. Whereas a porous, amorphous surface can degrade, a denser, highly crystalline surface resists dissolution and in vivo resorption.[55] Manufacturers of implant systems have refined techniques to obtain more consistent coatings with high crystallinity. One concern regarding coatings is the bond strength between the HA and the metal substrate. The bond strength between the HA and the bone is superior to the HA and the implant surface. Although there have been isolated reports of coating failures,[56,57] the overall clinical reliability has been extremely favorable.[58–60] Novel application techniques of HA have resulted in much thinner layers with lower risk of fracture from the substratum. The concern has also been raised that HA coatings increase the risk of developing peri-implantitis.[61] Induced peri-implantitis in animal studies suggests the rough surface could be a liability.[62] There are some clinical reports of favorable early implant survival with poorer long-term results.[63–65] The periodontally compromised patient may be more susceptible to failure.[66] Despite numerous other clinical studies documenting high survival rates, the controversy over the long-term clinical effectiveness of HA-coated dental implants persists.[58–60] The current clinical trend is to utilize moderately rough surfaced implants rather than rough plasma-sprayed implant surfaces.

Oxidation

All titanium implants have a surface oxide layer of approximately 5 nm in thickness.[2] Oxidized implants are prepared with a thicker oxide layer. This is developed through heat treatment or placing the implant as an anode (anodized) in a galvanic cell with a suitable electrolyte.[67]

When strong acids are used in the electrolyte solution, the oxide layer will be dissolved along current convection lines and thickened in other regions. The dissolution of the oxide layer along the current convection lines creates micropores on the titanium surface[68] (Figure 10-22). Anodized implants

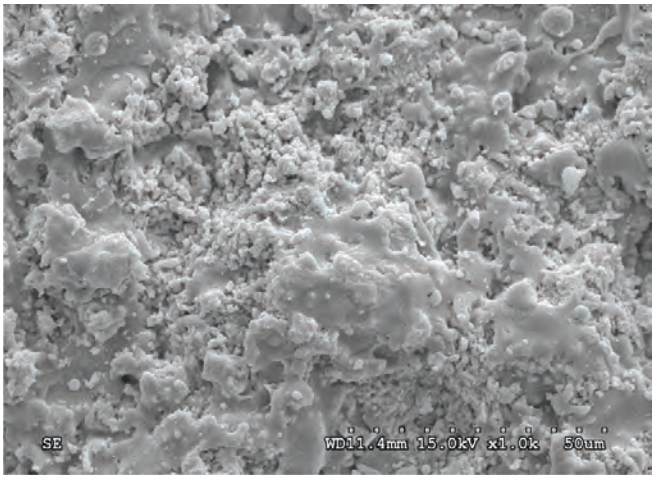


FIGURE 10-21. SEM photomicrograph (1000×) of an implant surface coated with plasma-sprayed HA.

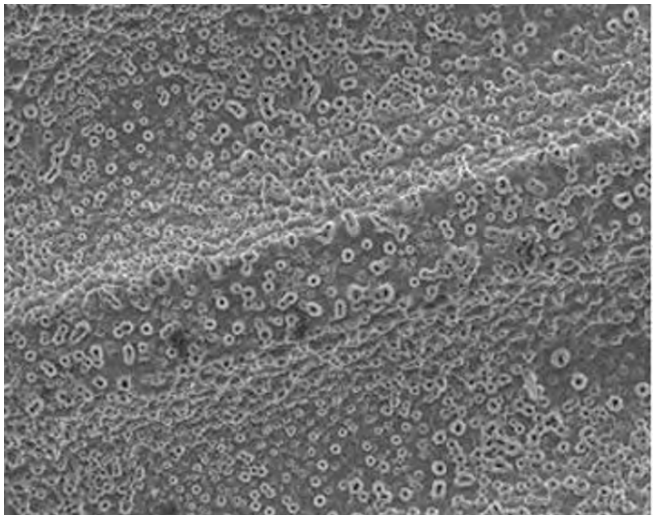


FIGURE 10-22. SEM photomicrograph (5000×) of an anodized implant surface (TiUnite, Nobel Biocare, Yorba Linda, CA).

treated with different voltages produce varying levels of surface roughness.[67] For oxidized surfaces, there is often no correlation between height deviation and bone integration.[16] Because anodized implants are usually considered minimally rough, the oxidation process may change topographic properties other than the height deviation.[16] Grade 1 titanium is currently used for commercially available anodized implants.[69] As such, implants with narrower diameters could be more prone to fracture. An interesting feature of heat-treated or anodized titanium is the ability to alter the color of the metal. The thicker oxide layer alters the reflection of light from the implant surface, producing colors such as green, blue, yellow, and magenta.

Human studies have concluded that anodized implants demonstrate more bone-to-implant contact and stronger anchorage than machined/turned surfaces.[70,71] In a clinical study on immediate implant loading, anodized implants had a higher success rate compared with machined/turned implants with an identical design.[72] However, a 5-year retrospective investigation of a mixed use of machined and anodized implants in patients found no significant difference in implant survival.[73] Although there were initial impressions that the anodized implant surface could also improve the soft tissue interface, there is no evidence to support this opinion.[24,74] Indeed, there are suggestions that extending the anodized surface superior to the bone may attract microbes and cause peri-implantitis.[75–77]

New Surface Modifications (Table 10-1)

Surface Energy

To further enhance the SLA surface, a change from a hydrophobic to a hydrophilic surface was developed.[78] Hydrophilic surfaces would seem more desirable than hydrophobic implants in view of their interactions with biologic fluids, cells, and tissues. Before acid etching, the implants are submerged in sodium chloride under nitrogen protection. This results in a very clean hydrophilic surface. Although the SLA surface topography is unchanged by the process, animal studies have shown more bone-to-implant contact at 2 to 4 weeks.[78] Clinical studies on this modified surface using a short healing protocol (3 wk) have found favorable results.[79,80]

TABLE 10-1. **Implant Manufacturers Surface Properties**

Manufacturer	Surface Trade Name	Process
Astra Tech	Osseospeed	Titanium blasting + fluoride
BioHorizons	RBT	Blasting, soluble HA particles
BIOMET 3I	Osseotite	Acid etch
BIOMET 3I	Nanotite	Acid etch + calcium phosphate deposition
Nobel Biocare	TiUnite	Oxidized titanium
Straumann	SLA	Sand blasting (large grit), acid etch
Zimmer	MTX	Blasting, soluble HA particles

HA = hydroxyapatite.

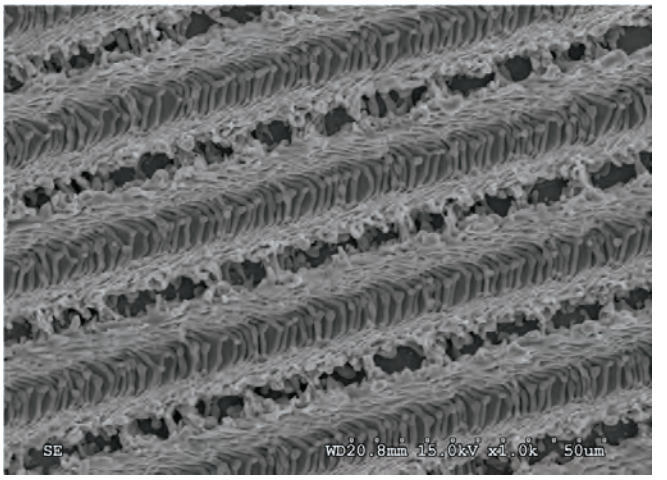


FIGURE 10-23. SEM photomicrograph of an implant neck with laser microchannels (Laser-Lok, BioHorizons, Birmingham, AL).

Ultraviolet light exposure of implants is also under investigation to increase the surface hydrophilic status.[81]

Laser Etching

A unique subtractive surface treatment is produced by precision laser etching microchannels into the implant neck. Linear grooved repeating patterns are created with the laser in the range of 8 to 12 μm (Figure 10-23). The laser microchannels have been found to inhibit epithelial down-growth and provide an area for connective tissue attachment on the collar of the implant[82] (Figure 10-24). This surface has also been shown to enhance bone attachment and reduce marginal bone loss compared with an identical implant design without the microchannels.[83,84]

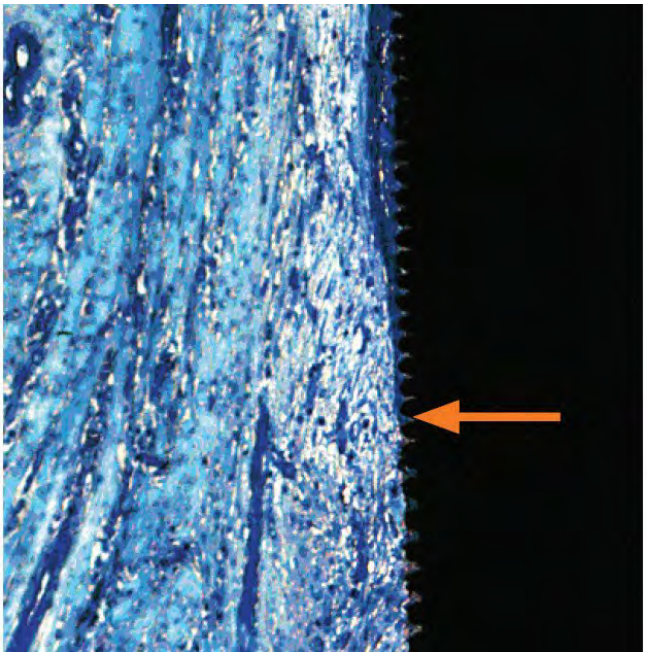


FIGURE 10-24. Photomicrograph of the connective tissue attachment to the laser microchannels.

Nanotechnology

Dental implants with nanometer-sized surface irregularities represent a new generation of surface treatments. There has been a growing interest in how the presence of nanometer structures on a dental implant surface influences the bone healing. Recent in vitro studies have revealed an increased osteoblast response to different nanophase surfaces.[85,86] The nanometer roughness may present more binding sites for proteins and adhesion of osteoblastic cells.[86] Some commercially available implant brands have been developed with nanometer structures. There is evidence of a stronger bone response to these newer implants compared with their predecessors.[19] However, there may be explanations for these findings other than the nanometer roughness.[19] In addition, the optimal surface nanotopography for enhanced biologic activity is unknown. A greater degree of nanometer-level roughness may be better, worse, or even irrelevant in the clinical results of an implant.[19]

Ions-Fluoride

Development of the TiO_2 grit-blasted implant surface has included chemical treatment with fluoride to further modify the topography.[87] Incorporation of small amounts of fluoride ions in the oxide layer, a slight increase on the micrometer scale in surface roughness, and the appearance of a nanoscale topography have been reported for this surface.[88,89] Animal and human studies indicate that the fluoride-treated surface leads to increased bone formation and stronger bone-to-implant bonding at shorter healing times than TiO_2 grit-blasted or machined titanium surfaces.[85,90] However, because the level of fluoride ions attached to the implant surface is quite low (<1%), the chemical effect of such a small amount has been questioned.[19]

Calcium Phosphate

In order to avoid the drawbacks of plasma-sprayed HA coatings, scientists have developed new methods. In one technique, calcium phosphate nanoparticles are applied to the implant surface using discrete crystalline deposition. The calcium phosphate on the implant is not a coating but consists of actual deposits of discrete crystals (Figure 10-25). A significant increase in osteoconduction has been found as a function of the enhanced surface nanotopography obtained by the nanocrystals and the known biologic benefits of calcium phosphate in bone formation and healing.[91] A human study using test implants in the maxilla found a stronger bone response to surfaces having nanometer-sized calcium phosphate deposits compared with acid-etched implants.[92] Although one cannot rule out the chemical effect of the attached HA, other factors may explain the enhanced bone response.[19] Clinical evaluations have found favorable outcomes with this surface technology.[93,94]

Another approach to applying calcium phosphate coatings to titanium implants mimics the natural process of biomineralization. One method involves the electrolytic deposition of calcium phosphate. The electrochemical deposition performed in simulated body fluids can produce a carbonated apatite coating directly on the titanium surfaces.[95] Simulated body fluids are rich in calcium, phosphorus, and other elements. This method makes possible perfect control of the thickness of the deposit on all kinds of complicated surfaces. A second method is based on the biomimetic precipitation of calcium phosphate on titanium surfaces by immersion in simulated body fluids.[96] This method involves the growth of bonelike crystals on the surface of the implant. It has been shown that such biomimetic coatings are more soluble in physiologic fluids and resorbable by osteoclastic cells than high temperature coatings such as plasma-sprayed HA.[97] Studies have demonstrated a higher bone-to-implant contact for biomimetic calcium phosphate coatings than for uncoated titanium implants.[98,99] However, studies need to be performed to compare this implant coating with other surface treatments.

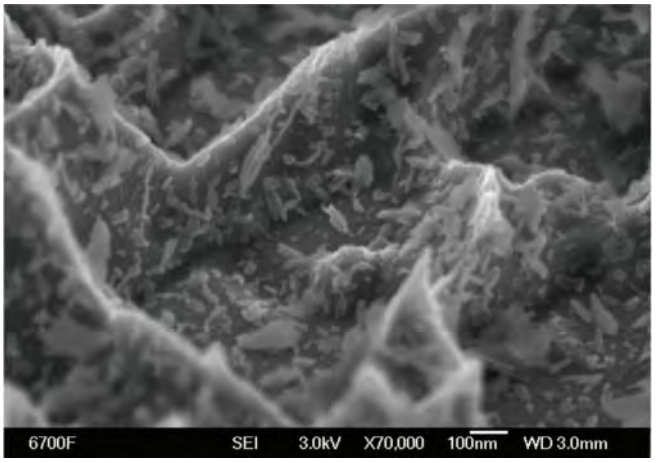


FIGURE 10-25. SEM photomicrograph of the an acid-etched implant surface with nanocrystals of calcium phosphate deposits.

Bioactive Proteins—Bone Morphogenetic Proteins

Future directions include the addition of bioactive proteins onto the dental implant surface. These agents can be used to enhance the bone healing process locally. Bone morphogenetic proteins (BMPs) are the most promising group with the greatest therapeutic potential. There are several types of BMPs but BMP-2 and BMP-7 have been the most studied because they have the significant osteoinductive properties.[100] Recombinant versions of the human BMP protein, such as rhBMP-2, are used for these applications. Additional research is also currently being performed on the use of rhBMP-2 for the repair of peri-implant bone defects (Figure 10-26).

Because the titanium surface possesses a paucity of reactive function groups for retention of the biomolecule, the metal must be modified to enable immobilization of the proteins. Conditions during plasma polymerization of allyl amine on titanium are varied to yield NH_2 surfaces for BMP binding as an alternative to the silane method used in other studies.[101] Surface modification with bioactive molecules on

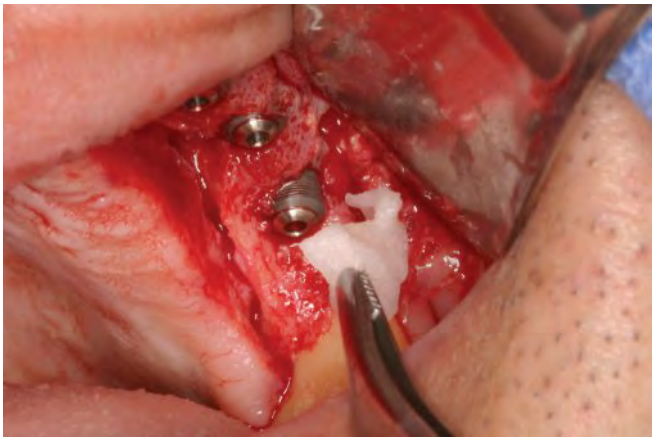


FIGURE 10-26. A collagen sponge infused with rhBMP-2 is used to repair the peri-implant bone defect.

a nanometer scale is a main field in current biomaterial research. Nanomechanical fixation of nucleic acid strands into an anodized titanium oxide layer allows bioactive molecules to conjugate and become immobilized.[102] The nanotubular structure formed by anodizing the titanium surface is a promising configuration for sustained BMP delivery.[103] Another limiting factor of coating implant surfaces with bioactive molecules is in their release kinetics. Ideally, they would be released gradually and not in a single burst. Incorporation of BMP into a biomimetic calcium-phosphate coating allowing gradual resorption has been suggested as one approach.[104] Effective dosing is also critical for the successful use of BMPs. One animal study found anodized titanium implants coated with recombinant human BMP-2 showed significant potential to stimulate local bone formation.[105,106] The therapeutic dosage appeared to be in the range of 0.75 to 1.5 mg/mL. Higher concentrations exhibited more immature bone, seroma formation, and bone remodeling, resulting in undesirable implant displacement.[106]

Bioactive Proteins—Cytokines

Numerous cytokines have been researched for incorporation onto implant surfaces including platelet-derived growth factor (PDGF), transforming growth factor-beta (TGF-β), basic fibroblast growth factor (FGF), insulin-like growth factor (IGF), growth hormone (GH), and many others.[107] Although these growth factors are not osteoinductive, they are important regulators of wound healing. The α-granules in the platelets contain several growth factors including PDGF and TGF-β. Platelet concentrates have been evaluated as a source of autologous growth factors for the coating of implants prior to insertion into the prepared bone. Animal studies have found accelerated osseous healing and increased bone-to-implant contact.[108–110] Recombinant technology is utilized to produce exogenous growth factors for experimental use. As previously discussed with BMP, the growth factors can be incorporated into carriers, such as calcium phosphate coatings or polymers. Although some research may find an enhanced bone response to a specific growth factor studied, other experiments are less encouraging. There is no unequivocal evidence available suggesting a positive effect on the implant-bone integration of growth factors coated on titanium dental implants.[111] These are indeed complex molecules with multiple interactions. Much additional research is required before human clinical trials on cytokine implant surfaces will result.

Pharmacologic Coatings

A different strategy to influence bone healing around dental implants is to utilize pharmacologic agents to control bone remodeling. The incorporation of bone antiresorptive drugs, such as bisphosphonates, may have a positive effect on implants placed in poor-quality bone. The antiresorptive agent acts as an inhibitor of osteoclast activity, which may result in more net osteoblastic activity. Early studies on bisphosphonates incorporated onto titanium implants found an increased density of the surrounding bone.[112] In addition, increased bone formation rate, greater bone-to-implant contact, and high removal torque have been noted.[113,114] A recent human study found implants coated with bisphosphonate placed in poor-quality sites had higher stability measurements than uncoated implants after healing.[115] Similar to the issues with using growth factors, drugs require a sustained release to have the desired effect.[116] Bisphosphonates have a high chemical affinity for calcium phosphate, so incorporation of the agent into a biomimetic coating may be a solution. A recent human study used a fibrinogen matrix to carry the drug.[115] The encouraging findings on the use of bisphosphonate coatings warrants further investigation.

THE BIOLOGY AND BIOMECHANICS OF OSSEOINTEGRATION

The application of engineering principles to dental implant therapy has unfolded a new era in diagnosis, treatment planning, and rehabilitation in patient treatment. Biomechanics is concerned with the response of biologic tissues to applied loads. In implant dentistry, the supporting tissue of interest is bone. In addition to the host response, we must also be concerned with the mechanics of the prosthetic replacement and examine the behavior of these devices when subjected to forces.

The dental sciences may be separated into biologic and biomechanical components. In dentistry, the biologic aspects of oral health are often emphasized. Common diseases related to the natural dentition are primarily of biologic origin, including periodontitis, dental caries, and endodontic problems.[117–120]

When dental implant complications are reported, the vast majority of problems are related to the mechanics, rather than to esthetics.[121] Unlike natural teeth, the biologic aspects of dental implants have relatively fewer complications. For example, the development of a direct bone-implant interface is largely a biologic event. Most contemporary reports indi-

cate dental implants form a successful bone-supporting interface more than 95% of the time, regardless of the implant system used.[122–124] The biologic aspects of implant therapy are quite predictable.

The most common implant-related complications are biomechanical in nature and usually occur after the implant is loaded. A literature review focusing on implant failure indicated these problems primarily occur within 18 months of initial implant loading.[121] These early implant-loading failures occur most often in the softest bone types (16% failure) or the shortest implant lengths (17% failure). These two failure groups are typically related to biomechanical factors. Softer bone may be too weak for the occlusal forces applied to the implants, and short implants have higher stresses at the bone-implant interface.

The most common technical complications associated with implant therapy that do not lead to the implant failure are also related to mechanics. For example, the incidence of problems with implant overdentures includes attachment complications (30%) and prosthesis fracture (12%).[121] For implant-supported fixed prostheses, common complications include acrylic veneer fracture (22%), screw loosening (7%–10%), porcelain fracture (≤30%), and substructure metal fracture (3%).[121] In addition to these prosthetic complications, implant components (2%–4%) and even implant bodies may fracture (1%–2%).[121] Mechanical complications often outnumber the biologic problems associated with dental implants.

TABLE 10-2. **Health Scale for Dental Implants**

Implant Quality Scale	Clinical Conditions
I. Success (optimum health)	a. No pain or tenderness upon function b. No mobility c. <2-mm radiographic bone loss from initial surgery d. No exudate
II. Satisfactory survival	a. No pain on function b. No mobility c. 2–4 mm radiographic bone loss d. No exudate
III. Compromised survival	a. May have sensitivity on function b. No mobility c. >4-mm radiographic bone loss (<half of implant) d. Probing depth > 7 mm e. May have exudate
IV. Failure (clinical failure)	Any of the following: a. Pain on function b. Mobility c. Radiographic bone loss > half length of implant d. Uncontrolled exudate e. No longer in the mouth

From Misch CE, Perel ML, Wang HL, et al. Implant success, survival, and failure: the International Congress of Oral Implantologists (ICOI) Pisa Consensus Conference. Implant Dent 2008;17:5–15.

Marginal Bone Loss

Some degree of marginal bone loss following dental implant placement is seen with all endosteal dental implants. Although the amount of early crestal bone loss may vary, it typically stabilizes after the first year. Adell and colleagues[125] were the first to quantify and report on marginal bone loss around osseointegrated implants. Their retrospective clinical study found a greater magnitude of bone loss during the first year of loading, averaging 1.5 mm (range 0–3.0 mm). Subsequent annual marginal bone loss after this first year was much less, in the region of 0.05 to 0.1.mm. Early bone loss is such a common occurrence that many implant success criteria do not even quantify the amount of bone lost in the first year of function[126–129] (Table 10-2). Following the initial bone remodeling around the implant, there is a steady state with stable marginal bone levels in healthy sites. There have been several theories on why the initial bone loss occurs including surgical trauma, biomechanical response to stress, and the implant-abutment microgap. Other contributing factors include disruption of the soft tissue attachment, bacterial infection, and implant design.

Surgical Trauma

The Brånemark group theorized that early marginal bone loss around dental implants was due to surgical trauma including interference with periosteal vascularization from elevating a flap and marginal bone preparation.[130] They thought, with refinement of the marginal bone cutting technique, the bone loss could be reduced.

There is a small amount of marginal bone loss reported with flap reflection around natural teeth.[131,132] It may be tempting to apply this concept to explain bone loss from implant surgery. Although some crestal bone cells may die from the initial trauma of periosteal reflection, the blood supply is reestablished as the periosteum regenerates. If there was bone loss from flap reflection, it would affect the entire exposed ridge in a generalized horizontal pattern and not vertical bone remodeling localized around the implant neck. In addition, bone loss would also be observed following implant insertion to second-stage surgery, which is rarely the case.

The heat generated from bone preparation during drilling was a concern during the development of osseointegrated dental implants. They advocated a graded series of sharp drills, profuse saline irrigation, and a maximum drill speed of 2000 rpm to prevent overheating.[130] Research had shown the threshold temperature for bone damage was 47°C for 1 minute.[133] As previously questioned, if surgical trauma from drilling caused early crestal bone loss, it should be evident at the second-stage surgery (Figure 10-27). There are implant systems that use countersink drills that prepare the denser cortical bone crest and this could possibly cause thermal injury. However, this theory does not explain the marginal bone changes seen after abutment connection and loading.

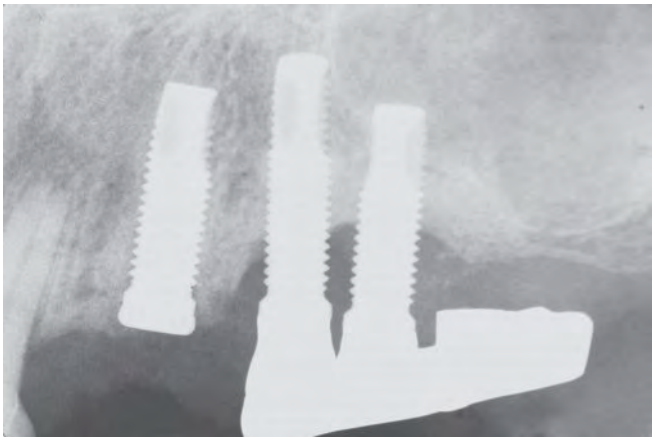


FIGURE 10-27. No marginal bone loss around the submerged implant after 6 months healing. Note the marginal bone loss around the implants loaded by a cantilevered bar.



FIGURE 10-28. Finite element stress analysis model reveals higher stress concentration around the neck of the loaded dental implant (Laser-Lok implant, BioHorizons, Birmingham, AL).

Biomechanical Response

A widely accepted theory on bone formation comes from the classic Wolff's law.[134] Wolff proposed that bone reacts to the forces placed upon it. Any change in the function of a bone leads to changes in its internal architecture and in its external form. His theory led to a load-deformation relationship for bone, in which *load* is defined as stress (force/unit area), and *deformation* is defined as strain (change of length). A stress-strain curve was developed to determine how much stress and strain is beneficial for a bone and how much is detrimental, resulting in bone loss.[135]

Engineering Principles

The relationship between stress and strain determines the modulus of elasticity (stiffness) of a material. The modulus conveys the amount of dimensional change in a material for a given stress level. The modulus of elasticity of a tooth is similar to cortical bone. Dental implants are typically fabricated from titanium or its alloy. The modulus of elasticity of titanium is 5 to 10 times greater than that of cortical bone. An engineering principle called the *composite beam analysis* states that when two materials of different elastic moduli are placed together with no intervening material and one is loaded, a stress contour increase will be observed where the two materials first come into contact.[136] The stress contour forms a V- or U-shaped pattern, with greater magnitude near the point of first contact. In the dental implant-bone interface model, these stresses are of greatest magnitude at the crestal bone. This phenomenon has also been observed in both photoelastic and three-dimensional finite element analysis studies when implants are loaded within a bone model[137–140] (Figure 10-28). The results of these analyses suggest that a certain amount of marginal resorption may be the result of biomechanical adaptation of bone to stress.

Implant Design Biomechanics

Different amounts of marginal bone loss have been reported for different implant body designs. The macro design and surface conditions of the implant body may affect the amount of strain distributed to an implant-bone interface. Press-fit, cylinder implants lacking threads, require a rough surface (TPS, HA) to improve integration for biomechanical stress transfer. Threaded implants not only attain better initial stability in bone but also have the ability to transfer forces to the supporting bone through their thread geometry. The thread shape can influence the type of force that is transmitted at the interface (compressive, tensile, shear). Compressive forces tend to maintain the integrity of the bone-implant interface, whereas tensile and shear forces are disruptive. Cortical bone is strongest in compression and weakest in shear.[141] Therefore, an implant design that maximizes compressive loading and minimizes tensile and shear forces may offer advantages. Under axial loading, an implant with V-shaped threads has approximately 10 times greater shear component of force than a square thread.[142] In addition to thread shape, the functional surface area of the bone-implant interface is important. *Functional surface area* is defined as the area of the implant that actively serves to dissipate loads into the supporting bone.[143] The important portion of the thread is the area that participates in compressive load distribution under an axial occlusal load. Because most of the stress to the implant-bone interface is concentrated at the crest, this zone is critical for optimal stress distribution. Although apical threads are important for achieving primary stability, there is minimal load transfer through this portion of the implant.

One strategy for enhancing stress distribution around the implant neck is to incorporate designs for improved force distribution. A polished or machined implant collar not only reduces initial bone formation and bone contact around the neck but also is a poor biomechanical interface transmitting primarily shear stress, because such marginal bone will typically remodel below the base of the collar to the threaded region or textured surface[144,145] (Figures 10-29 and 10-30).



FIGURE 10-29. Implant with a 1.5-mm machined collar placed into the anterior maxilla.



FIGURE 10-31. Microthreads around the implant collar have been proposed to improve stress distribution.

The bone remodels to this area because these mechanical features are more efficient in force transfer. Microthreads have been placed around the implant neck of some implant systems in an attempt to improve load distribution and decrease stress peaks[146,147] (Figure 10-31). Although clinical studies have shown minimal bone loss in implants incorporating this design feature, there may be other variables explaining this finding.[148,149]



FIGURE 10-30. Periapical radiograph reveals marginal bone loss to the base of the implant collar.

Cellular Biomechanics

Bone remodeling at the cellular level is controlled by the mechanical environment of strain.[150] Bone cells in conjunction with the extracellular matrix compose the strain-sensitive population. It has been theorized that the actual strain that is perceived by the bone tissue initiates a chain of events that result in a biologic response. Based on this rationale, a dental implant should be designed to establish an optimum strain environment at the bony interface that maintains osseointegration.

The amount of strain in a material is directly related to the amount of stress applied.[135] Occlusal stress applied through the implant prosthesis transmits stress to the supporting bone-implant interface. The amount of bone strain at the bone-implant interface is directly related to the amount of stress applied through the implant prosthesis. Frost[151] described the reactions of bone to microstrain levels as disuse atrophy, adaptation, mild overload, pathologic overload, and fracture (Figure 10-32). There is an optimum microstrain for bone maintenance. Increased levels above the adaptive

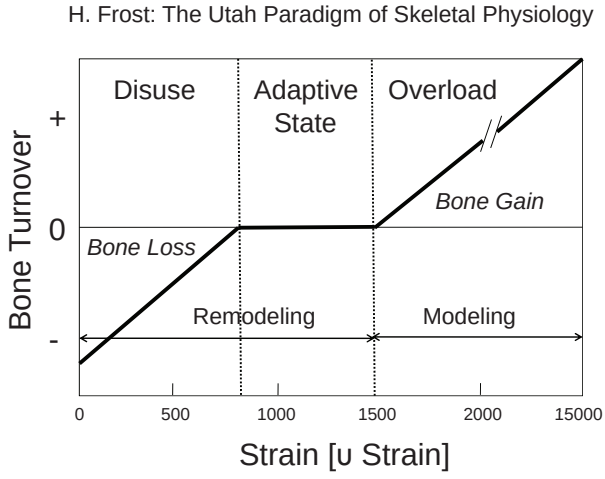


FIGURE 10-32. Frost's mechanostat theory on the reaction of bone to microstrain.

state can result in modeling and bone gain. An example would be the increase bone volume seen in the body of the mandible following improved masticatory function with an implant prosthesis.[152] Strain levels that are too high can result in bone loss around a dental implant. High strain at the crest beyond physiologic limits (pathologic overload) may cause microfractures of bone and result in resorption.[153,154]

Bone Density

The structure of bone is described in terms of quality or density, which reflects the strength of bone.[155] Bone density is directly related to the strength and elastic modulus of bone.[156] In denser bone, there is less strain under a given load compared with softer bone. As a result, there is less bone remodeling in denser bone compared with softer bone under similar load conditions.[151] A decrease in functional bone remodeling could result in a reduction of bone loss. In a prospective human study, Manz[157] observed the amount of marginal bone loss around implants was related to the bone density. Six months after implant uncovering, radiographic marginal bone loss was found to be gradually greater because the bone quality scale (1–4) was rated poorer. However, implant design may overcome some of the biomechanical influence of bone density.[124,158]

Animal Studies

Several animal studies in the literature demonstrate the response of bone to dental implant loading. Hoshaw and associates[159] investigated the hypothesis that mechanical loading of implants influences bone remodeling at the interface. They placed titanium dental implants into canine tibiae and allowed them to integrate. Thereafter, a short-term loading protocol was designed to challenge the bone but remain within normal biting ranges. Significant differences in coronal bone loss were observed between loaded and unloaded implants. The authors concluded the response was consistent with the current understanding of microdamage formation and repair around implants. Duyck and coworkers[160] used a dog model to evaluate the crestal bone loss around screw-type dental implants with no load (control), a static load, and dynamic loads. The dynamic-loaded implants were the only group to demonstrate crestal bone loss. Because the only variable in these two animal studies was the intensity or type of occlusal load applied to the implants, these reports imply dynamic occlusal loading may be a factor in crestal bone loss around dental implants. Other animal studies have shown increased marginal bone loss from occlusal overloading.[161,162]

Clinical Studies

The length of a bridge cantilever and occlusal stress on the nearest abutment are directly related.[163] Clinical reports have shown an increase in marginal bone loss around implants closest to a cantilever used to restore the lost dentition.[164–166] This suggests that an increase in marginal bone loss may be related to occlusal stress. The incorporation of cantilevers into implant prostheses may also be associated with a higher incidence of mechanical technical complications such as

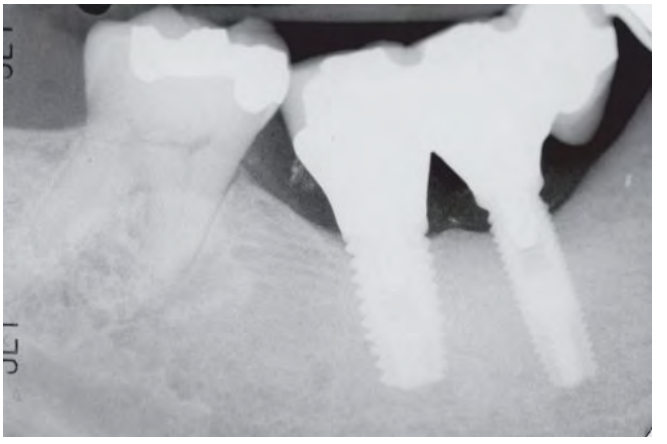


FIGURE 10-33. Cantilevered fixed bridge supported by two implants in the posterior mandible.

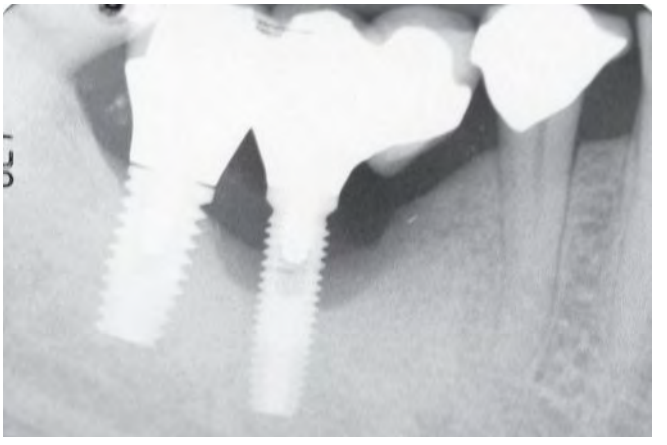


FIGURE 10-34. Marginal bone loss around the anterior standard-diameter implant supporting a cantilevered bridge.



FIGURE 10-35. Fracture of the anterior standard-diameter implant and posterior abutment screw.

screw loosening and bridge fracture[167] (Figures 10-33 to 10-35). Quirynen and colleagues[168] evaluated 69 implant patients with various implant restorations and concluded excessive marginal bone loss after the first year of loading correlated well with the presence of overload due to a lack of anterior contact, the presence of parafunctional activity, and full fixed implant prostheses in both jaws. Although these clinical reports did not provide any statistical analyses to demonstrate a clear link between occlusal stress and bone loss, they offer clinical observations on the relationship between occlusal overload and marginal bone loss.

Biologic Width

It has been suggested that soft tissues around implants form biologic structures similar to teeth that may serve as a protective mechanism against bacterial invasion of the underlying bone.[169] The dentogingival and peri-implant complexes have anatomic similarities and distinct differences. Although an epithelial attachment is present in both the teeth and implants, the mechanism of attachment differs. Whereas the attachment of the junctional epithelium to teeth is mediated by glycoproteins, a pseudo-attachment via a hemidesmosome complex exists around dental implants.[170] The epithelial attachment to teeth is more tenacious and resistant to mechanical and bacteriologic insult. Inferior to the epithelial attachment, connective tissue fibers directly insert into the cementum of the root surface. The connective tissue does not insert into the implant surface but forms a tight circumferential cuff. Animal studies show the connective tissue appears to be similar to scarlike tissue with a horizontal circular collagen fiber network around the implant neck.[171,172] The obvious difference between teeth and osseointegrated implants is in their supporting tissues. The roots of the teeth are surrounded by a periodontal membrane, whereas implants have a direct bone-to-implant interface.

Biologic width has been described as the distance established by the junctional epithelium and connective tissue attachment to the root surface of a tooth. Gargiulo and associates[173] found this average distance is 2.04 mm, of which 1.07 mm is occupied by the connective tissue attachment and another approximate 0.97 mm occupied by the junctional epithelium. However, the dimension of biologic width is not constant and it depends on several variables. The major part of the information about biologic width around implants is derived from animal studies. Although the soft tissue interface differs anatomically, a biologic width also exists around dental implants. However, the dimensions of this soft tissue complex may vary slightly compared with teeth and depending on the type of implant (one-piece vs. two-piece).[174–176] The biologic width physiologically forms around a permucosal implant following nonsubmerged placement or second-stage exposure and is a stable complex.[174]

It has been hypothesized that following nonsubmerged implant placement or abutment connection, a certain width of the peri-implant mucosa is required to enable the development of a biologic width. If this soft tissue dimension is not adequate, bone resorption may occur to ensure the establishment of this soft tissue complex.[177] In a controlled experiment on dogs, implants were placed bilaterally in the posterior mandible.[178] Following adequate healing, the control side implants were exposed and abutments were inserted in a conventional manner. On the test side, the gingiva was dissected and a critical amount of connective tissue on the inside of the flap was excised prior to abutment connection. The histology showed that in the test implants with thinner flaps, bone resorption was consistently observed after soft tissue healing, whereas the total biologic width was not statistically significant between the test and the control implants. They concluded that bone resorption may take place to allow a stable soft tissue attachment to form. The process of biologic width formation around implants was also described by Berglundh and coworkers[178] in another dog study. They observed that the morphogenesis of the mucosal attachment involved loss of marginal bone. However, there have been no human histologic studies on the formation of biologic width or the influence of mucosal thickness on bone resorption.[179] A recent clinical study on initial tissue thickness did find more marginal bone loss around implants placed in thin tissue compared with thicker mucosa.[180]

It has been suggested that the repeated removal of the implant healing abutment during prosthetic treatment can result in disruption of the epithelial seal[181,182] (Figure 10-36). This mechanical injury to the soft tissue may cause an inflammatory response and result in an inferior epithelial migration along the implant. The reestablishment of biologic width more apically may explain the crestal bone loss seen in one animal study.[181] A similar study found a single disconnection of the healing abutment to insertion of the prosthetic abutment did not cause this additional bone loss.[182] A clinical study evaluating the mucosal condition and marginal bone levels after placing healing abutments and prosthetic treatment found no evidence of adverse outcomes from the abutment exchange.[183] However, there was no control group or randomization in this retrospective review. It may be prudent

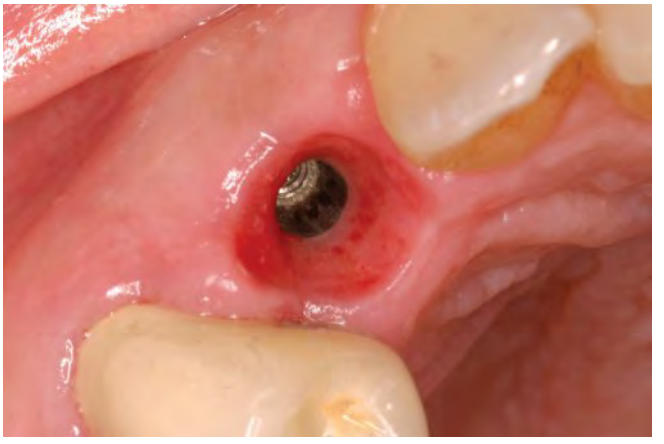


FIGURE 10-36. Disruption of the epithelial attachment from abutment removal. Note slight bleeding from the implant sulcus.



FIGURE 10-37. Gingival recession from repeated removal and replacement of implant components exposes the implant abutment.



FIGURE 10-38. One-stage implant design with the implant-abutment microgap and crown margin above the bone level.

to limit the number of abutment disconnections in thinner mucosa and high esthetic areas (Figure 10-37).

Although the structure and function of the soft tissue complex around dental implants is well documented, the direct influence of biologic width development on marginal bone remodeling is still unclear. Some clinicians have taken the concept of biologic width used in establishing restorative crown margins on teeth and compared this situation to the implant-abutment junction.[184] The influence of an implant-abutment interface on bone remodeling is discussed in the following section.

Implant-Abutment Microgap

From a mechanical engineering perspective, microgaps between components are inevitable when different machined parts are fitted together. The majority of two-piece dental implant systems have a microgap at the implant-abutment interface.[185] This area is prone to microbial leakage with colonization of bacteria from the oral cavity.[186,187] Because the implant-abutment interface is subgingival at the bone level, the bacterial reservoir can result in an inflammatory reaction in the surrounding tissues.[188] It has been hypothesized that this zone of inflammation from the microgap may contribute to marginal bone loss. The additional influence of biologic width development below the microgap on bone loss is only speculative. Animal studies have been conducted that appear to show a correlation between the presence and location of a microgap and marginal bone loss.[189–192] When the microgap is moved, supracrestal bone loss may be reduced[192] (Figures 10-38 and 10-39). Furthermore, when using one-piece implants (no microgap), no significant inflammatory cell infiltrate was seen and minimal early bone resorption was found[189,191] (Figure 10-40). The rough/smooth border on one-piece nonsubmerged implants determines the initial bone-to-implant contact.

Loading forces on implants may also contribute to the bacterial colonization of the microgap. Under function, the components can separate, opening a space between the

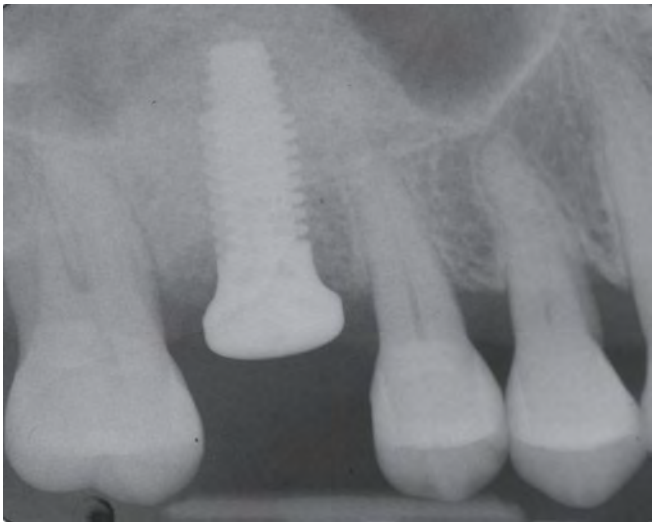


FIGURE 10-39. Periapical radiograph of the one-stage implant placed with the machined collar above the marginal bone.

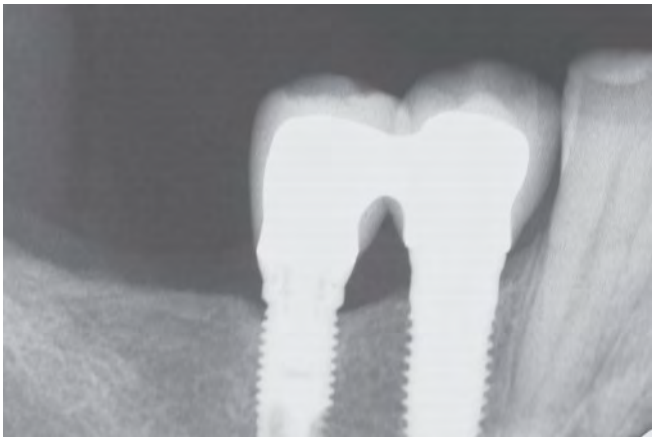


FIGURE 10-40. Marginal bone loss differs between the two implant designs. The posterior implant is a two-piece external hex implant. The anterior implant is a one-piece implant with no microgap.

implant and the abutment and causing percolation of the bacterial reservoir. An in vitro experiment evaluating bacterial leakage along the microgap under loading forces found statistically significant differences between five implant systems with respect to the number of chewing cycles and bacterial colonization.[193] Zipprich and colleagues[194] evaluated microbial colonization and the dynamic behavior of dental implants with different designs of the implant-abutment connection. They found the micromovement between some of the implant-abutment complexes when implants were loaded off axis. The authors speculated that certain implant designs may reduce the pumping effect between the implant and the abutment, thus preventing microbial colonization of this interface. The potential colonization of oral bacteria within the implant-abutment microgap may be impacted by conditions such as the precision fit between implant components, torquing of the abutment screw when components are connected, and implant loading forces under function (Figure 10-41).

Although the microgap theory explaining marginal bone loss seems quite plausible and several animal studies suggest a positive correlation, there is research that finds inconclusive or contrary results. Todescan and associates[195] placed two-piece implants in dogs using a submerged healing technique. Implants were grouped into placement above, level, and below the bone. Although there was a clear tendency of the epithelium and connective tissue to be longer the deeper the implants were placed, additional bone loss below the microgap did not occur. In a clinical study on edentulous patients, two implants were placed using three approaches: two-piece, nonsubmerged implants; two-piece, submerged implants; and one-piece, nonsubmerged implants.[196] After 5 years of functioning, no significant clinical, radiologic, or microbiologic differences were found among the three groups. No association was found between the level of the microgap and the amount of bone loss. In the evaluation of a microgap on marginal bone loss, there are many variables that make definitive conclusions regarding all implant systems difficult (one- vs. two-piece implants, type of implant-abutment connection, implant surface, gingival biotype). Indeed, some implant designs with tight abutment interfaces or connections remote from the bone may not induce significant marginal bone changes[197] (Figure 10-42).



FIGURE 10-41. Plaque is found within the abutment screw threads indicating bacterial leakage between the abutment and the implant.



FIGURE 10-42. An implant with an internal conical seal connection can reduce bacterial microleakage.

Platform Shifting

The concept of platform shifting proposes that moving the implant-abutment interface medial and away from the marginal bone may help protect the bone from the inflammatory cell infiltrate and minimize crestal bone resorption. Although some implant systems originally had this feature incorporated into their design, the concept was first noted serendipitously when abutments narrower in diameter than the implant platform were used.[198] The term "platform switching" was introduced as the abutment size was switched from a matching to a smaller diameter to alter the horizontal position of

the microgap.[199] Implant manufacturers that already had this design incorporated into their system had shown low levels of marginal bone loss[200,201] (Figure 10-43). Several clinical studies have found that marginal bone levels appear to be better maintained with implants incorporating a platform shift compared with standard connections.[202–204] However, other studies have found no significant bone maintenance benefit from platform shifting. An animal study examined conventional and platform-shifted implants during healing at different time periods and histomorphometric analysis found bone remodeling was minimal in both groups with no significant difference.[205]

Crespi and coworkers[206] performed a clinical study on implants with a platform shift versus conventional implants in extraction sockets with immediately restoration. They also found minimal bone loss and no differences in bone level changes between platform-switched and conventional external-hexed implants after 24 months. A clinical study on Astra Tech implants with a platform shift versus Brånemark implants with an external hex found small marginal bone changes with both systems and no significant differences.[207]

Atieh and colleagues[208] performed a systematic review of the literature on the topic. Ten studies with 1239 implants were included. They determined marginal bone loss around platform-switched implants was significantly less than around platform-matched implants. The degree of marginal bone resorption appeared inversely related to the extent of the implant-abutment mismatch. They recommended further long-term, well-conducted, randomized, controlled studies to confirm the validity of this concept. A recent consensus workshop concluded that studies applying the platform-shifting concept did not have adequate follow-up to meet their inclusion criteria.[209]

Although there is evidence that platform shifting may reduce marginal bone loss, other factors may contribute to this finding including the implant-abutment connection, neck design, and the topography of the implant surface.[210] In addition, this technique has some clinical limitations and not all implant systems can utilize this concept. Because the abutment is narrower than the implant body, there must be adequate vertical height through the tissues to develop proper emergence profile of the restoration.

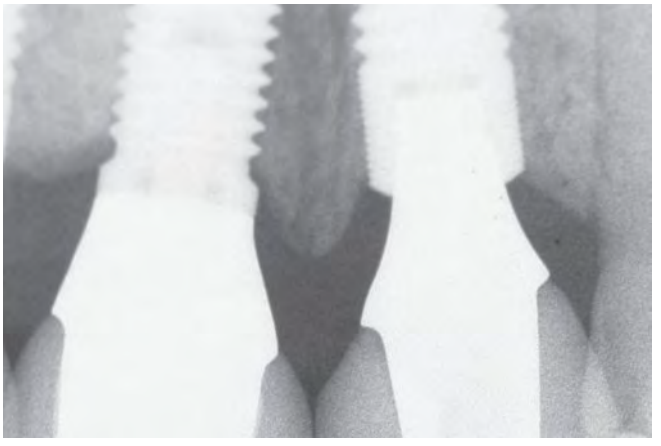


FIGURE 10-43. The left Brånemark implant has marginal bone loss to the first thread area. The right Astra Tech implant with a platform shift has stable marginal bone level with the implant plateau.

Conclusion

The cause of marginal bone loss around dental implants has been a debated topic. Literature from cellular biomechanics, engineering principles, animal studies, and clinical reports all substantiate that occlusal overload may be an etiology of peri-implant bone loss. Although this occlusal overload concept does not negate other factors related to marginal bone loss, it is more clinician-dependent than most other parameters. Therefore, treatment plans that emphasize occlusal stress reduction to the prosthesis are mandated. Thin peri-implant tissues may impact early marginal bone remodeling during the development of the biologic width. A microgap between the implant and the abutment may be a contributing factor to marginal bone loss in some implant designs.

Implant companies and clinicians have expended considerable effort to find ways to control crestal bone loss. Bone loss can have a negative impact on implant esthetics, health, and biomechanics and may have long-term consequences that ultimately compromise the prosthesis and/or implant longevity. A careful documentation of marginal bone level changes should be mandatory for all implant systems before being marketed for clinical use.

Early Loading Failure

On occasion, an implant may fail shortly after it has initially integrated to the bone. The implant can appear to have rigid fixation and all clinical indicators may indicate normal healing. However, once the implant is placed into function, it loses anchorage and becomes mobile. This has been termed *early loading failure* and usually occurs within an 18-month time period.[211] The causes of early loading failure include poor bone density, inadequate bone volume, and/or excessive stress within the bone-implant interface.[212–214] Occlusal overload increases the risk for microfractures at the implant-bone interface, which can result in significant marginal bone loss and even failure[215] (Figure 10-44). There is ample evidence that occlusal factors are related to marginal bone defects and failure of dental implants.[216,217]

It would be difficult to perform human clinical studies on biomechanical implant overload. Animal models have provided information on this topic. Isidor[218] evaluated the influence of occlusal overload and plaque accumulation in monkeys restored with implant prostheses. Following implant integration, bridges were attached to some of the implants and placed into excessive premature occlusal contact. The remaining implants were not cleaned and a cotton ligature was placed around the abutments to promote place accumulation. After 18 months, six out of eight implants with occlusal overload became loose. All implants with plaque accumulation remained integrated but exhibited increased



FIGURE 10-44. Severe marginal bone loss noted 1 year after delivery of the fixed implant prosthesis.

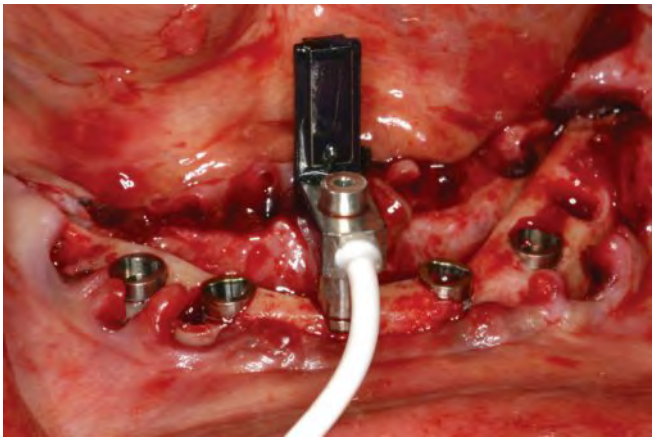


FIGURE 10-45. Resonance frequency analysis can measure the stability of the implant-bone interface.

marginal bone loss. This study concluded occlusal stress was a greater risk factor for early implant failure than bacterial plaque accumulation.

A beagle dog study examined implant overloading in the presence of healthy or inflamed peri-implant tissues.[219] Implant abutments were placed in either supra- or infraocclusal contact. Ligatures were placed around half of the abutments to promote plaque accumulation and the others received routine cleaning. The study found in the presence of uninflamed peri-implant mucosa, overloading of implants increased interfacial bone formation and reduced marginal bone levels. Occlusal overloading aggravated the plaque-induced marginal bone resorption when peri-implant inflammation was present. Another monkey study investigated bone level changes in an inflammation-free state when various levels of traumatic force were exerted.[220] The supraoccluding implant prostheses were defined as excessively high by 100 µm, 180 µm, and 250 µm. The results showed that bone resorption around implants tended to increase with 180 µm or more excessive height of the superstructure, suggesting a threshold of premature contact. It is also implies there is a possibility of bone resorption around the implants caused by excess occlusal trauma, even when there is no inflammation in peri-implant tissue. Other animal studies have been less conclusive.[221–223]

Early loading failure is devastating because it often occurs after delivery of the final prosthesis. This has significant treatment time and cost implications because repair or remake of the prosthesis is necessary. It has been estimated that early failure may affect more than 15% of implant restorations.[224] Early implant failure from biomechanical overload, as high as 40%, has been reported in the softest bone types.[214] No reports in the literature correlate such high incidence extreme of early implant failure rates related to the biologic-related complications. Because routine clinical evaluation and radiographic studies may not expose implants at higher risk for early failure, other diagnostic methods should also be considered. The use of resonance frequency analysis to measure implant stability following implant healing may provide useful information regarding implant survival[225] (Figure 10-45). Although it appears late implant failures are more related to chronic peri-implantitis, the etiology and influencing factors can be difficult to specify[217,226] (Figures 10-46 and 10-47). Excess cement



FIGURE 10-46. Periodontal probing finds peri-implantitis with deep pocketing, heavy bleeding, and purulent exudate.

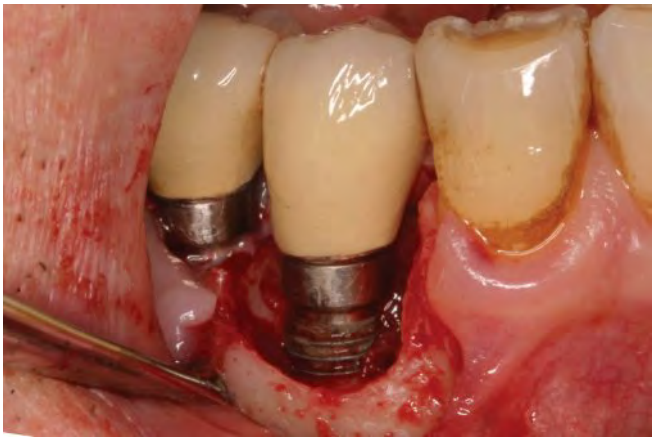


FIGURE 10-47. Surgical exposure finds significant marginal bone loss.

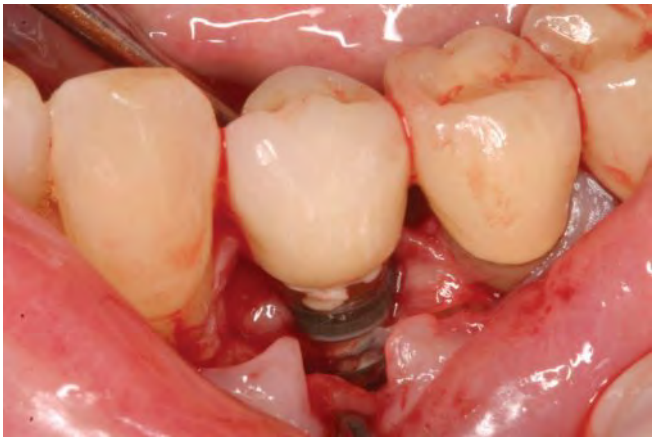


FIGURE 10-48. Excess subgingival cement caused inflammation and significant marginal bone loss.

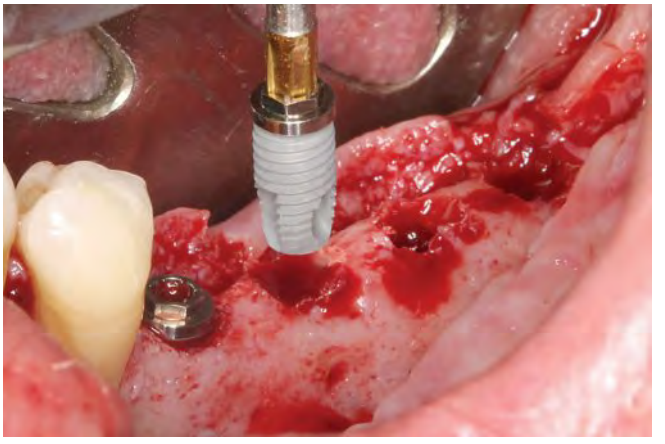


FIGURE 10-49. Placement of short implants in the posterior mandible.

has also been implicated as a cause of significant marginal bone loss and failure (Figure 10-48). There are case reports on occlusal overload following long -term integration that have been successfully managed with correction of the traumatic occlusion.[227,228]

THE SCIENCE OF OSSEOINTEGRATION

Treatment Planning

As previously noted, dental implant complications are often related to mechanical problems.[229] Overload and high stress can cause loss of integration, implant fracture, screw loosening, and fracture or damage to the prosthesis. As such, treatment planning for implant prostheses should focus on reducing any undesirable mechanical disadvantages. Strategies should be incorporated to minimize stress and provide long-term function. Patient risk factors such as parafunctional habits and strong masticatory dynamics need to be identified preoperatively. Mechanical force magnifiers, such as cantilevers and greater crown height, can influence stresses on the system. Several other parameters are in control of the clinician in managing stress and preventing overload.

Implant Number

The definition of *stress* is force divided by the area over which the force is applied. The mechanical distribution of stress occurs primarily where bone is in contact with the implant. One biomechanical approach to decrease stress is to increase the surface area of the implant support system.[230] The most effective method to increase the surface area of the system is to increase the number of implants to support the prosthesis (Figures 10-49 to 10-51). A biomechanical study found force distributed over three abutments results in less localized stress to the crestal bone than two abutments.[231] Other than single tooth replacement, there is no consensus on the number of implants needed to support a particular type of

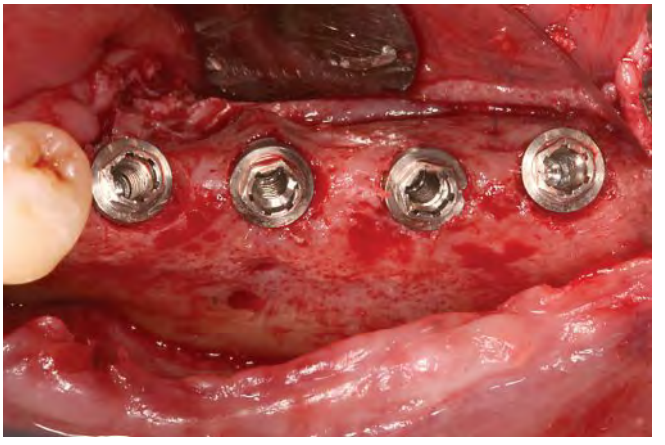


FIGURE 10-50. Four short implants placed in the posterior mandible.

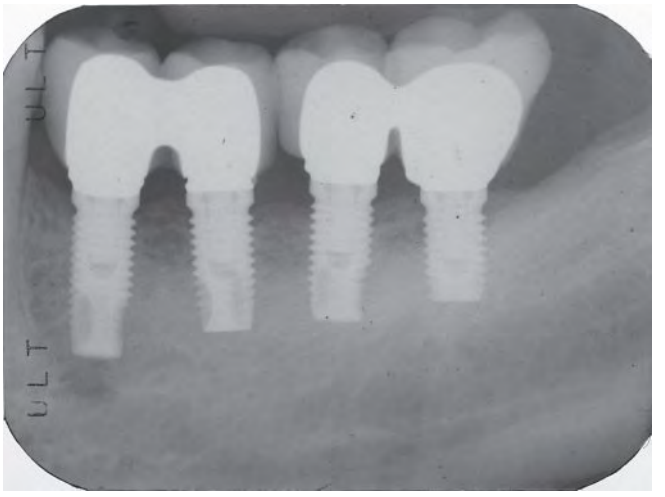


FIGURE 10-51. Periapical radiograph of short implants restored with crowns.

prosthesis. Too many variables must be considered in each patient, making it a case-by-case decision. However, when force factors are higher, the clinician should consider increasing the number of implant abutments. This may be even more critical when immediate implant loading is considered, especially in the maxilla.[232,233] General guidelines for implant numbers are discussed further in the "Treatment Planning" section.

Abutment Positions

Implant positioning is also related to the number of implants. The position of the implants supporting a prosthesis will determine the distribution of the occlusal load. To improve load transfer and decrease stress, the number of pontics should be limited. In addition, planning should consider terminal implant abutments at each end of the prosthesis to avoid cantilevered pontics. There may be anatomic limitations to this rule, such as the position of the maxillary sinus or mandibular canal. Bone augmentation may be needed to develop terminal abutment sites.

A greater span between abutments increases the flexibility or deflection of the prosthesis.[234] The flexibility of a prosthesis or connecting bar varies directly with the cube of its length. Therefore, a bridge with three pontics flexes 27 times as much as a bridge with one pontic. This greater deflection of the prosthesis can cause porcelain or acrylic veneer fracture, metal substructure fracture, cement failure, or screw loosening.[235,236] The replacement of three adjacent missing teeth with a fixed bridge is usually contraindicated.[231,237] Not only is bridge flexure a concern, the load distribution is concentrated on fewer abutments (Figures 10-52 to 10-54). A guideline is a limit of two pontics between implants in a fixed prosthesis.[231]

Treatment plans should address the number of teeth planned for prosthetic replacement. Very often, second molar replacement is considered optional in full-arch prostheses and single tooth crowns. Second premolar occlusion, or a shortened dental arch, is considered the minimum prosthetic replacement for temporomandibular joint and occlusal stabil-



FIGURE 10-53. Removal of the failed pterygoid implant.



FIGURE 10-54. Failed pterygoid implant with the long-span bridge segment.



FIGURE 10-52. Occlusal view of long-span implant bridge (four pontics).



FIGURE 10-55. Preoperative view of patient with maxillary denture and missing mandibular posterior teeth.



FIGURE 10-56. Maxillary immediate load conversion prosthesis opposing mandibular premolar implants (shortened dental arch).

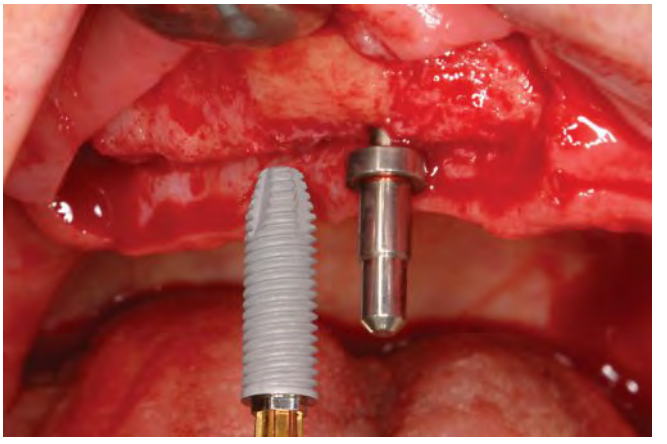


FIGURE 10-58. Longer implants (15.0 mm) used for immediate load maxillary fixed prosthesis seen in Figures 10-57 to 10-59.

ity as well as function[238] (Figures 10-55 and 10-56). This prosthetic plan may be useful when significant bone augmentation would be required for molar implant placement or a reduction in treatment costs is desired.

Implant Size

Many surgeons make the assumption that a longer implant provides more favorable mechanical support for a prosthesis. As previously noted, stresses from occlusal loading are of greatest magnitude in the crestal bone around the neck of the implant. Therefore, increasing implant length does little to decrease the stress that occurs at the implant neck.[239–241] However, increased implant length may provide better initial stability and increased bone-implant surface area. When immediate implant loading is considered, a longer implant is well advised for achieving primary stability (Figures 10-57 and 10-58).

The surface area of an implant is directly related to the width of the implant. Wider-diameter implants have a greater surface area of bone contact than narrow implants of similar design. A 0.25-mm increase in implant diameter increases the overall surface area of the implant by 5% to 10%.[231] Finite element stress analysis studies have shown that the effect of implant diameter on stress distribution in bone was more significant than the effect of the implant's length or its geometr.[241] The use of wider implants may be better to dissipate the acting forces and thus reduce the stress in the bone surrounding the implant. An increase in implant diameter has also been suggested to be more effective than buccal-lingual implant staggering in reducing tensile stresses.[242] The atrophic residual ridge may require width augmentation to avoid placing a smaller-diameter implant in the posterior jaws (Figures 10-59 and 10-60). Because the platform of a wider-diameter implant is larger, less force is transmitted to the abutment screw during loading. This may result in a lower incidence of screw loosening and fracture.[243] The use of a wider-diameter implant is especially important with single tooth molar replacement to avoid complications (Figure 10-61). However, the insertion of a wider-diameter implant into a narrowed residual ridge may be counterproductive. If there is

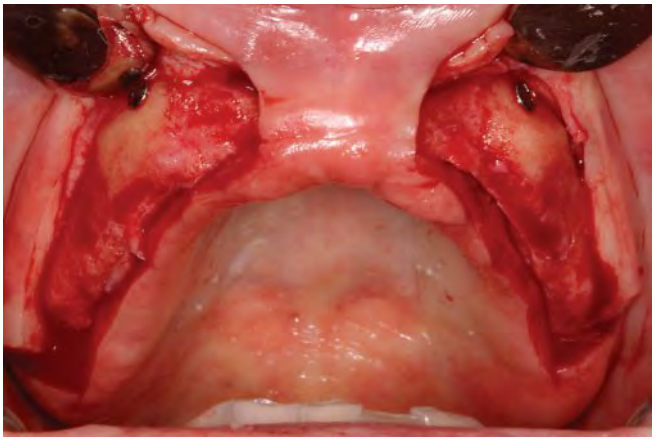


FIGURE 10-57. Reconstructed maxillary ridge from case seen in Figures 10-55 and 10-56.



FIGURE 10-59. Resorbed residual ridge has limited width for standard-diameter implants.

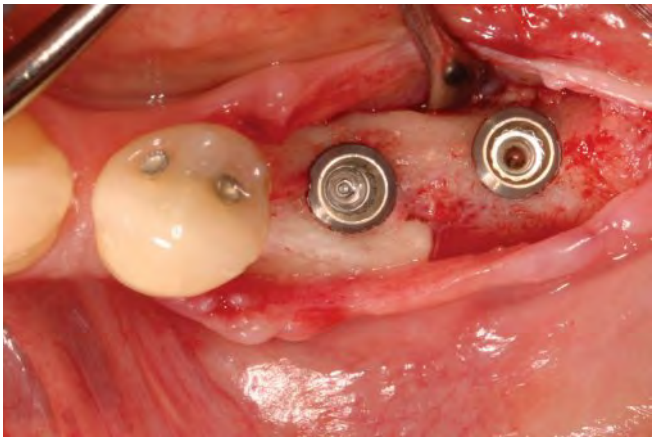


FIGURE 10-60. The atrophic ridge was augmented with a cortical block graft to allow placement of standard-diameter (4.0-mm) implants.

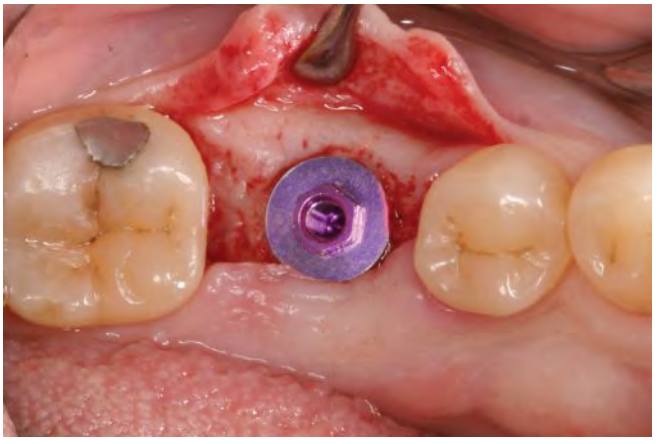


FIGURE 10-61. Wide-diameter implant (6.0-mm) used for mandibular first molar replacement.

inadequate bone at the crest to support the implant, concentrated stresses can cause greater marginal bone loss.[244,245]

Mechanical Risk Factors

An important aspect of diagnosis and treatment planning for implant prostheses is to identify mechanical risk factors that place higher demands on the system.

The manner in which forces are applied to implant restorations dictates the likelihood of system failure. Forces may be described in magnitude, duration, type, and direction.[246] The duration of a force may negatively affect the implant system. Even relatively low-magnitude forces, applied repeatedly over time, may result in fatigue failure of an implant or prosthesis. Stress concentrations may develop if insufficient area is present to adequately dissipate high-magnitude forces. This can lead to structural failure of system components. Forces applied to areas of the prosthesis that are unsupported, such as a cantilever, can cause moment loads resulting in bending or torsional failure.

Cantilevers

Cantilevers are force magnifiers and present a considerable risk factor for implant prostheses. Cantilevers result in rotational or bending moments on the supporting implants. The moment of a force is defined as a vector (M). In the cantilevered prosthesis model, the magnitude of the moment equals the product of the force applied to the cantilever multiplied by the perpendicular distance (or moment arm) from the implant support to the point of force application.[231] For example, if a 100-N force is applied to a cantilevered segment that is 1.5 cm from the center of the implant, the moment of the force (M) is 150 N-cm. The force is multiplied by the length of the cantilever. Torquing imposed on the cantilevered implant prosthesis can result in bone loss, implant failure, implant breakage, screw loosening, and bridge fracture[247–250] (see Figures 10-33 to 10-35). Eliminating cantilevered segments or reducing their length can decrease destructive forces on the implant system.

Recommendations have been made regarding the design of an implant prosthesis when cantilevering is planned. In full-arch cases, the greater the anteroposterior (AP) distance between the supporting implants (AP spread), the smaller the resultant load of the implant system.[251] It has been suggested that the distal cantilever should not extend 2.5 times the AP spread under ideal conditions[252] (Figure 10-62). The length of the cantilever should be reduced when four rather than six implants are used to support a full-arch prosthesis and in the maxilla where softer bone is often found.[253,254] When there is adequate implant support, short-span prostheses with limited cantilevers have performed well.[250] In these unilateral partially edentulous cases, Takayama[255] has suggested that the cantilever should not extend beyond the distance between the implants. Because a common distance separating implants is 7.0 to 8.0 mm, the size of the cantilever should not be greater than a premolar[231] (Figure 10-63). In addition, a mesial cantilever would have to withstand less occlusal force than a posterior cantilever. When cantilevers are planned in the final prosthesis, the occlusion on the cantilevered pontics should



FIGURE 10-62. The anteroposterior spread is used as a guide for the cantilevered distance in a fixed prosthesis.

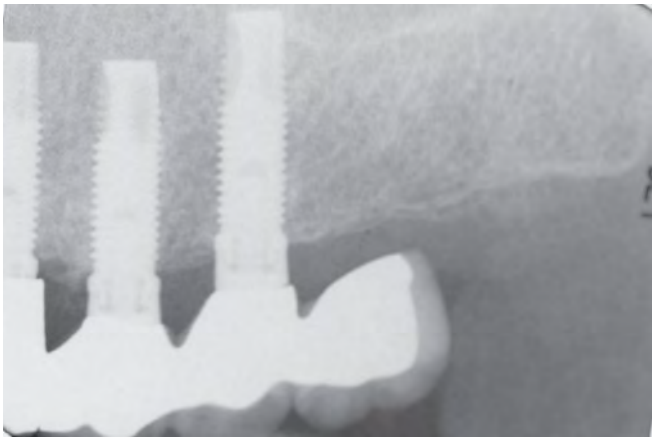


FIGURE 10-63. A premolar size pontic as a posterior cantilever from three maxillary implants.



FIGURE 10-65. Patient with parafunctional habits and worn dentition.

be slightly reduced, with no contacts in mandibular excursions.[231] When higher occlusal loads are expected, it may be prudent to consider using a natural tooth abutment for a bridge and thereby eliminating the unsupported pontic. Although there have been theoretical concerns with connecting teeth and implants in a bridge, this design has proved to be a viable prosthetic alternative.[231,256] Nonfunctional esthetic cantilevers have also been proposed when space is limited for the placement of two adjacent implants in the esthetic zone[257] (Figure 10-64).

Parafunction

Parafunctional habits can be the most destructive forces clinicians and patients have to contend with in the compromise of prosthesis longevity. Parafunctional activities, such as bruxism and clenching, are characterized by habitual repeated and/or sustained occlusal contacts. Bruxism changes normal masticatory forces by magnitude (higher bite force), duration (hr vs. min), direction (lateral > vertical), and type (shear > compression). Parafunction has been implicated in higher implant failure rates.[258,259] In a study of 589 consecutive implants, Naert and associates[260] suggested overload from parafunctional habits may be the most probable cause of implant failure and marginal bone loss after loading. They found patients with implant-fixed bridges in both jaws showed significantly more marginal bone loss than did those with only one fixed implant prosthesis opposed by either natural dentition or a complete denture. In patients with severe bruxism, the long-term function of the prosthesis will be diminished. Patients must understand that they will likely have more technical complications with repeated visits for repair and servicing of their prosthesis[236,261] (Figures 10-65 to 10-67). The treatment plan is altered to minimize the negative impact of the pathologic forces on the restoration, abutments, implants, and supporting bone. Full-mouth rehabilitation may be needed to reestablish proper anterior guidance and dental relationships. Greater numbers of implants may be planned as well as avoiding narrower-diameter implants, especially in posterior regions. Occlusal materials can be selected to avoid excessive wear or fracture. For example, if both arches are restored, porcelain against porcelain occlusion is discouraged.[262] It may be prudent to consider acrylic or composite prosthetic teeth in one arch to minimize



FIGURE 10-64. A lateral incisor pontic is cantilevered from a cuspid implant where there is inadequate space for two implants.



FIGURE 10-66. The patient was rehabilitated with maxillary and mandibular fixed hybrid (acrylic to metal) prostheses.



FIGURE 10-67. The acrylic veneer material has fractured from the cantilevered segment.

the risk of fracture and impact forces. The benefit of a removable implant prosthesis is that it may be taken out at night to avoid nocturnal forces. The routine use of an occlusal guard with fixed prostheses is highly recommended[231,262] (Figure 10-68).

Masticatory Dynamics

Clinicians should consider the influence of greater loads on the implant system and plan accordingly. The muscles of mastication generate the occlusal forces placed on the implant system. Several factors influence masticatory dynamics including patient size, gender, age, and skeletal relationships. Larger athletic patients may generate higher forces.[263] Age and gender are associated with structural and functional alterations in the muscles of the stomatognathic system. Older patients typically record lower biting forces than younger patients, and the bite force of men is on average 30% higher than women.[264,265] The brachycephalic patient presents with a short head, skeletal deep bite, and less convex facial profile. They may generate three times the bite force of normocephalic patients.[266] Dolichocephalic patients tend to have weaker bite force. The maximum bite force is greater in the molar regions and decreases anteriorly.[267] A biomechanical approach to planning would be to consider wider-diameter implants in the posterior areas of the jaws. It is well known that conventional denture wearers have lower maximum bite forces than dentate subjects.[268] In addition, muscle atrophy occurs in patients with longer periods of edentulism. Therefore, treatment plans for tooth replacement against an opposing complete denture may be less mechanically demanding than natural dentition.



FIGURE 10-68. A bite splint is used to protect the implant connecting bar and opposing dentition from nocturnal parafunctional activity.

Implant prostheses have been shown to improve masticatory function and biting force.[269] However, the tactile sensibility of dental implants is reduced compared with natural teeth.[270] Concern has been raised that a lack of proprioception may generate higher loads, especially with parafunction.[231] However, there is a recovery of osseoperception compared with complete denture wearers.[271]

Treatment Planning

It is difficult to provide strict guidelines for the number and distribution of implants to support a particular prosthesis. However, generalizations may be made based on biomechanical support, long-term studies, and clinical experience. When the minimum number of implants is used, the prosthesis is placed at risk if a complication occurs. The philosophy of "protect the prosthesis" is a prudent guideline in treatment planning and designing of the implant support system.[231]

Edentulous Maxilla

Although the use of two implants for overdenture support is a well-supported option in the mandible, this approach is poorly documented in the maxilla and infrequently recommended.[272] Many patients with minimal atrophy of the upper jaw are able to tolerate a maxillary complete denture. It is not until the maxilla becomes more resorbed that the patient experiences problems with stability and retention of the prosthesis. With increased atrophy, less bone is available for implant placement and less residual ridge for prosthetic support. As such, the use of only two implants may not provide the desired improvement in denture function. In addition, higher loads are placed on the two independent implants in softer quality bone.[273] Studies have found higher implant failure rates and greater levels of marginal bone loss around independent maxillary implants supporting overdenture prostheses[274–277] (Figures 10-69 and 10-70). Although the cost is lower, compared with that of using a connecting bar, the mechanical advantage of implant splinting is preferred.[278]

Clinical studies have found that maxillary overdenture failure rates are often significantly higher than any other type of implant prosthetic option.[277,279] As previously noted, this may due to less bone volume and poorer bone quality as well implant distribution, prosthetic design, and the opposing occlusion. The prescription of an overdenture is sometimes an unplanned alternative when complications prevent the consideration of a fixed prosthesis. As such, the implant numbers and distribution may not be optimal for support. The minimum number of implants for support of a maxillary



FIGURE 10-69. Atrophic maxilla with two independent implants and attachments.

overdenture is four. These implants may often be placed anterior to the maxillary sinuses (Figures 10-71 and 10-72). The four implants should be splinted with an implant connecting bar[231,276] (Figure 10-73). No significant distal cantilevering of the bar should be designed in these cases.[280] Implant placement in the midline area is typically avoided to allow use of anterior prosthetic attachments and prevent bulky overdenture contours in the midpalatal region. The palate and posterior areas of the maxillary ridge are used for additional prosthetic support (Figure 10-74). A maxillary overdenture with six implants may be removable yet completely implant-supported. Sinus bone grafting is often required to place posterior implants. Reduction of palatal coverage is often a desired benefit of the additional implant support (Figure 10-75). The six implants are cross-arch–splinted with a connecting bar. As previously described, implants in the midline area are avoided. Although two separate connecting bars have been proposed, this design is prosthetically unfavorable because the overdenture has a tendency to rock from the lack of anterior support (Figure 10-76).



FIGURE 10-70. Failure of the maxillary right implant 6 months after prosthesis delivery.

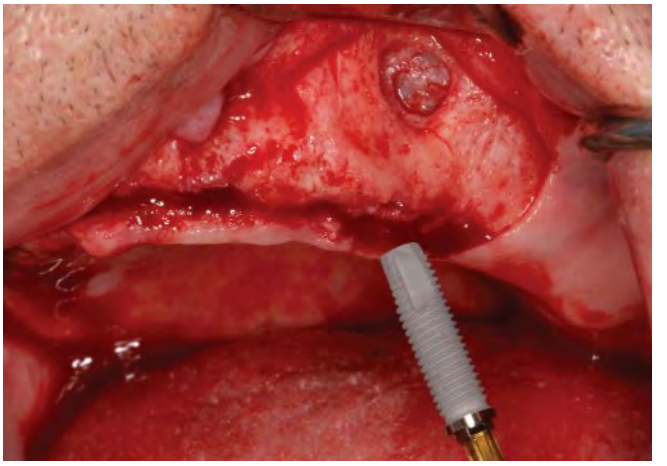


FIGURE 10-71. The posterior implants are tilted away from the maxillary sinus.

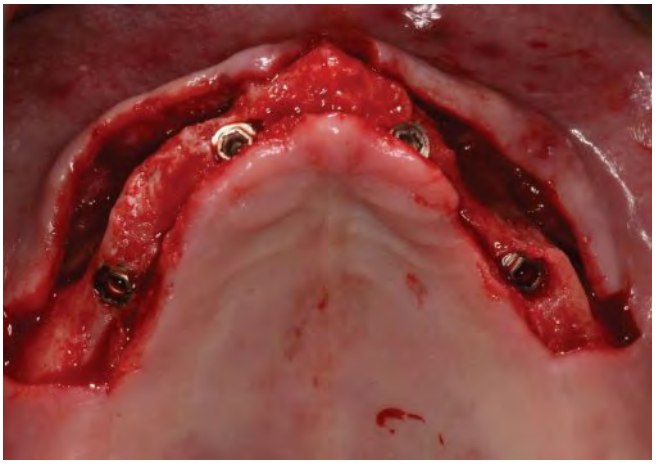


FIGURE 10-72. The four maxillary implants planned for overdenture support are placed anterior to the sinuses.



FIGURE 10-73. The four implants are splinted with a connecting bar. The midline area is avoided to allow placement of an attachment.



FIGURE 10-74. The maxillary overdenture is retained by the attachments and supported by the bar as well as the palate and posterior ridges.

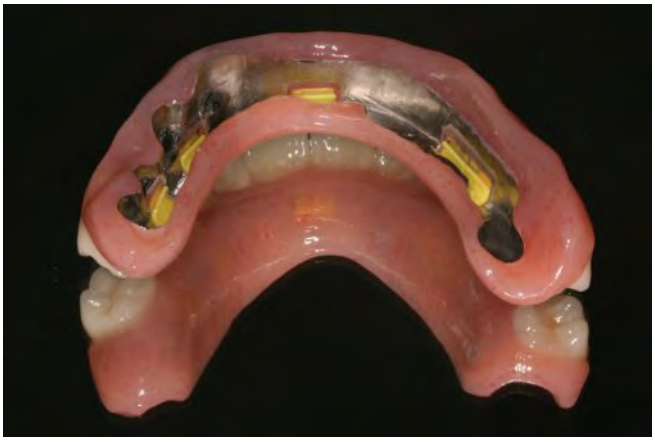


FIGURE 10-75. A maxillary overdenture supported by six implants is completely implant-supported with no palatal coverage and retained with attachments.

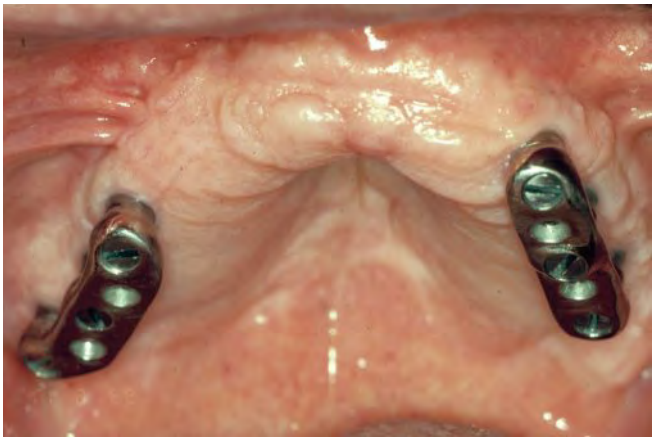


FIGURE 10-76. Two separate implant bars are discouraged because the maxillary overdenture has a tendency to tilt with anterior biting forces.



FIGURE 10-77. Six maxillary implants planned for support of a fixed prosthesis.

The minimum number of implants proposed for support of a maxillary fixed prosthesis is four. Although the early Swedish literature documented maxillary cases with as few as four implants, they more often recommended six.[281] The implants were placed anterior to the maxillary sinuses. A higher failure rate was noted when only four implants were utilized.[282] More recently, the "All on Four" concept was introduced for treatment of the edentulous maxilla using four implants for a fixed prosthesis.[283] The posterior implants are placed in a tilted manner to follow the anterior sinus border. This extends the posterior position of the distal implants. Although this approach has shown favorable early results, the obvious significant disadvantage is that the loss of one implant results in complete prosthetic failure. A more prudent approach is to consider six implants as the minimum number for a maxillary fixed prosthesis (Figures 10-77 to 10-79). Greater numbers of implants (7–12) may be recommended when mechanical risk factors are higher, bone volume is compromised, and/or bone density is poor.[231] Bone



FIGURE 10-78. The maxillary fixed hybrid (acrylic to metal) prosthesis supported by six implants.



FIGURE 10-79. The maxillary fixed prosthesis supported by six implants opposed by a shortened dental arch.



FIGURE 10-81. Guided surgery template used to prepare the implant sites. Eight implants were planned because the bone quality was less dense.

augmentation is often required in the atrophic maxilla to provide the preferred numbers and distribution of implants (Figures 10-80 to 10-83). Greater numbers of implants (>6) are also advised when immediate loading with a fixed prosthesis is planned[232,284] (Figures 10-84 and 10-85).

Edentulous Mandible

The limitations and difficulties the lower denture patient experiences are well appreciated. The standard of care for treatment of the edentulous mandible demands the use of dental implants to minimize atrophy and improve function.[285] The minimum number of implants utilized for mandibular overdenture support is two (Figures 10-86 and 10-87). Two independent implants in the anterior mandible for overdenture retention is a well-documented protocol for prosthetic treatment. However, this plan is primarily used when cost is a primary factor. The posterior residual ridges can still resorb



FIGURE 10-82. The eight implants integrated to support the fixed prosthesis.

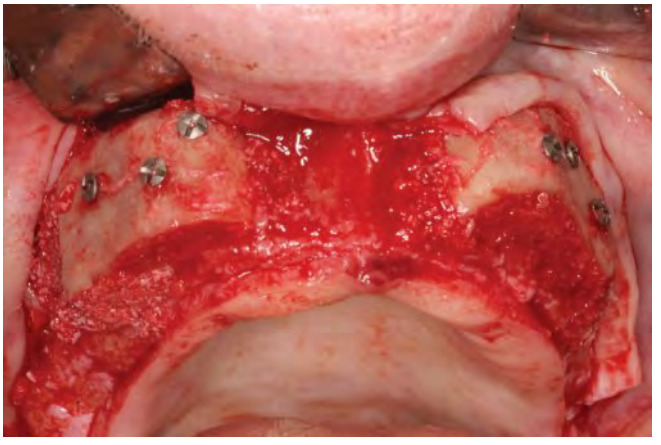


FIGURE 10-80. Atrophic maxilla reconstructed with iliac bone graft.



FIGURE 10-83. The maxillary fixed prosthesis supported by eight implants.

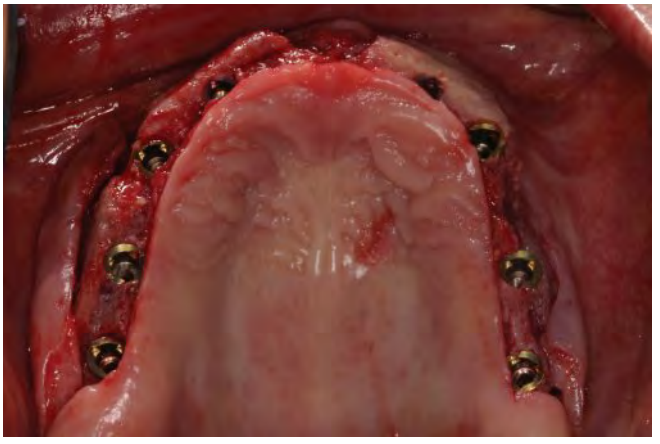


FIGURE 10-84. Eight maxillary implants placed for support of an immediate load prosthesis.



FIGURE 10-87. The mandibular overdenture has two attachments used to retain the prosthesis.



FIGURE 10-85. The maxillary immediate load implant acrylic bridge.

under the distal extensions of the soft tissue borne prosthesis. Adding additional independent implants can provide greater overdenture retention if needed.

Although a bar-overdenture may be constructed on only two implants, a more common approach is to use four implants for this type of prosthesis.[231] Four implants provide more posterior support and greater stability. The four implants are distributed in the anterior mandible between the mental foramen areas (Figure 10-88). It is important the surgeon understands the added space requirements for this type of prosthesis and that significant osteoplasty may be needed. An implant connecting bar splints the implants and incorporates attachments for overdenture retention (Figure 10-89). The midline area is typically avoided to allow placement of an attachment within the bar. A short cantilevered extension of the bar may be considered bilaterally to provide added posterior support.



FIGURE 10-86. Two mandibular implants with locator abutments used to retain an overdenture.



FIGURE 10-88. Four implants in the anterior mandible used to support a connecting bar with attachments.



FIGURE 10-89. The internal aspect of the mandibular overdenture with retentive attachments.

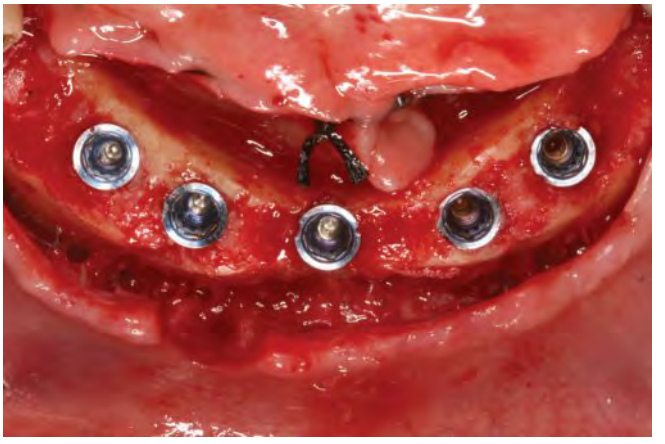


FIGURE 10-91. A poor anteroposterior spread of the implants necessitates a compromised length of the posterior cantilever.

In the classic Brånemark approach to the edentulous mandible, four to six implants were inserted for a fixed prosthesis.[281] The implants were placed in the anterior mandible between the mental foramen areas. The fixed prosthesis had cantilevered extensions to replace the posterior dentition. This treatment protocol provided very favorable long-term results.[282,286] A more systematic treatment protocol for implant placement in the anterior mandible was introduced by Misch.[287] The available bone in the anterior mandible is divided into five equal columns of bone labeled A, B, C, D, and E (starting on the patient's right side). The A and E implants are placed just anterior to the mental foramen areas and the C implant is inserted into the midline. The B and D implants split the difference between the other implants (Figure 10-90). This provides an even spacing with adequate distance between the implants for favorable load distribution, marginal bone maintenance, prosthetic procedures, and oral hygiene. As previously discussed, the length of the posterior cantilevered segments are determined by the AP spread (Figure 10-91). The greater the AP spread, the further the distal cantilever may be extended to replace the missing posterior teeth. However, if stress factors are high (i.e., parafunction, strong musculature, opposing natural dentition), the cantilevers should be reduced (Figure 10-92). Another consideration is thickness of the cantilevered segment because reduced interarch space can compromise strength of the prosthesis. Fatigue failure of the cantilever usually occurs distal to the last supporting implant. Although early studies on immediate loading of mandibular implants use multiple implants, favorable results with fewer implants (5–6) in the anterior mandible have been reported[288,289] (Figures 10-93 to 10-95).

Another option that may eliminate or reduce the distal cantilever is to place posterior mandible implants. There needs to be adequate available bone volume superior to the mandibular canal. This clinical scenario is more common with immediate extractions or recent edentulism with minimal atrophy (Figures 10-96 to 10-98). There is a theoretical concern with rigid splinting of anterior and posterior mandible implants based on mandibular flexure under function.[231] Distal to the mental foramen areas, the mandible can exhibit

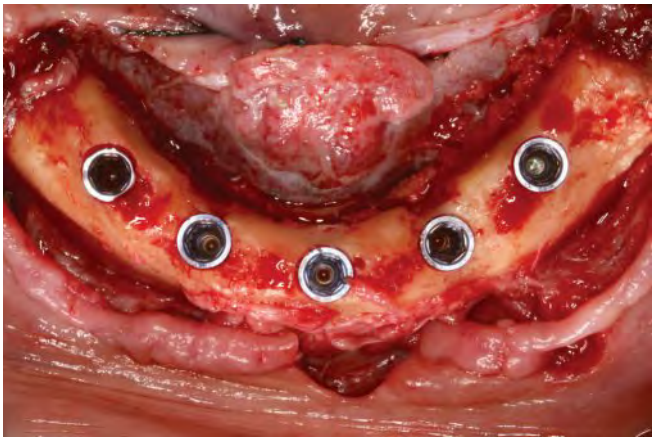


FIGURE 10-90. Five implants are evenly spaced between the mental foramen areas in the A, B, C, D, and E positions.



FIGURE 10-92. The cantilevered segments are too long for this implant distribution. The posterior abutment screws fractured under the increased load.



FIGURE 10-93. Preoperative view of hopeless mandibular dentition.

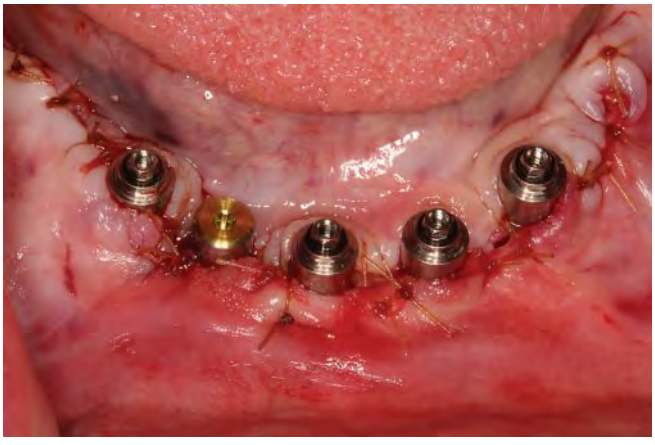


FIGURE 10-94. Mandibular implant abutments placed for an immediate prosthesis.



FIGURE 10-95. Delivery of a mandibular immediate conversion prosthesis.

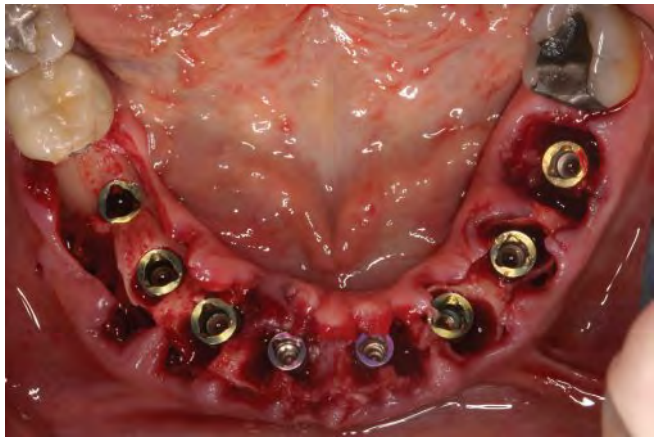


FIGURE 10-96. Anterior and posterior implants placed following extractions for support of an immediate load prosthesis.



FIGURE 10-97. Mandibular immediate load acrylic bridge.



FIGURE 10-98. The final porcelain to metal mandibular implant bridge.

considerable movement toward the midline with jaw opening.[290] In addition, torsion of the mandibular body during mastication has also been documented.[291] The further posterior to the mental foramen areas, the greater these dynamic movements are of concern. If molar implants are splinted to anterior implants, the mandibular movement may place greater stresses on the implants, abutment screws, and prosthesis.[231] It may be prudent to place only unilateral posterior implants (eliminating the cantilever on one side) or divide the prosthesis into anterior and posterior segments to diminish the influence of mandibular mechanics.

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CHAPTER 11

Comprehensive Implant Site Preparation

Ole T. Jensen, DDS, MS

Edentulous or partially edentulous reconstruction of the mandible must be based on the dental restorative need.[1] If more teeth, and more alveolar bone are missing, then the range of treatment options is more extensive. In general, specific sites require significant treatment variation, which can be categorized as follows:

- Single missing teeth—incisors, cuspid-bicuspid, molar.
- Multiple missing teeth—two to six contiguous missing teeth, free end saddles, anterior saddle.
- Edentulous—immediate extraction and implant placement, modest resorption, marked resorption, extreme resorption.

For each restorative location, there is a specific hard tissue finding, often not ideal, that must be addressed. But the desired implant treatment plan must first be decided on before hard tissue modification for functional osseointegration is deemed necessary.[2] The following treatment schemes are based on common treatment planning with an underlying philosophy efficient for the number of implants placed relative to the desired dental restoration.

SINGLE MISSING TEETH

In the mandible, there are three basic single-tooth sites that entail completely different approaches to implant placement. The clinician should consider the use of minimal flaps, minimal bone grafting, and sometimes, connective tissue grafting as indicated. The anterior incisor site, the cuspid-bicuspid site, and the molar site present with completely different problems related to implant fixation and bone augmentation requirements.

Mandibular Incisor Sites

There is controversy in how to treat a single missing anterior incisor in the mandible.[3] Usually, there is minimal space for implant placement as well as reduced bone mass. If the surgeon accepts the dictum that at least 1 mm of bone needs to be present circumferentially around the implant, a site must be 5 mm in diameter to house a 3.0-mm implant and 5.5 mm for the most commonly used 3.5-mm diameter implant.[4] The use of the 2.0- to 2.5-mm one-piece implants is also a consideration but has not been fully vetted in the literature.[5] In situations of oligodontia, the lower anterior incisors may not be present, and in this setting, the caudal alveolar process never fully forms.[6] This requires guided bone regeneration sometimes using both lingual and facial flaps (Figure 11-1). Figure 11-2 demonstrates a missing lower central incisor tooth treated with a single implant, which encroaches upon the periodontal ligament space. When bone and space for implants is minimal, consideration should be given to conventional dentistry or a more aggressive approach of removal of adjacent incisors to make room for implants. Two missing teeth have also been treated with a single implant. Treatment plans that sometimes make more sense are three to four tooth bridges, often best accomplished at the time of dental extraction using guided bone regeneration (GBR) principles in a submerged treatment scheme. Sometimes perfectly good teeth are removed in order for the restoration to become feasible using a dental implant approach. The use of immediate loading in these settings should be cautioned against because early osseointegration may fail with minimal loading in such compromised sites. When one-piece implants are employed, care should be taken that deflective occlusal contacts are not present in anterior profusive movements.

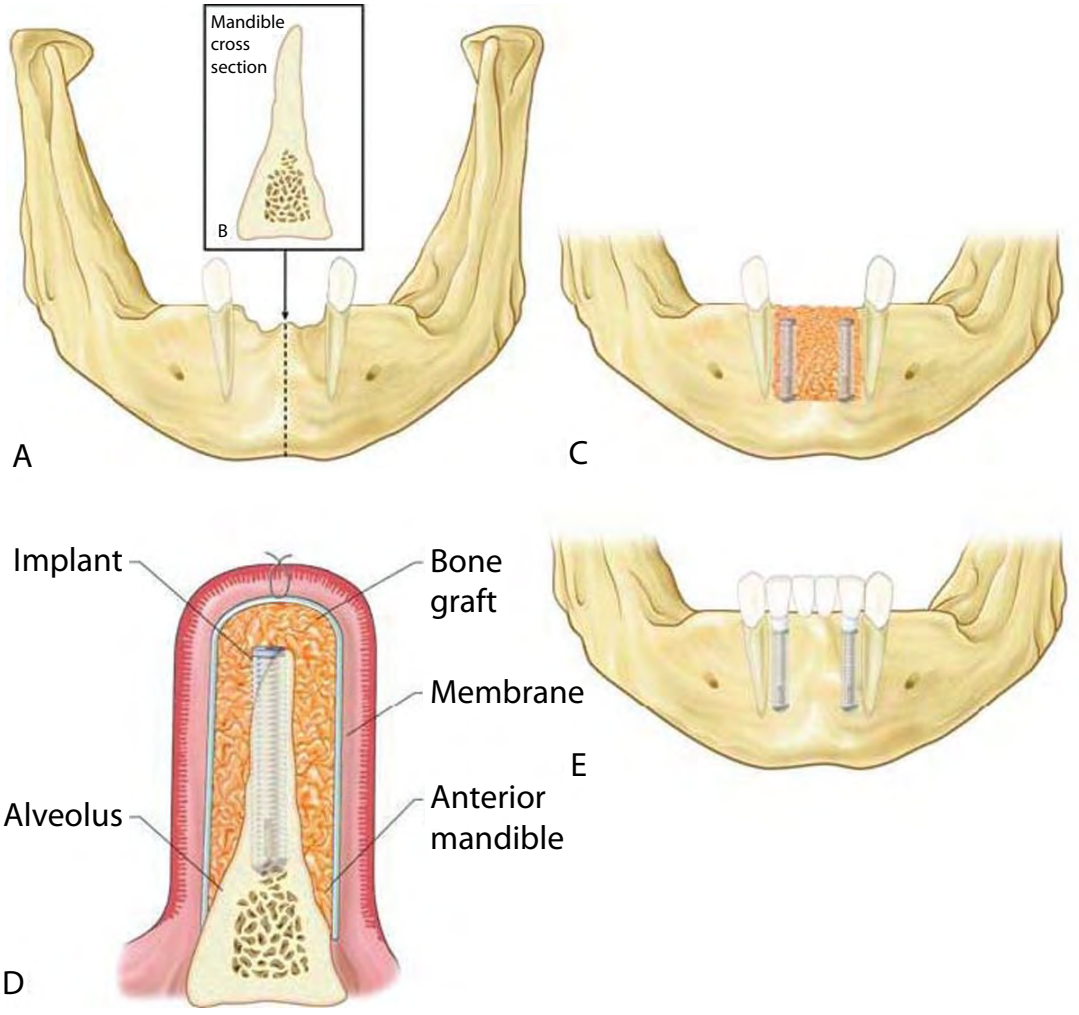


FIGURE 11-1. **A** and **B,** Anterior mandibular alveolar atrophy can present as a very thin alveolus, too deficient for implant treatment without bone grafting. **C,** This area can sometimes be treated with narrow-diameter implant placement and guided bone regeneration (GBR) if the implant can fixate into basal bone. **D,** This can require bone grafting both lingual and facial with barrier membrane coverage. **E,** Final implant healing follows usually with the requirement of a splinted restoration.

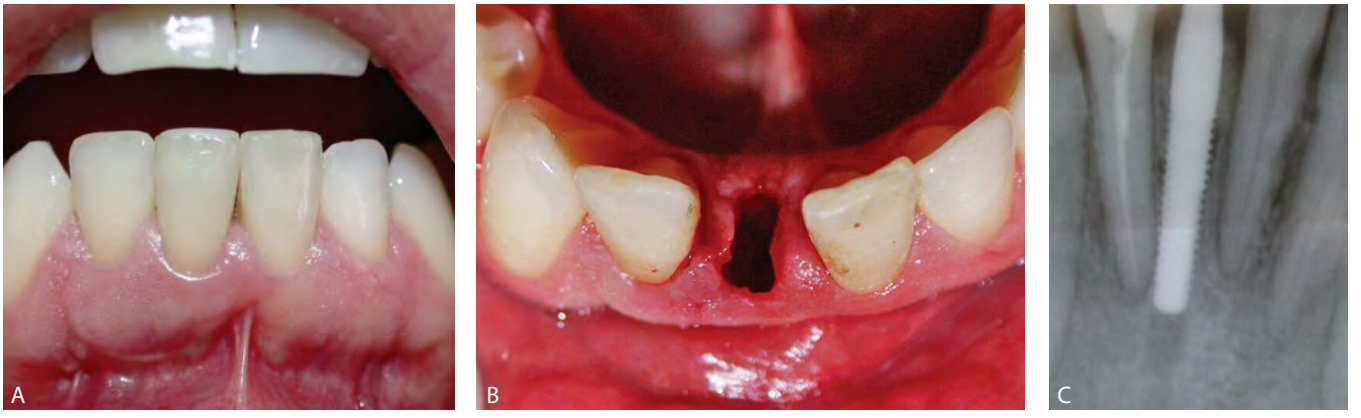


FIGURE 11-2. **A,** One of the dilemmas for implant therapy is what to do with a single missing lower incisor. These sites often have minimal facial bone, mucogingival deficiency, and adjacent root proximity. **B,** Atraumatic extraction with intrasocket grafting, keeping the implant away from the facial plate, is advisable. **C,** Despite near-ideal placement, this 3-mm one-piece implant encroaches upon the periodontal ligament space.

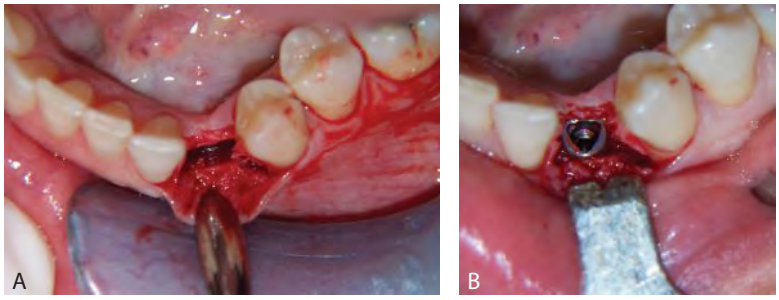


FIGURE 11-3. **A,** The canine-bicuspid zone in the mandible is often moderately horizontally deficient especially in healed sites. **B,** Alveolar split grafting with immediate implant placement using a bone flap can establish 2 mm of facial bone in front of a newly placed implant.

A missing lower central incisor case entails placement of a single implant that has at least 1 mm of bone circumferential around the implant. Therefore, if a 3-mm implant is used, a 5-mm-wide site must be present. Placement of implants into periodontal ligament space may compromise adjacent teeth and often prevents the formation of adequate osseointegration.[7] For this reason, some practitioners have even suggested the use of mini (2- to 2.5-mm-diameter) implants.[5]

In general, the best time to place implants in the anterior incisor region is at the time of extraction, which is often a setting of full facial dehiscence and best treated with GBR and a staged technique. Connective tissue grafts can be placed as needed as part of the implant restoration.[8]

Canine-Bicuspid

The canine-bicuspid zone can often be treated with immediate implant placement at the time of dental extraction. When the socket defect is large, greater than 3 mm, intrasocket grafting is advisable, though not entirely necessary for osseointegration. Intrasocket grafting will help maintain alveolar width, which otherwise will resorb centripetally, sometimes affecting osseous coverage on the facial surface of the implant.[9] This is especially true in the bicuspid zone, which can often be horizontally deficient.

For healed extraction sites, GBR can be used when there is a small dehiscence present at the time of implant placement. When the alveolus is narrow, alveolar split grafting can be done before or sometimes at the time of implant placement. Though split grafting is usually done with an open flap approach, the use of osteoperiosteal flaps such as the book flap (Figure 11-3) provides another option for alveolar widening when there is sufficient width to do a split, usually 4 mm minimum, such that 2 mm of facial plate results from the splitting process.[10] When the alveolus is slanted, pyramidal in shape, it can be split using an open flap technique, left to heal for 4 weeks and then split again using a closed wound osteoperiosteal flap approach with simultaneous implant insertion.[11] This technique, however, is usually reserved for segmental deficiencies and is not always possible or advisable in single-tooth sites (Figure 11-4).[11]

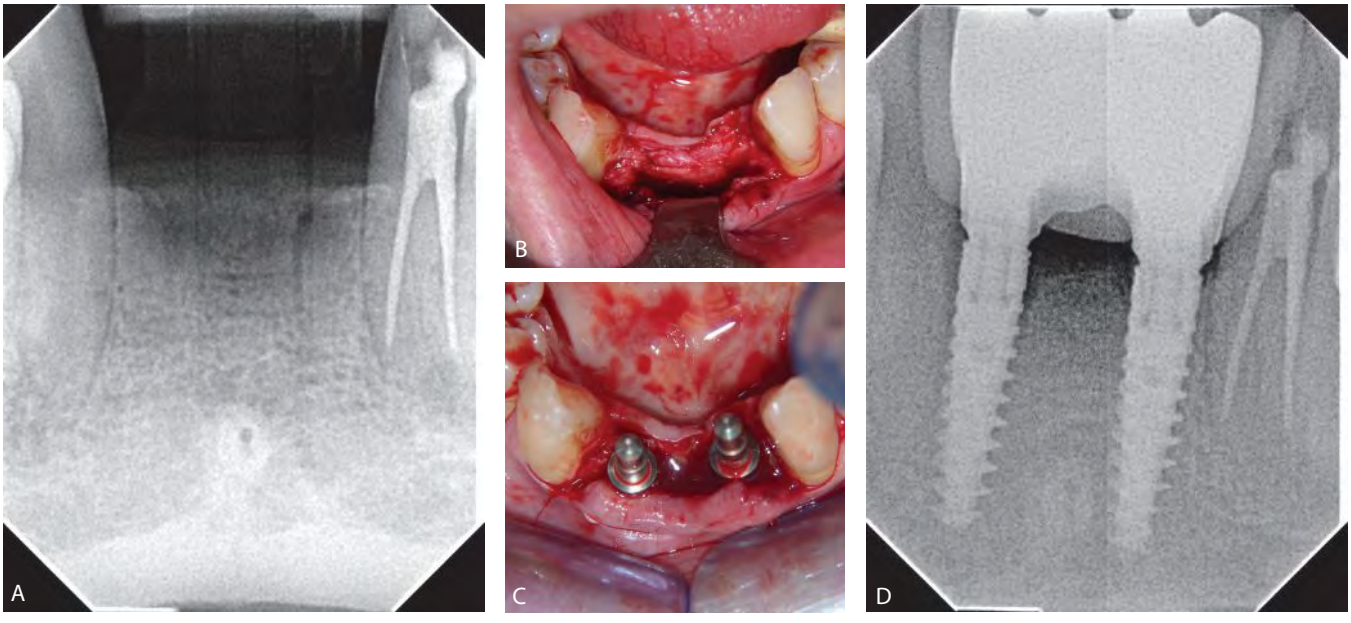


FIGURE 11-4. **A,** When all of the anterior mandible incisors are missing, a common finding is a moderately narrow alveolus not amenable to implants without augmentation grafting. **B,** If the site is 4 mm in width, it can easily be split using a osteoperiosteal flap approach. **C,** Long implants are placed simultaneously if they are able to engage fixation deep to the split site, which usually out-fractures at about a 10-mm depth. **D,** One and one half years later, a two-tooth restoration on two implants appears well integrated with good vertical maintenance of alveolar bone.

Single-Molar Sites

The molar site in the mandible is generally not a good site for immediate placement of implants after tooth extraction unless large-diameter implants are placed (Figure 11-5). Great care must be taken in avoiding the inferior alveolar nerve. A good rule of thumb is to stay 2, or even 3, mm superior to the nerve.[12] This then becomes the foundation point for implant placement and all associated grafting schemes in the posterior mandible.

One of the key issues of molar extraction sites is the presence of the buccal plate. When there is a substantial buccal plate of bone, the large-diameter implant can be placed, which should not remove or disturb the buccal plate as it is placed. This applies to the lingual plate as well. Large-diameter implants can then be placed with little or no bone grafting using relatively short lengths such as 7.5 to 9.0 mm.[13] When 4- to 6-mm-diameter implants are used in molar extraction sites, bone grafting and resorbable membranes are recommended in a staged protocol.[14]

In healed molar sites for single teeth, often lateral or vertical osseous deficiency is present. Because most horizontal deficiency still allows plenty of osseous volume for placement of an implant, usually on the lingual side of the alveolar crest, GBR can be used to improve the osseous contour as needed to develop alveolar form and not necessarily to gain osseointegration. When the alveolus is extremely thin or vertically deficient and more than 3 to 4 mm, vertical augmentation is recommended, block grafting, bone morphogenetic protein-2 (BMP-2) mesh grafting or perhaps a small sandwich osteotomy graft (Figure 11-6) can be done. Of these approaches, the sandwich graft is the simplest, easily gaining 5 mm of vertical height with little morbidity.[15]

SEGMENTAL MANDIBULAR DEFECTS

In general, mandibular defects in partially edentulous patients present as either anterior or posterior saddle defects. In partially edentulous states, the anterior teeth may be lost,

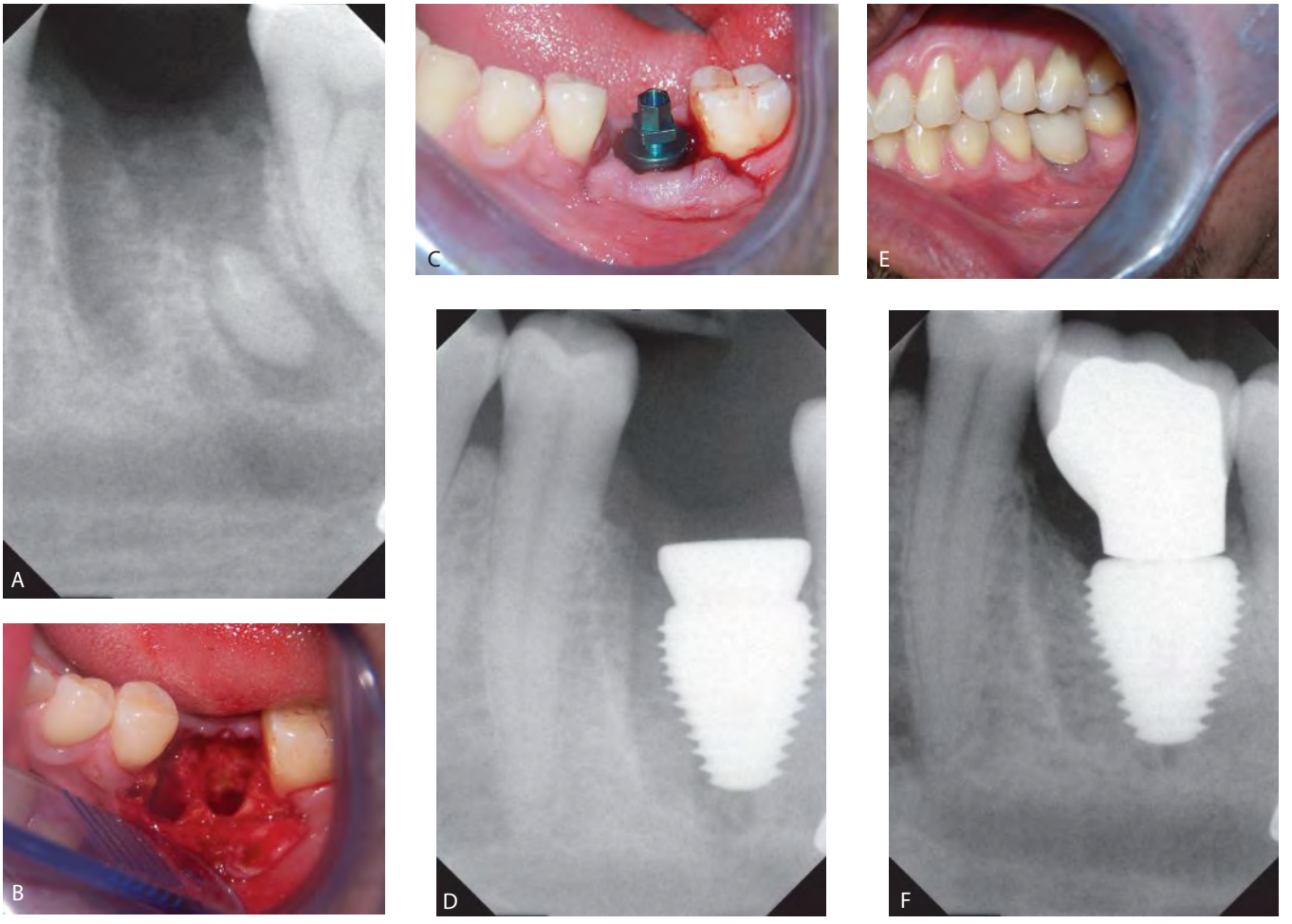


FIGURE 11-5. **A,** Single-molar sites in the mandible can be treated with immediate implants *without* bone grafting when a wide-diameter (7- to 9-mm) implant is used that can gain primary stability. **B,** A typical molar extraction site may still have buccal and lingual plate integrity. **C,** Wide-body implant insertion should be in the center of the extraction defect. **D,** Four months later, the implant, placed without bone grafting, appears to have osseous coverage of all implant threads. **E,** Final restoration. **F,** Bone findings 10 months after implant insertion.

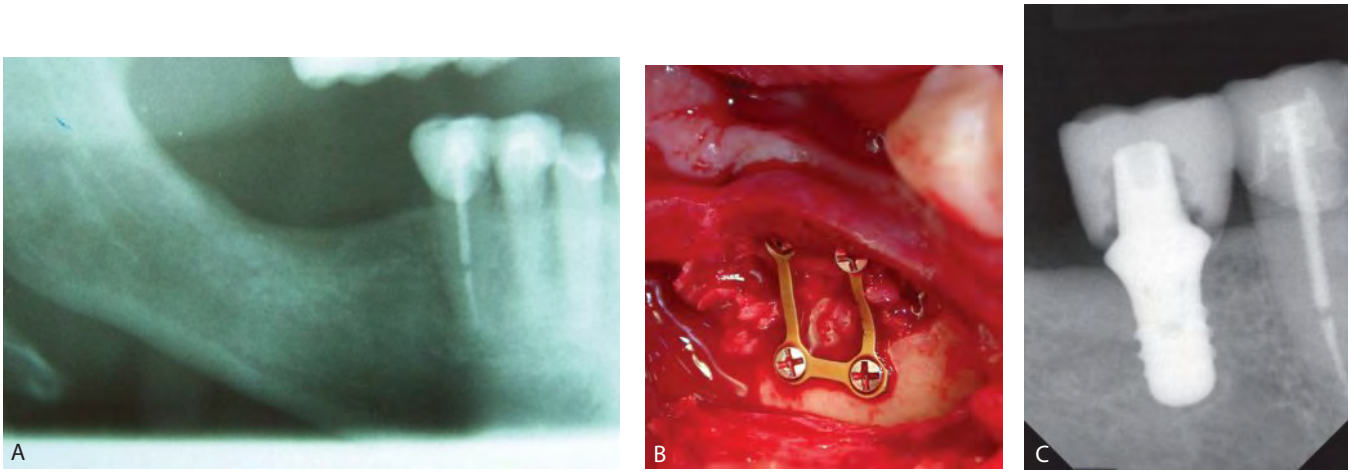


FIGURE 11-6. **A,** The posterior mandible that is both vertically and horizontally deficient can often be treated by an "alveolar lift" osteotomy procedure. **B,** By elevating the segment above the alveolar plane, subsequent flap exposure for implant placement 4 months later can reduce a thin residual ridge and place an implant with either little or no further grafting. **C,** Final implant restoration with a wider alveolus at the appropriate alveolar plane.

often due to trauma, or conversely, the posterior segment may be edentulous from periodontal loss with retained anterior dentition. As contiguous teeth are lost, segmental resorption commonly develops leading to combined vertical and horizontal deficiency. Bone augmentation solutions then take on certain patterns of treatment based on the extent of the bone loss.

Anterior Segmental

The extent of bone deficiency can be categorized as mild, in which GBR procedures are suggested; moderate, treated by block grafting, interpositional techniques, or titanium mesh procedures; or severe lesions, treated by iliac corticocancellous block grafts or larger titanium mesh procedures often using morphogens (Figure 11-7). In complete loss, continuity defect treatment may proceed with reanastomosis of free fibula grafts, iliac bone source grafting, or BMP-2 procedures depending on the length of the discontinuity.

The most common anterior mandibular lesions, however, are the moderate to severe lesions due to trauma or periodontal loss. Once the vertical loss reaches 5 mm, one method to easily treat this lesion, even up to 10 mm of deficiency, is the sandwich osteotomy bone graft. This procedure will take care of the vast majority of vertical osseous deficiency states not amenable to simple GBR. Figure 11-8 shows a surgical series demonstrating interpositional grafting with an "alveolar lift" of 4 mm with a segmental internal alveolar split to widen a narrow alveolus. Lifting a narrow alveolus that may even have adequate height can often bring the wider part of the alveolus crestally. Excess vertical height, after healing, is then reduced at the time of implant placement. Access to the osteotomy is made through the vestibule with the lateral osteotomy being made using piezosurgery superficially and deep with a sagittal saw. Inlay material is used to fill the gap and a bone plate is used to reposition the segment facially as the segment tends to displace lingually using a sandwich technique.[15]

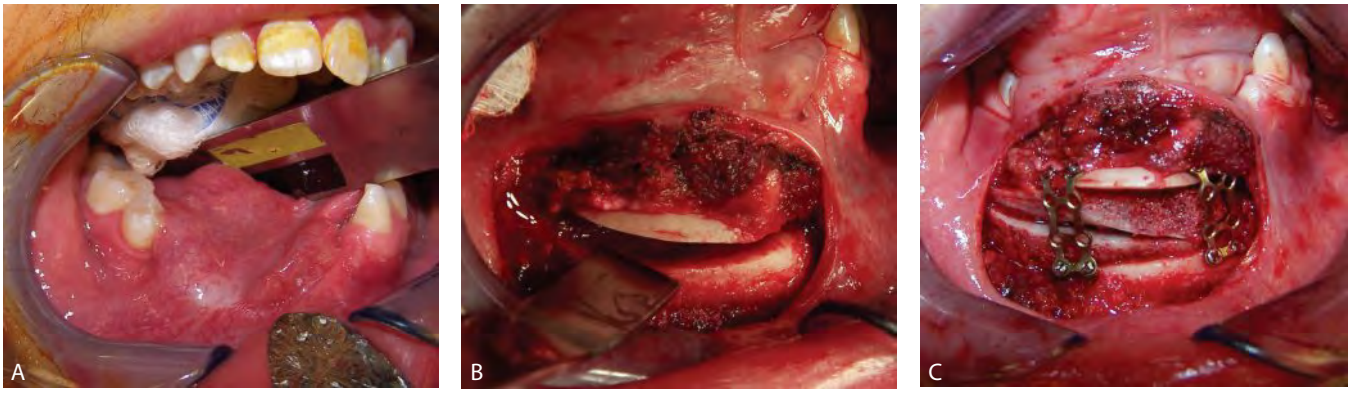
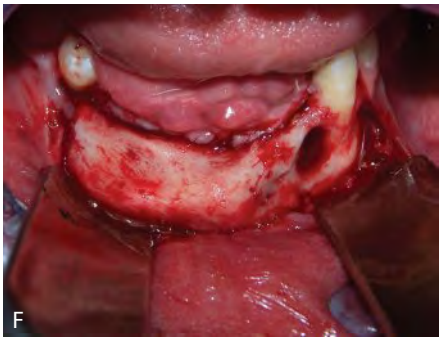


FIGURE 11-7. **A,** A 19-year-old patient status postmandibular tumor resection from 10 years ago now presents for reconstructive surgery. The floor of the mouth mucosa extends to the buccal mucosa. **B,** An osteotomy segment is elevated 2 cm using a sandwich osteotomy from a vestibular incision. **C,** A 15-mm interpositional iliac bone graft was placed (in two layers) with particulate overgrafting.

(Continued)







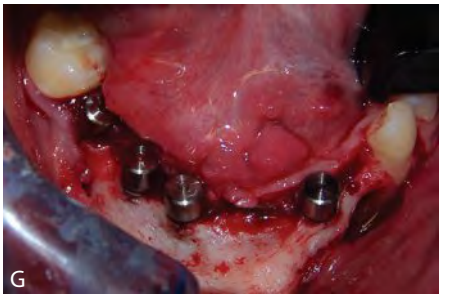


FIGURE 11-7. *(Continued)* **D,** Six months later, the graft appeared to be consolidated. **E,** A connective tissue graft helped establish keratinization. **F,** Exposure of the site reveals a well-healed graft with one relatively small defect. **G,** Implants were placed with high insertion torque and good stability.

One variation of the sandwich technique is the internal alveolar split osteotomy used to gain both height and width of the alveolus, but there must be substantial width available for dividing the alveolus, which is not often the case[16] (Figure 11-9).

When the site is 10 mm or more deficient, the use of alveolar vertical distraction should be considered[17] (Figure 11-10).

Mandibular Posterior Segment

The free end saddle is often accompanied by six to eight sound anterior teeth such that removal of the anterior teeth to perform a full -arch fixed hybrid restoration is contraindicated. These sites often have marked atrophy with close inferior alveolar nerve approximation. These sites are most often treated with block bone grafting, but iliac or mandibular block source bone resorbs at a high rate in this location so the graft should be overbuilt to take this into account.[18] The development of unique GBR strategies are still frequently being done using high -profile internal structure supports such as screws or even implants with particulate graft.[19] One new technique is the "bone-wedge technique" in which small autogenous wedges of bone are inserted into basal bone in high profile and then grafted between and covered by a barrier membrane (Figure 11-11). The posterior edentulous segment is also well suited for the sandwich osteotomy done curved in a smile shape, creating a small chamber (Figure 11-12).

FULL ARCH

Controversy continues regarding full-arch reconstruction. Early use of iliac block grafting to reconstruct jaw bone height, sometimes done in conjunction with implant placement, has become an infrequently done procedure even though these procedures have passed the test of time[20,21] (Figure 11-13). The use of short implants that perforate the inferior border of the mandible or implants done without any grafting for fixed hybrid bridges confound conventional wisdom often undergoing appositional growth of bone mass at both the inferior and the superior borders of the mandible. This suggests that there may be a limited need for bone grafting at all for the edentulous mandible[22] (Figure 11-14). The use of the V-4 technique is an example of the use of only four implants placed in mandibles as vertically deficient as 5 mm of height when immediate functional loading is done.[22] Total edentulous jaw distraction[23] (Figure 11-15), tent pole iliac grafting from the inferior approach, and nerve transposition to improve implant positioning for anteroposterior spread (Figure 11-16) are alternatives.[24] When the mandible is in discontinuity due to ablation, free fibula grafts and/or iliac grafting and—now recently BMP-2—allograft with confining cribs have been used successfully.

Discussion

The surgeon's philosophical template for lower jaw bone reconstruction with dental implants is about to undergo a



FIGURE 11-8. **A** and **B,** The anterior edentulous zone can present with marked alveolar width deficiency and as a retrodisplaced alveolar process. **C** and **D,** Despite adequate height, a sandwich osteotomy used to lift the segment vertically then internally split cannot only improve segment anteroposterior position but improve alveolar width. **E,** Overall segment bone mass for implants are enhanced. **F–H,** Implants are placed in conjunction with bone grafting. **I–K,** One year later, implants and the final restoration appears well healed and functioning with short incisor crowns, sometimes difficult to achieve with traditional grafting methods such as GBR.

significant paradigm shift to accommodate the use of BMP.[25] Much of this experimental work is based on the concept of grafting around high-profile implants using guided bone regeneration techniques. Because harvesting of intraoral or extraoral autogenous bone constitutes a certain amount of morbidity, the development of a BMP-2 model for grafting becomes highly desirable.[26] Also, because the use of xenograft and allograft products, even in conjunction with the use of

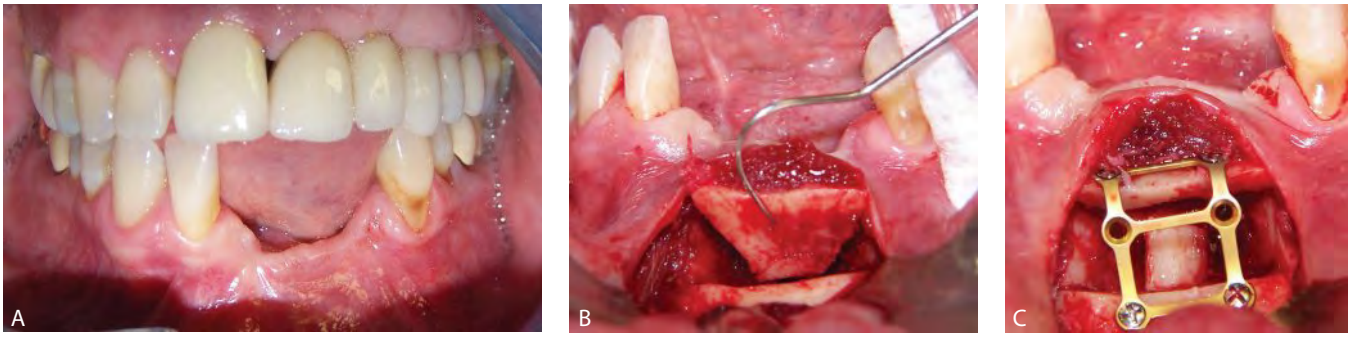


FIGURE 11-9. **A,** A significant combination of alveolar ablation, vertical and horizontal. **B** and **C,** A sandwich graft with internal alveolar split will both raise and widen the alveolus substantially—in this case, 10-mm increase in height and 5-mm increase in width using bone morphogenetic protein-2 (BMP-2) as grafting material.

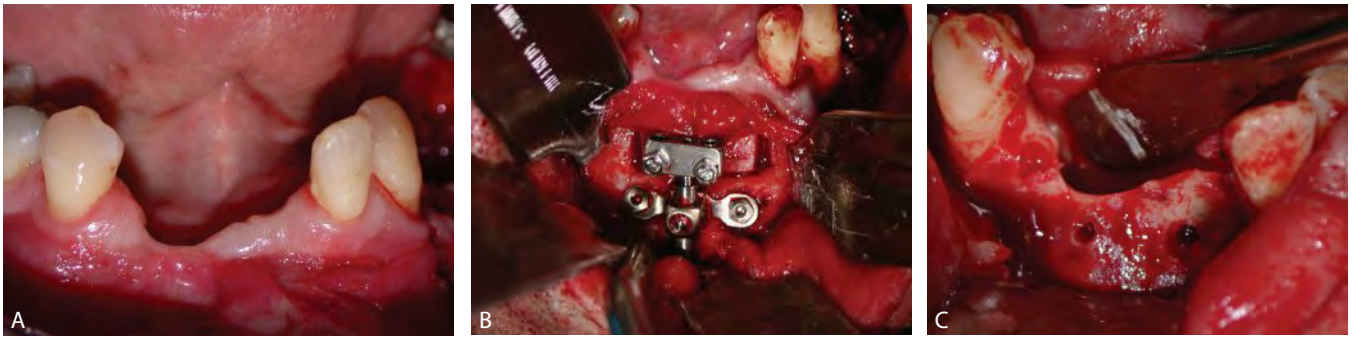


FIGURE 11-10. **A,** An anterior defect of uncertain morphology caused by trauma can be planned for alveolar distraction. **B,** A small Mommaerts-Laster distractor is placed. **C,** At the time of device removal, implants and further bone grafting are done in conjunction with reduction of excess vertical height.

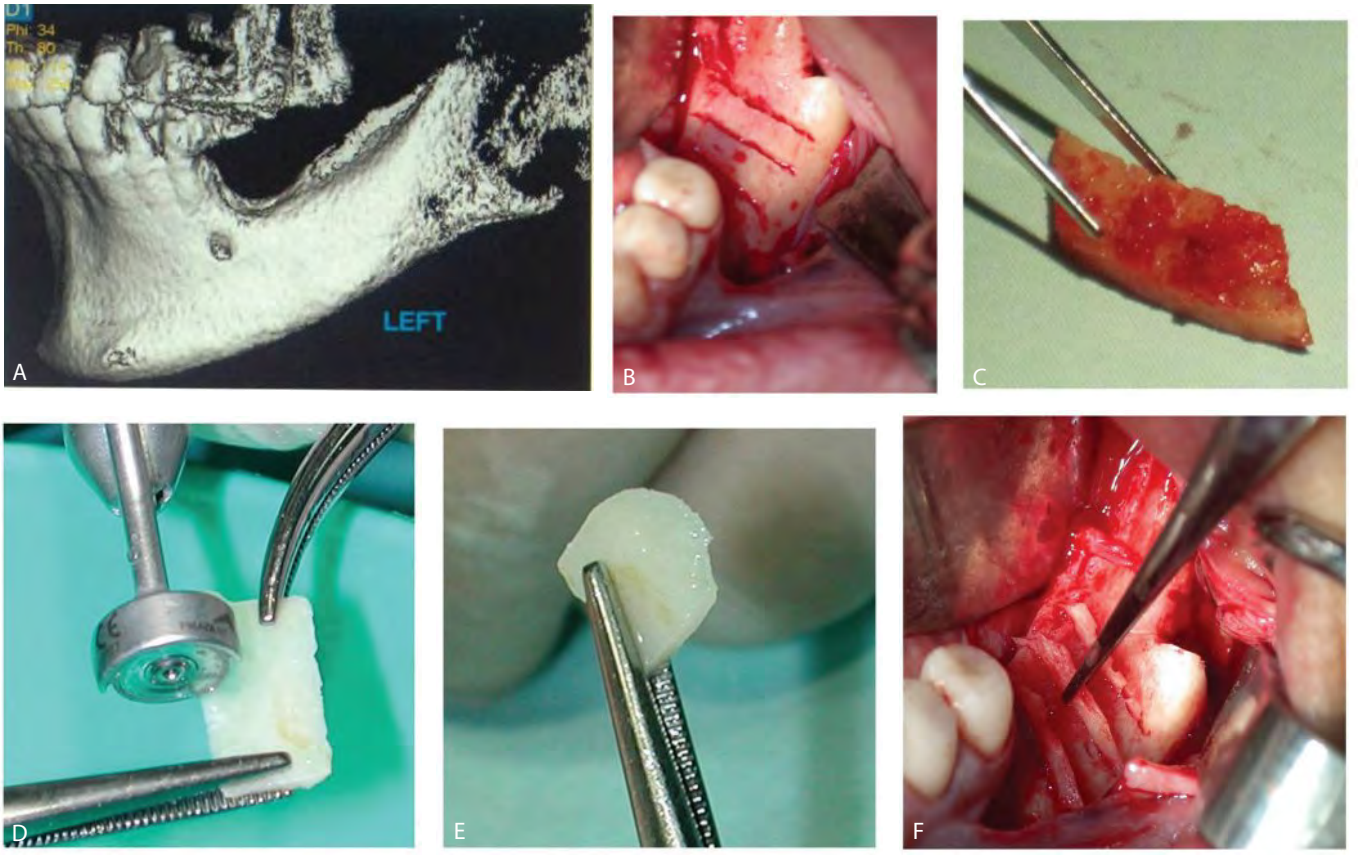


FIGURE 11-11. *(Continued)*



FIGURE 11-11. **A,** Posterior mandibular atrophy is complicated by nerve proximinity. **B,** The site is flapped and scored with a fine-tip bur (surgeons Fares Kablah and Zvi Laster). **C–E,** The external oblique ridge cortex is removed and fashioned into wedges that are tapered at one edge. **F** and **G,** The wedges are tapped into the scored sites and grafted and covered with a membrane. **H** and **I,** Implants are placed 4 months later into a maintained vertical dimension.



FIGURE 11-12. **A,** Posterior mandibular vertical height loss. **B,** If there is sufficient room for an osteotomy, an alveolar lift can be done. **C,** The segment can be easily elevated several millimeters using a smile-shaped sandwich osteotomy.

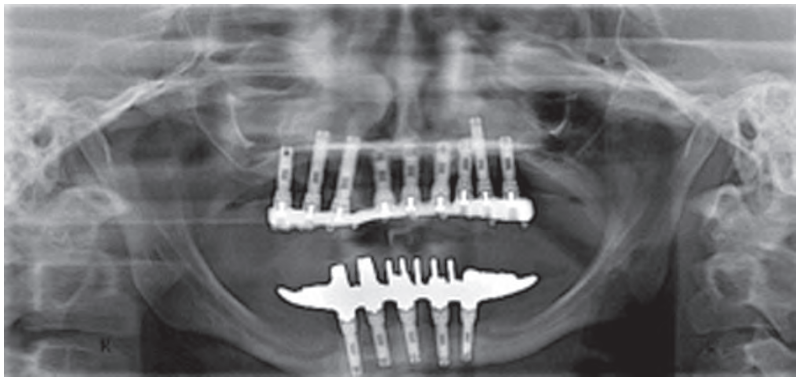


FIGURE 11-13. A 20-year result of vertical iliac block graft placed in both arches with implants placed 6 months later shows stable bone and secure implants with areas of actual bone gain found in the mandible.

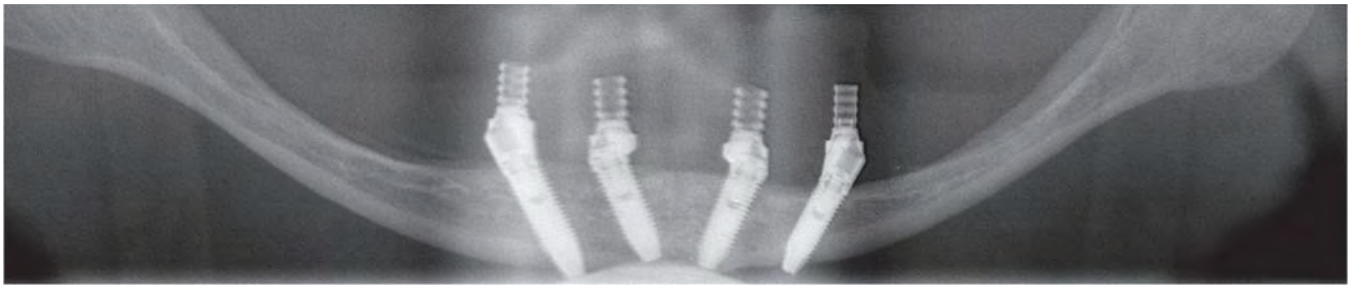


FIGURE 11-14. Atrophic edentulous mandibles may not require vertical bone grafting at all, especially in the aged patients. Implants can be placed using a V technique to avoid convergence of the osteotomy holes, which can weaken the mandible. Implants can perforate the inferior border of the mandible a few millimeters and provoke periosteal osseous apposition.

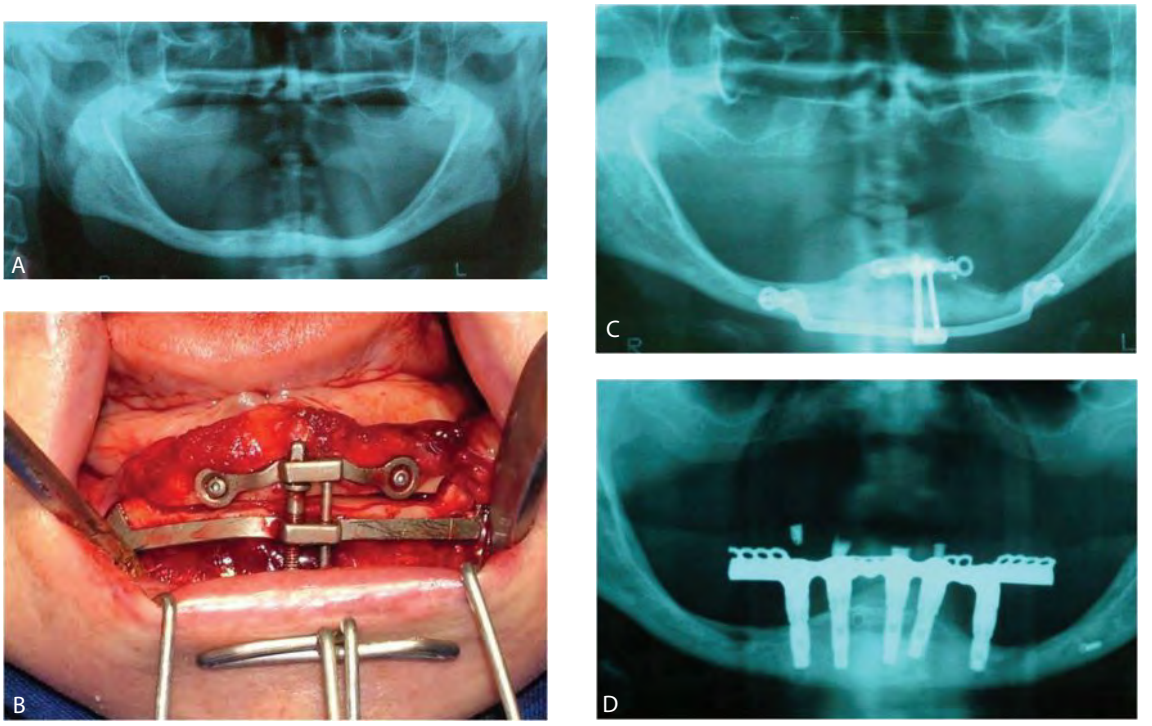


FIGURE 11-15. **A,** The presence of an atrophic mandible in younger patients may justify the use of an augmentation procedure such as distraction osteogenesis. **B,** This distraction device, developed by Dr. Marcelo Soares, protects the alveolus from fracture perioperatively. **C,** After consolidation, implants are placed at greater length and a more robust mandible is achieved. **D,** Long-term osseointegration with longer implants in a fixed hybrid restoration.

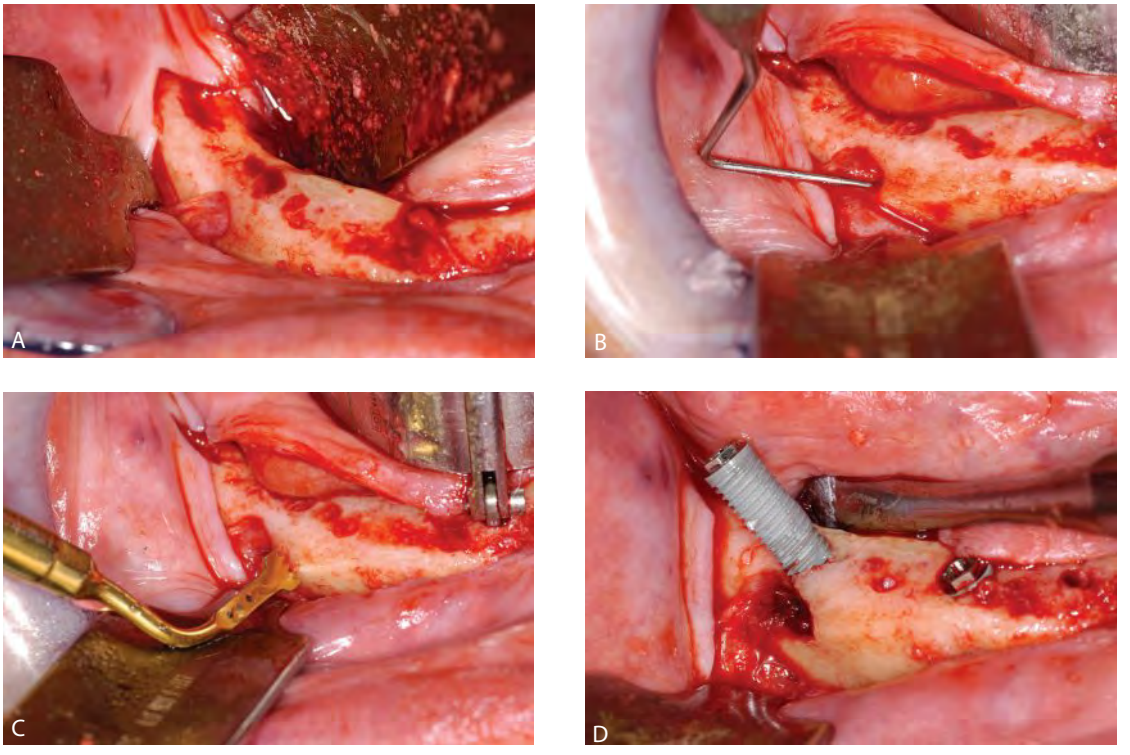


FIGURE 11-16. **A,** Nerve repositioning is a consideration only if the nerve is located anteriorally. **B,** The anterior loop of the nerve (usually 1mm or less) at the mental foramen can be determined using a periodontal probe. **C** and **D,** Using piezosurgery, the anterior loop can be distalized, permitting placement of an implant more posterior in the arch to increase anteroposterior spread.

platelet-rich plasma (PRP) or platelet-derived growth factor-BB (PDGF-BB), have not been as successful at forming de novo bone as has BMP-2 clinically. The oral maxillofacial surgeon looks forward to development of this field.[27]

Several studies have now evaluated the use of BMP-2 in various treatment strategies in both animal and clinical settings. Wikesjo and associates[29] showed that BMP, when placed under a confining membrane around high-profile implants, consistently made bone in a canine model. Also, using a calcium phosphate cement impregnated with BMP-2 without a barrier once again showed bone formation in supra-alveolar implants, this time with greater bone density.[29] In a similar canine model, Sigurdsson and coworkers[30] showed that there was no difference in osseointegration content (55%) in BMP or native bone. Importantly, Javonovic and colleagues[31] showed that high-profile implants grafted with BMP in a canine then restored and left in function for 12 months maintained bone around the implants with *no discernable difference* between native and BMP-2–induced bone (Figure 11-17).

These animal research findings are basically a recapitulation of canine studies using iliac bone or allogeneic bone with and without membranes over high-profile implants done in 1990[21,32] (Figure 11-18). There is a proof of principle here that cannot be denied and now without having to resort to the tibia or ilium for graft material. Now, it is simply for the oral maxillofacial surgeon to investigate and validate clinically the appropriate system for implant fixation, regenerative grafting, and morphologic space maintenance that is suggested by as yet unpublished preclinical work[33] (Figure 11-19) and clinical research[34,35] (Figure 11-20).

SUMMARY

The reconstruction of the mandible must be addressed with a technique that matches the extent of the defect present. But implant placement is determined by the overall restorative plan. Current restorative treatment plans have become more efficient for number of implants placed, often allowing

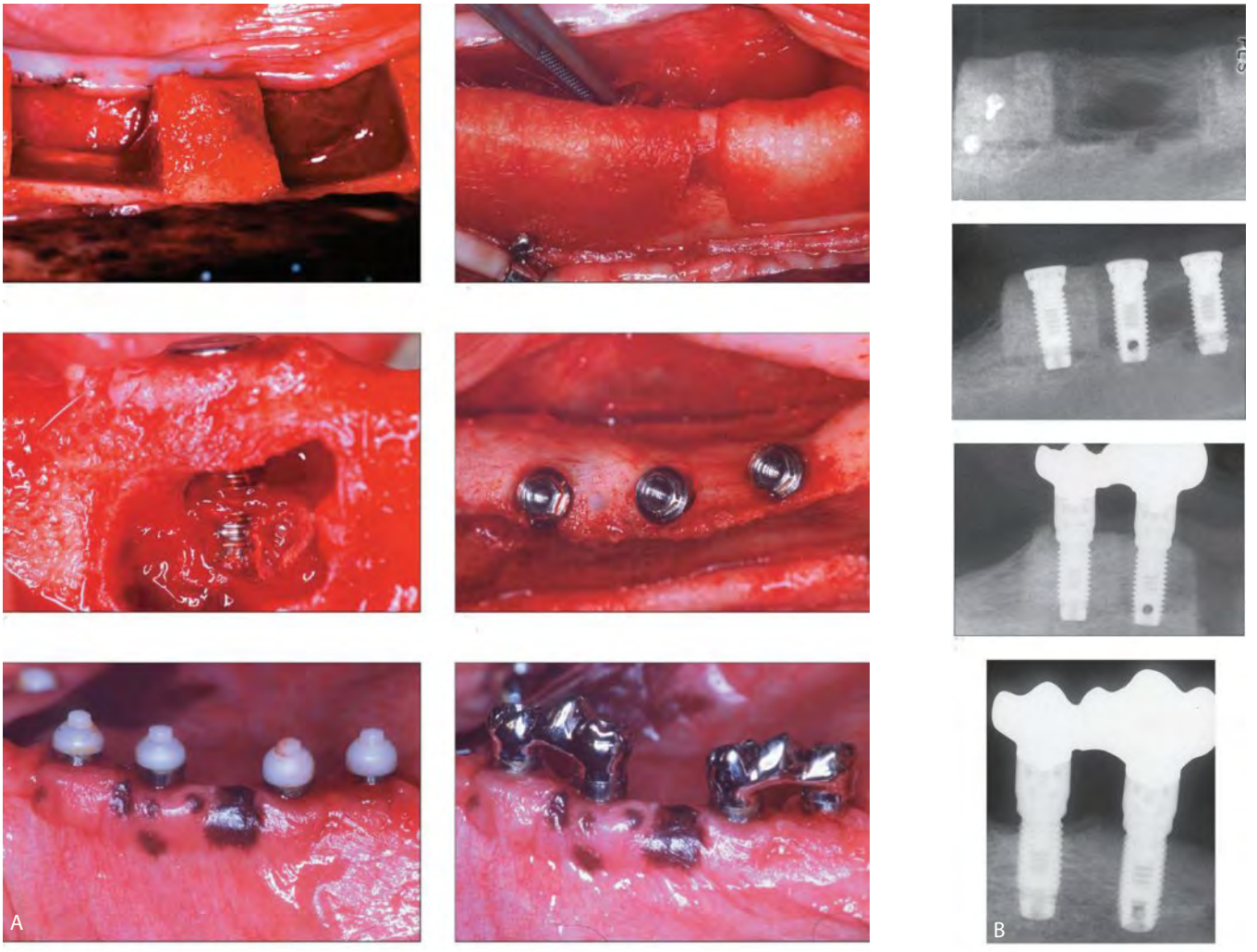


FIGURE 11-17. **A,** Dr. Sacha Javonovic used a dog mandible model to determine whether BMP-2 bone can function long term. Defects were made and filled with BMP-2, then implanted 4 months later and observed histologically for osseointegration after 1 year. **B,** Findings after BMP-2 grafting, implant placement, and loading.

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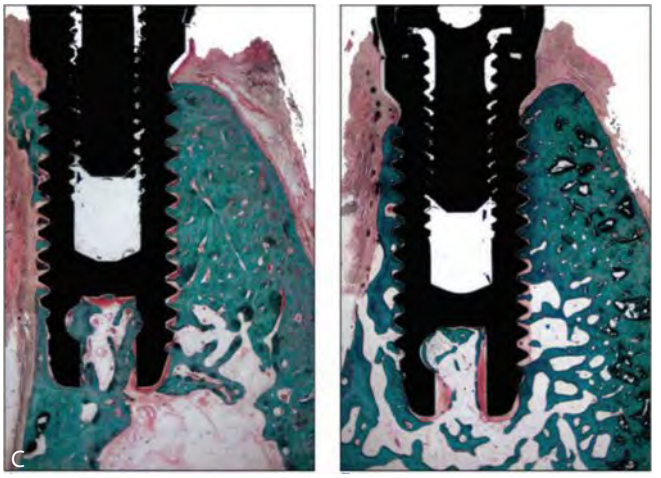
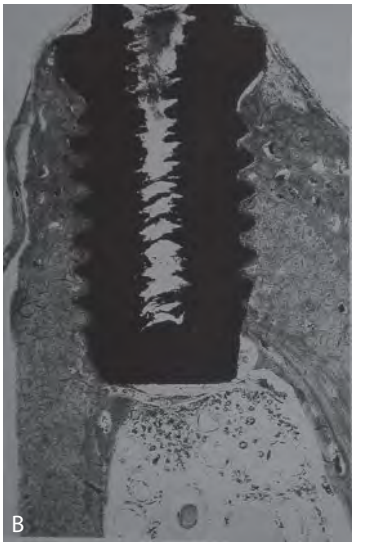
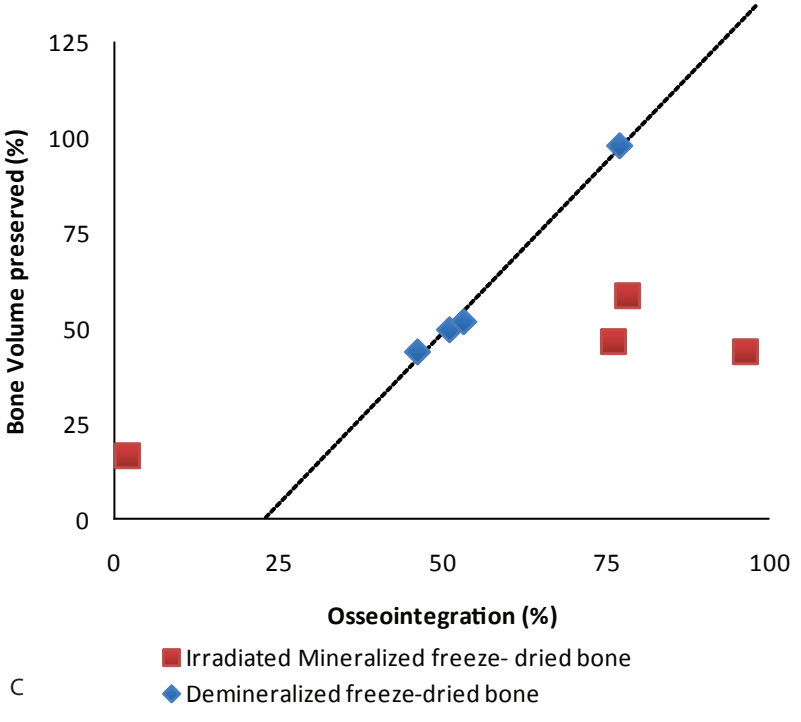


FIGURE 11-17. *(Continued)* **C,** Osseointegration findings were the same for implants placed into natural or BMP-2–generated bone.







C

FIGURE 11-18. **A,** Barrier membrane use in high-profile implants has been shown to be imperative for bone maintenance. This specimen is a dog mandible, both sides treated with high-profile implants and iliac block auto graft. The right side was covered with an expanded polytetrafluoroethylene (ePTFE) membrane and the left was not, which resorbed almost entirely after 4 months. **B,** Membrane-protected iliac block graft of a high-profile Branemark implant. **C,** Non–membrane-protected iliac bone graft of a high-profile Branemark implant shows marked bone resorption.

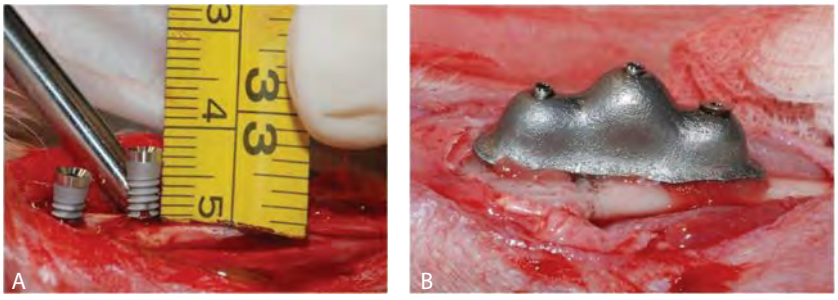


FIGURE 11-19. **A** and **B,** Recent efforts to use BMP-2 for high-profile implants have used titanium shells to protect the graft.

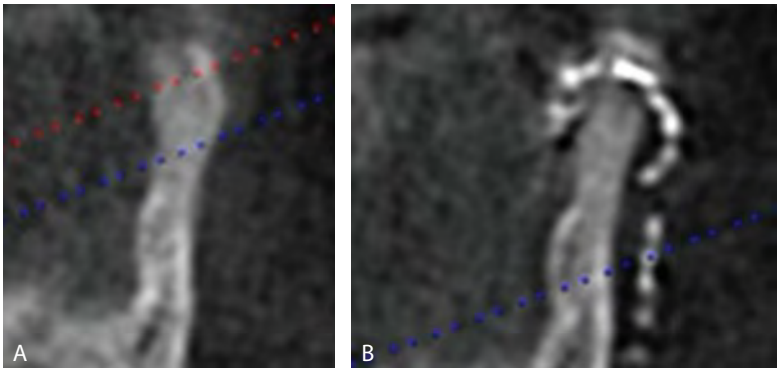


FIGURE 11-20. **A** and **B,** The use of shells or titanium mesh to treat narrow arches using BMP-2 is just now being studied clinically.

the surgeon to avoid compromised locations, thereby obviating the need for extensive bone graft reconstruction. Care should be taken to design appropriate therapy according to the age of the patient. Elderly patients contraindicated for extensive reconstruction should be considered instead for the simple over-denture approach or nongrafting All-on-Four.[22] Bone morphogenetic grafting is no longer a dream of the future. Well-documented preclinical science is beginning to supplement common practices for clinical mandibular reconstruction.

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12

CHAPTER

Soft Tissue Management in Implant Therapy

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SOFT TISSUE INTEGRATION

The term *soft tissue integration* describes the biologic processes that occur during the formation and maturation of the structural relationship between the soft tissues (connective tissue and epithelium) and the transmucosal portion of an implant. Although experimental and clinical research have only recently begun to focus on improving our understanding of the factors that can affect this soft tissue environment, our current knowledge indicates that the maintenance of a healthy soft tissue barrier is as important as osseointegration itself for the long-term success of an implant-supported prosthesis. As such, the implant surgeon must be well acquainted with various surgical techniques and approaches for successfully managing peri-implant soft tissues in commonly encountered clinical situations. Furthermore, when an inadequate quantity or quality of soft tissue is available to secure a stable peri-implant environment, the implant surgeon must know the principles and techniques to successfully reconstruct these components. This chapter focuses on basic principles and surgical techniques to manage and, when indicated, reconstruct peri-implant soft tissues to enhance the long-term predictability and aesthetic outcomes achieved in implant therapy.

FLAP MANAGEMENT CONSIDERATIONS

The primary goal of implant soft tissue management is to establish a healthy peri-implant soft tissue environment. This goal is accomplished by obtaining circumferential adaptation of attached tissues around the transmucosal implant structures, thereby providing the connective tissue and epithelium needed for the formation of a protective soft tissue seal.[1] In addition, when implant therapy is performed in aesthetic areas, re-creating natural-appearing soft tissue architecture and topography at the prosthetic recipient site is often necessary. To achieve these goals, the surgeon must carefully preserve and manipulate existing soft tissues at the implant site and perform soft tissue augmentation, when indicated. The quantity, quality, and positioning of the existing attached tissues relative to the planned implant emergence should be evaluated before implant surgery. The flap should be designed to ensure that an adequate band of attached, good-quality tissue is always available lingual or palatal to the planned implant emergence. Designing the flap in this fashion is practical because subsequent correction of soft tissue problems occurring in lingual and palatal areas is difficult. Preoperative evaluation using a surgical template helps the surgeon visualize whether adequate tissue quality and volume are available in the area critical for prosthetic emergence. The surgeon can then decide where the incisions will have to be made or how the existing soft tissues must be manipulated with specific surgical maneuvers to establish a stable peri-implant soft tissue environment in each individual case.

Design for Submerged Implant Placement

When placing a submerged implant, the buccal flap must be designed to preserve both the blood supply to the implant site and the topography of the alveolar ridge and mucobuccal fold. The access flap is outlined by a pericrestal incision and one or more linear or curvilinear vertical releasing incisions that extend onto the buccal aspect of the alveolar ridge. The pericrestal incision is beveled to the lingual or palatal aspects (Figure 12-1). The incision is initiated over the lingual or palatal aspects of the ridge crest, and the scalpel blade is angled to make contact with the underlying bone. Typically, linear vertical releasing incisions are used in edentulous

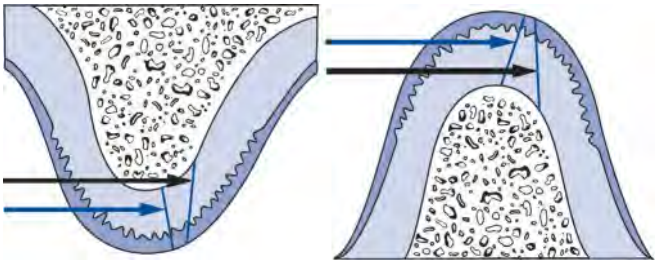


FIGURE 12-1. Beveled pericrestal incisions. The *black arrows* represent the path of the palatal and lingual beveled pericrestal incisions recommended for submerged implant placement in the maxilla and mandible. The *blue arrows* represent the buccal beveled incisions recommended for abutment connection and nonsubmerged implant placement in the maxilla and mandible. (Adapted from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

situations and curvilinear beveled incisions are used in partially edentulous situations. In either case, reflection of the buccal flap exposes the entire ridge crest and provides ample access for implant instrumentation. This is accomplished with minimal lingual or palatal flap elevation, thus preserving periosteal circulation and providing attached tissue to anchor the buccal flap during subsequent wound closure. The stability of the postoperative wound complex is improved, and the topography of the alveolar ridge and mucobuccal fold is preserved. As a result, wound dehiscence is decreased and the use of a provisional prosthesis during the osseointegration period is facilitated.

Design for Abutment Connection and Nonsubmerged Implant Placement

Except for the location and bevel of the pericrestal incisions, the same flap design is used for an abutment connection to submerged implants as for placement of nonsubmerged implants (see Figure 12-1). The pericrestal incision is initiated in a position that ensures the maintenance of approximately a 3-mm apicocoronal dimension of attached lingual tissue or good-quality palatal mucosa (free of rugae) for readaptation around the emerging implant structures. The quantity and position of the existing soft tissues guide the location of the incision. In general, this incision is located closer to the midcrestal position than the one made for submerged implant placement. The scalpel blade is held so as to create a buccal bevel to facilitate abutment connection and implant placement while preserving periosteal blood supply by minimizing the need for a lingual or palatal flap reflection. In addition, the buccal bevel maximizes the amount of attached tissue reflected with the buccal flap (see Figure 12-1).

As suggested previously, by adjusting the location and bevel of pericrestal incisions and precisely locating linear or curvilinear vertical releasing incisions, the implant surgeon is equipped with practical flap designs for submerged implant placement, abutment connection, and nonsubmerged implant placement in edentulous and partially edentulous and aesthetic case types (Figures 12-2 to 12-6).

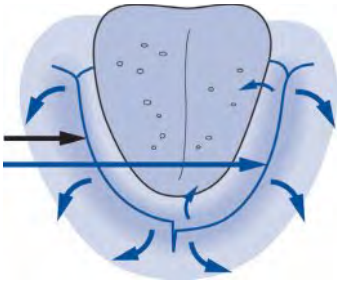


FIGURE 12-2. The flap design for implant placement in the edentulous mandible incorporates a midline vertical releasing incision and distal vertical releasing incisions made well beyond the area planned for the implant placement. The *black arrow* indicates the location of the pericrestal incision used for submerged implant placement. The location of the incision used for abutment connection and nonsubmerged implant placement is indicated by the *straight blue arrow.* (Adapted from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

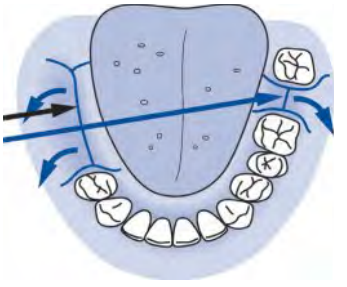


FIGURE 12-3. The flap design for implant placement in the partially edentulous mandible is outlined by pericrestal and curvilinear beveled vertical releasing incisions. The *black arrow* indicates the location of the pericrestal incision used for submerged implant placement. The location of the incision used for abutment connection and nonsubmerged implant placement is indicated by the *straight blue arrow.* (Adapted from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

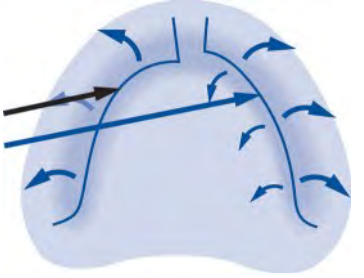


FIGURE 12-4. The flap design for implant placement in the edentulous maxilla incorporates paramidline vertical releasing incisions and distal vertical releasing incisions made well beyond the area planned for the implant placement. The *black arrow* indicates the location of the pericrestal incision used for submerged implant placement. The location of the incision used for abutment connection and nonsubmerged implant placement are indicated by the *straight blue arrow.* (Adapted from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

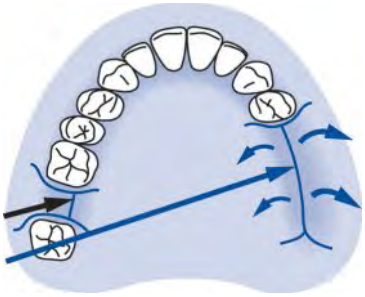


FIGURE 12-5. The flap design for an implant placement in the partially edentulous maxilla is outlined by pericrestal and curvilinear beveled vertical releasing incisions. The *black arrow* indicates the location of the pericrestal incision used for submerged implant placement. The location of the incision used for abutment connection and nonsubmerged implant placement is indicated by the *straight blue arrow.* (Adapted from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

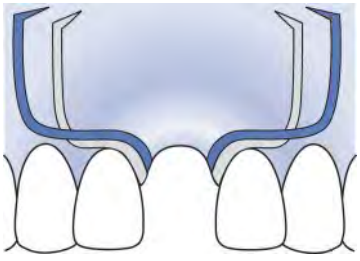


FIGURE 12-6. The flap design for aesthetic implant therapy is outlined by pericrestal and curvilinear beveled releasing incisions. The vertical legs of the releasing incisions are made in the adjacent interdental areas, thereby providing the opportunity to camouflage within interdental grooves and the mucogingival junction. This flap design incorporates greater amounts of mucosal tissues, improving the overall elasticity of the flap. When combined with tension-releasing cutback incisions, coronal advancement is facilitated without an embarrassment of circulation to the flap margin. The flap design is exaggerated by moving the releasing incisions farther away from the site when reconstruction of large-volume aesthetic ridge defects is necessary or for implant placement at sites where vestibular depth is inadequate. (Adapted from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

Surgical Maneuvers for Management of Peri-implant Soft Tissues

Once the flap has been outlined in a manner that ensures an optimal lingual and palatal soft tissue environment, the surgical maneuvers that are used for managing the resulting buccal flap during abutment connection and nonsubmerged implant placement can be determined, for the most part, by the apicocoronal dimension of the attached tissue remaining on the buccal flap margin. Three distinct soft tissue surgical maneuvers are commonly used during abutment connection or nonsubmerged implant placement to achieve the desired outcome of obtaining primary closure with circumferential adaptation of attached tissues around emerging implant structures: *resective contouring*, *papilla regeneration*, and *lateral flap advancement.*

Although the minimum width of attached tissue necessary to establish a stable peri-implant soft tissue environment has yet to be established, the following guidelines for using each of the soft tissue maneuvers provide consistent results in most clinical situations. It is important to note that the use of a specific maneuver is based primarily on the apicocoronal dimension of the attached tissue remaining along the buccal flap margin at each implant site. A combination of these surgical maneuvers is often indicated because the width of attached tissue remaining on the buccal flap varies as a result of necessary adjustments made in the path of the crestal incision to maintain an adequate width of attached tissue on the lingual or palatal flap.

Resective Contouring

When the width of the gingival tissues remaining on the buccal flap is 5 to 6 mm, resective contouring facilitates circumferential adaptation of the soft tissues around the emerging implant structures. A fine scalpel blade held in a round handle is used to perform a gingivectomy on the buccal flap corresponding in shape and position to the anteriormost abutment or nonsubmerged implant neck. After resective contouring, the tissue is adapted around the emerging implant structure; this process is then repeated sequentially around each implant (Figure 12-7). The contoured flap is then repositioned apically and secured around the abutments with a suture passing through each interimplant area; additional sutures are placed to close the curvilinear releasing incisions.

Papilla Regeneration

When the width of the gingival tissues remaining on the buccal flap is 4 to 5 mm, use of the papilla regeneration maneuver is indicated. Advocated by Palacci and colleagues,[2] this maneuver facilitates primary closure and circumferential adaptation around the transmucosal implant structures while preserving an adequate band of attached tissue around the

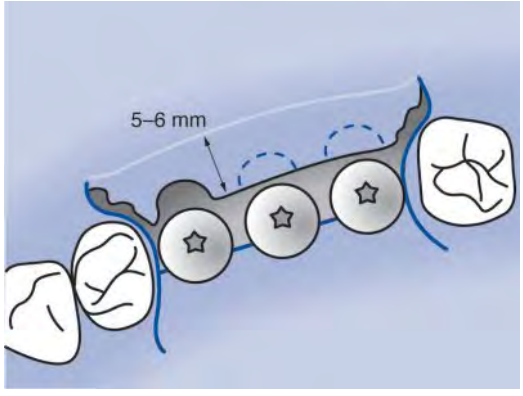


FIGURE 12-7. Resective contouring maneuver. When the apicocoronal dimension of the attached tissue remaining on the buccal flap used for the abutment connection or a nonsubmerged implant placement is between 5 and 6 mm, resective contouring is used to facilitate circumferential adaptation of the soft tissues around the emerging implant structures. (Adapted from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

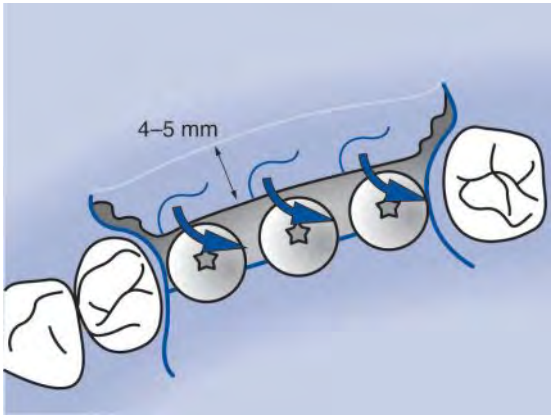


FIGURE 12-8. Papilla regeneration maneuver. When the apicocoronal dimension of the attached tissue remaining on the buccal flap used for an abutment connection or a nonsubmerged implant placement is between 4 and 5 mm, the papilla regeneration maneuver is used to facilitate circumferential adaptation of the soft tissues around the emerging implant structures. (Adapted from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

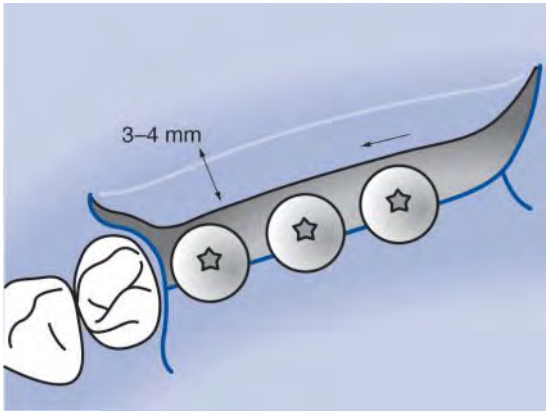


FIGURE 12-9. Lateral flap advancement maneuver. When the apicocoronal dimension of the attached tissue remaining on the buccal flap used for an abutment connection or a nonsubmerged implant placement is between 3 and 4 mm, lateral flap advancement is used to facilitate circumferential adaptation of the soft tissues around the emerging implant structures. (Adapted from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

emerging implant structures. In addition, attached mucosa is taken from the top of the ridge and moved in a buccal direction while approximately 3 mm of attached lingual or palatal tissues is preserved. A fine scalpel is subsequently used to sharply dissect the tissues to create pedicles in the buccal flap, which are passively rotated to fill the interimplant spaces (Figure 12-8). Passive adaptation of the pedicles in the interimplant space may require reverse cutback incisions made away from the base of the pedicle. The tissues are sutured, avoiding tension within the pedicles, usually using a figure-of-eight horizontal mattress suture. Alternatively, a simple interrupted suture passed through the buccal flap in a fashion that passively advances the pedicle into the interimplant space is effective in many situations. Care must be taken to avoid placement of the suture through the pedicle because this would reduce circulation to the pedicle. Another variation of this technique uses pedicles created in the palatal flap, which can also be rotated to fill the interimplant spaces, and is especially useful in maxillary situations where thick palatal tissues exist.[3]

Lateral Flap Advancement

When the width of the gingival tissues remaining on the buccal flap is 3 to 4 mm, the use of the lateral flap advancement maneuver facilitates primary closure and circumferential adaptation of attached tissues around the emerging implant structures (Figure 12-9).[3] This maneuver is especially suited for completely edentulous or posterior partially edentulous implant case types, in which an adequate band of attached tissue exists adjacent to the implant site. Attached tissues available from adjacent areas are simply repositioned to obtain primary closure with attached tissues around the emerging implant structures.

This maneuver requires that the flap be designed to extend beyond the area of implant placement to include the attached tissues present in adjacent edentulous areas. As the closure progresses, the flap advances, resulting in primary closure around the implants and the creation of a denuded area that will heal by secondary intention at the distal extent of the dissection. This surgical maneuver is useful in edentulous situations and in Kennedy classes I and II partially edentulous situations.

RATIONALE FOR SOFT TISSUE GRAFTING WITH IMPLANTS

The rationale for soft tissue augmentation around dental implants is related to the need for soft tissue around natural dentition. In general, experienced clinicians agree that an adequate zone of attached tissue around a natural tooth or implant prosthesis is desirable to better withstand the functional stresses resulting from mastication and oral hygiene. Moreover, a certain amount of attached tissue is needed to withstand the potential mechanical and bacterial challenges presented by aesthetic restorations that extend below the free gingival margin. Potential mechanical challenges include tooth preparation, soft tissue retraction, impression procedures, cementation of provisional and permanent restorations, removal of implant healing abutments, replacement of healing abutments with permanent abutments, taking of implant-level impressions, and placement of provisional and permanent implant restorations.

After the final restoration, the intracrevicular aesthetic restorative margins may continue to present a permanent inflammatory challenge to the surrounding soft tissue attachment apparatus. Some implant practitioners believe that the microgap at the site of the abutment connection to two-piece implants may present a similar challenge. Whether these challenges result in an initial apical displacement of the marginal tissues or possibly even progressive loss of attachment depends on multiple factors, including the following[3]:

- Age of the patient.
- General health of the patient.
- Host resistance factors.
- Effects of systemic medications.
- Periodontal phenotype.
- Technique and effectiveness of oral hygiene.
- Frequency and technique of professional oral hygiene care.
- Operative technique.
- Choice of restorative materials.
- Initial location of restorative margin vis-à-vis circumferential biologic width requirements.
- Prominence of the implant position in the alveolus.
- Preexisting bony dehiscence.
- Design and surface characteristics of the implant.
- Depth of implant placement.
- Thickness and apicocoronal dimension of the attached tissue.

Because multiple factors influence the health of the marginal tissues, prospective or retrospective experimental or clinical studies are difficult to design and conduct, much less interpret. Certainly, studies that primarily consider the apicocoronal dimension of attached tissue and its effect on marginal soft tissue health, without considering the other factors, are inconclusive at best. Therefore, the rationale for soft tissue augmentation around natural dentition or a dental implant prosthesis should be based on clinical experience rather than on results from experimental or clinical studies.[3]

CLINICAL GUIDELINES FOR SOFT TISSUE AUGMENTATION

When the apicocoronal dimension of attached tissue remaining on the buccal flap will be less than 3 mm, the surgeon should consider soft tissue augmentation. Other factors to consider include tissue thickness, tissue quality, the presence of soft tissue inflammation or pathology, the type of implant restoration planned, and the aesthetic importance of the site. In a nonaesthetic area, the surgeon can use the various surgical maneuvers described previously to obtain primary closure and then reevaluate the need for soft tissue grafting based on the health and volume of peri-implant attached tissues obtained after initial healing. In contrast, when the total width of attached tissue present is less than 3 mm in an aesthetic area, soft tissue augmentation is indicated before implant placement. In most instances, this can be accomplished with an epithelialized palatal mucosal graft, which quickly provides an improvement in the quality of the soft tissues.

Similarly, in aesthetic areas, small-volume soft tissue aesthetic ridge defects can be corrected simultaneously with submerged or nonsubmerged implant placement with subepithelial connective tissue grafting, whereas large-volume soft tissue aesthetic ridge defects are most predictably reconstructed before implant placement with a series of subepithelial connective tissue grafts. Large-volume soft tissue defects can also be corrected with the use of a vascularized interpositional periosteal connective tissue (VIP-CT) flap, which in ideal circumstances, allows for predictable reconstruction synchronous with implant placement.

PRINCIPLES OF ORAL SOFT TISSUE GRAFTING

The first principle of oral soft tissue grafting is that the recipient site must provide for graft vascularization. It is understood that free grafts initially survive by plasmatic diffusion and are subsequently vascularized as capillaries and arterioles form a vascular network providing the permanent circulation for the graft. When a recipient site is partially avascular (e.g., a denuded root surface, an exposed implant abutment, or an area recently reconstructed with a block bone graft), the dissection should be extended to provide a peripheral source of circulation to support the free graft over the avascular or poorly vascularized areas. Although pedicle grafts and flaps maintain their blood supply, it is also good surgical practice to prepare a recipient site that can contribute circulation to ensure optimal results in the event of a reduction of circulation to a portion (most commonly, the margin) of the pedicle graft or flap.

The second principle of oral soft tissue grafting is that the recipient site must provide a means for rigid immobilization of the graft tissue. Initial graft survival requires that the graft be immobilized and intimately adapted to the recipient site. Mobility of the graft during initial healing can interfere with its early nourishment through plasmatic diffusion or can disrupt the newly forming circulatory supply to the graft, resulting in excessive shrinkage or sloughing of the graft.

The third principle is that adequate hemostasis must be obtained at the recipient site. Active hemorrhage at the site prevents the intimate adaptation of the graft to the recipient site. Hemorrhage also interferes with the maintenance of the thin layer of fibrin between the graft and the recipient site, which serves to physically attach the graft to the recipient site and provides for the plasmatic diffusion that initially nourishes the graft before its vascularization. Preparation of a recipient site with a uniform surface enhances the intimate adaptation with the graft. The periosteum is generally considered to be an excellent recipient site for oral soft tissue grafts because it fulfills all of the requirements discussed previously. In addition, decorticated alveolar bone can support and nourish a free soft tissue graft, although immobilizing the graft at the site is more troublesome.

The fourth principle of oral soft tissue grafting involves the size and thickness of the donor tissue. The donor tissue must be large enough to facilitate immobilization at the recipient site and to take advantage of peripheral circulation when root or abutment coverage is the goal. The graft also must be large enough and thick enough to achieve the desired volume augmentation after secondary contraction has occurred. In addition, the donor tissue should be harvested to

ensure a uniform graft surface that facilitates intimate adaptation to the recipient site. Thicker grafts (>1.25 mm) are especially useful for root and abutment coverage when graft healing over the central portion of the avascular surface is characterized by necrosis. The necrotic graft is gradually overtaken by granulation tissue from the periphery and ultimately forms a scar. Thicker grafts are better able to maintain their physical integrity during this process, which can take as long as 4 to 6 weeks. In summary, harvesting a graft that is too small or too thin should be avoided by evaluating the donor site before surgery and by applying the foregoing principles during recipient- and donor-site surgery.

Although failure to adhere to these surgical principles may not result in the loss of the soft tissue graft, increased complications such as inadequate volume yield, graft sloughing, wound breakdown, infection, and patient discomfort can be expected.

EPITHELIALIZED PALATAL GRAFT TECHNIQUE FOR DENTAL IMPLANTS

General Considerations

The use of an epithelialized palatal graft for the treatment of a mucogingival defect has enjoyed a long history of predictable success.[4–6] This versatile technique can be used not only to increase the dimensions of attached tissue around the natural dentition and dental implants but also as a predictable method for covering denuded root or abutment surfaces. Although the term *free gingival graft* is a misnomer, it is commonly used to describe the transfer of epithelialized tissue harvested from the palate. When the contemporary surgical technique is used as described later, thick split-thickness grafts (>1.25 mm) or full-thickness grafts are preferred around both natural dentition and dental implants.

Contemporary Surgical Technique

The surgical technique for gingival grafting around dental implants is essentially the same as the technique used around natural dentition.[3–7] When gingival grafting is performed after implant abutment connection or delivery of the final restoration, a horizontal incision is made through the interimplant papilla coronal to the desired final tissue position. This facilitates abutment coverage with the gingival graft. When gingival grafting is performed at second-stage surgery or simultaneously with nonsubmerged implant placement, the horizontal incision is made at the mucogingival junction, and any existing gingival tissues are repositioned to the lingual or palatal aspect of the implants (Figure 12-10A). This step is extremely important when implants are placed in the mandible because subsequent lingual soft tissue defects in this area are difficult to correct. A split-thickness dissection is then carried apically to create a uniform periosteal site. In the edentulous mandible, care must be taken to avoid damage to the mental nerve with the vertical releasing incisions that typically outline the mesial and distal extents of the recipient site in the dentate patient. Instead, in these instances, a midline vertical releasing incision and sharp dissection are used to create an adequate recipient site (>5 mm apicocoronal dimension) with a half-moon shape, as shown in Figure 12-10B. Subsequently, the mucosal flaps are excised and residual elastic or muscular tissue is removed with tissue scissors or nippers. When working in a severely atrophic mandible, the mucosal flaps are preserved and sutured to the periosteum at the base of the dissection. The technique for graft immobilization is the same regardless of whether gingival grafting is performed around natural dentition, at second-stage surgery for submerged implants, or at the time of nonsubmerged implant placement. The graft is sutured to each papilla or interimplant area coronally and then to the periosteum peripherally to rigidly immobilize the graft at the recipient site (Figures 12-11 and 12-12; see also Figure 12-10C). The following graft immobilization pressure is

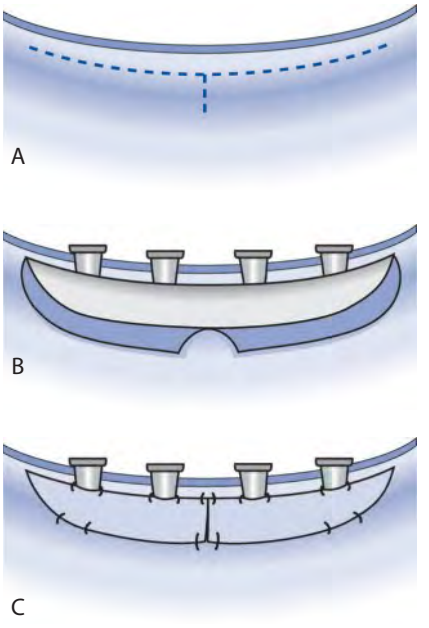


FIGURE 12-10. Surgical technique for gingival grafting simultaneous with abutment connection or nonsubmerged implant placement. **A,** A full-thickness horizontal incision is made at the mucogingival junction, and a partial-thickness vertical releasing incision is made at the midline. **B,** Full-thickness elevation of the flap lingually exposes the ridge crest and allows repositioning of the keratinized tissues lingually for abutment connection or nonsubmerged implant placement. **C,** Split-thickness dissection on the buccal aspect of the alveolar ridge provides a recipient site for rigid immobilization of the donor graft, which is adapted around the emerging implant structures and secured to the lingual tissues and to the periosteum peripherally. The dissection is limited distally to avoid unwanted injury to the mental nerve. (**A–C,** Adapted from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

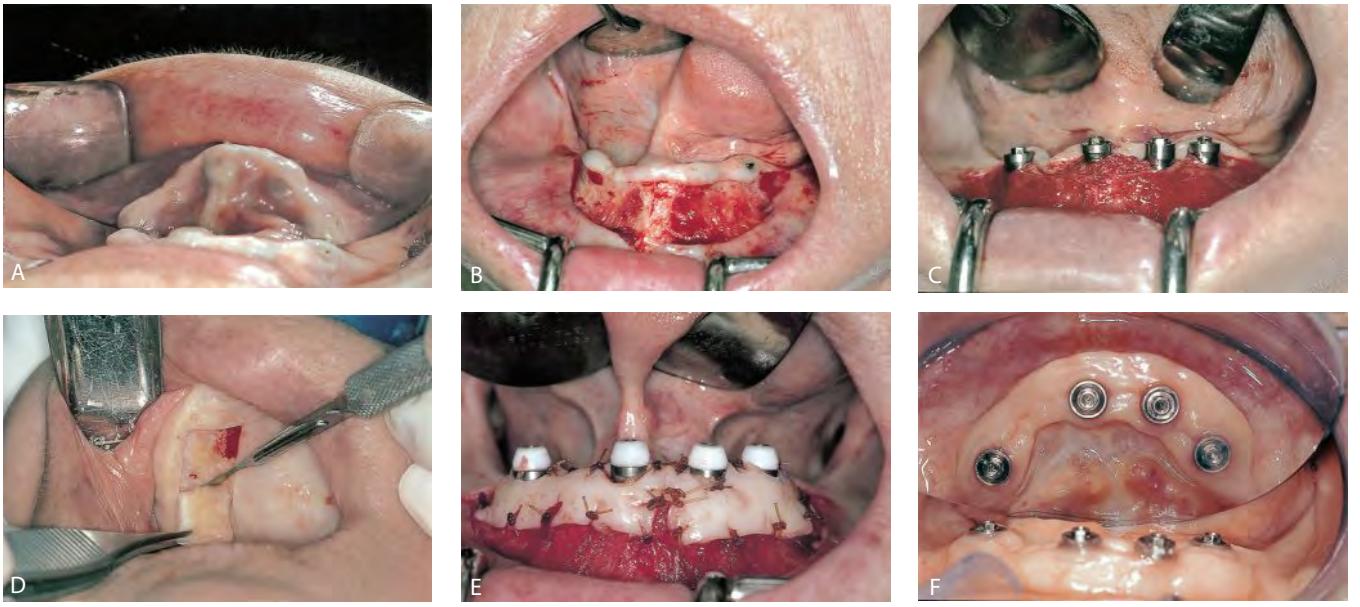


FIGURE 12-11. **A**, Preoperative view of four submerged implants ready for abutment connection. The amount of attached tissue is inadequate to ensure a stable peri-implant soft tissue environment. **B,** Split-thickness dissection is performed to create a uniform periosteal recipient site. **C,** Full-thickness elevation of the attached tissues exposes the implants for abutment connection; the existing keratinized tissue has been repositioned to the lingual aspect of the emerging abutments. **D,** A palatal mucosal graft (gingival graft) is harvested from each side of the palate. **E,** The grafts have been contoured for precise adaptation around the abutments and secured to the lingual tissues and periosteum peripherally. **F,** This 2-month postoperative view demonstrates a tremendous volume yield from the gingival grafting procedure. A stable peri-implant soft tissue environment has been obtained. (**A–F,** Reproduced with permission from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

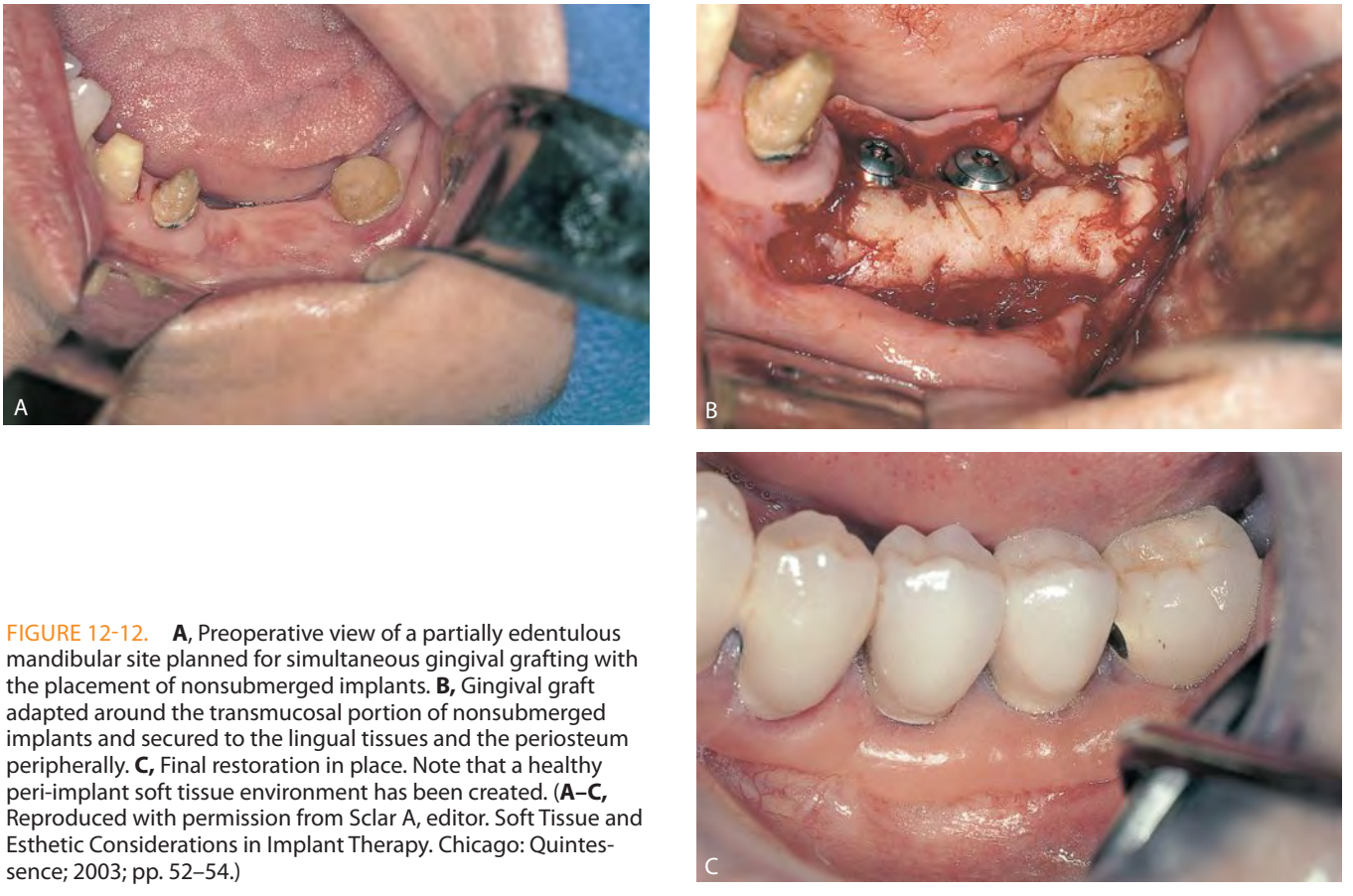


FIGURE 12-12. **A**, Preoperative view of a partially edentulous mandibular site planned for simultaneous gingival grafting with the placement of nonsubmerged implants. **B,** Gingival graft adapted around the transmucosal portion of nonsubmerged implants and secured to the lingual tissues and the periosteum peripherally. **C,** Final restoration in place. Note that a healthy peri-implant soft tissue environment has been created. (**A–C,** Reproduced with permission from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

applied with a moistened saline gauze for 10 minutes. Although a periodontal dressing is not necessary for the recipient site, a protective dressing for the donor site is recommended.

Gingival grafting is indicated before implant placement in the severely atrophic maxilla or mandible that is less than 10 mm in height and has less than 3 mm of attached tissue. In this clinical situation, the surgeon should avoid significant dissection of the palatal or lingual tissues. Instead, a large recipient bed is created on the buccal aspect of the site, extending far enough apically from the midcrest to re-create the buccal vestibular fold. The graft is then harvested and rigidly immobilized with sutures placed approximately 5 mm apart to avoid unnecessary trauma and hematoma formation at the periphery. During subsequent implant surgery, a 3-mm or greater portion of the mature grafted tissue is repositioned lingually, providing good-quality gingival tissue for wound closure over submerged implants and circumferential adaptation of attached tissue around emerging implant abutments or nonsubmerged implants.

SUBEPITHELIAL CONNECTIVE TISSUE GRAFTING FOR DENTAL IMPLANTS

General Considerations

The subepithelial connective tissue graft is an extremely versatile procedure that can be used to enhance soft tissue contours around the natural dentition and dental implants (Figures 12-13 to 12-15). The procedure combines the use of a free soft tissue autograft harvested from the palate that is interposed beneath a partial-thickness pedicle flap at the recipient site (i.e., open approach). Alternatively, the graft

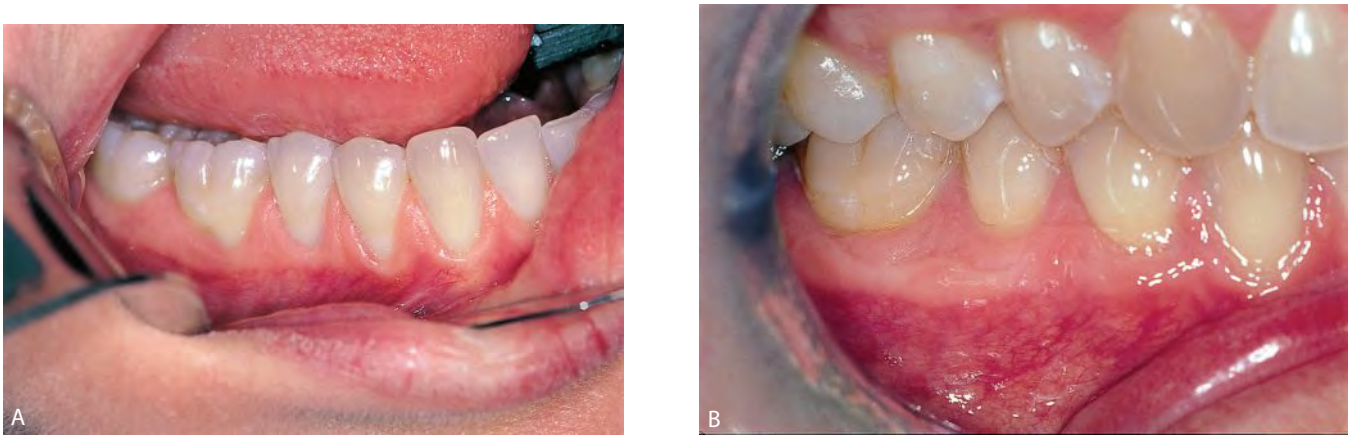


FIGURE 12-13. **A** and **B,** Progressive soft tissue recession involving the mandibular bicuspids and a first molar tooth was successfully corrected with a root coverage procedure using a subepithelial connective tissue graft. (**A** and **B,** Reproduced with permission from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)



FIGURE 12-14. **A,** The progressive soft tissue recession around this lateral incisor implant restoration jeopardized its long-term success. **B,** A subepithelial connective tissue graft was performed via a closed pouch recipient site, resulting in the restoration of soft tissue aesthetics and stability for this patient with a thin scalloped periodontium. Prophylactic soft tissue grafting would have prevented the recession from occurring and is indicated when intracrevicular restorations are planned for patients who present with thin periodontal tissues. (**A** and **B,** Reproduced with permission from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)



FIGURE 12-15. **A,** Preoperative view of central incisor implant site with a small-volume soft tissue aesthetic ridge defect. **B,** An open flap approach involving full-thickness dissection at the ridge crest and partial-thickness dissection on the buccal aspect of the alveolar ridge was used for the implant placement and synchronous subepithelial connective tissue grafting. Coronal advancement of the cover flap enabled further soft tissue volume enhancement via submersion of the one-piece nonsubmerged implant, thus expanding the "soft tissue envelope." **C,** After conservative exposure and insertion of a custom abutment and provisional restoration, the soft tissues were allowed to stabilize before the delivery of the final restoration, which demonstrates pleasing soft tissue aesthetics. (**A–C,** Reproduced with permission from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

can be secured in a split-thickness pouch prepared at the recipient site (i.e., closed approach). The graft is harvested internally from the palate, resulting in a partial-thickness donor-site pouch that allows for primary closure and, thus, a more comfortable palatal wound. Because the graft is positioned between the periosteum and a partial-thickness cover flap or pouch at the recipient site, it enjoys the advantage of a dual blood supply to support graft revascularization. Because of the abundant blood supply available for healing, the connective tissue graft is less technique-sensitive, easier to perform, and more predictable than the gingival graft. The connective tissue graft also results in superior color matching and aesthetic blending at the recipient site. The subepithelial connective tissue graft can be used during initial implant-site development before implant placement or simultaneous with submerged implant placement for the correction of small-volume soft tissue aesthetic ridge defects. Similarly, the connective tissue graft can be performed simultaneously with an abutment connection or nonsubmerged implant placement to reconstruct these small-volume soft tissue defects or for the correction of soft tissue recession defects that develop in the recall period. Finally, whenever a large-volume soft tissue aesthetic ridge defect is present, a series of connective tissue grafts is usually required for reconstruction of these aesthetic ridge defects before implant placement.[3]

Surgical Technique: Donor-Site Surgery

The technique for harvesting subepithelial connective tissue grafts from the premolar region of the palate has two variations: the *single-incision approach* and the *dual-incision approach*.[7,8] In either case, the donor-site surgery begins with a full-thickness curvilinear incision made through the palatal tissues approximately 2 to 3 mm apical to the gingival margin of the premolars (Figure 12-16A). This incision can be made perpendicular to the surface of the palatal tissue or it can be slightly beveled. When it is made perpendicular to the palatal tissues, the thickness of the coronal portion of the graft is maximized; however, this usually prevents passive primary closure. In contrast, beveling the first incision limits the thickness of the coronal portion of the graft but, in many cases, enables a passive primary closure.

When using the dual-incision approach, a partial-thickness curvilinear incision is made approximately 2 mm apical to the first incision to complete an ellipse (see Figure 12-16B). This incision defines the thickness of the subepithelial connective tissue graft to be harvested. The incision should be approximately 1 mm deep to ensure adequate thickness of the remaining cover tissue and to minimize the incidence of sloughing at the donor site. The scalpel is then oriented parallel to the surface of the palatal tissue, and sharp dissection is used to create a rectangular pouch. The apical extent of the dissection is determined by the height of the palate. The mesiodistal extent of the dissection is determined by the length of the first and second incisions, which in turn, are determined by the overall size of the palate and the width of the premolars. The scalpel blade is then used to complete the outline of the donor connective tissue graft with incisions that pass through the underlying connective tissue and periosteum just short of the mesial and distal extent of the pocket. Unnecessary trauma to the overlying palatal tissues is thus avoided when the scalpel is turned perpendicular to the surface of the donor tissue. A Buser periosteal elevator and membrane-placement instrument are then used to carefully begin subperiosteal elevation of donor tissue at the coronal aspect of the dissection. Once the coronal aspect of the graft has been elevated, it is carefully supported with tissue forceps and the subperiosteal elevation is extended to the apical portion of the pouch. Next, gentle traction is placed on the elevated tissue with forceps, and a horizontal incision is made through the apical aspect of the donor tissue from within the pouch. The harvested tissue, which contains epithelium, connective tissue, and periosteum, is then transferred with tissue forceps to the recipient site or temporarily placed on sterile gauze moistened with saline. If the graft is submerged under the recipient's site flap, curved Iris tissue

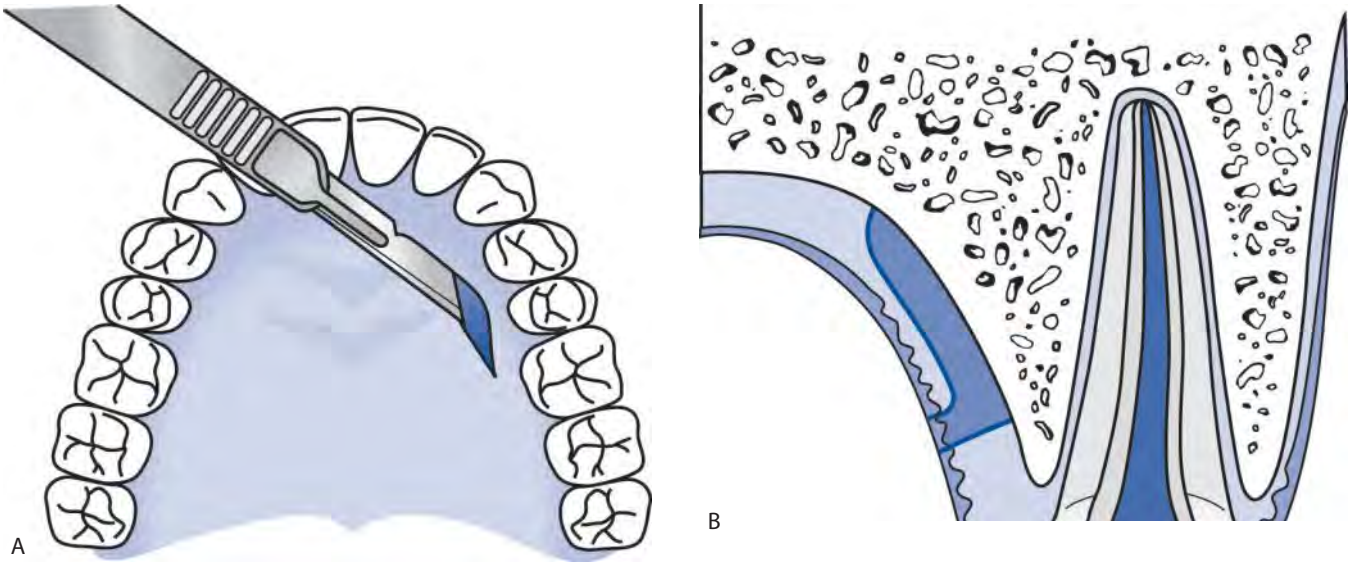


FIGURE 12-16. Subepithelial connective tissue grafting donor-site surgery via a dual-incision approach. **A,** The occlusal view demonstrates the location and orientation of the full-thickness and partial-thickness incisions. **B,** The cross-sectional view demonstrates the pathways of the incisions for donor-site harvest via the dual-incision approach. The *shaded area* represents the resultant donor graft, consisting of both connective tissue and periosteum. (**A** and **B,** Adapted from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

scissors should be used to remove the epithelial tissue. Hemostasis is then obtained at the donor site by placing an absorbable collagen dressing, such as CollaPlug, and applying pressure with saline-moistened gauze. The donor site is closed using interrupted 4-0 chromic gut sutures on a P3 needle passed through the interproximal areas.

The single-incision technique differs in that only one incision is used to establish access to both the subperiosteal and the subepithelial planes of dissection. This approach begins with a full-thickness curvilinear incision, as described previously. Next, the scalpel is reoriented within the incision until it is parallel to the surface of the palatal tissue. Subepithelial dissection that parallels the external surface of the palatal tissue is accomplished to create a rectangular pouch. After making the first incision, the surgeon may find it useful to perform subperiosteal elevation coronally. This improves visualization of available soft tissue thickness (Figure 12-17), thereby aiding the surgeon in establishing the appropriate subepithelial plane of dissection. The remainder of the surgical procedure is identical to the procedure described previously for the dual-incision technique.

The advantage of the dual-incision approach is that it is easier to perform. Because the thickness of the donor tissue is defined by the second incision, the result is the harvesting of a graft of uniform thickness. The disadvantage of this approach is that primary closure is seldom possible, and therefore, the palatal wound can be uncomfortable. Nevertheless, this approach is usually recommended for the novice surgeon. Although harvesting a donor graft of uniform thickness is technically more challenging when the single-incision approach is used, primary closure of the palatal wound results in greater patient comfort. As a result, most experienced surgeons prefer this approach.

Surgical Technique: Recipient-Site Surgery

Preparation of the recipient site involves either the elevation of a split-thickness flap through supraperiosteal dissection (open technique) or a supraperiosteal dissection, which avoids vertical releasing incisions to create an envelope or pouch (closed technique). The decision of which technique to use when grafting around a natural tooth or an implant restoration depends on several factors. The open technique allows direct visualization during dissection, which ensures the preparation of a uniform recipient site. This approach also allows for significant coronal advancement when vertical soft tissue augmentation is needed over an exposed root or abutment surface. The vertical releasing incisions used in the open technique sacrifice some circulation. However, the use of a curvilinear beveled flap with tension-releasing cutback incisions avoids embarrassment of circulation to the flap margin and allows for greater coronal flap advancement than do traditional trapezoidal flaps that require periosteal releasing incisions to allow even limited coronal advancement.

In contrast, the closed technique avoids the need for vertical incisions, thus preserving the blood supply to the site and optimizing aesthetic results. However, as a "blind" technique, it can be technically more demanding. Also, because it does not allow for significant coronal advancement of the cover



FIGURE 12-17. Subepithelial connective tissue grafting donor-site surgery via the single-incision approach. The cross-sectional view demonstrates the pathways of the incision and the dissection for the donor-site harvest. The *shaded area* represents the resultant donor graft consisting of both connective tissue and periosteum. (Adapted from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

flap, this technique is of limited use when significant vertical soft tissue augmentation is needed, and it is contraindicated whenever vestibular depth limits the preparation of an adequately sized recipient site. In general, the closed recipient site is preferred when the abutment or root exposure is less than 4 mm apicocoronally or when there is a significant risk of sloughing of the cover flap because of poor vascularity at the site.

Closed Technique

The technique for closed recipient-site preparation is the same whether it is performed around a natural tooth or an implant restoration. A horizontal incision is extended to the mesial and distal aspects of the soft tissue defect just coronal to the level of the root or abutment coverage desired (Figure 12-18). Using a no. 15C scalpel, the surgeon makes this incision at a right angle to the epithelium at a depth of approximately 1 mm. The horizontal incisions not only mark the graft's final coronal position but also facilitate the pouch dissection and subsequent immobilization of the graft.

Next, the scalpel is oriented parallel to the tissue surface, and the horizontal incisions are extended into the sulcus to create the entrance to the recipient site. The split-thickness dissection is extended apically beyond the mucogingival junction at the mesial and distal aspects of the site before crossing the midline. To ensure that the recipient site can contribute adequate peripheral blood supply to sustain the graft, the dissection must extend well beyond the width of the soft tissue defect being corrected. As a general rule, the width of the recipient site should be three times that of the exposed root or abutment, which can be accomplished by extending the defect mesially and distally. The surgeon must take care to avoid perforating or tearing the overlying tissues with the scalpel; a meticulous technique is required to ensure a uniform recipient-site surface.

The blunt end of a membrane-placement instrument is then used to probe the resultant pouch and confirm that the dissection is complete. Occasionally, strands or webs of tissue extending from the overlying tissues to the periosteum are detected in the apical extent of the dissection. If not released with sharp dissection, these tissue strands prevent proper positioning and passive adaptation of the connective tissue graft within the pouch. A periodontal probe is then used to measure the dimensions of the recipient pouch and to guide the surgeon in the donor harvest, and pressure is applied with saline-moistened gauze to obtain hemostasis.

Once the recipient site has been prepared and the donor tissue has been harvested, the donor tissue should be intimately adapted and rigidly immobilized at the recipient site. When a closed recipient site is used, the dimensions of the donor connective tissue should closely match those of the recipient-site pouch. Curved Iris tissue scissors are used to size the graft before securing it in the pouch. The graft should always be oriented so that the periosteal side faces down at the recipient site. A 4-0 chromic suture on a P3 or FS2 needle is used to place a horizontal mattress suture that enters the apical portion of the recipient pouch, engages the graft, and exits the pouch apically. This suture is used to gently "pull" the graft into the recipient pouch and secure the graft apically, thereby resisting subsequent coronal displacement. First, the suture needle is passed through the vestibular mucosa into the recipient pouch and retrieved with forceps. The suture needle is then passed through the connective tissue side of the graft and back through the periosteal side of the graft. Next, the membrane-placement instrument is used to identify the apical extent of the recipient site, and the suture needle is passed back through the mucosal tissue to exit the pouch several millimeters lateral of where it entered. A fine hemostat is clamped across equal lengths (~7.5 cm) of the suture material, and suture scissors are used to cut away the remaining suture and needle.

Subsequently, the surgeon uses the clamped suture material to slowly pull the graft into the recipient pouch, taking care not to tear the overlying tissue. The paddle end of the membrane-placement instrument is used like a shoehorn to guide the graft into the entrance of the recipient pouch. The flat portion of the instrument is moistened with saline and placed between the graft and the overlying tissue as the

graft is gently pulled into the pouch. This technique prevents bunching of the graft at the entrance of the recipient pouch as well as excessive stretching of, and damage to, the overlying tissues. The spiked end of the membrane-placement instrument is then used to gently "push" the graft further into the pouch entrance, while the clamped suture material is used to "pull" the graft apically. A triple tie secures the graft in the pouch.

The graft is secured coronally, either with interrupted sutures that pass through the graft and interproximal tissues (see Figure 12-18) or with a sling suture. Interrupted sutures in the papillary area are then used to secure the cover tissue pouch. Additional sutures can be carefully placed to approximate the coronal margins of the pouch in an effort to cover more of the exposed graft. Nevertheless, because significant coronal advancement of the overlying tissues is not possible, a portion of the graft will remain uncovered. Whenever possible, it is recommended that two thirds or more of the graft be secured within the recipient-site pouch. Gentle pressure is applied over the graft site with saline-moistened gauze for a minimum of 10 minutes.

Open Technique

Again, the technique for open recipient-site preparation is the essentially the same whether it is performed around a natural tooth or an implant restoration or to improve soft tissue contours during implant-site development. This approach is useful for a moderate amount of vertical soft tissue augmentation, making it applicable for abutment coverage procedures and for improving soft tissue contours during implant-site development or when performed over a submerged implant (Figure 12-19). The dissection begins by outlining the recipient site with partial-thickness horizontal and vertical incisions using a no. 15C scalpel blade on a round handle. The horizontal incision, which is performed first, extends mesial and distal to the soft tissue defect at a level just coronal to the final soft tissue position desired after augmentation. Exaggerated curvilinear beveled incisions with tension-releasing cutback incisions are then initiated apically well beyond the mucogingival junction to outline the cover flap. Next, sharp dissection is used to elevate a split-thickness flap. The dissection is initiated coronally with a no. 15C scalpel blade. Flap elevation is continued apically under direct vision with sharp dissection under tension, which is carefully maintained with the use of micro-Adson tissue forceps. The goal is to maximize the thickness of the overlying tissue flap, leaving only a thin layer of immobile periosteum. When coronal advancement of the cover flap is performed, the adjacent papillary areas are deepithelialized with a fresh no. 15C scalpel. This further extends the wound margin, thereby reducing flap retraction and greatly enhancing incision line aesthetics. It also eliminates the possibility that the undersurface of the coronally advanced flap will be coapted over an epithelial surface, which would prevent initial wound healing and could result in dehiscence along the incision. The dimensions of the recipient site are then measured with a periodontal probe, and hemostasis is obtained by applying gentle pressure with saline-moistened gauze.

Once the donor graft has been harvested, it is usually trimmed to be slightly smaller than the open recipient site. This facilitates immobilization of the graft and suturing of the cover flap into position without unwanted engagement of the underlying graft, which can cause graft dislodgment secondary to swelling or retraction of the cover flap. Whether grafting around natural dentition or an implant restoration(s), the graft is first secured coronally with sutures passed through the adjacent papillary areas using a 4-0 chromic gut suture on a P3 needle. Alternatively, sling sutures can be used for this purpose. Next, the graft is secured laterally and apically to the periosteum with additional sutures. The goal is to gently stretch the tissue, thus improving its adaptation to the recipient site.

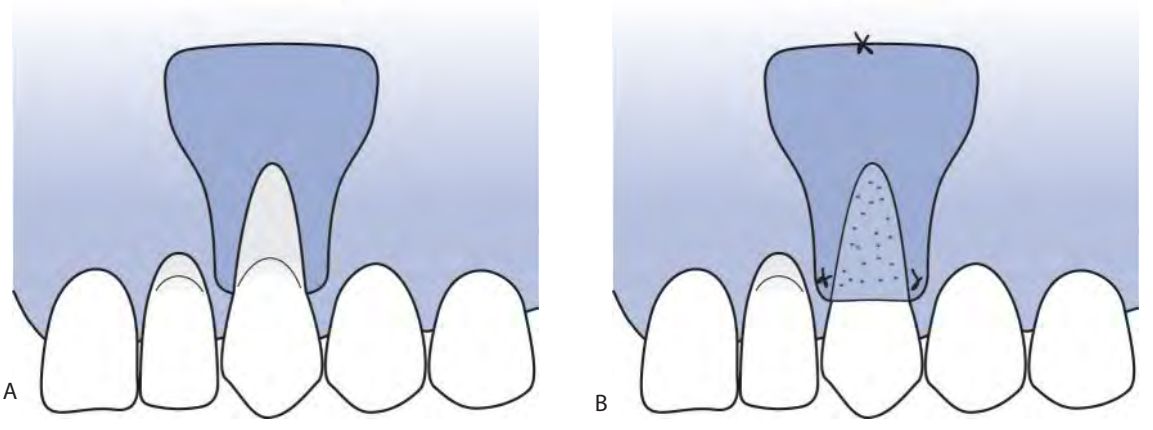


FIGURE 12-18. Closed "pouch" technique for the preparation of a recipient site for a subepithelial connective tissue graft to improve soft tissue contours around a natural tooth or an implant restoration. **A,** Split-thickness dissection *(shaded area)*. **B,** Graft mobilization apically and coronally. (**A** and **B,** Adapted from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

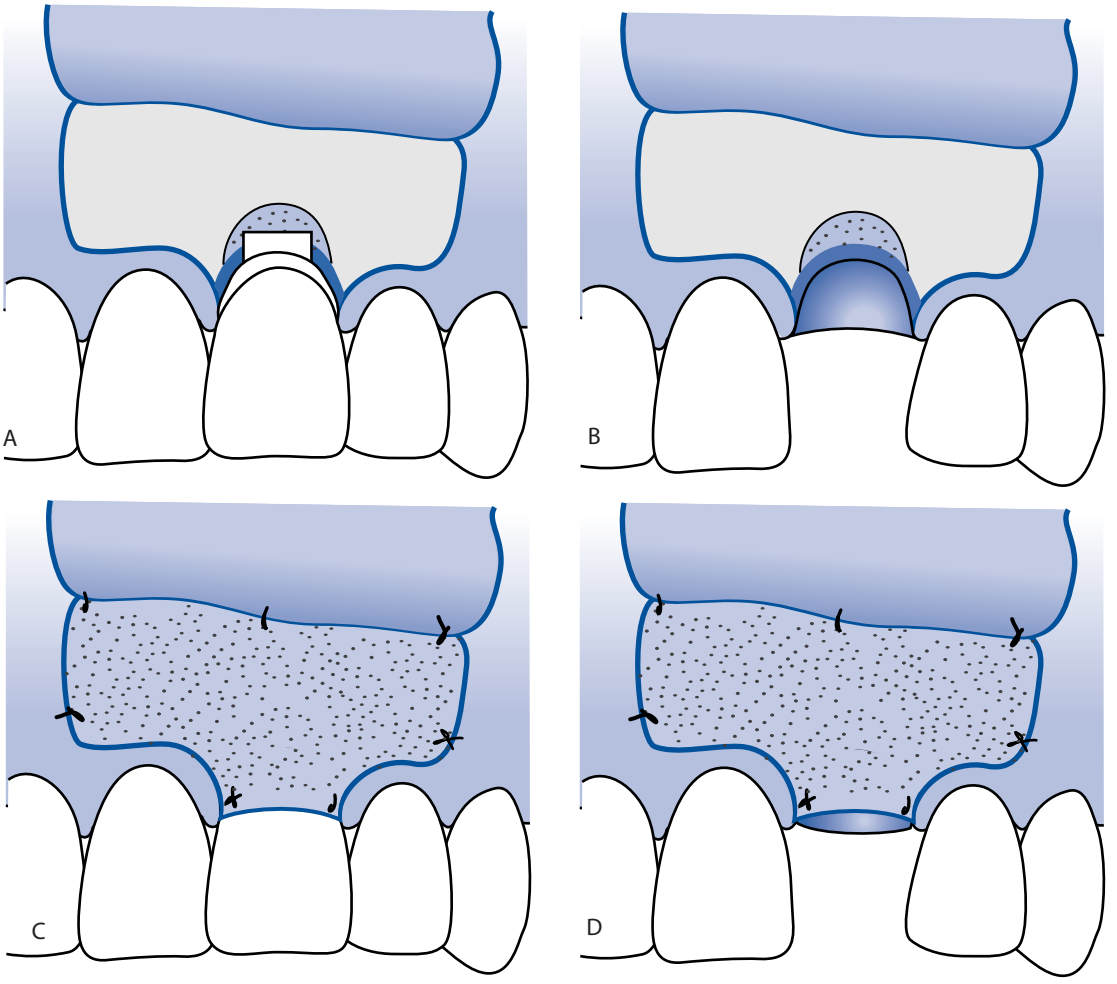


FIGURE 12-19. Open flap technique for the preparation of a recipient site for a subepithelial connective tissue graft to improve soft tissue contours at an implant site. This approach is useful at the time of abutment connection (**A** and **B**) and over a submerged implant (**C** and **D**). (**A–D,** Adapted from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

Next, the cover flap is secured coronally with interrupted sutures passing through the papillae. These sutures should pass through the facial flap and the deepithelialized papillary tissue and then return under the contact points, where they are tied facially. Alternatively, a sling suture can be used. In this case, the suture passes through the flap and the papillary tissue on the first pass; it then passes under the contact points as it returns to the facial aspect, where it is tied. Depending on the thickness of the cover flap tissue, 4-0 or 5-0 chromic gut suture on a P3 needle is used. Next, the cover flap is secured laterally. The use of exaggerated curvilinear beveled incisions to outline the cover flap not only extends the recipient site, providing additional circulation to sustain the graft, but also facilitates immobilization of the graft and closure of the cover flap.

The suture needle should be perpendicular to the beveled incision as it passes through the tissue. It also should be oriented in an apicocoronal direction as it is passed through the flap and adjacent tissue. A single pass is recommended to ensure precise positioning of the cover flap. The attached tissue contained in the flap is first precisely repositioned and secured with sutures placed laterally. The sutures then are placed apical to the mucogingival junction. When performed as part of implant-site development or when grafting over a submerged implant, the recipient site is extended further onto the palatal or lingual surface of the alveolar ridge via split-thickness dissection, and the graft is secured in a similar fashion before closing the cover flaps, as described previously. Moistened saline gauze is used to apply gentle pressure at the site for 10 minutes; a periodontal dressing is not usually needed.

VASCULARIZED INTERPOSITIONAL PERIOSTEAL CONNECTIVE TISSUE FLAP

General Considerations

The VIP-CT flap is an innovative technique that provides for reconstruction of large-volume soft tissue aesthetic ridge defects with a single procedure.[3] In addition, the pedicled

blood supply derived from the connective tissue-periosteal plexus within the flap provides the biologic basis for predictable simultaneous hard and soft tissue grafting procedures during aesthetic implant-site development, even at compromised sites. Additional advantages of the technique include negligible postoperative soft tissue shrinkage; enhanced results realized from hard tissue grafting procedures owing to the supplemental source of circulation and the contribution to phase-two bone graft healing provided by the mesenchymal cells transferred with the flap; and when hard and soft tissue site-development procedures are necessary, reduced treatment time and patient inconvenience.

Although the amount of horizontal soft tissue augmentation obtained with the VIP-CT flap is consistently greater than that obtained with free soft tissue grafting techniques, the amount of vertical soft tissue augmentation typically obtained exceeds that obtainable even when several free soft tissue grafts are performed, which has allowed the re-creation of positive gingival architecture, even in situations in which previous hard and soft tissue site-development techniques have fallen short. This technique has also proved useful in the treatment of compromised sites in which existing soft tissues were poor in quality and severely scarred, rendering them inadequate to support required hard tissue implant-site development (Figure 12-20). It is a predictable means of resubmerging an implant in the anterior area when an unexpected soft tissue dehiscence compromises the final aesthetic result.

The volume of tissue transfer routinely obtained with the VIP-CT flap has also allowed the camouflaging of small-volume combination hard and soft tissue ridge defects as well as the correction of large-volume soft tissue defects simultaneously with implant placement (Figures 12-21 and 12-22), as previously discussed.

Of greatest significance, this technique provides the implant surgeon with a proven technique for predictable simultaneous hard and soft tissue aesthetic implant-site development at compromised anterior sites with large-volume combination aesthetic ridge defects (Figure 12-23). These enhanced results are directly related to maintenance of intact circulation to the flap and decreased postsurgical contraction.

Surgical Technique

As in the previously described techniques, the surgeon begins by outlining and preparing the recipient site and then proceeds to donor-site preparation. An exaggerated curvilinear beveled flap design is used at the recipient site. Abbreviated vertical releasing incisions are extended over the alveolar crest onto the palatal surface at both the mesial and the distal aspects of the recipient site. This allows full exposure of the ridge crest for hard tissue grafting or implant placement. The palatal incision at the distal aspect of the recipient site parallels the gingival margin on the oral aspect of the adjacent tooth (Figure 12-24A).

After recipient-site preparation, donor-site preparation begins by extending this incision horizontally to the distal aspect of the second premolar. To facilitate subsequent closure of the donor site, the orientation of this incision should be slightly beveled and follow a path approximately 2 mm apical to the free gingival margins of the canine and premolar teeth (see Figure 12-24A). Sharp dissection is then used internally to create a split-thickness palatal flap in the premolar area. The subepithelial dissection is carried mesially toward the distal aspect of the canine. The surgeon should be careful to maintain an adequate thickness of the palatal cover flap to avoid sloughing. In most cases, the dissection has to be deeper in the area of the palatal rugae to avoid perforating the cover flap. Next, a vertical incision is made internally through the connective tissue and periosteum at the distal extent of the subepithelial dissection, as far apically as is possible without damaging the greater palatine neurovascular structures. This incision defines the margin of the flap. Using a Buser periosteal elevator and a membrane-placement instrument, the surgeon then carefully elevates the resultant periosteal-connective tissue layer, beginning in the second premolar area and working toward the anterior extent of the



FIGURE 12-20. Use of the vascularized interpositional periosteal connective tissue (VIP-CT) flap to restore soft tissue volume and health at a severely compromised site. **A,** Preoperative view of a severely compromised lateral incisor site after a failed bone graft that resulted in the loss of col and papilla on the adjacent central incisor and severely scarred and inelastic soft tissue cover at the site. **B,** A VIP-CT flap was performed to provide sufficient volume of good-quality tissue to support the subsequent bone graft. **C,** The final result after subsequent bone grafting demonstrates the complete reconstruction of natural ridge contours and the successful restoration of the adjacent col and papilla, a remarkable result that is not always obtainable even with the VIP-CT flap. (**A–C,** Reproduced with permission from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

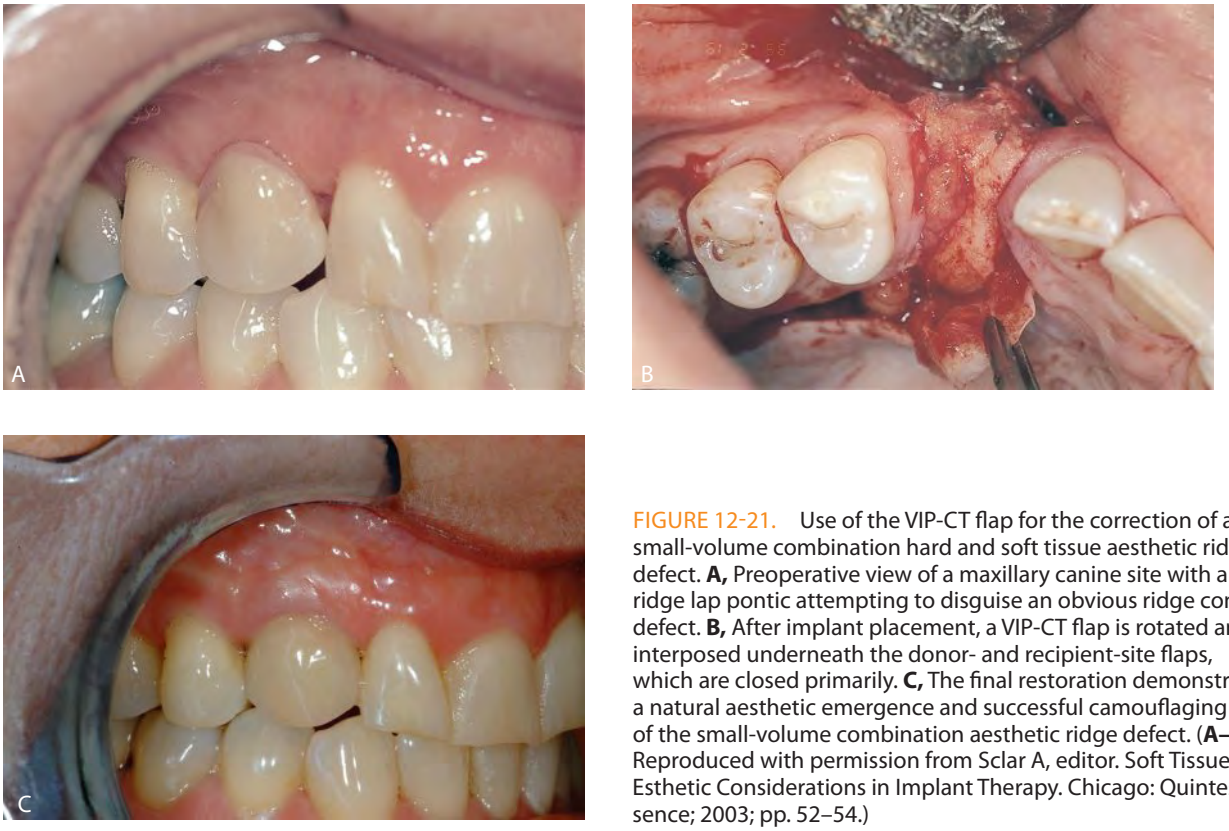


FIGURE 12-21. Use of the VIP-CT flap for the correction of a small-volume combination hard and soft tissue aesthetic ridge defect. **A,** Preoperative view of a maxillary canine site with a ridge lap pontic attempting to disguise an obvious ridge contour defect. **B,** After implant placement, a VIP-CT flap is rotated and interposed underneath the donor- and recipient-site flaps, which are closed primarily. **C,** The final restoration demonstrates a natural aesthetic emergence and successful camouflaging of the small-volume combination aesthetic ridge defect. (**A–C,** Reproduced with permission from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

dissection. Usually, this careful subperiosteal dissection yields intact periosteum on the undersurface of the pedicle, which aids in subsequent rigid immobilization of the graft. Furthermore, intact periosteum potentially provides osteoblastic activity if applied over a bone graft when simultaneous hard and soft tissue site development is performed. A second incision is then initiated under tension internally at the apical extent of the previous vertical incision and extended horizontally anterior to the distal aspect of the canine. The outline of the periosteal-connective tissue pedicle is now complete. Limiting the incisions to the anatomic landmarks given ensures that the margin of the pedicle is safely harvested from the palatal area, where the thickest amount of connective tissue is available, without risk of damage to adjacent neurovas-

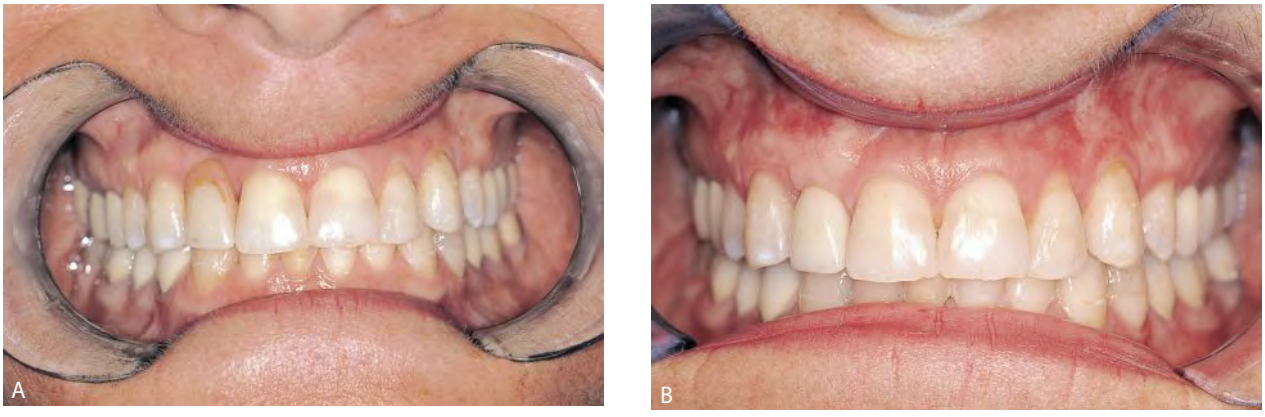


FIGURE 12-22. Use of the VIP-CT flap for the correction of a large-volume soft tissue aesthetic ridge defect simultaneous with a submerged implant placement. **A,** Preoperative view of a lateral incisor implant site with removable partial denture with a tissue-colored flange used to disguise the large-volume soft tissue defect at the site. **B,** The final restoration demonstrates a natural emergence and soft tissue aesthetics after the implant placement and synchronous use of the VIP-CT flap. Typically, several free soft tissue grafts are necessary to restore a large-volume soft tissue defect. (**A** and **B,** Reproduced with permission from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

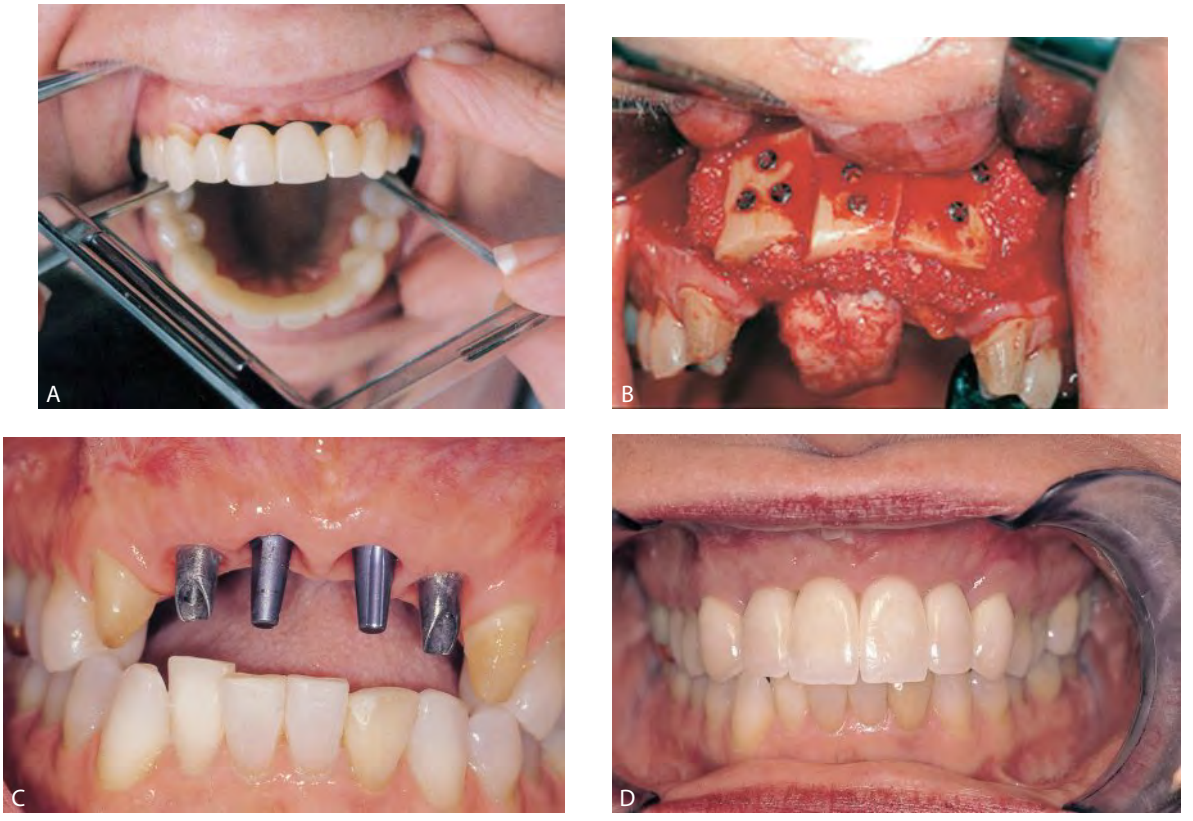


FIGURE 12-23. Simultaneous reconstruction of a large-volume combination hard and soft tissue aesthetic ridge defect for the replacement of four maxillary incisors. **A,** Preoperative view of the compromised site secondary to multiple interventions leading to tooth loss and a previously failed attempt at bone graft reconstruction. **B,** Intraoperative view after rigid fixation of corticocancellous block bone grafts and condensation of particulate bone graft material. The VIP-CT flaps have been prepared and are ready for rotation over the block bone graft, thereby improving the volume of the soft tissue in the areas critical for prosthetic emergence and supplementing the circulation of the soft tissue cover for enhanced bone graft healing. **C,** Nonsubmerged central and lateral incisor implants were placed after 4 months of healing with customized tooth-form healing abutments. The final restorative abutments, pictured in this clinical photograph, were delivered after an additional 4 months. Note that use of the VIP-CT flap simultaneous with the block bone grafting procedure resulted in a significant vertical soft tissue augmentation and the restoration of the natural soft tissue architecture at the site. **D,** The final restorations are harmonious in appearance, and pleasing gingival aesthetics are evident. (**A–D,** Reproduced with permission from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

cular structures. Next, a Buser periosteal elevator is used to carefully elevate the periosteal-connective tissue pedicle and undermine the full thickness of the palatal mucosa and periosteum at the base of the pedicle, just beyond the midline of the palate (see Figure 12-24B). This subperiosteal elevation or undermining begins at the distal aspect of the dissection in the area of the second premolar and is carried anteriorly toward, but short of, the incisive foramen so as to avoid compromise to the neurovascular structures in this area. Doing so provides additional elasticity at the base of the pedicle to allow passive rotation to the recipient site without the need for a tension-releasing cutback incision. Essentially, the two distinct planes of dissection performed define the interpositional periosteal-connective tissue pedicle flap without disrupting its circulation. The subepithelial plane is superficial to the greater palatine vessels but deep enough to avoid sloughing of the palatal cover flap. The subperiosteal plane is deep to the greater palatine vessels and is limited anteriorly and posteriorly to avoid damage to the neurovascular structures as they course through the palate.

Tension-releasing cutback incisions extended into the base of the pedicle flap are rarely necessary when subperiosteal undermining is performed. When unavoidable, these relaxing incisions are initiated at the pivot point of flap rotation along the line of greatest tension. Although the line of greatest tension is the radius of the rotation arc created by the apical horizontal incision, the pivot point may not coincide with the termination of that incision. This is because the periosteal undermining causes a favorable displacement of the flap's pivot point and, in most cases, allows for tension-free rotation of the flap into the maxillary anterior area without the need for a tension-releasing cutback incision. Nevertheless, when a tension-releasing cutback incision is necessary despite undermining, the surgeon must be careful to limit the length of the incision to avoid embarrassing the circulation. An intraoperative assessment of the area of great-



FIGURE 12-24. Surgical technique for the VIP-CT flap. **A,** Occlusal view of incisions that outline the donor and recipient sites. Note that the preparation of the recipient site involves deepithelialization of the adjacent col and papillary areas. **B,** After split-thickness recipient-site preparation, deepithelialization of the attached tissue on the buccal aspect of the ridge as well as the adjacent col and papillary areas is performed, and implant placement is completed. Subsequently, the VIP-CT flap is developed via subepithelial and subperiosteal dissections performed within the bicuspid region of the palate. **C,** Subperiosteal undermining is extended to the midline, allowing the flap to passively rotate to the midline, where it is secured to the deepithelialized areas and periosteum at a split-thickness recipient site or over a block bone graft when simultaneous reconstruction is performed. (**A–C,** Adapted from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)

est tension will guide the placement of releasing incisions. Next, the flap is rotated into the recipient site and rigidly immobilized with sutures placed apically and/or laterally (see Figure 12-24C). Alternatively, the flap can be secured directly to a block bone graft using sutures passed through transosseous perforations in the bone graft. An absorbable collagen dressing, such as CollaPlug, is used as an aid to hemostasis and to eliminate dead space in the donor harvest area. Finally, the donor and recipient sites are closed primarily with absorbable sutures, and gentle pressure is applied with saline-moistened gauze for 10 minutes.

ORAL SOFT TISSUE GRAFTING WITH ACELLULAR DERMAL MATRIX

General Considerations

Acellular dermal matrix (AlloDerm) has been used as an alternative to harvesting autogenous epithelialized palatal grafts and subepithelial connective tissue grafts in periodontal surgery since 1996. AlloDerm grafts are composed of freeze-dried allograft skin processed to remove all immunogenic cellular components (epidermis and dermal cells), leaving a useful acellular dermal matrix for soft tissue augmentation. AlloDerm can be used to increase the width of attached tissue around the natural dentition and implants, obtain root or abutment coverage, and correct small-volume soft tissue ridge defects. The advantages of using AlloDerm include the elimination of donor-site surgery for greater patient comfort, unlimited tissue supply, excellent handling characteristics, and decreased surgical time. Disadvantages include greater secondary shrinkage and slower healing at the recipient sites when used as an onlay graft or when complete coverage of an interpositional AlloDerm graft is not obtainable. Predictable root or abutment coverage requires coverage of the AlloDerm graft with good-quality cover flap tissue.

Surgical Technique

The surgical technique for using AlloDerm is essentially the same as that described previously for the gingival and subepithelial connective tissue grafts. The AlloDerm graft must be rehydrated for 10 minutes before use. Two distinct sides of the AlloDerm graft are identified by applying the patient's blood to each surface and rinsing with sterile saline. The connective tissue side will retain the red coloration, whereas the basement membrane side will appear white. The connective tissue side contains preexisting vascular channels that allow for cellular infiltration and revascularization. When used as an onlay graft to increase the width of attached tissues, the connective tissue side should be oriented toward and intimately adapted to the recipient site (Figure 12-25). When used for root or abutment coverage, the basement membrane side of the graft should be oriented toward the exposed root or abutment (Figure 12-26). The basement membrane side of the AlloDerm graft facilitates epithelial cell migration and attachment. Wherever possible, the author recommends preparing a larger recipient site (6–8 mm apicocoronal dimension) and immobilizing a larger AlloDerm graft than what is used when an autogenous gingival graft is performed. This offsets the additional shrinkage observed with AlloDerm onlay grafts.

Improvement has been observed in the rate of incorporation of AlloDerm onlay and interpositional grafts when platelet-rich plasma (PRP) is incorporated into the surgical protocol.[3] In these instances, the AlloDerm graft is first rehydrated in nonactivated anticoagulated PRP solution before its immobilization at the recipient site. Subsequently,

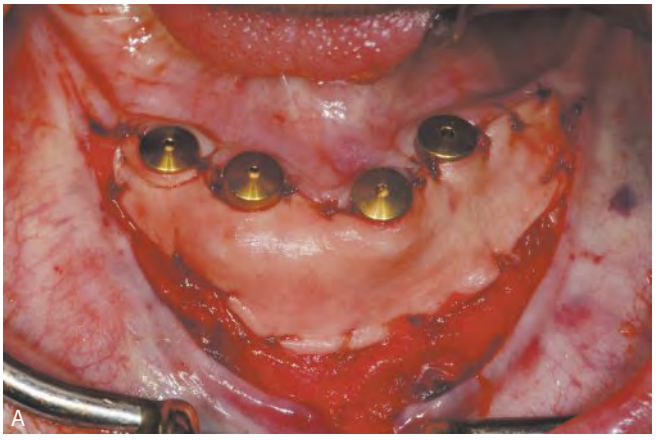




FIGURE 12-25. Use of AlloDerm (a freeze-dried allograft skin processed to remove all immunogenic cellular components [epidermis and dermal cells]) to increase the width of attached tissue around an implant restoration. **A,** Intraoperative view of the use of an AlloDerm graft simultaneous with the placement of four nonsubmerged implants in an edentulous mandible to improve the peri-implant soft tissue environment and to eliminate mobile mucosal tissues in the area while increasing vestibular depth. **B,** The 2-month postoperative view demonstrates a sufficient area of attached nonmobile peri-implant soft tissues to ensure a healthy soft tissue environment and ample access for oral hygiene maintenance. (**A** and **B,** Reproduced with permission from Sclar A, editor. Soft Tissue and Esthetic Considerations in Implant Therapy. Chicago: Quintessence; 2003; pp. 52–54.)





FIGURE 12-26. Use of AlloDerm (a freeze-dried allograft skin processed to remove all immunogenic cellular components [epidermis and dermal cells]) for root- or abutment-coverage procedures. **A,** Preoperative view of generalized progressive periodontal soft tissue recession treated with AlloDerm grafts. **B,** The postoperative view demonstrates successful root coverage at sites amenable to such a result and an increased width of attached tissue at those sites not amenable to complete root coverage.

activated PRP is used topically at the recipient site as a growth factor–enriched wound dressing. Whenever PRP is used with AlloDerm or autogenous soft tissue grafts, care must be taken to avoid the formation of a PRP blood clot between the soft tissue graft and the periosteal recipient site or the cover flap.[3]

CONCLUSION

This chapter provides the implant surgeon with the basic information necessary for successful management of peri-implant soft tissues in the most common clinical scenarios. In addition, it presents principles of oral soft tissue grafting and surgical details of the most commonly used oral soft tissue grafting techniques. However, because limited information concerning the indications, advantages, and expected outcomes of the individual surgical approaches and techniques has been presented, further study by the reader is encouraged.

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13

CHAPTER

Craniofacial Implant Surgery

Douglas P. Sinn, DDS, Edmond Bedrossian, DDS, and Allison Vest, MBBS

Reconstruction of acquired or congenitally absent facial structures such as ears, eyes, the nose, and other structures is a challenging task for the reconstructive surgeon. Often, inadequate soft tissue, cartilaginous, or osseous structure exists for a reconstruction that is both functional and aesthetic and is achieved with a reasonable effort on the part of the surgeon and patient.

The use of external titanium cranial implants for prosthetic reconstruction in the head and neck region was developed owing to pioneering work by Brånemark, Briene, Adell, and others in the late 1960s and early 1970s.[1–5] Since this technology emerged as a reliable reconstruction method for the maxillofacial/oral region, early work began regarding extraoral applications of the titanium osseointegrated implant. Initially, concern regarding long-term stability and recurrent infection was vocalized by many authors. Subsequently, however, work in the late 1970s and early 1980s by Tjellström, Albrektsson, Brånemark, and Lindström revealed that the extraoral application of titanium implants for prosthetic reconstruction, bone-anchored conductive hearing aids, and other applications was a reliable technique.[6–12] Following the initial application of this technology for auricular reconstruction, other reconstructive procedures utilizing osseointegrated retention such as orbital, nasal, and frontal prostheses have been evaluated in the literature.

PROSTHETIC RECONSTRUCTION

Most authors agree that prosthetic reconstruction of the ear results in a cosmetically superior result when compared with autogenous reconstruction. This is not to imply that traditional reconstructive techniques cannot achieve an excellent aesthetic result; however, the complex anatomy of structures such as the ear and nose can be extremely difficult to reconstruct and nearly impossible to replicate with traditional reconstructive surgery. Implant-retained prostheses offer an excellent reconstructive option that provides for excellent symmetry, color, and anatomic detail. Further, prosthetic reconstruction offers a rescue option for unacceptable or failed autogenous grafting procedures.[12–14]

Implant-retained prostheses offer several advantages over more traditional prosthetic techniques. Cranial implants provide secure attachment of the prosthesis, which obviates adhesives, double-sided tape, glasses, or other more traditional fixation methods that may compromise prosthetic stability. They enhance the patient's quality of life via improved self-image, greater activity level owing to superior retention, and ease of prosthesis management. Traditional adhesives have several disadvantages such as discoloration of the prosthesis, skin reactions (especially in radiated areas), and poor performance during activity or perspiration.[12,13,15] Another significant advantage of cranial implantation is that the technique avoids distortion of tissues inherent in traditional surgical reconstruction, which allows for superior tumor surveillance. It has been suggested, despite difficulties with osseointegration in irradiated bone, that cranial implants may have an advantage in the postirradiation patient due to poor-quality soft tissues available for reconstruction.[16]

Several disadvantages to prosthetic reconstruction exist including the necessity of prosthetic/implant maintenance due to normal wear and discoloration and depending on the level of the patient's activity. The prosthesis may be dislodged at inopportune times such as social or athletic events, and some authors have noted that some patients may have adverse psychological effects related to the prosthesis.[12–15]

AUTOGENOUS RECONSTRUCTION

The advantages of using autogenous tissue in head and neck reconstruction follow the generally accepted principles of reconstruction, that is, stable long-term reconstruction with living tissue with an intact blood supply. An inherent advantage with autogenous reconstructions is the potential to fight infection and heal.[14,15] In addition, the cartilaginous

framework may have some growth potential in younger patients.[17] Traditional techniques allow for the reconstruction of partial deformities (preserving local tissue), whereas prosthetic reconstruction is usually reserved for total loss of the structure and may actually require removal of local tissues to facilitate prosthetic rehabilitation. Traditional reconstruction may be a superior option for the poorly compliant patient as well. An added advantage is the elimination of prosthetic support/maintenance that can be a significant expense to the patient.

The primary disadvantage to autogenous reconstruction is that the final result is often less than satisfactory to the surgeon and the patient in terms of aesthetic outcome.[18] In reference to auricular reconstruction, Wilkes and Wolfaardt noted that "the final reconstructed ear is acceptable aesthetically but less likely to exactly match the contralateral side when compared to a prosthetic ear."[14] Autogenous reconstruction generally requires multiple-staged procedures and may necessitate tissue expansion, multiple grafts, or adjacent tissue transfers (such as temporoparietal flap). Such procedures are technically more demanding and may increase the risk of surgical complications such as flap necrosis, nerve injury, alopecia, and infection. Greater surgical and donor site morbidity (i.e., costochondral grafting) are further disadvantages to traditional reconstructive surgery. The time required for reconstruction is another issue, with the average time of the classic four-stage reconstruction being around 9 months (with an additional 3 months if tissue expansion is required) versus 4 to 5 months for prosthetic reconstruction (in the nonirradiated patient) with cranial implants.[14]

TECHNICAL CONSIDERATIONS

The most important technical aspect to consider when placing titanium implants in the craniofacial skeleton is appropriately positioning the implants to allow for successful prosthetic reconstruction. This necessitates close consultation with the maxillofacial anaplastologist/prosthetist during the treatment planning phase in order to avoid inaccurate implant placement resulting in unusable points of fixation. The quantity and quality of bone in the proposed implant site should be evaluated with appropriate imaging before placement of cranial implants. Axial and coronal computed tomography (CT) provide excellent osseous detail and allow visualization of anatomic structures related to the implant site. A prefabricated surgical template is recommended for accurate placement at the time of surgery (Figure 13-1). The most common surgical sites selected for the placement of cranial implants are the temporal bone, the orbit, and the maxilla/nasal region. Technical considerations related to these individual sites are discussed later.

Temporal Implants

In the case of temporal cranial implants for auricular reconstruction, most authors prefer a subperiosteal-, anterior-, or inferior-based flap. Careful dissection to avoid perforation is necessary, especially in the previously operated patient or in the event that preoperative radiation was administered to the region. The subcutaneous region of the flap is meticulously thinned in order to prevent soft tissue mobility around the implant. The presence of soft tissue mobility at the implant/soft tissue interface may lead to significant soft tissue reactions.[19] An appropriate (~≥25 mm) amount of the flap width is maintained to ensure vascularity of the pedicle. Lundgren and coworkers[20] proposed ideal positioning parameters and recommended placement approximately 18 mm from the external auditory canal at the 6:00, 9:00, and 12:00 o'clock positions for the right ear and the 12:00, 3:00, and 6:00 o'clock positions for the left ear. The distance between implants should be approximately 11 mm (center to center). Generally, we have found these estimates to be accurate; however, local anatomic considerations often necessitate placement of the implants into nonideal locations. Prosthetic reconstruction should still be possible provided the surgeon does not exceed the parameters outlined by the surgical guide. The implant-retaining magnets must be contained within the confines of the final prosthesis in order to achieve an optimal outcome.

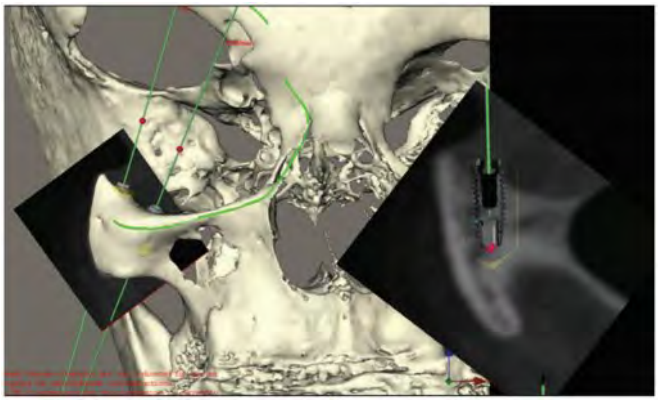


FIGURE 13-1. Preservation of the body of the zygoma during resection surgery is preferred because it serves as an important anchorage site during the reconstruction surgery.

The complication rate is extremely low, although some patients may suffer from dural exposure during the surgical procedure. They are managed conservatively and usually heal uneventfully. In general, middle cranial fossa or sigmoid sinus exposures do not create problems in most cases. Injury to aberrant anatomic variants of the intratemporal portion of the facial nerve is rare but should be considered when operating on younger patients or patients with craniofacial anomalies.[19,21,22]

Orbital Implants

Owing to the osseous anatomy of the orbit, orbital implants must be placed radially within the orbital rim to provide adequate bone thickness for retention. Generally, implant placement within the lateral rim is recommended owing to

the increased thickness of the bone in this region. We have found the medial orbit to be problematic in most cases secondary to lack of adequate bone and increased anatomic complexity due to the lacrima fossa. Unfortunately, this means that the desired axial loading of the implants is impossible in this region, which is a less favorable biomechanical situation when compared with other craniofacial implant regions. Therefore, meticulous technique and consideration for staged bone grafting may be required for a successful implant-retained orbital prosthesis. Usually, three to four implants are placed in the lateral rim to provide adequate prosthetic stability. Further, it is important to note that the implants must be placed sufficiently within the orbit, slightly behind the rim, to allow adequate prosthetic thickness to provide camouflage for the implant fixtures.

Nasal Implants

Implantation of the nasal region can be technically challenging owing to the poor availability of quality bone. The more complex anatomy of the nasal cavity and the thin friable tissue in the area add to the difficulty of cranial implantation in this region. This is especially true in the irradiated patient. Implants are generally placed in a triangular arrangement with one fixture superior (radix) and two placed in a lateral position with the frontal process of the maxilla. The implants must be placed slightly within the nasal cavity to engage adequate bone and, as in the case of the orbital reconstruction, provide for adequate prosthetic thickness.

SURGICAL TECHNIQUE

When considering the placement of maxillofacial implants in any type of maxillofacial defect, the fixtures should be placed with the planned prosthetic framework in mind. The angle of the implants should provide an emergence profile allowing for proper prosthetic design without interfering with the ideal sculpture of the prosthesis. Misplaced implants may cause poor aesthetic outcome. Proper spacing and angles of the implants is necessary to allow manipulation of the prosthetic components.

Pretreatment Criteria

1. Collaboration with the oncologic surgeons before the resection of maxillofacial lesions.
2. Presence of adequate residual bony volume for support of maxillofacial implants.
3. Consideration of the thickness of the overlying soft tissues.
4. Close planning with the maxillofacial prosthodontist and/or anaplastologist allowing for the proper *emergence profile* of the prosthesis.

The preceding points are a few of the preoperative considerations that will allow predicable outcomes for the treatment of patients with simple and complex maxillofacial defects.

Pre-Resection Collaboration

Planning with the oncologic surgeons before resection of patients with maxillary tumors is essential in order to maximize the residual volume of zygomatic bone remaining after the tumor ablation surgery.

Communication of the contours of soft tissue reconstruction immediately after the resection of the tumors is also important. In order to allow adequate for the emergence profile of the abutments as well as the retaining components of the maxillofacial prosthesis, consideration of the "depth" of the defect is critical. In cases of orbital exoneration, the free flap reconstruction must allow for a residual "concave" defect. If the depth of the defect is not adequate, preprosthetic soft tissue surgery may be necessary in consultation with the maxillofacial prosthodontist and/or the anaplastologist. Close cooperation between the oncologic surgeons and the maxillofacial surgeon is paramount.

Residual Bony Volume

The need for adequate residual bony volume for placement of the maxillofacial implants is obvious. The conventional preoperative evaluation has relied on traditional radiographs and/or two-dimensional CT scans for planning of the implant positions. The surgeons would use the limited information gained from these radiographs and, in conjunction with intraoperative "trial and error," locate at times the extremely limited bony volume for the placement of maxillofacial implants in the attempt to avoid hollow spaces such as the frontal, ethmoidal, and maxillary sinuses. The advent of computer-guided software has allowed the adaptation of this technology for the treatment of maxillofacial defects.

Computer-Guided Treatment Planning

The Maxillofacial Concept Software allows collaboration between the surgeon, the maxillofacial prosthodontist, and/or the anaplastologist in planning treatment for the patient with maxillofacial defects before initiation of the treatment. The two-dimensional DICOM files of the patient are converted into three-dimensional format, which allows better visualization of the remaining osseous tissues by the surgical team. The patient's soft tissue can also be reformatted and superimposed onto the reconfigured three-dimensional bony volume showing the topography of the patient's remaining facial and intraoral architecture.

The maxillofacial prosthodontist and the anaplastologist can assess the thickness of the soft tissues and guide the surgical team in the best location for the implants and, consequently, the abutments and the prosthetic frame work design. By using the information provided by the Maxillofacial Concept Software, the entire team can plan the position of the implants that best complies with the surgical and the prosthetic principles. The surgical team preoperatively can now position the implants, evaluate their trajectory (Figure 13-2), select final abutments (Figure 13-3), and

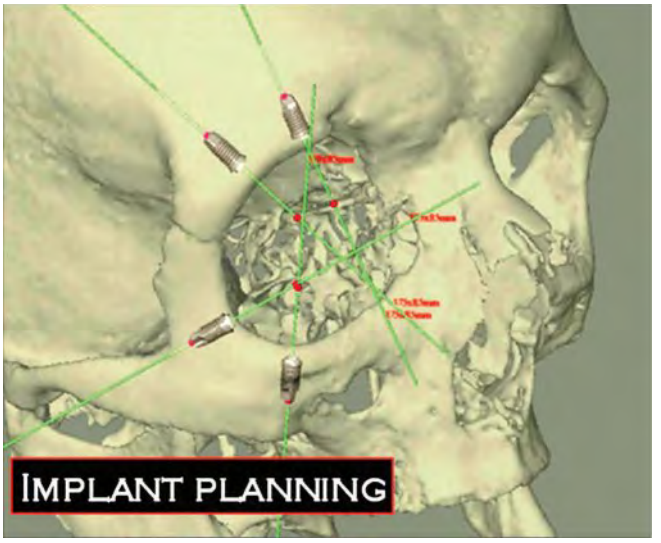


FIGURE 13-2. The maxillofacial software allows the three-dimensional, "active" positioning of the implants during treatment planning.

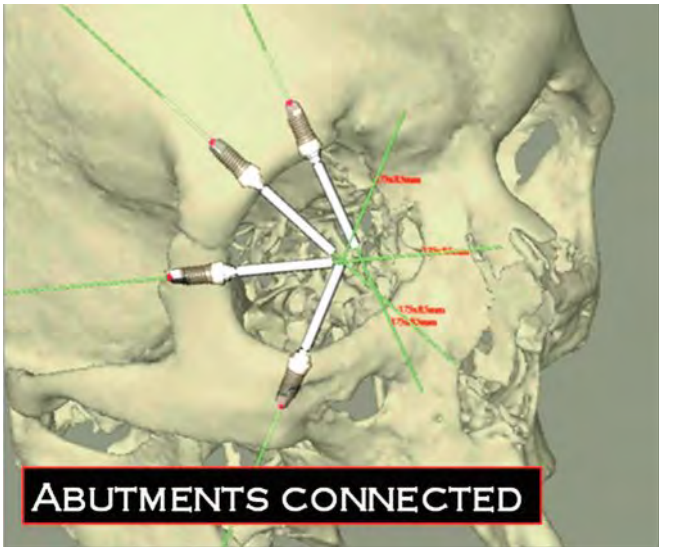


FIGURE 13-3. Addition of abutments to the planned implant positions allows for better visualization of the trajectory of the implants.

modify the treatment plan several times until the best implant positions for the support of the maxillofacial prosthesis leading to the most transparent transition line and aesthetic emergence profile are reached (Figure 13-4). The final treatment plan can then be taken to the operating room and the "virtual surgery" can be translated to the actual patient (Figure 13-5).

Overlying Soft Tissues

Equal importance is given to the presence of healthy overlying and surrounding peri-implant soft tissues. Whether cutaneous or mucosal tissue, the clinicians must be very aware of the thickness of the tissues in order to allow a maintainable environment around the prosthetic substructures by the

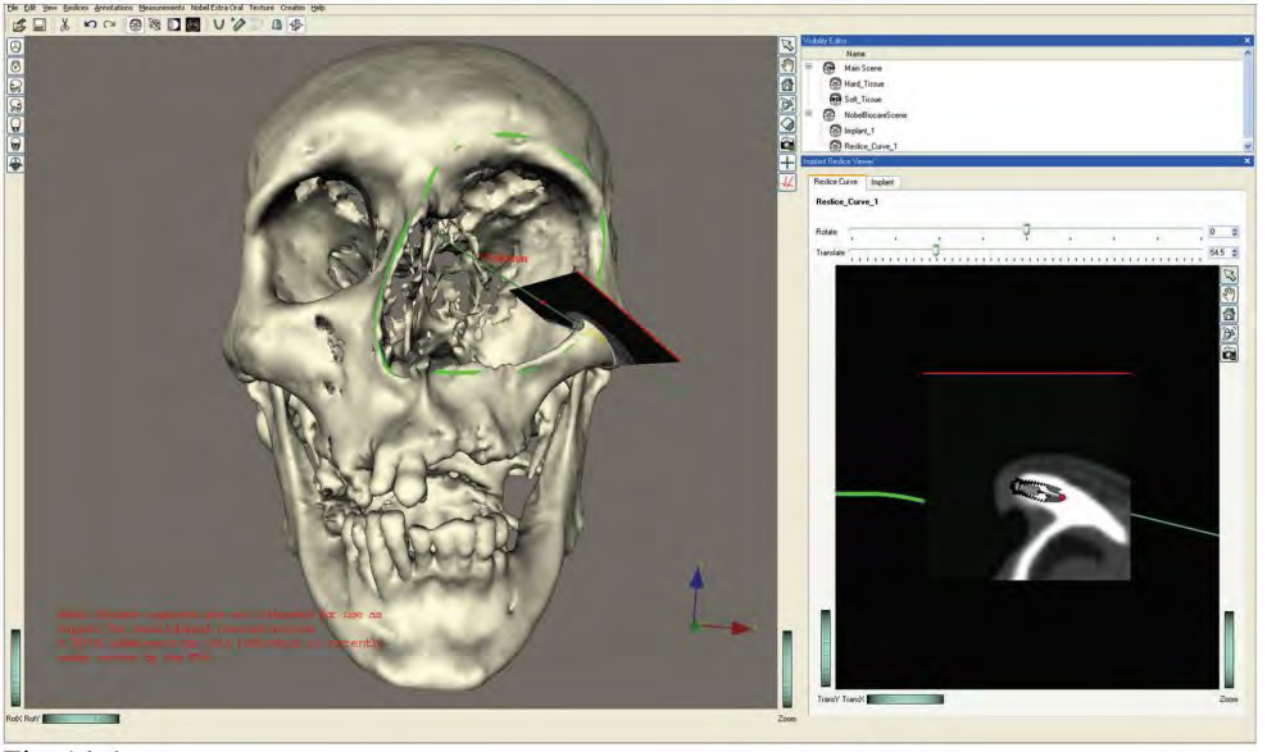


FIGURE 13-4. The final planned position of the implants can be evaluated in three-dimensional as well as two-dimensional windows simultaneously using the maxillofacial software.

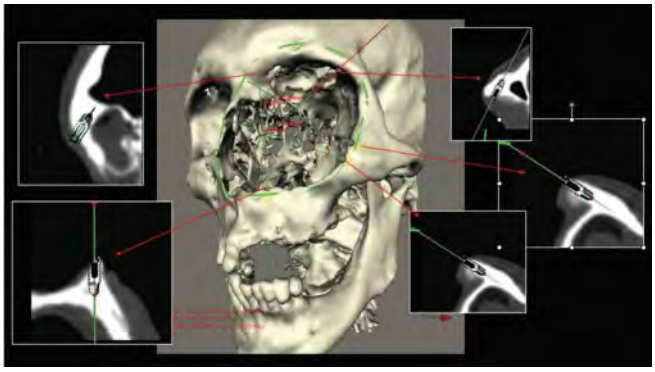


FIGURE 13-5. "Virtual surgery" of a complex maxillofacial defect.

patient and the maxillofacial prosthodontist. The management of the peri-abutment soft tissue is very similar to the intraoral management of soft tissue surrounding abutments. Thinning of the flaps is usually necessary to allow a maintainable depth of soft tissue around maxillofacial abutments.

Transition Line

The transition line of the prosthesis with the patient's skin should be as subtle and low profile as possible to allow for a lifelike appearance (Figure 13-6). In any type of defect, the fixtures should be placed with the planned prosthetic framework in mind. The angle of the implants, the trajectory, and its long axis during its placement should provide for adequate emergence profile allowing for proper bar design and/or magnet placement without interfering with the ideal sculpturing of the prosthesis. Proper spacing and angles of the implants are also necessary to allow the manipulation of the prosthetic screws and drivers needed during fabrication and delivery of the maxillofacial prosthesis.



FIGURE 13-6. "Feathered" margin of the prosthesis allows for a subtle transition line between the prosthesis and the patient's skin.

Surgical Considerations

Treatment of patients with maxillofacial defects may involve the intraoral and extraoral structures. It is important to define the type of implants used for the treatment of the various clinical cases.

Extraoral Implants

The implants used are the regular platform implants. The extraoral implants used to date are the machined surface regular platform Brånemark, 4.0-mm implants. Extraoral implants used for the bone-anchored hearing aid (BAHA) appliance or to support a prosthetic ear are not the regular platform Brånemark implants. The implants used for ear prosthesis are specially designed to support the ear reconstruction or the BAHA appliance. Vistafix implants developed also by Brånemark and more currently revised are the implant of choice for ear prosthetic stability and BAHAs (Figure 13-7). They are available in 3.0- and 4.0-mm lengths with a 1.5-mm collar that is slightly submerged in the bone to increase the surface area of bone contact (Figure 13-8).

Intraoral and Intranasal Implants

Intraoral and intranasal implants are also the machined-surface, regular platform implants or conical cranial implants available from Straumann. In cases in which the zygomatic bone is used for anchoring implants, several approaches were considered. For patients having had partial or total maxillectomies, regular platform implants were used in the remaining portion of the zygomatic body to allow for contralateral point stabilization of the prosthetic framework.

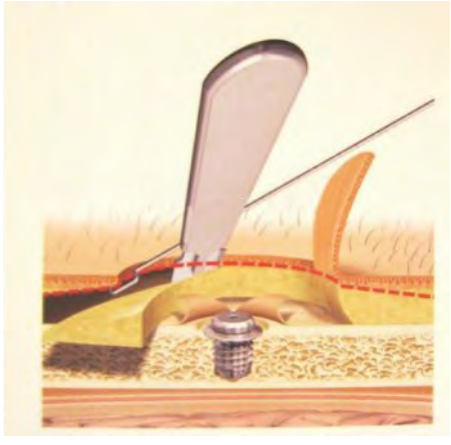


FIGURE 13-7. Vistafix cranial implant for temporal bone with collar to increase surface area owing to 3- and 4-mm lengths with limited surface area.

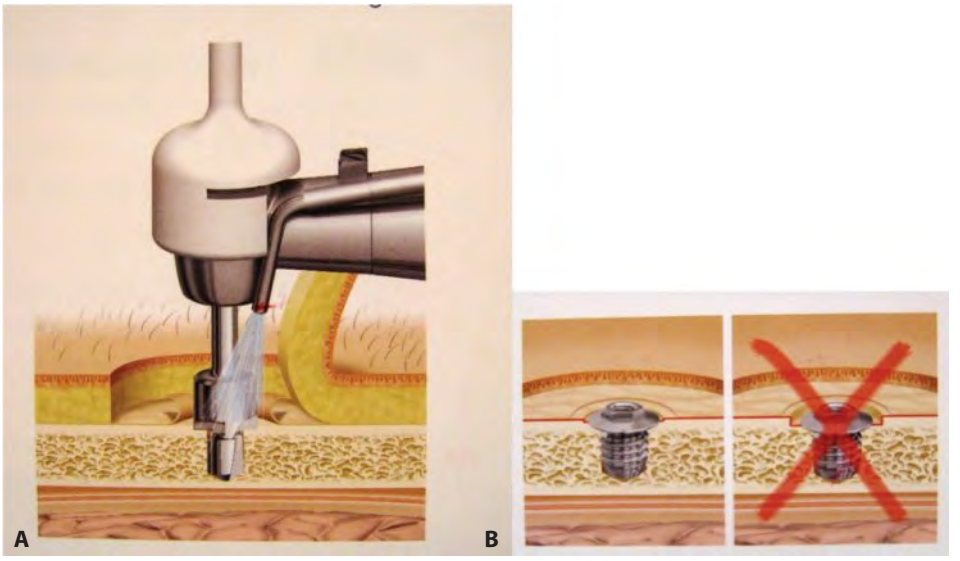


FIGURE 13-8. **A** and **B,** Vistafix drill bit creates the implant osteotomy and countersink for collar simultaneously.

In cases in which the maxillary residual arch is intact, the Brånemark zygomatic implant with the 45-degree angulated platform can be used.

Preparing the Osteotomy

Most cases are treated in the operating room under general anesthesia. After sterile preparation and draping of the patient, lidocaine with epinephrine is administered to the surgical field to allow better control of hemostasis. Sharp dissection through the epithelium, connective tissue, and periosteum exposes the residual bony defect. The drilling sequence to prepare the osteotomy is the same as for conventional intraoral implants. The surgeon has to clinically judge the quality of bone while preparing the osteotomy. When using cranial implants, either Brånemark or Straumann, the osteotomy can be completed with a specially designed drill that is either 3 or 4 mm in length and create the collar osteotomy simultaneously (see Figure 13-8).

Orbital Defects

Placement of two to three implants is generally adequate for support of the substructure and the orbital prosthesis. The preferred site is the lateral supraorbital rim, if it has not been resected. However, implants may be placed in the residual periorbital bony rim. Special consideration for treatment planning the orbital prosthesis is the available depth of the orbital defect. Inadequate orbital depths will not allow the tipping of the trajectory of the implant into the center of the orbit, resulting in inadequate space for the fabrication of the substructure and the orbital prosthesis. Virtual planning of the implants is performed (Figure 13-9), and the orbital depth is established intraoperatively by *debulking* of the orbital contents. The implants are placed as planned at 35-N/cm (Figure 13-10). The immediate postoperative implant positioning is in concert with the proposed virtual planning of the implants (Figure 13-11). The computer-guided treatment planning allows for the positioning of the implants in appropriate bony volume in

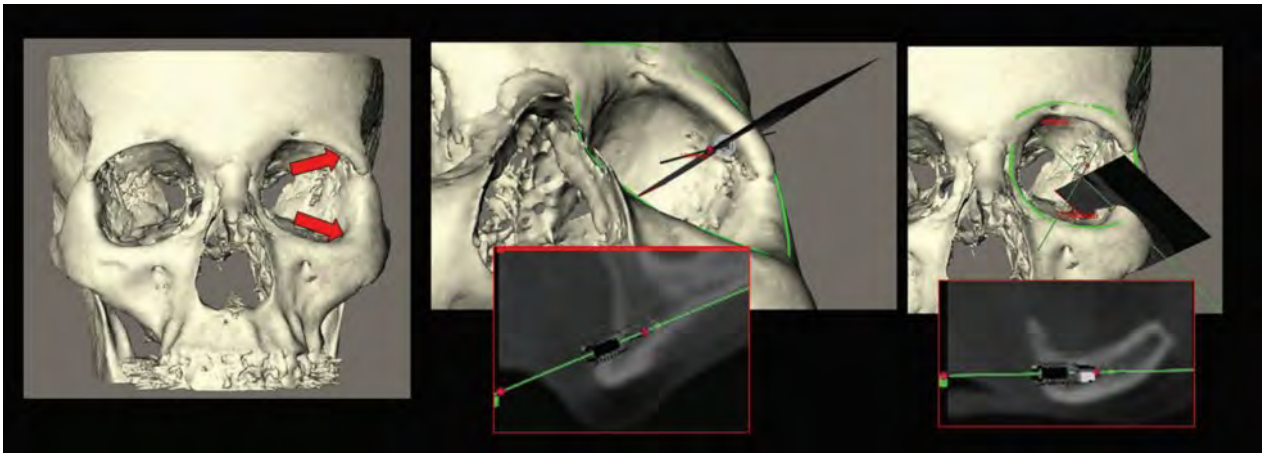


FIGURE 13-9. Two implants in the lateral-inferior corner of the orbital rim complete the virtual planning of the proposed maxillofacial implants for retention of an orbital prosthesis.

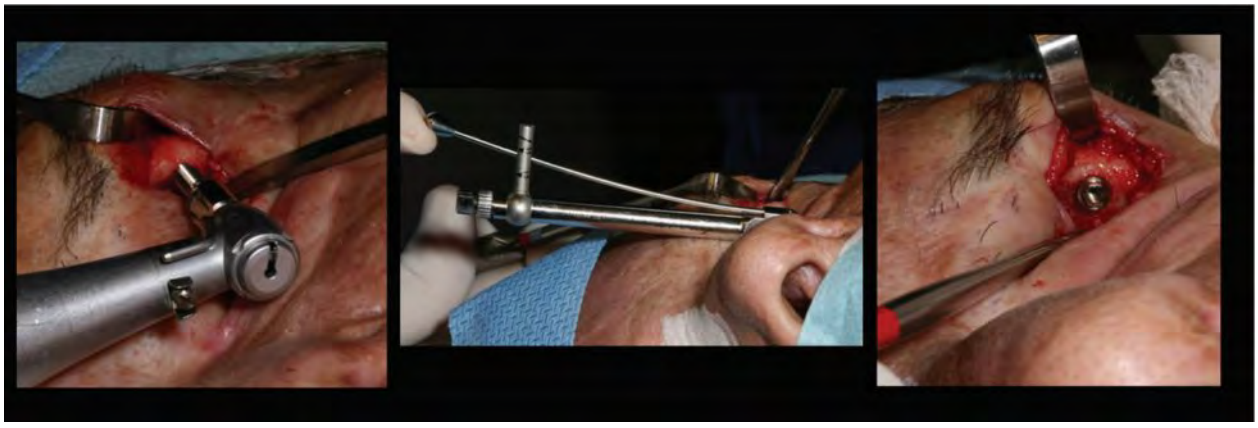


FIGURE 13-10. The implants are placed with 35-N/cm insertion torque.

concert with the proper trajectory for reconstruction of the maxillofacial prosthesis (Figure 13-12). After exposure of the orbital rim, a round bur is used to identify the position of the implant (Figure 13-13). A paralleling pin is placed upon completion of the 2-mm osteotomy to better evaluate the trajectory of the implant (Figure 13-14). The final osteotomy depth is 3 mm followed by the insertion of the implant. Most procedures follow the one-step protocol in which the temporary healing abutment is placed at the time of the implant surgery with adaptation of the soft tissues around the temporary healing abutment (Figures 13-15 and 13-16). Once the second planned implant is placed, the position of the actual surgery can be compared with the position of the implants in the virtual surgery using the computer-guided treatment planning software. Triple antibiotic ointment and peri-implant

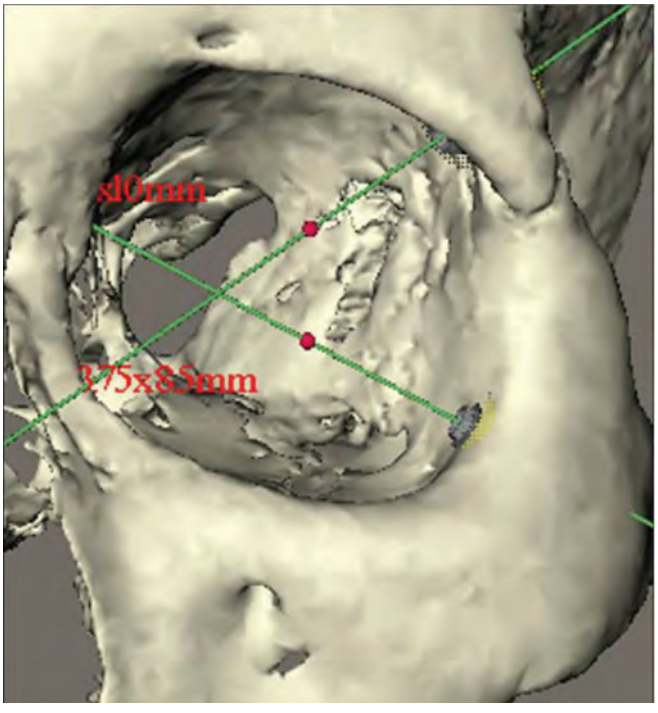


FIGURE 13-11. "Virtual" positioning of the orbital implants.



FIGURE 13-12. Preoperative virtual planning of a 30-year-old male for implants to support an orbital prosthesis.



FIGURE 13-13. Use a round bur to initiate the osteotomy.



FIGURE 13-14. A paralleling pin demonstrates the trajectory of the 2-mm osteotomy.



FIGURE 13-15. Insertion of the 4-mm, RP Brånemark implant into the osteotomy site.



FIGURE 13-16. Placement of a healing abutment completes the one-stage protocol.



FIGURE 13-17. Metal framework with a magnet used as the retentive component for the prosthesis.



FIGURE 13-18. Final magnet-retained orbital prosthesis.

surface pressure dressings complete the surgical treatment. Three to 4 months of osseointegration period is allowed followed by fabrication of the substructure and the prosthetic eye (Figures 13-17 and 13-18).

Nasal Defects

Placement of the implants for support of a nasal prosthesis is unique because access by the maxillofacial prosthodontist must be considered during planning of the position of the implants. The generally accessible bone volumes that can support the implants are in the premaxilla. Placing two implants through the nasal floor in positions corresponding with dental positions nos. 7 and 10 are recommended (Figure 13-19). During treatment planning, especially in the fully dentate patient, consideration of vital structures including the teeth and the neurovascular content of the nasopalatine canal is essential. The use of the maxillofacial planning software allows for visualization of the existing vital structures avoiding their collision with the proposed implants

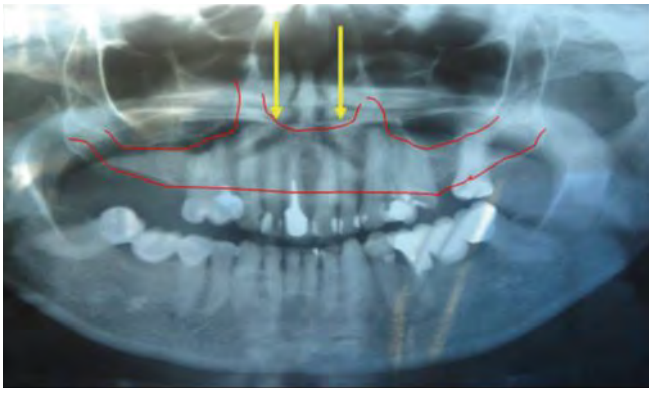


FIGURE 13-19. Implants to retain a nasal prosthesis are placed through the nasal floor in proximity to teeth nos. 7 and 10.

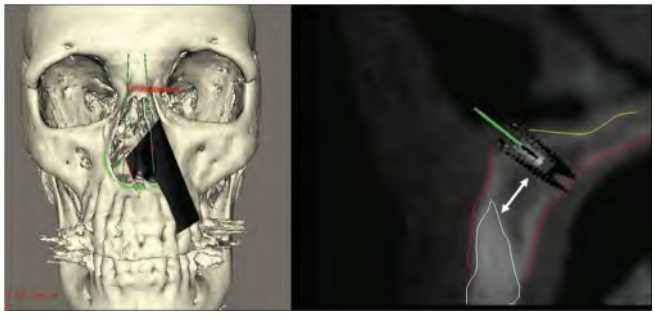


FIGURE 13-20. The nasal floor, the hard palate, and the position of existing teeth are readily visualized during the virtual planning. A safe distance is maintained between the vital teeth and the implant.

(Figure 13-20). Aesthetic considerations of the final prosthesis must also be taken into account. In cases in which the oncologic resection was not orchestrated with the maxillofacial reconstruction phase of the patient's treatment, an excess of remaining soft tissue hinders an aesthetic prosthetic outcome (Figure 13-21). Three general paranasal areas should be considered when planning a nasal prosthesis for a predictable aesthetic outcome (Figure 13-22):

1. The upper lip drape.
2. The position of the nasal cartilage.
3. The immediate paranasal-alar width and bulk.

The surgical procedure may begin by addressing the upper lip drape. A 2- to 3-mm area of incisal tooth display is aesthetic at rest for the female patient. In order to establish the proper lip drape, a "gull-wing" resection of the upper lip immediately below the nasal apertures is performed through the skin, connective tissues, and periosteum (Figure 13-23). The repositioning of the inferior portion of the resection allows for the visual evaluation of the adequacy of the resection. In order to allow room for the fabrication of the metal substructure supporting the prosthetic nose, excess portion of the nasal cartilage is trimmed. Debulking of the paranasal connective tissues allows for the maxillofacial prosthesis *artistic license* to create favorable contours for a thin *finish line* of the prosthesis.

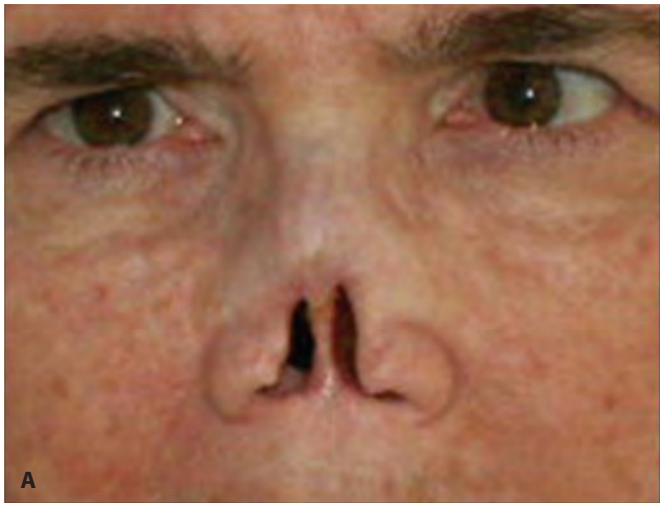




FIGURE 13-21. **A** and **B,** Inadequate resection and site preparation result in an unaesthetic transition line between the prosthesis and the patient's skin.



FIGURE 13-22. The length of the upper lip *(red arrow),* the width and the bulk of the paranasal soft tissues, and the position of the nasal septum/cartilage are the three preoperative considerations in treatment planning for nasal implants.

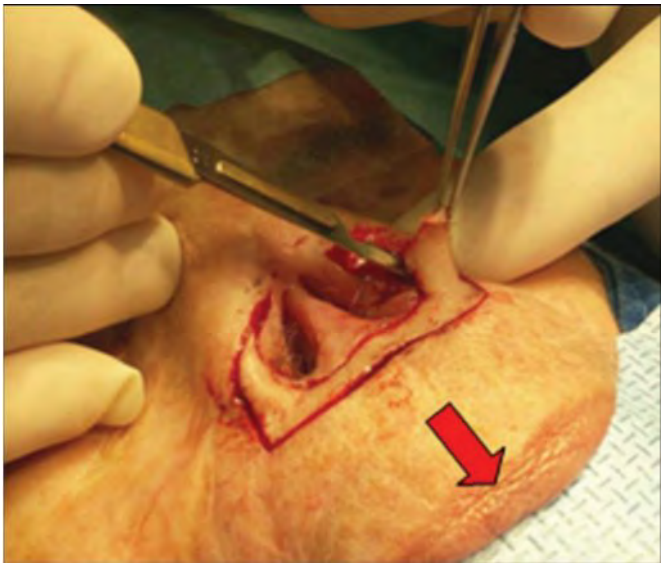


FIGURE 13-23. "Gull-wing" resection of the upper lip allows for the shortening of the upper lip length.

Following the virtual treatment planning, the two implants are placed with the implant platforms tilted outward to allow ease of access by the maxillofacial prosthodontist. The one-stage surgical protocol is followed. However, it is prudent to place the final abutment at the time of stage I surgery. Placement of the final abutment and the impression coping at time of surgery in conjunction with packing with triple antibiotic pressure dressing allows for the adaptation of the nasal mucosa to the nasal floor. It is not advisable to place the third implant in the nasal bone as the nasal prosthesis is implant retained; therefore, there are no biomechanical advantages to the third implant. The emergence profile of the third implant platform generally interferes with the natural contours of the prosthetic nasal bridge. After allowing 3 to 4 months for osseointegration, the magnet-retained prosthesis is fabricated (Figure 13-24).

Complex Maxillofacial Defects

Complex maxillofacial defects, a combination of orbital and nasal defects with or without intraoral involvement, need

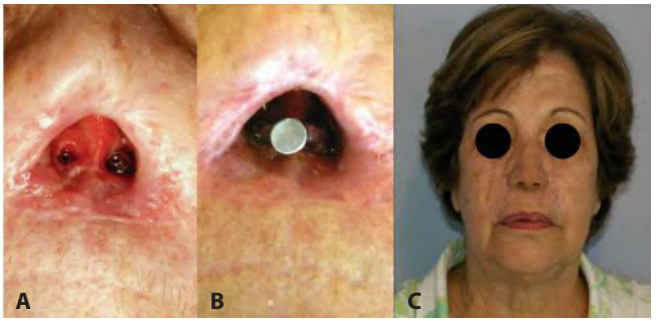


FIGURE 13-24. **A–C,** Abutment level impression is taken after osseointegration of the implants followed by fabrication of the superstructure and the final magnet-retained nasal prosthesis.



FIGURE 13-25. Complex orbital-nasal defect with a portion of the anterior cheek missing.

case-by-case planning. However, the concept for the positioning of the implants remains the same; using the least number of implants to allow adequate support of the prosthesis by the metal substructure is the guide to planning in this group of patients.

Combination of orbital and nasal defects (Figure 13-25) may be treatment planned as an orbital defect plus a nasal defect. The lateral orbital rim is the preferred position for the support of the orbital prosthesis. In the planning process, care must be taken to appreciate the final emergence profile of the orbital process, thereby positioning the trajectory of the implant into the orbital defect (Figure 13-26). The premaxillary floor of the nose is the preferred area for placement of implants to support the nasal prosthesis (Figure 13-27). The use of the medial portion of the superior orbital

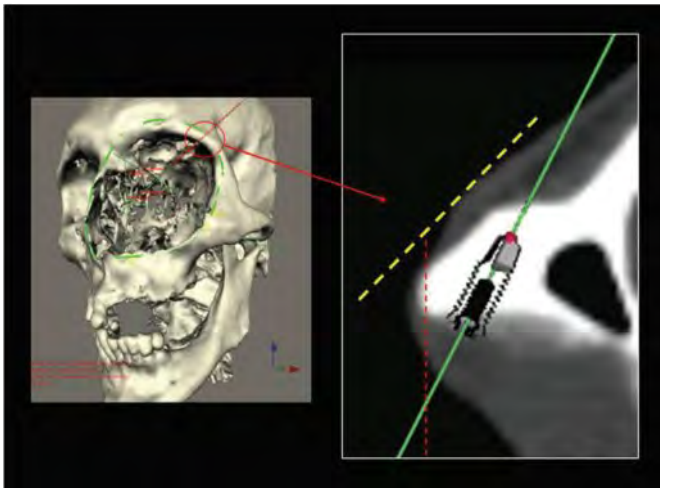


FIGURE 13-26. The trajectory of the implants into the defect is essential for an aesthetic outcome.

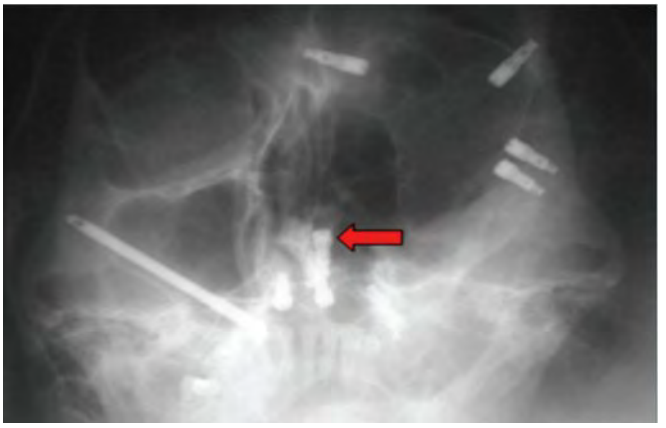


FIGURE 13-27. The *red arrow* of the immediate postoperative radiograph identifies the nasal floor implant needed for the support of the complex bar to support the maxillofacial prosthesis. The patient's edentulous maxilla was treated using the combination of the tilted and the zygoma concept.

rim should be avoided, if possible, because the natural concavity of the upper eye lid in this area does not allow for fabrication of the superstructure (Figure 13-28). The final orbital-nasal prosthesis may be retained by one or two magnets (Figure 13-29).

Unusual Maxillofacial Defects

Multiple variations of maxillofacial defects exist, and therefore, it is paramount for the maxillofacial surgeon and the oncologic surgeon to collaborate before the resection of the tumor with the reconstructive phase in mind. Preservation of the zygomaticomaxillary complex, in total or in portion, aids in the future reconstruction of patients with orbital defects

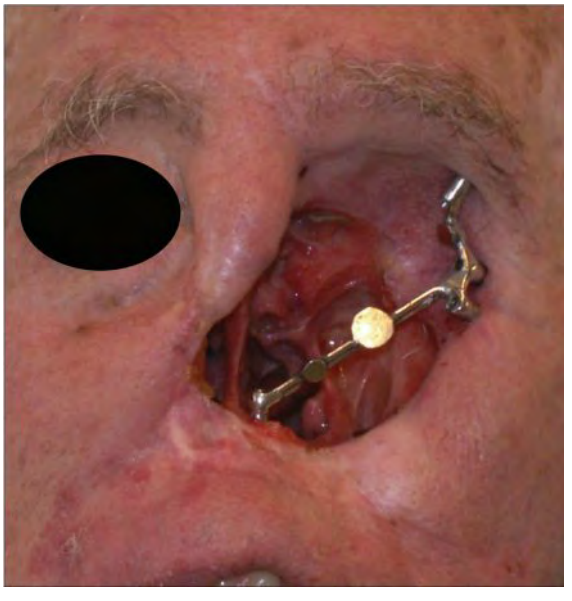


FIGURE 13-28. The final bar is fabricated to retain the orbital-nasal prosthesis.

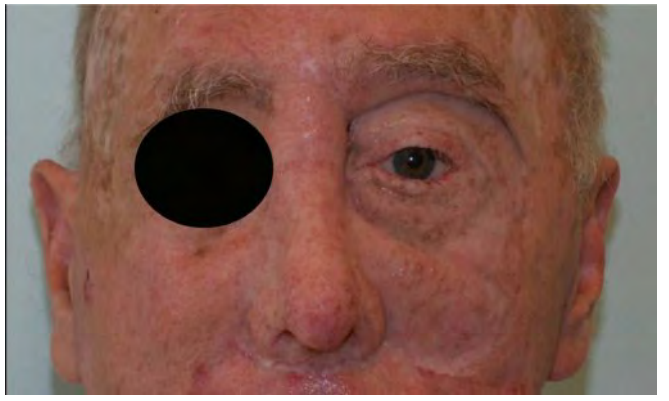


FIGURE 13-29. Completed magnet-retained prosthesis on left.

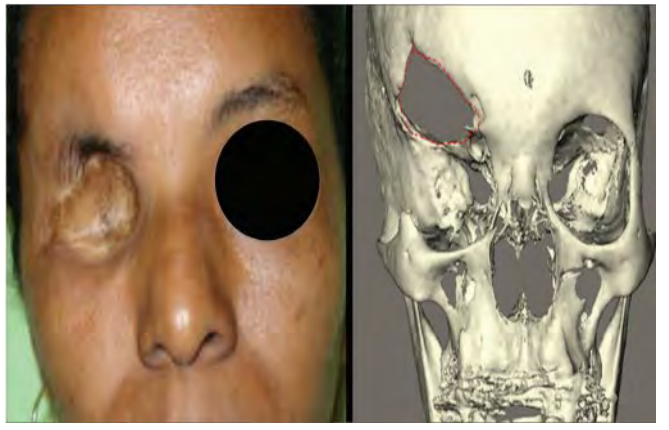


FIGURE 13-30. The preservation of the inferior portion of the zygomatic-maxillary complex allows for placement of implants.

(Figure 13-30). Postresection, virtual planning allows for identification of optimal surgical and prosthetic sites for the placement of the implants (Figure 13-31). The resultant clinical implant positions are predictably gained using the preoperative computer-guided software (Figure 13-32).

HEALING PERIOD

The traditional two-stage or the one-stage surgical protocol may be adopted per the surgical teams' preference. *For the two-stage approach*, after installation of the implants, cover screws are placed and the implants are submerged. Stage II surgery; exposure of the extraoral implants through the cutaneous tissue, is performed 3 to 6 months after the stage I surgery.

Abutment Connection and Impression Taking

The final abutments are placed approximately 2 to 4 weeks after the stage II surgery. This will allow the resolution of the soft tissue swelling around the temporary healing abutments installed at stage II surgery. The impression can be taken at this time.

For the one-stage approach, temporary healing abutments or the final abutments are secured to the implants

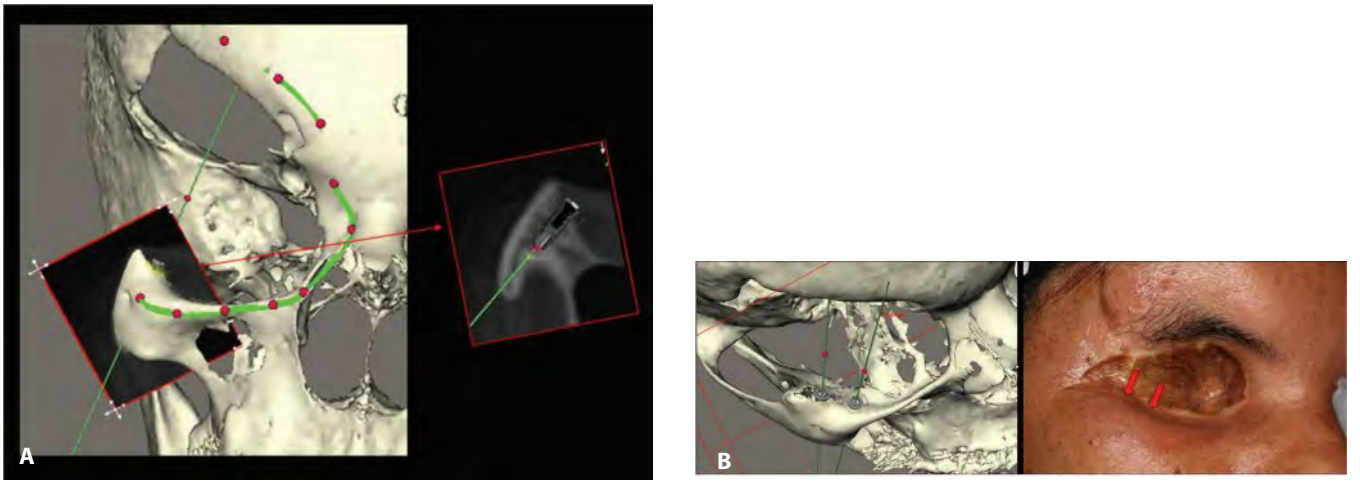


FIGURE 13-31. **A** and **B,** The maxillofacial software allows for the planning of two implants in the inferior-lateral portion of the remaining portion of the patient's zygoma.

immediately after their insertion. The peri-abutment soft tissues are maintained by daily hygiene using warm water with occasional application of triple antibiotic if necessary. For the one-stage protocol, impressions may be taken at the stage II appointment, 3 to -4 months after placement of the implants.

Soft Tissue Reactions and Infections

The rate of soft tissue reactions around percutaneous implants has been reported between 3% and 60%, with significant variation reported depending on the stated definitions used to classify a reaction.[23,24] Most authors have noted soft tissue reactions of approximately 3% to 7% of percutaneous implants, with the majority of the reactions being mild erythema or irritation.[16,25] In a total of 2624 postoperative implant observations in 309 cranial implants (follow-up from 1–12 yr) Westin and colleagues[19] noted a 3% incidence of significant skin reactions. Abu-Serriah and associates[26] found soft tissue infection to be the most common complication in a group of 150 cranial implants. They also note that decreased after the first 2 years of service in the temporal region; however, implants in the orbital and nasal regions exhibited a constant rate of soft tissue complications when observed over time.

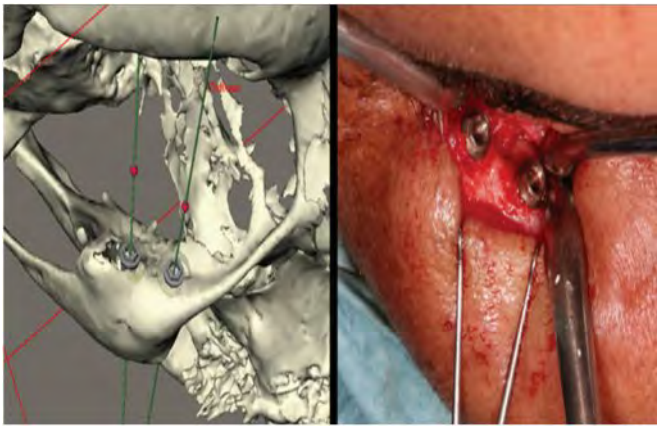


FIGURE 13-32. The "actual" position of the implants placed is the same as their position during the "virtual" treatment planning.

Fourteen percent of the implants included in the current study exhibited some form of soft tissue reaction at some point during the follow-up period. These ranged from mild to significant reactions including granulation tissue and infection and, in some cases, resulted in loss of the implant. Holgers and Tjellström[24] utilized a classification scale for soft tissue reactions surrounding percutaneous titanium implants. Utilizing Holgers and Tjellström's classification system, we found that 95.6% (109/114) of the cranial implants tissue reactions fell within classes 0 to 3 and required only local measures to control soft tissue reactions. In the event that local revision was necessary, electrocautery was utilized to remove granulation tissue and sharp dissection was used to thin the tissue surrounding the implant interface. Patients who underwent revision were also placed on appropriate empirical antibiotics. The patients were then instructed regarding home care and followed-up monthly, with the majority having improvement of the peri-implant inflammation. The remaining implants (5) exhibited significant reactions, which despite efforts and multiple revisions, resulted in loss of the implants.

The most common isolates from peri-implant cultures obtained from skin-penetrating cranial implants are *Staphylococcus* (most commonly *Staphylococcus aureus*) and *Streptococci* species, gram-negative bacilli, and yeast species such as *Candida parapsilosis*.[27] Routine culture of peri-implant tissues is not beneficial owing to contaminants from normal skin flora and the rarity of frank infection surrounding the implants. In the event that empirical antibiotic therapy fails or clinical suspicion of an unusual pathogen exists, culture-directed antibiotic therapy may be beneficial. Soft

tissue reactions around the skin-penetration implant can be minimized with excellent hygiene and meticulous surgical technique (e.g., appropriate flap thinning and maintaining adequate blood supply by maintaining a robust pedicle). We do not recommend routine use of antibiotic ointments for transcutaneous titanium implants. Medications such as steroid cream or antibiotic ointment should be reserved for short-term therapy, and long-standing soft tissue reactions should be evaluated for peri-implant infection or soft tissue revision surgery as noted earlier. Long-term utilization of antibacterial ointments around skin-penetrating implants may also have the adverse effect of selection of resistant strains of bacteria or yeast species.[27–30]

LONGEVITY OF CRANIAL IMPLANTS

The successful placement and osseointegration of cranial implants have been evaluated by multiple authors. Histologically, osseointegration of cranial implants appears to closely resemble that of intraoral titanium dental implants.[31] It is generally accepted that the success rate differs dramatically relative to the location in which the implant is placed. The most predictable site for cranial implant placement is the temporal bone, by a significant margin. Long-term success rates in the temporal region have been reported ranging from 92% to 98% in larger studies.[12,13,16,20–22,25,26]

Success rates in orbital implantation have been reported from 91% to 96%.[16,21,22] In the present study, a total of 31 orbital implants were placed with a success rate of 87.1%.

Cranial implants placed in the nasal region are especially difficult owing to the low availability of adequate bone in the region and difficulty with soft tissue management and hygiene. Success rates reported in the literature when implanting the nasal region range from approximately 70% to 80%.[16,21,22]

RADIATION IN CRANIAL IMPLANTATION

When utilizing cranial implants for reconstruction of oncologic defects of the facial skeleton, placement of implants into radiated bone is inevitable. As with all radiated tissues, soft tissue fibrosis coupled with the loss of the microvasculature occurs in the recipient bed. The resulting decreased oxygen tension has a negative effect on the ability to place titanium implants and obtain successful integration. Most authors report significantly increased failure rates (range 17%–42%) when placing cranial implants into radiated bone.[16,21,32–35] When examining 81 cranial implants, Jacobsson and coworkers[25] noted a success rate of 62.7% (as compared to 92.1% in nonirradiated bone). Implants lost in irradiated bone were placed after a 12-month period, generally successfully. The orbit is an especially difficult location to achieve implant integration after radiation therapy.[36] In a group of 24 orbital implants, Roumanas and colleagues[22] observed a high rate of loss in the irradiated orbit and noticed a 100% complication rate including failure to integrate and late failure, tissue inflammation, and soft tissue recession. Successful implantation of the orbit is significantly more difficult in the radiated patient, with most authors reporting success rates in the range of 50% to 66%.[22,25,32] Despite the adverse effect of radiation regarding cranial implant osseointegration, the risk of craniofacial osteoradionecrosis is quite low and seldom observed.[16,22,32,36] The timing of implant placement at the conclusion of radiation therapy remains controversial, with most authors recommending a delay of 6 to 19 months before implant placement.[22,26,32]

The utilization of hyperbaric oxygen (HBO) has been advocated by multiple authors in the literature to improve integration rates and optimize soft tissue healing when placing cranial implants into irradiated bone.[16,26,32] When examining 125 irradiated cranial implants, Granstrom and associates[32] noted 38.4% of irradiated implants were lost versus 17% in the nonirradiated group. Of the irradiated implants, 45 received preoperative HBO therapy and no implants were lost in this group. The authors concluded that HBO was extremely beneficial in the radiated field before placement of cranial implants. In reference to the proposed soft tissue benefits of HBO, the authors also observed no statistical difference in local skin reactions when comparing patients who received HBO therapy and those who did not.

PROSTHETIC CONSIDERATIONS

Stable and consistent prosthetic attachment is crucial to the successful rehabilitation of the maxillofacial patient. For the anaplastologist or practitioner who is designing and fabricating prostheses, implementing cranial implants can significantly help meet the needs of the patient. *Anaplastology* (from the Greek *ana*, again, and *plastos*, something made or formed) is commonly defined as the application of prosthetic materials for reconstruction of an absent, disfigured, or missing body part. "The anaplastologist is charged with the formidable task of restoring the delicate beauty of the human ear."[37] This task is assigned and applied to all facial prosthetic reconstructions.

Preoperative Planning

Preoperative appointments with the anaplastologist are considered an integral part of the treatment planning process for patients having prosthetic reconstruction.[38] At the initial consultation, before and after photos of previous cases can be shown to help the patient visualize possible results and treatment options. The anaplastologist can effectively plan to achieve the highest level of realism and symmetry attainable under given circumstances. When possible, presurgical impressions capturing natural anatomy before planned resections or tumor excision are very helpful. This record of the patient's own facial features can help to establish a prosthetic design that the patient will ultimately recognize and relate to. Discussion of all retention types should be initiated at this stage with the advantages of each clearly explained.

Options without Cranial Implant Surgery

A patient's lifespan, poor hygiene, or psychosocial implications can be contraindications for an implant-retained prosthesis. In these cases, an adhesive-retained prosthesis can be designed relatively quickly without requiring any surgery or osseointegration waiting time. However, prosthetic adhesives can be cumbersome especially to older patients who have limited dexterity or impaired eyesight. An adhesive-retained prosthesis requires skill in locating it exactly on the soft tissue as designed by the anaplastologist or proper fit and aesthetics are compromised. The effectiveness of prosthetic adhesives can be severely diminished owing to soft tissue movement, oily skin types, and environmental factors such as extremely humid climates.

Options with Cranial Implant Surgery

An implant-retained prosthesis provides a secure and consistent method of attachment. The mechanical connection between patient and prosthesis can alleviate psychological concerns that the prosthesis will become loose or dislodge at any time.[39] It also ensures exact positioning without the need of a mirror or care giver. In addition, the lifespan of an implant-retained prosthesis is typically longer than one requiring adhesive owing to less wear and tear associated with the adhesive removal process.

When the surgically placed, implant-retained type is chosen, the bar and clip versus magnetic attachment option should be decided presurgically (Figures 13-33 and 13-34). This decision will determine how many exposed implants are needed. Experience has shown that the gold bar arrangement proves more difficult for many patients to clean than the freestanding abutments for magnetic attachment. The magnetic attachment requires little manual dexterity; the magnetic force can actually help to guide the prosthesis into place.[40] Conversely, both the gold bar and the clips can break or bend, rendering attachment either difficult or impossible. Additional space within the prosthetic form for the acrylic housing containing the clips can also be compromising to the final design. The recently developed O-ring magnet by Technovent Ltd "provides far superior retention to that of conventional magnets."[41] In the author's experience, using only one of these O-ring magnets in a prosthesis meets or surpasses the retention of a bar clip design.

To aid in the treatment planning for a prosthesis supported by cranial implants, bone quality should be examined with applicable imaging techniques before cranial implant placement.

Prosthetic Surgical Considerations

Successful prosthetic reconstruction using cranial implants is contingent upon proper positioning of the implants. When deciding how many implants to place, several factors should be taken into account. What is the minimum needed to support secure attachment for the patient's activity level? How many can be placed in the most ideal locations according to the surgical template? Does this allow adequate space between the abutments for proper cleaning? If an implant fails, will the remaining be sufficient for retention? If the patient has been irradiated or bone quality is questionable, placing an additional implant should be considered.



FIGURE 13-33. The freestanding magnetic attachment method.

The location and implication of hair-bearing tissue should be identified before surgery. Hair in the proposed peri-abutment region presents potential for infection.[38] Removing hair follicles or utilizing split-thickness skin grafts or laser hair ablation is the preferred way to manage unwanted hair.

Auricular Considerations

Advancements in three-dimensional technology such as scanning and milling machines provide for accurate reproduction of contralateral auricular forms. Utilizing such devices helps the practitioner accurately and quickly achieve a certain level of symmetry (Figure 13-35). The patient's existing auricular cast is scanned, digitally mirror-imaged, and milled in three dimensions. This shape gives the anaplastologist a reliable reference in which to design the final auricular prosthesis.

In congenital cases, there are often several options to consider. Cartilaginous remnants from failed autogenous reconstructions can be retained or sculpted and repositioned for the construction of a neotragus. The presence of a tragus allows the anterior prosthetic margins to be elegantly concealed behind this anatomic feature (Figure 13-36). Sym-



FIGURE 13-34. The gold bar and clip attachment method.

metrically and well-positioned microtic tissue can remain underneath the implant-retained auricular prosthesis. Conversely, microtic tissue compromising the end aesthetic result can be partially or entirely removed at the time of implant placement. This should be clearly discussed with the patient before surgery, allowing plenty of time for decision making. It is not uncommon for the patient to feel emotionally attached to her or his microtic tissue.



FIGURE 13-35. Patient cast, prototyped model, and final prosthesis.



FIGURE 13-36. Modification of cartilaginous tissue for the construction of a neotragus and prosthetic reconstruction.

Templates

Implants should be placed in an area of the prosthesis that is thick enough to contain and naturally disguise the retentive hardware. Designing a prototype prosthesis before surgery will determine these areas of thickness and other important features. The prototype can then be used in the construction of the surgical template. The template should designate preferred implant locations (Figure 13-37) and other optional sites in case the favored locations are not of acceptable bone quality. It should also denote ideal spacing between implant sites appropriate for the chosen attachment method (gold bar

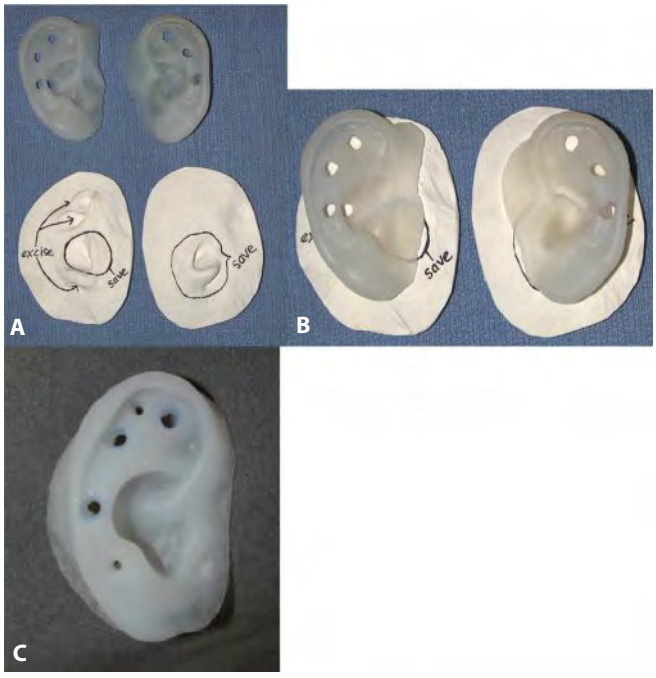


FIGURE 13-37. **A–C,** Ideal locations within the antihelix portion of the surgical template.

or free-standing magnetic). In addition, the orientation or angle of the desired implants should be denoted. This angle should be conducive to cleaning around the abutments.

All templates should passively, but precisely, register onto the patient during surgery. Nearby anatomic features should be built into the template's design and, therefore, ensure proper placement. Various materials such as acrylic or silicone can be used in template fabrication. The chosen material must be properly sterilized before use in surgery. Even though the custom-designed template is provided to the surgeon, it is useful for the anaplastologist to be present at the implant surgery.[39] This facilitates consultation and ensures that the prosthetic result will not be compromised should unforeseen circumstances arise.

Auricular Templates

The basic reference mark in locating potential auricular implant sites is the middle of external auditory canal. When this landmark is available and symmetrical to the contralateral side, a clock-positioning guide can be referenced. In this technique, the ideal location is approximately 20 mm from the center of the external auditory meatus. Positions are 8:00 o'clock and 10:30 for the right side and 1:30 and 4 o'clock on the left (see Figure 13-37). Utilizing these locations within the template should allow for the retentive hardware to be concealed in the antihelix portion of the prosthesis (see Figure 13-37). Any cartilaginous tissue intended for a neotragus can be indicated in the same template.

Construction of the Prosthesis

The final visual prosthetic result depends on achieving a delicate balance of many factors during all stages of construction. Fabrication begins with capturing an accurate impression. Soft tissue movement, areas of sensitivity, and hair surrounding the site should be taken into account. The range of motion in the soft tissue should be evaluated before capturing impressions. Impressions should be taken with the soft tissue in a natural state, which allows for tightly fitting prosthetic margins. For auricular impressions, having the patient open and close the jaw will demonstrate the range of motion of the temporomandibular joint. This is important for taking into account soft tissue movement during actions such as talking, chewing, and yawning.

An accurate impression material must be used to precisely register the abutments and record soft tissue. Applying two layers of polyvinyl siloxane material will achieve the required exactness. A thin, light-bodied layer is applied followed by a heavy-bodied material to stabilize the impression hardware (Figure 13-38). For the magnetic-attachment type shown in Figure 13-38, the transfer magnet will remain within the impression and be used for registering the laboratory analog abutments within the cast.

The resulting cast from this captured impression will serve as the canvas upon which to build the definitive prosthesis. Many tools are available for the practitioner to use when sculpting and designing the final form. Input and feedback from the patient should be especially encouraged during this stage. The prototype form should be tried on and examined by both patient and anaplastologist.



FIGURE 13-38. **A,** Transfer magnets in location. **B,** Two-layered impression technique.

Prosthetic margins should utilize existing anatomic structures such as wrinkles, scar lines, and hair for disguise. Being a very prominent feature in the middle of the face, a nasal defect may not offer any of these attributes to take advantage of; thin, delicate tapering of margins must be planned for. The boundary of an orbital prosthesis can be effectively disguised behind the edges of the patient's spectacles (Figure 13-39). Careful selection of the frames before prosthetic treatment can result in better camouflaging of the margins.



FIGURE 13-39. Margins of an orbital prosthesis disguised behind the patient's frames.

Nasal Prostheses

For the nasal patient, it is important to ask for photographic references upon which to base the prosthetic design. Both profile and frontal photographs should be employed. This will ensure that the final result looks familiar and recognizable to the patient. Adequate airflow must also be accommodated within the prosthetic design. The required retentive hardware can consume a lot of space on the posterior side of the prosthesis (Figure 13-40). Incorporating sufficient airflow is important for passive breathing and should be tested while the prosthesis is still in its prototype stage.

Color and Tinting of the Prosthesis

Creating realistic skin colors in silicone requires selecting a combination of colorant that mimics the absorption and transmission of human skin.[42] To achieve a high level of realism, several factors must be taken into consideration: opacity/translucency of the silicone, the correct hue (color), value (too dark or too light), and chroma (intensity).[37] Various lighting conditions can alter any of these aspects of color. This phenomenon, known as *metamerism*, affects the camouflaging ability of the prosthesis. Therefore, the light source should be taken into account while mixing a patient's silicone color match. A wide range of color-corrective and daylight-simulating fixtures can be employed.

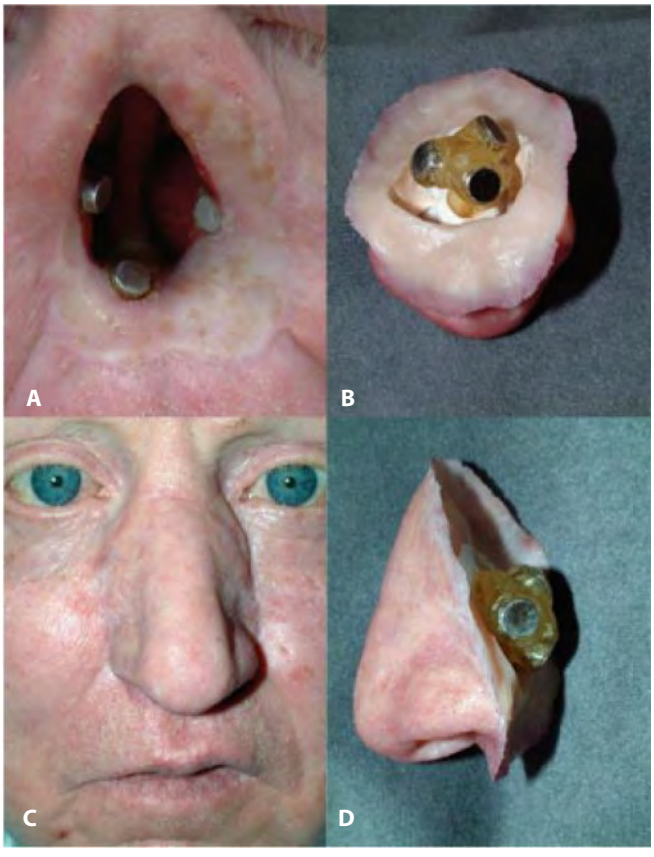


FIGURE 13-40. **A,** Implants placement. **B,** Magnetic attachment and posterior side of the prosthesis. **C,** Final result. **D,** Prosthesis and retentive design from the profile. (**A–D,** Photos courtesy of Edward Ellis III, DDS.)

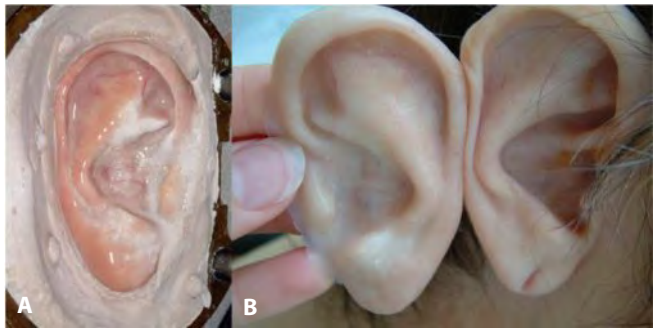


FIGURE 13-41. **A,** Intrinsic colors painted into the mold. **B,** Cured silicone removed from the mold and compared with the patient's skin.

It is important to take into account the patient's skin color as a whole and any skin grafts or scarring in the adjacent soft tissue. If part of the prosthesis will be blending into a darker skin graft, for example, two or more silicone base shades should be mixed for transitioning into each area. Many different glazes, shadow, and highlight colors must be custom-blended to achieve realism (Figure 13-41). When the intrinsic colors are finalized, the silicone should be catalyzed according to the manufacturer's instructions.

The final result should be undetected at a normal conversational distance and release the patient from having his or her condition recognized in social situations. This will transform the patient's self-esteem and overall presentation. Symmetry, organic form, and attention to color detail will contribute to the patient's acceptance of the prosthetic silicone feature.

DISCUSSION

Retention Components

Once the implants have been confirmed to be osseointegrated, the framework for the prosthesis is fabricated. The framework for the maxillofacial prosthesis may be a simple or a complex gold bar. The two types of retention components used are magnets or clips. Prosthetic ears are secured to the underlying bar using clips. Magnets are preferred for ear prosthesis owing to ease of application and cleanliness of the area. Bar and clips do not allow access for cleaning and as good maintenance of the implants. For the isolated prosthetic eye or prosthetic nose, the use of clips or magnets can be considered. For the more complex and larger defect involving a prosthesis that replaces the eye-nose and a portion of the patient's cheek, a combination of clips and magnets may be desired.

Management of the Skin Tissues around the Extraoral Abutments

Minimal soft tissue trauma during the surgical phase of the reconstruction in conjunction with the thinning of the subcutaneous tissues is critical for the long-term maintenance of the extraoral implants. Minimizing motion around the

abutments by application of a pressure dressing will also help in stabilizing the soft tissue adaptation around the extraoral abutments. The use of antibiotics and hydrocortisone applied to the pressure dressing will aid in reducing the inflammation and prevent bacterial colonization.

Long-Term Maintenance

Extraoral Application

In order to increase the long-term success of the extraoral implants, maintenance of healthy cutaneous cuff around the extraoral abutments is crucial. Daily cleansing of the percutaneous abutments using cotton swabs moistened with water and diluted peroxide followed by application of chlorhexadine has shown to be sufficient. A soft-bristled brush may also help in removing debris. It is important to avoid the use of sharp or metal instruments around the percutaneous abutments. The prosthesis should also be cleansed daily using soap and water. Care should be taken to maintain a clean surface in order to avoid damage to the prosthesis. Bacteria and/or fungal colonization on the surface of the prosthetic may cause skin irritation. Sleeping with the prosthesis removed allows ventilation and revascularization around the percutaneous abutments and should be encouraged.

Extraoral cranial implant-retained prosthetic reconstruction has proved to be successful. Replacement of eyes, ears, nose, and larger areas including combined midface defects frequently have no other options available. Burn patients and those with congenital defects are good candidates for this type of reconstruction, especially after autogenous attempts have been inadequate.

In conclusion, use of cranial implant prosthetic reconstruction should be considered in difficult maxillofacial and cranial defects.

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SECTION 3

Maxillofacial Trauma

CHAPTER 14

Initial Management of the Trauma Patient

Michael P. Powers, DDS, MS, and John R. Gusz, MD

The initial assessment and management of a patient's injuries must be completed in an accurate and systematic manner to quickly establish the extent of any injury to vital life support systems. Nearly 25% to 33% of deaths caused by injury can be prevented when an organized and systematic approach is used.[1]

Significant data exists to suggest that death from trauma has a trimodal distribution.[2] The first peak on a linear distribution of deaths is within seconds or minutes of the injury. Invariably, these deaths are due to lacerations of the brain, brainstem, upper spinal cord, heart, aorta, or other large vessels. Few of these patients can be saved, although in areas with rapid transport and some fortunate circumstances, a few of these deaths have been avoided. The second death peak occurs within the first few hours after injury. The period after injury has been called the "golden hour" because these patients may be saved with rapid assessment and management of their injuries. Death is usually due to central nervous system (CNS) injury or hemorrhage. Analysis of trauma system efficacy suggests that trauma deaths could be reduced by at least 10% through organized trauma systems.[3] These patients, whose numbers are significant, benefit most from regionalized trauma care.[3] The third death peak occurs days or weeks after the injury and is usually due to sepsis, multiple organ failure, or pulmonary embolism.[4]

Patients are assessed and treatment priorities are established after an initial evaluation of the patients' injuries and the stability of the vital signs. In any emergency involving a critical injury, logical and sequential treatment priorities must be established on the basis of overall patient assessment. Injuries can be divided into three general categories: severe, urgent, and nonurgent.[2] Severe injuries are immediately life-threatening and interfere with vital physiologic functions; examples are compromised airway, inadequate breathing, hemorrhage, and circulatory system damage or shock. These injuries constitute approximately 5% of patient injuries but represent over 50% of injuries associated with all trauma deaths. Urgent injuries make up approximately 10% to 15% of all injuries and offer no immediate threat to life. These patients may have injuries to the abdomen, orofacial structures, chest, or extremities that require surgical intervention or repair, but their vital signs are stable. Nonurgent injuries account for approximately 80% of all injuries and are not immediately life-threatening. This group of patients eventually requires surgical or medical management, although the exact nature of the injury may not become apparent until after significant evaluation and observation. Laboratory studies, additional physical findings, radiographic examinations, and observation for several days or weeks may be required.[5] The goal of initial emergency care is to recognize life-threatening injuries and to provide lifesaving and support measures until definitive care can be initiated.

ASSESSMENT OF THE SEVERITY OF INJURY

The primary goal of triage is to prioritize victims according to the severity and urgency of their injuries and the availability of the required care. With regional trauma centers in modern trauma systems, the goal of triage is to rapidly and accurately identify patients with life-threatening injuries and to manage these patients with the available resources to achieve the greatest possible outcome, while at the same time avoiding unnecessary immediate transport of less severely injured patients (Figure 14-1).[6–8] Many scales and scoring systems have been developed as tools to predict outcomes based on several criteria.

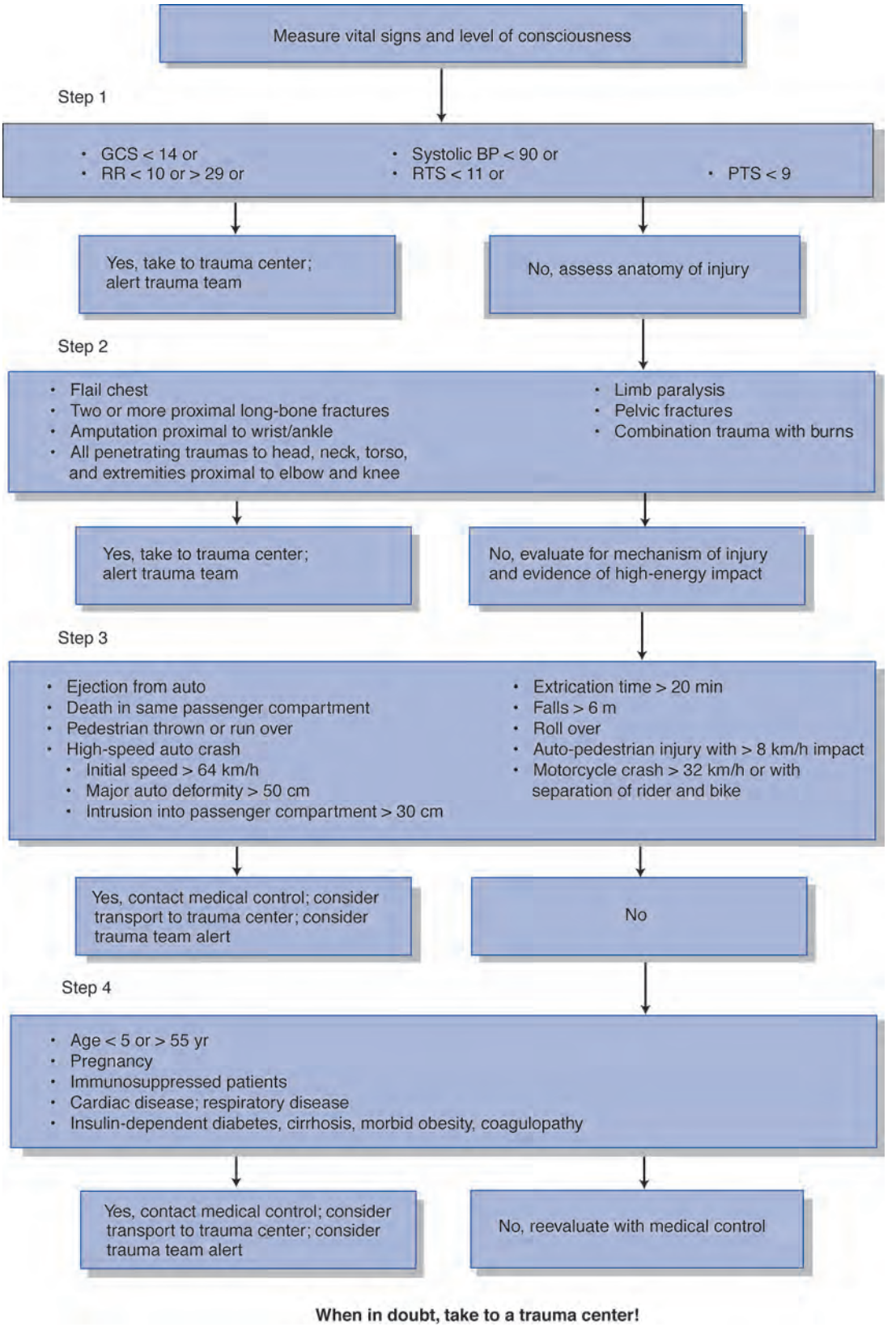


FIGURE 14-1. Triage decision scheme. BP = blood pressure; GCS = Glasgow Coma Scale; PTS = Pediatric Trauma Score; RR = respiratory rate; RTS = Revised Trauma Score. (Adapted from American College of Surgeons Committee on Trauma. Advanced Trauma Life Support for Doctors: Student Course Manual. 6th ed. Chicago: American College of Surgeons; 1997:24.)

TABLE 14-1. **Glasgow Coma Scale**

Action	Score*
Eye Opening	
Spontaneously	4
To speech	3
To pain	2
None	1
Motor Response	
Obeys	6
Localizes pain	5
Withdraws from pain	4
Flexion to pain	3
Extension to pain	2
None	1
Verbal Response	
Oriented	5
Confused	4
Inappropriate	3
Incomprehensible	2
None	1

* Patient's score determines category of neurologic impairment: 15 = normal; 13 or 14 = mild injury; 9–12 = moderate injury; 3–8 = severe injury.
Adapted from Teasdale G, Jennett B. Assessment of coma and impaired consciousness: a practical scale. Lancet 1974;2:81–84.

Glasgow Coma Scale

The Glasgow Coma Scale (GCS) was developed in 1974 by Teasdale and Jennet.[9] It was the first attempt to quantify the severity of head injury. The three variables included were best motor response, best verbal response, and eye opening (Table 14-1). Best motor response is a reflection of the level of CNS function, best verbal response shows the CNS's ability to integrate information, and eye opening is a function of brainstem activity. Scores range from 3 to 15, with a higher number representing an increased degree of consciousness. The use of the letter T designates that the patient was intubated at the time of the examination.

In a prospective multicenter study, patients with a head injury who had an admission GCS of 9 or less correlated with higher mortality rates, regardless of center volume, mechanism of injury, or treatment[10] ; therefore, this scale can be used to predict outcomes. The GCS has weaknesses in that it does not take into account focal or lateralizing signs, diffuse metabolic processes, or intoxication.

Trauma Score and Revised Trauma Score

The Trauma Score was developed by Champion and colleagues[11] to quickly assess the extent of injury to vital systems and the severity of the injury to provide proper triage and treatment of the patient. It was later modified by Champion and coworkers[12] to become the Revised Trauma Score in 1989.

The Trauma Score provided a means of characterizing the physiologic status of injured patients' cardiovascular, respiratory, and neurologic systems. The Trauma Score incorporated five variables: GCS, respiratory rate, respiratory expansion, systolic blood pressure, and capillary refill. The Revised Trauma Score omitted respiratory expansion and capillary refill owing to difficulty assessing these elements in the field and the wide margin for interpretation.

TABLE 14-2. **Revised Trauma Score Variables**

Glasgow Coma Scale	Systolic Blood Pressure (mm Hg)	Respiratory Rate	Coded Value
13–15	> 89	10–29	4
9–12	76–89	> 29	3
6–8	50–75	6–9	2
4 or 5	1–49	1–5	1
3	0	0	0

Adapted from Champion HR, Sacco WJ, Copes WS, et al. A revision of the trauma score. J Trauma 1989;29:623–629.

With the original Trauma Score, the total points added to give a trauma score of 1 to 15; the higher the score, the better the prognosis. Thus, an injured patient who exhibits eye opening to painful stimulus (score 2), a verbal response that is incomprehensible (score 2), and withdrawal from a painful stimulus (score 4) would have a GCS of 8 points and would contribute 3 points to the trauma score.

The Revised Trauma Score has a coded value for each of three variables (Table 14-2). A value of 0 to 4 is assigned for each variable to give a total range of 0 to 12, with lower scores representing an increasing severity of injury. Trauma scores of around 8 indicate an approximate 33% probability for mortality (Table 14-3).[13,14] In 1989, Champion and coworkers[12] performed the Major Trauma Outcome Study, consisting of an analysis of 33,308 trauma patients whose cases were submitted by 89 hospitals across the United States and Canada, with survival probabilities associated with admission trauma scores determined for 25,327 patients. They concluded that patients likely to benefit from prompt diagnosis and definitive care at level I trauma centers are those with an original trauma score of 12 or less.

Injury Severity Score

The Injury Severity Score was developed to deal with multiple traumatic injuries. It compares death rates from blunt

TABLE 14-3. **Predicting Mortality Using the Revised Trauma Score**

Trauma Score	Mortality Rate (%)
12	< 1
10	12
8	33
6	37
4	66
2	70
0	>99

Adapted from Senkowski CK, McKenney MG. Trauma scoring systems: a review. J Am Coll Surg 1999;189:491–503.

TABLE 14-4. **Mortality Rates for Various Injury Severity Scores by Age Groups**

		MORTALITY RATES FOR SCORES (%)				
Age (yr)	*N*	15	25	35	45	55
0–49	1,540	3	8	32	61	89
50–69	316	5	21	56	68	100
70+	109	16	45	82	100	100

Adapted from Powers M. Initial assessment and management of the trauma patient. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 1. Philadelphia: JB Lippincott; 1992; pp. 269–310.

trauma using data that rate the severity of injury in each of the three most severely injured organ systems. Each injury is evaluated and categorized according to the injured organ system (respiratory, CNS, cardiovascular, abdominal, extremities, and skin) and graded according to the severity of the injury: 1 is minor; 2 moderate; 3 severe non–life-threatening; 4 life-threatening, survival probable; 5 survival not probable; 6 fatal cardiovascular, CNS, or burn injuries. The three highest scores for organ systems are then squared and added; the highest possible Injury Severity Score is 108 ($6^2 + 6^2 + 6^2$). Mortality rates have been found to increase with greater severity of injury and age (Table 14-4).[15]

In addition to the field scales that measure abnormal physiologic signs for assessment of injury for triage decisions, mechanism of injury factors and anatomic factors are also important considerations. Mechanism of injury factors can provide insight to a possible significant injury that has not yet resulted in significant changes in vital signs. Although data evaluation is important, it is important to remember that clinical evaluation always trumps mechanism of injury data when the vital signs are stable. Those factors that have a high correlation with life-threatening injuries include the following:[16]

- Evidence of a collision involving high-energy dissipation or rapid deceleration.
- A fall of 6 m or more.
- Evidence that the patient was in a dangerous environment when injured (e.g., a burning building or icy water).
- An automobile accident in which it takes more than 20 minutes to remove the patient, there is significant damage to the passenger compartment, rearward displacement of the front axle has occurred, the patient is ejected from the vehicle, a rollover occurs, or other passengers have died.

Anatomic factors that correlate with mortality include penetrating trauma to the head, neck, torso, groin, or thigh; flail chest; major burns; amputations; two or more proximal long bone fractures; and paralysis. Concurrent disease or factors such as age of younger than 5 years or older than 55 years and known cardiac or respiratory disease may sharply worsen a patient's prognosis, even in the presence of only a moderately severe injury.[17]

The American College of Surgeons Committee on Trauma Subcommittee on Advanced Trauma Life Support has developed a schematic orderly assessment of injured patients. The Advanced Trauma Life Support (ATLS) system consists of rapid primary evaluation, resuscitation of vital functions, a detailed secondary assessment, and finally, the initiation of definitive care (see Figure 14-1).[7]

Other Scoring Systems

Many other scoring systems and tools have been created in attempts to accurately aid triage and to predict outcomes, including the Pediatric Trauma Score,[18] the Trauma and Injury Severity Score,[19] and A Severity Characteristic of Trauma score[20] ; scales using the ninth edition of International Classification of Diseases nomenclature have been implemented including an International Classification of Disease-Based Injury Severity Score.[21]

PRIMARY SURVEY: ABCs

An algorithm for the initial systemic evaluation and stabilization of the multiply injured patient is presented in Figure 14-2. During the primary survey, life-threatening conditions are identified and reversed quickly. This period calls for quick and efficient evaluation of the patient's injuries and almost simultaneous lifesaving intervention. The primary survey progresses in a logical manner based on the ABCs: *a*irway maintenance with cervical spine control, *b*reathing and adequate ventilation, and *c*irculation with control of hemorrhage. Letters D and E have also been added: a brief neurologic examination to establish *d*egree of consciousness and *e*xposure of the patient via complete undressing to avoid injuries being missed because they are camouflaged by clothing. With exposure of the patient, temperature preservation of the patient is extremely important.

Airway Maintenance with Cervical Spine Control

The highest priority in the initial assessment of the trauma patient is the establishment and maintenance of a patent airway. In the trauma patient, upper airway obstruction may be due to bleeding from oral or facial structures, aspiration of foreign materials, facial fractures, airway structure trauma, or regurgitation of stomach contents. Commonly, the upper airway is obstructed by the position of the tongue, especially in the unconscious patient (Figure 14-3). Initially a chin-lift or jaw-thrust procedure may position the tongue and open the airway. The chin-lift procedure is performed by placing the thumb over the incisal edges of the mandibular anterior teeth and wrapping the fingers tightly around the symphysis of the mandible. The chin is then lifted gently anteriorly and the mouth opened, if possible. This method should not hyperextend the neck.[8] The other hand can be used to assist with access to the oral cavity, using the fingers in a sweeping

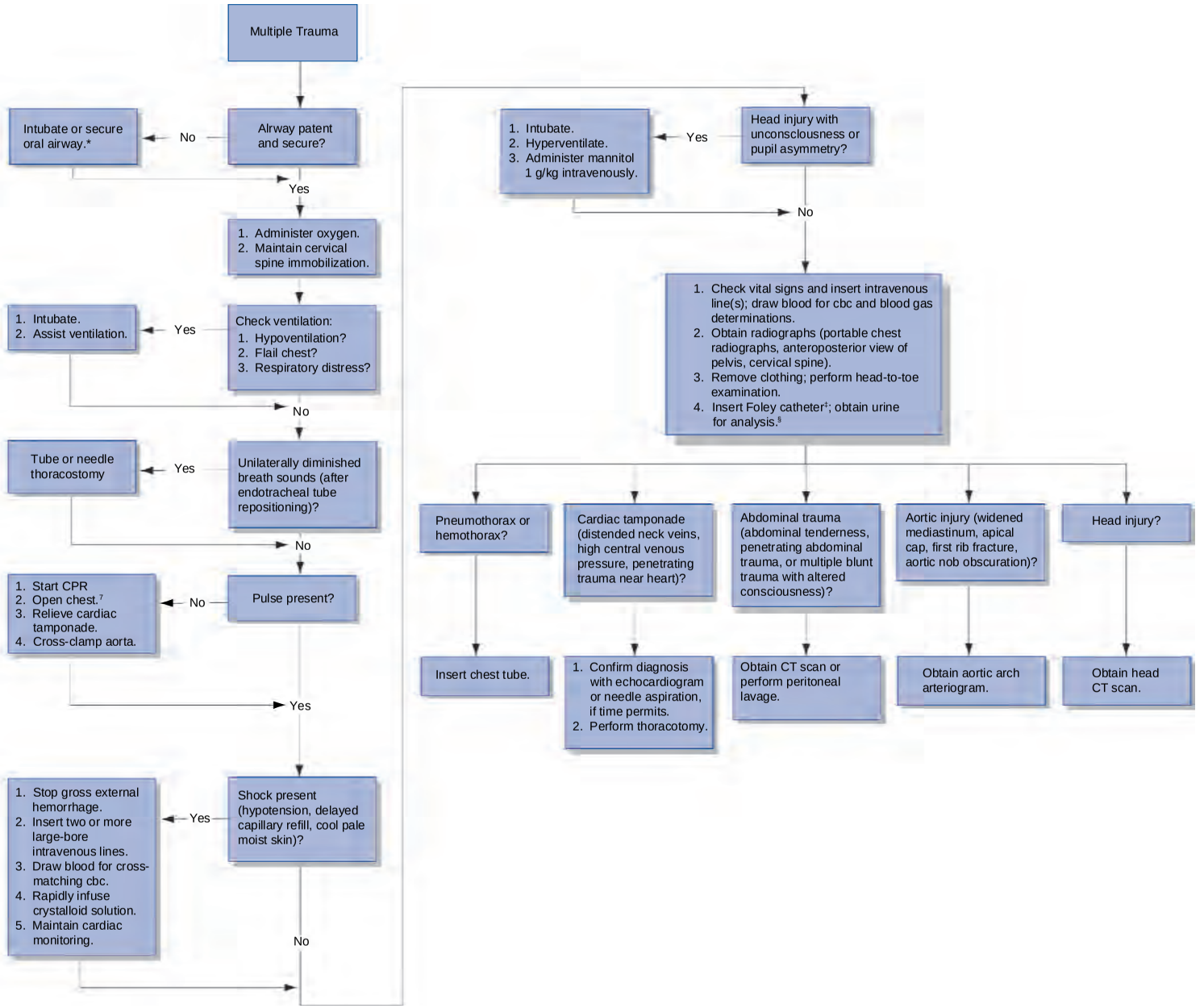


FIGURE 14-2. Multiple trauma algorithm. cbc = complete blood count; CT = computed tomography. *Maintain cervical spine precautions. Nasotracheal intubation (preferred) or orotracheal intubation with axial head traction. † Unlikely to be of benefit for blunt trauma with asystole. Perform only if experienced with the procedure and if there is adequate surgical support. ‡ If not contraindicated (i.e., high-riding prostate, meatal blood, scrotal hematoma). § If not contraindicated (i.e., midface or cribriform plate fracture). (Adapted from Trunkey DD. The Multiply Injured Patient. In Ho M, Saunders CE, editors. Current Emergency Diagnosis and Treatment. 3rd ed. Norwalk, CT: Lange; 1990:43.)

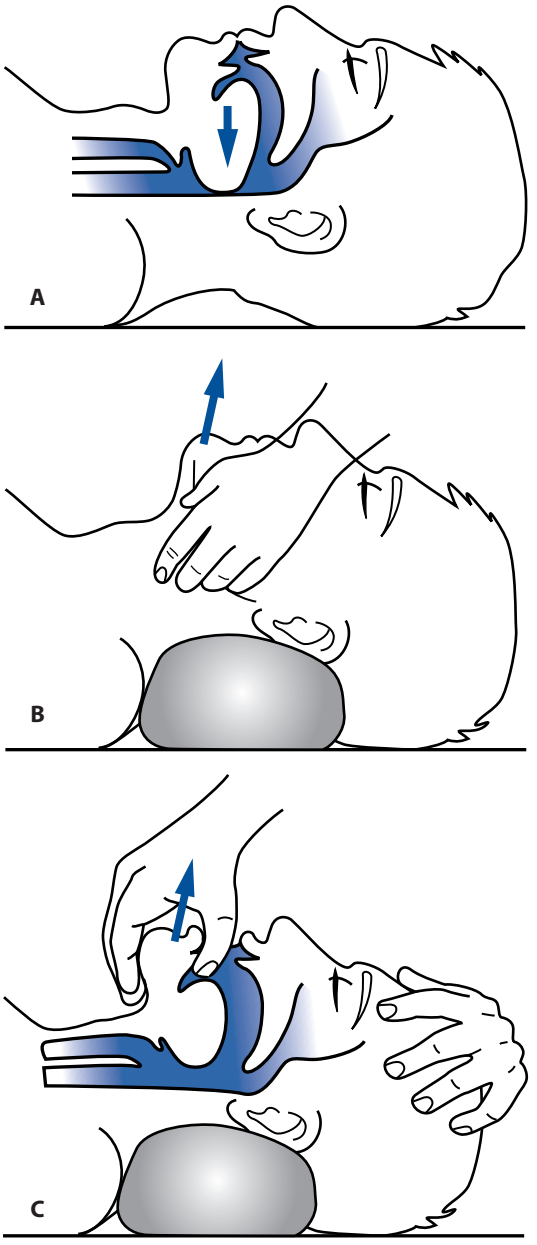


FIGURE 14-3. **A,** Common in the unconscious patient, the tongue drops posteriorly to occlude the airway. This may be especially true in the patient with mandibular fractures because the tongue loses support. A patient with a suspected maxillofacial or head trauma must have the head stabilized at all times to prevent hyperflexion of an injured cervical spine until the possibility of injury has been ruled out. **B,** With the cervical spine stabilized, a jaw-thrust may be used. **C,** A chin-lift procedure also may be helpful to open the airway. (**A–C,** Adapted from Powers M. Initial assessment and management of the trauma patient. In Peterson L J, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 1. Philadelphia: JB Lippincott; 1992; pp. 269–310.)

motion to remove such things as debris, vomitus, blood, and dentures that may be responsible for the obstruction. A tonsillar suction tip is helpful to remove accumulations from the pharynx. Patients with facial injuries who may have basilar skull fractures or fractures of the cribriform plate may, with the routine use of a soft suction catheter or nasogastric tube, be compromised because these tubes may inadvertently be passed into the contents of the cranial vault during attempts at a pharyngeal suction.

The jaw-thrust procedure requires the placement of both hands along the ascending ramus of the mandible at the mandibular angle. The fingers are placed behind the inferior border of the angle, and the thumbs are placed over the teeth or chin. The mandible is then gently pulled forward with the fingers at the angle and rotated inferiorly with pressure from the thumbs. The elbows may be placed on the surface alongside the patient to assist with stability. The jaw-thrust procedure is the safest method of jaw manipulation in a patient with a suspected cervical injury. The jaw-thrust procedure does require two hands, and assistance must be available to clear the debris and other obstructions. After the jaw is opened, it may be possible to place a bite block or large suction device to wedge the teeth open. An oral or nasal airway should be placed to elevate the base of the tongue and to maintain the patent airway.

With any patient sustaining injuries above the clavicle or with decreased levels of consciousness, one should assume there may be a cervical spine injury and avoid hyperextension or hyperflexion of the patient's neck during attempts to establish an airway. Excessive movement of the cervical spine can turn a fracture without neurologic damage into a fracture that causes paralysis. Maintenance of the cervical spine in the neutral position is best achieved with the use of a backboard, bindings, and purpose-built head immobilizers. The use of soft or semirigid collars allows, at best, only 50% stabilization of movement.[22] Cervical spine injury should be assumed present and protected against until the patient can be stabilized and cervical injury can be ruled out during the secondary survey. Oral airway devices are usually preferred with patients with decreased levels of consciousness.

Breathing

With establishment of an adequate airway, the pulmonary status must be evaluated. If the patient is breathing spontaneously—confirmed by feeling and listening for air movement at the nostrils and mouth— supplemental oxygen may be delivered by facemask. The exchange of air does not guarantee adequate ventilation. The chest wall of a patient with a pneumothorax, flail chest, or hemothorax may move but not ventilate effectively. Also, shallow breaths with minimal tidal volumes do not ventilate the lungs effectively. Very slow or rapid rates of respiration usually suggest poor ventilation. The patient's status should be reevaluated constantly. If signs of adequate ventilation deteriorate, a secure airway should be placed (ideally an endotracheal tube) and assisted ventilation should be started. If the patient is not breathing after establishment of an airway, artificial ventilation should be provided with a bag-valve mask or a bag attached to an endotracheal tube. The patient who requires assisted positive-

pressure ventilation from an Ambu bag or ventilator must be carefully monitored if the chest status has not been completely evaluated. Changes in intrathoracic pressure may convert a simple pneumothorax into a tension pneumothorax. The chest should be exposed and inspected for obvious injuries and open wounds. There should be equal expansion of the chest wall without intercostal and supraclavicular muscle retractions during spontaneous respiration. The rate of breathing should be evaluated for tachypnea or other abnormal breathing patterns. Signs of chest injury or impending hypoxia are frequently subtle and include anxiety, an increased rate of breathing, and a change in breathing pattern, frequently toward shallower respirations.[7] The chest wall should also be inspected for bruising, flail chest, and bleeding, and the neck should be evaluated for evidence of tracheal deviation, subcutaneous emphysema, and distended jugular veins. The chest should be palpated for the presence of rib or sternal fractures, subcutaneous emphysema, and wounds. Auscultation of the chest may reveal a lack of breath sounds in an area, suggestive of inadequate ventilation. Distant heart sounds and distended neck veins are suggestive of cardiac tamponade. Arterial oxygen tension (PaN_2) should be maintained between 80 and 100 mmHg. Aside from airway obstruction, the causes of inadequate ventilation in the trauma victim result from altered chest wall mechanics. Open pneumothorax, flail chest, tension pneumothorax, and massive hemothorax are immediate life-threatening conditions and should be quickly identified and treated.

Open Pneumothorax

An *open pneumothorax* is due to a defect in the chest wall, allowing air to be moved in and out of the pleural cavity with each respiration (Figure 14-4). Because of the loss of chest wall integrity, equilibrium develops between intrathoracic pressure and atmospheric pressure. The involved lung collapses on inspiration and slightly expands on expiration, causing air to be sucked in and out of the wound; this is referred to as a *sucking chest wound*. If the opening in the chest wall is approximately two thirds of the diameter of the trachea, air will pass through the path of least resistance—the chest wall defect. With the collapse of the involved lung and a loss of negative pleural pressure, the expired air from the normal lung passes to the involved lung instead of out of the trachea, and it returns to the normal lung on inspiration. This eventually results in a large functional dead space in the normal lung and, combined with loss of the involved lung, may develop into a severe ventilation-perfusion problem.

An open pneumothorax should be treated with coverage of the defect with a sterile occlusive dressing that is secured to the chest on three sides of the dressing. The unsecured side of the dressing acts as a one-way valve, allowing air to escape the pleural cavity on expiration. Secure taping of *all* edges of the dressing results in an accumulation of air within the thoracic cavity and a subsequent tension pneumothorax. Occlusive dressings such as petrolatum gauze may be used as a temporary measure during initial examination or over large defects. A chest tube must be placed in a distant site on the affected chest wall to avoid development of a tension pneumothorax, and the wound must eventually be closed in the operating room. If the lung does not expand after closure of the defect or if signs of poor ventilation persist, the patient should be placed on a ventilator with positive end-expiratory pressure (PEEP) to expand the lung. The patient should be carefully monitored and have a chest tube in place to avoid the development of a tension pneumothorax caused by a tear

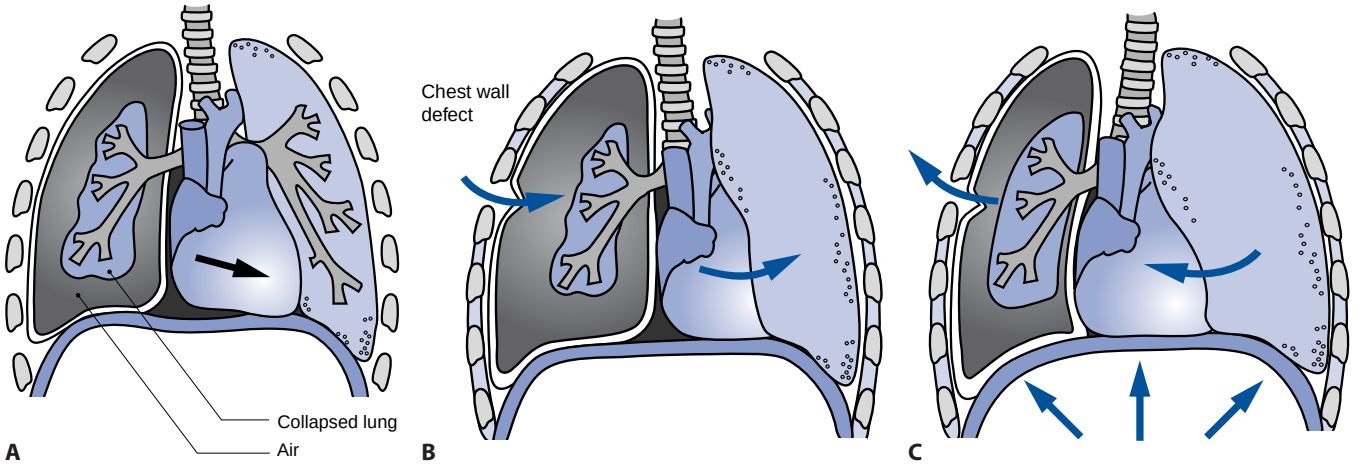


FIGURE 14-4. **A,** A pneumothorax develops from damage to the chest wall or laceration of the lung pleura, with a resulting loss of negative intrapleural pressure. A pneumothorax may be graded as small (15–60%) or large (>60%). **B** and **C,** An open or communicating chest wound occurs when there is an open wound in the chest wall. Air can often be heard moving in and out of the wound during respirations; the condition may be referred to as a *sucking chest wound.* An open pneumothorax may be converted to a simple pneumothorax with the use of an occlusive dressing over the chest wall wound. Care must be taken not to create a trapdoor effect and cause a tension pneumothorax to develop. (**A–C,** Adapted from Powers M. Initial assessment and management of the trauma patient. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 1. Philadelphia: JB Lippincott; 1992; pp. 269–310.)

in one of the bronchi or in the lung parenchyma. Signs of a tension pneumothorax in patients on ventilators include increased airway resistance and diminished tidal volume.

A closed pneumothorax may develop from blunt trauma to the chest or a lung laceration, possibly from a fractured rib. Air escapes from the lung into the pleural space. As the pressures equalize, the affected lung collapses. A ventilation-perfusion deficit occurs because the blood circulated to the affected lung is not oxygenated. With a pneumothorax, percussion of the chest shows hyperresonance. Breath sounds are usually distant or absent; however, in the noisy trauma bay during the initial examination of the victim, breath sounds may be difficult to appreciate. Pneumothorax is confirmed and evaluated with upright chest radiographs. An open pneumothorax that has a dressing placed over the chest wound becomes a closed pneumothorax.

Pneumothoraces that are traumatically induced are usually treated with a tube thoracostomy to correct any respiratory compromise. A small pneumothorax may be treated by hospitalization and careful observation if the patient is otherwise healthy, is symptom free, and does not need general anesthesia or positive-pressure ventilation and if the size of the pneumothorax is not increasing as measured on serial 24-hour chest radiographs.[23,24] This is rarely the case with the trauma victim, and a chest tube should be placed immediately in the multiply injured patient with a pneumothorax. Consideration should also be given to placing a chest tube in patients with chest trauma who will be transferred between facilities, especially by helicopter or airplane (Figure 14-5).

A moderate-sized chest tube (32–40 Fr in adults or 26–30 Fr in children) is generally placed in the fifth intercostal space midaxillary line. The midaxillary line is generally preferred for cosmetic reasons, and if the tube is positioned properly superiorly toward the apex of the lung, it can effectively remove both fluid and air. In initial trauma management, the placement of the chest tube is usually in the fifth intercostal space.

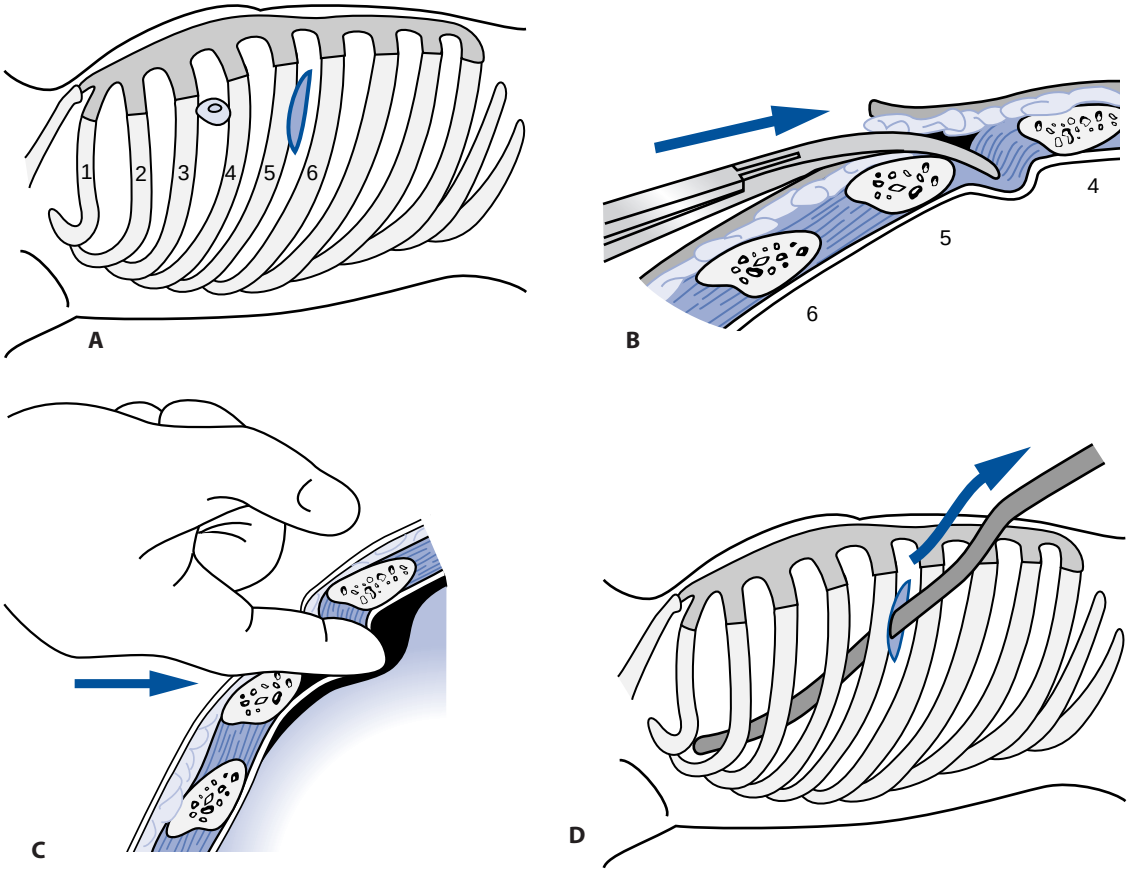


FIGURE 14-5. Chest tube placement. The patient should be supine with the arm positioned superiorly to assist with access to the midaxillary line. **A,** An incision is made through the skin and subcutaneous tissue along the inferior aspect of the fifth rib. **B,** A large Kelly clamp is used, with the tips placed inferiorly, to bluntly dissect over the fifth rib into the intercostal space between the fourth and the fifth ribs. **C,** A gloved finger should be used to enter the pleural space to avoid possible laceration of structures within the pleural space, such as the lung, or possible disruption of abdominal contents in case of a ruptured diaphragm. **D,** The chest tube is then passed along the finger, superiorly and posteriorly within the pleural cavity. The tube should be secured to the chest with sutures, covered with an occlusive dressing, and then connected to an underwater sealed drainage, which creates suction, following verification of tube position by chest radiographs. (**A–D,** Adapted from Powers M. Initial assessment and management of the trauma patient. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 1. Philadelphia: JB Lippincott; 1992; pp. 269–310.)

A skin incision of approximately 3 cm in length is made one intercostal space below the intended placement of the tube. If the tube is to be placed through the fifth intercostal space, an incision is made through the skin along the sixth intercostal space. A gloved finger is used to tunnel transversely through the subcutaneous tissue to the inferior margin of the fifth rib. The intercostal muscles are separated with a large Kelly clamp, and the chest tube is inserted over the superior margin of the fifth rib to avoid the neurovascular bundle traveling on the inferior margin of the fourth rib and advanced superiorly and posteriorly into the pleural cavity. The tube should be secured to the skin with sutures, and an occlusive dressing should be used to cover the defect around the tube. The tube is then connected to an underwater sealed drainage to remove the air or fluid. Upright posteroanterior and lateral chest radiographs should be taken to confirm the position of the chest tube, the position of the last drainage hole on the tube, and the position and amount of air or fluid remaining in the pleural cavity. Daily physical examination and radiographs should be performed to monitor progress of removal of air or fluid. If the tube becomes blocked and significant fluid or air remains, a new chest tube should be placed.

Tension Pneumothorax

A *tension pneumothorax* develops when the injury acts as a one-way valve through the chest wall or from the lung into the pleural cavity without equilibration with the outside atmosphere (Figure 14-6). A dangerous progressive increase of intrapleural pressure develops as air enters the pleural cavity on inspiration but cannot escape on expiration, causing complete collapse of the affected lung. As the pressure increases, the trachea and mediastinum are displaced to the opposite pleural cavity and impinge on the normal lung. The positive intrapleural pressure compresses the vena cava, leading to decreased cardiac output. The compression of the normal lung causes shunting of blood to nonventilated areas and severe ventilatory disturbances. These changes develop into a rapid onset of hypoxia, acidosis, and shock.[24]

The most common causes of tension pneumothorax are mechanical ventilation with PEEP, spontaneous pneumothorax in which emphysematous bullae have failed to seal, and blunt chest trauma in which the parenchymal lung injury has failed to seal. Occasionally, traumatic defects in the chest wall may lead to tension pneumothorax.[7] The presence of a pneumothorax should be considered in patients who rapidly become acutely ill; develop severe respiratory distress; and exhibit decreased breath sounds, hyperresonance on one side of the chest, distended neck veins, and deviation of the trachea away from the involved side. If untreated, a tension pneumothorax results quickly in death. If a developing tension pneumothorax is suspected, the positive intrapleural pressure should be released as quickly as possible. The pressure can be released by inserting a large-bore needle (14–16 gauge) anteriorly into the affected hemithorax through the second or third intercostal space in the midclavicular line. This quickly converts the tension pneumothorax to a pneumothorax, which can be treated with placement of a chest tube (Figure 14-7).

Hemothorax

Hemothorax is the collection of blood in the pleural cavity. It is commonly the result of penetrating injuries that disrupt the vasculature, but it can result from blunt trauma that tears the vasculature. The initial loss of blood collected in the pleural cavity may come from lung injuries, but because of

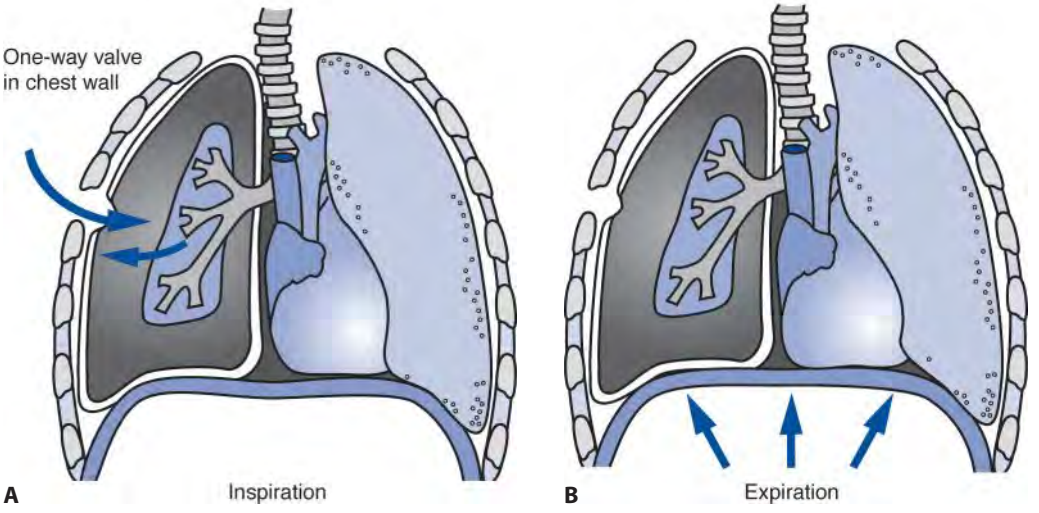


FIGURE 14-6. A tension pneumothorax develops as air enters the pleural cavity on inspiration but cannot leave during expiration **(A),** resulting in a progressive increase in intrapleural air pressure **(B).** The injury in the chest wall or trachea acts like a one-way valve, and the increasing intrapleural pressure results in a shift of the trachea and mediastinal structures away from the injury. The pressure on the vena cava does not allow for an adequate return of blood to the heart, and compression of the opposite lung (added to the injured lung) causes severe ventilatory disturbance. (**A** and **B,** Adapted from Vukich DJ, Markovchick VJ. Pulmonary and chest wall injuries. In Rosen R, editor. Emergency Medicine: Concepts and Clinical Practice. St. Louis: CV Mosby; 1988.)

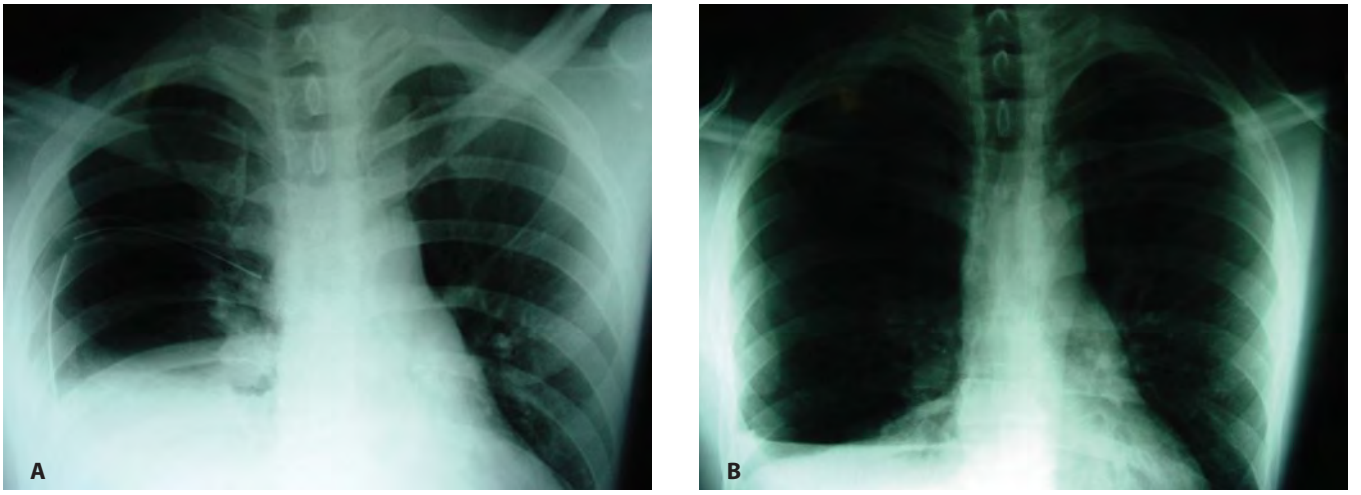


FIGURE 14-7. **A,** Right pneumothorax. A closed pneumothorax may develop from blunt trauma to the chest or a lung laceration, possibly from a fractured rib. Air from the lung to the pleural space equalizes the pressures, and the lung collapses. A ventilation-perfusion deficit occurs because the blood circulated to the affected lung is not oxygenated. With a pneumothorax, percussion of the chest shows hyperresonance. Breath sounds are usually distant or absent. Management of the pneumothorax is confirmed and evaluated with upright chest radiographs. **B,** Right pneumothorax after chest tube placement. A chest tube should be placed immediately in the multiply injured patient with a pneumothorax. A moderate-sized chest tube (32–40 Fr in adults or 26–30 Fr in children) is generally placed either anteriorly in the second intercostal space midclavicular line or in the fourth or fifth intercostal space midaxillary line.

low pulmonary arterial pressure, the blood loss is usually slowed. Massive hemothorax usually results from injuries to the aortic arch or pulmonary hilum; it may also result from injuries to the internal mammary arteries or intercostal arteries, which are branches of the aorta. A hemothorax may dangerously reduce the vital capacity of the lung and contribute to hypovolemic shock. A hemothorax is usually associated with a pneumothorax, and the subsequent blood loss causes hypotension, a decreased cardiac output, and metabolic acidosis, which when combined with the ventilatory compromise, results in hypoxia and respiratory acidosis.

A hemothorax should be suspected after penetrating or blunt chest trauma if the patient is in shock with reduced breath sounds and with a chest dull to percussion on one side. The neck veins may be flat because of severe hypovolemia or distended as a result of the mechanical effects of a chest full of blood.[7] With the loss of a small amount of blood (<400 mL), the diagnosis is difficult because there may be little or no change in the patient's appearance, vital signs, or physical findings. Fluid collections greater than 200 to 300 mL can usually be seen on a good upright chest radiograph as blunting of the costophrenic angle. The supine radiograph is less accurate.[24]

Treatment of a hemothorax consists of restoration of the circulating blood volume with transfusion of fluids or blood products through large-bore intravenous lines; control of the airway and support of the ventilation as required; and drainage of the accumulated blood from the pleural cavity. A large chest tube (36–40 Fr) should be inserted in the fifth or sixth intercostal space in the midaxillary line and directed posteriorly and superiorly to avoid damage to a possibly elevated diaphragm. The chest tube should be connected to an underwater seal and steady suction (20–30 cmH_2O). If the chest tube becomes clotted and fails to drain, another chest tube should be placed rather than an attempt made to irrigate the first tube. With massive bleeding, autotransfusion of the drained blood is possible until banked blood is available.[25]

Persistent hemorrhage requires surgical exploration. Thoracotomy for intrathoracic bleeding is indicated for the following: initial thoracostomy tube drainage greater than 1500 mL of blood; persistent bleeding at a rate greater than 200 mL; increasing hemothorax seen on chest radiographic studies; or persistant hypotension despite blood replacement with other sites of blood loss having been ruled out or the patient decompensating after an initial response to resuscitation.[24] In a few instances, emergency thoracotomy in the emergency room may be necessary for control of blood loss. However, mortality from this procedure is very high.

Flail Chest

A *flail chest* results when there are multiple rib fractures, usually at several sites along the rib (Figure 14-8). The resulting unstable segment of chest wall moves paradoxically during respirations—inward with inspiration and outward with expiration. A flail chest may affect respiratory ability to the point at which hypoxemia occurs. The pain associated with the respiratory effort may also compromise the ventilatory compliance of the patient. The fractured ribs may have punctured the lung, causing a tension pneumothorax or hemithorax. A problem with flail chest and hypoxemia is the underlying pulmonary contusion from the injury. The contused lung may be asymptomatic in the initial presentation but develop complications later with gas exchange. Little abnormal breathing may be apparent immediately after the injury. Later, as fluid moves into the lung with the developing

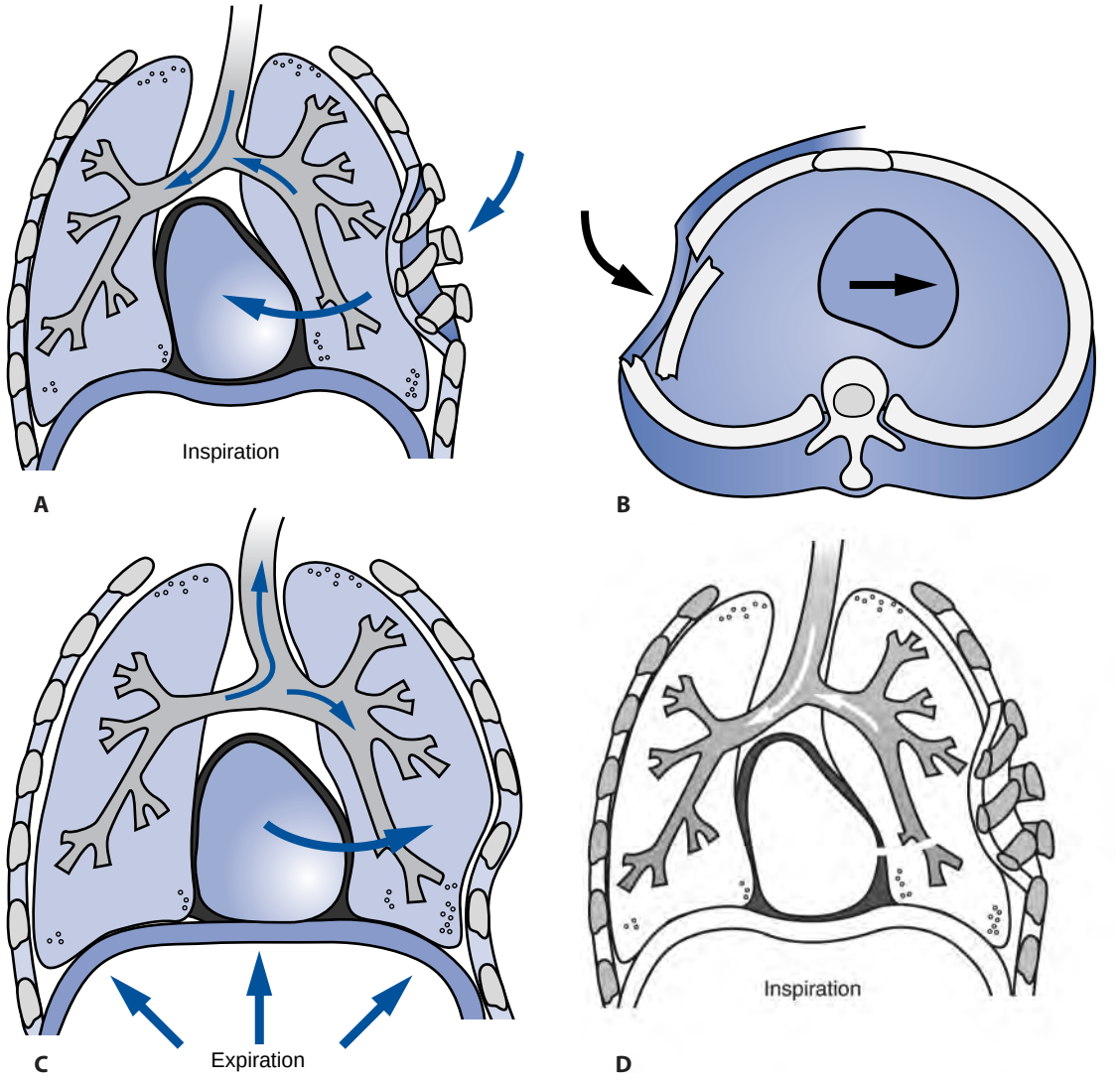


FIGURE 14-8. A flail chest occurs when three or more adjacent ribs are fractured in at least two locations, resulting in a freely moving segment of the chest wall during respirations. The chest wall moves paradoxically during inspiration and expiration owing to the flail segment. **A,** Upon inspiration, the flail segment sinks inward as the chest wall expands, impairing the ability to produce negative intrapleural pressure. **B,** The heart and other contents of the mediastinum shift toward the noninjured side. **C** and **D,** During expiration, the flail segment is pushed outward, and the chest wall cannot efficiently force air from the lungs. Air may shift uselessly from lung to lung. (**A–D,** Adapted from Vukich DJ, Markovchick VJ. Pulmonary and chest wall injuries. In Rosen P, editor. Emergency Medicine: Concepts and Clinical Practice. St. Louis: CV Mosby; 1988; p. 477.)

contusion, lung compliance falls, and more pressure is needed to inflate the lungs. The pulmonary contusion underlying major chest wall injuries may be the primary cause of hypoxia and morbidity in patients with flail chest. Mortality in patients sustaining severe blunt chest trauma remains relatively high at 12% to 50%.[26]

A flail chest is usually apparent on visual examination of the unconscious patient. It may not be initially apparent in the conscious patient because of splinting of the chest wall. The patient moves air poorly as a result of paradoxical breathing, and movement of the thorax is asymmetrical and uncoordinated. The region of the fractures may be tender to palpation.

Recommended management of flail chest involves three treatment considerations—pain management, supplemental oxygen delivery, and ventilation control. Prolonged relief is best obtained with intercostal nerve blocks to block the pain from the fractured ribs, thereby allowing the patient to breathe deeply and cough. The use of narcotic medications must be limited to avoid respiratory depression. A volume-cycled respirator with endotracheal intubation is indicated to provide PEEP ventilation and intermittent mechanical ventilation (IMV). This "internal splinting" with ventilatory support effectively manages the inadequate depth of ventilation, improves oxygen absorption in the segments of pulmonary contusion, and decreases atelectasis. If proper management with ventilatory assistance is initiated early, the respiratory support may be required for only 2 to 4 days. If management is delayed until the patient demonstrates

respiratory difficulty, prolonged therapy for up to 14 days may be necessary.[26] Care and constant monitoring of the patient is always important but especially with the use of mechanical ventilation that may cause further injury resulting in pneumothorax, airway injury, alveolar damage, or ventilator-associated pneumonia.

Oxygenation

After establishment of a patent airway and sustained breathing, the patient should be given supplemental oxygen. The patient may have diminished oxygen-carrying capacity as a result of injuries to the pulmonary or cardiovascular system; respiratory compromise may be due to a head injury and disruption of cerebellar reflex systems, airway distress from maxillofacial or neck injuries, or pulmonary injuries such as pulmonary contusion, flail chest, and a tension or open pneumothorax that mechanically prevents the proper delivery of oxygen to the cardiovascular system. Oxygen should be delivered through a facemask or endotracheal tube. A person breathing 100% oxygen can move five times more oxygen into the alveoli with each breath as when breathing normal air. Oxygen therapy can increase available oxygen by as much as 400% above normal.[27]

Administered oxygen can increase the inspired oxygen to 8 L/min and can increase the fraction of inspired oxygen (FiO_2). A higher FiO_2 can be delivered by a Venturi mask, with the proper application of a bag-and-mask system. The greatest difficulty with this system is maintaining an adequate seal between the mask and the face. The thumb and index finger are placed over the mask to hold the mask securely over the mouth and nose, and the other fingers are curled beneath the inferior border of the mandible. The FiO_2 can be increased in a bag-and-mask system with a rebreathing mask and an oxygen accumulator to deliver a high concentration of oxygen. Ventilation with the bag-and-mask system is difficult in patients with possible maxillofacial, cervical spine, or thoracic injuries, and the patient's airway must be stabilized with either endotrachial intubation or tracheotomy.

Endotracheal intubation helps to protect the airway and facilitates adequate lung inflation with high FiO_2 in the injured patient. Oxygen administered through the endotracheal tube should be increased to an FiO_2 of 100% (especially if the patient is comatose) until arterial blood gas measurements confirm hemoglobin saturation (PaN_2 > 60–70 mmHg), at which point FiO_2 can be lowered to between 40% and 60%.[28] Pulmonary oxygen toxicity may result if 100% oxygen is administered continuously for 24 hours; therefore, 100% oxygen delivery is acceptable only until PaN_2 levels can be ascertained. Some concern exists about the suppression of the respiratory drive with oxygen therapy, but the hypoxic drive can be reestablished after stabilization of the injured patient.

The most important mechanism of delivery of oxygen to the tissues is the hemoglobin within the erythrocytes in the cardiovascular system. In a traumatized patient, hemorrhage may decrease the available hemoglobin to the point of hypooxygenation of vital organ tissues and cell death. A normal hemoglobin of 15 g/100 mL provides transport of 20% volume of oxygen, whereas a hemoglobin of 7 g/100 mL carries only a 10% volume of oxygen, which is the critical reserve level of oxygen consumption for most tissues, especially the myocardium and brain.[27] The treatment of shock in the patient with multisystem injuries is directed toward restoring cellular and organ perfusion with adequately oxygenated blood, rather than merely restoring the patient's blood pressure and pulse rate.[8]

Circulation

Following establishment of an adequate airway and breathing in the injured patient, the cardiovascular system of the patient must be assessed and control of baseline circulation to the tissues must be quickly restored. The most common cause of shock in the traumatized patient is hypovolemia caused by hemorrhage, either externally or internally into body cavities. Assessment of the degree of shock is important because inadequate tissue perfusion can cause irreversible damage to vital organs such as the brain or kidneys in a short time period. During the primary assessment, a minimum of two large-bore (14–16 gauge) intravenous catheters should be placed peripherally if fluid resuscitation is required. At the time of placement of an intravenous catheter, blood should be drawn from the catheter to allow for typing, cross-matching, and baseline hematologic and chemical studies. If there is any doubt of adequate ventilation, arterial blood should be obtained for blood gas analysis.

Tissue perfusion and oxygenation are dependent on cardiac output and are best initially evaluated by physical examination of skin perfusion, pulse rate, and mental status of the patient. Blood pressure levels are commonly used as a surrogate for cardiac output and to suspect hypovolemia, but in the emergency situation, blood pressure measurement may be an unreliable indicator of developing shock. The response of the blood pressure level to intravascular loss is nonlinear because compensatory mechanisms of increased cardiac rate and contractility, along with venous and arteriolar vasoconstriction, maintain the blood pressure in the young healthy adult during the first 15% to 20% of intravascular blood loss. After a blood loss of 20%, the blood pressure level may drop significantly. (In the elderly patient with less efficient compensating mechanisms, the decline in blood pressure levels may begin to develop after a 10% to 15% blood loss.) The patient may arrest at an intravascular blood loss of 40%.[29]

Blood pressure level may be insensitive to the early signs of shock, and a patient's blood pressure level may quickly drop after the initial assessment because compensatory mechanisms can no longer keep up with intravascular volume loss. Also, the usual baseline blood pressure level of the patient is often unknown. A patient who has a systolic pressure of 120 mmHg but is normally hypertensive may have a significant loss, whereas a healthy young athlete may have a

normal systolic pressure of 90 mmHg and the blood loss might be assumed to be greater than it is.

Skin perfusion is the most reliable indicator of poor tissue perfusion during the initial evaluation of the patient. The early physiologic compensation for volume loss is vasoconstriction of the vessels to the skin and muscles. The cutaneous capillary beds are one of the first areas to shut down in response to hypovolemia because of stimulus from the sympathetic nervous system and the adrenal gland through epinephrine and norepinephrine release. The release of the catecholamines causes sweating, and during palpation, the skin may feel cool and damp. The lower extremities are usually first to be affected, and the first indication of intravascular loss may be paleness and coolness of the skin over the feet and kneecaps. A check of the capillary filling time by performing a blanch test gives an estimate of the amount of blood flowing to the capillary beds. In this test, pressure is placed on the fingernail, toenail, or hypothenar eminence of the hand (to evacuate blood from the capillary beds), followed by a quick release of the pressure. The time required for the blood to return to the capillary beds, represented by the restoration of normal tissue color, is usually less than 2 seconds in the normovolemic patient. This indicates that the capillary beds are receiving adequate circulation.[30]

The rate and character of the pulse should be assessed and are a good measure of the cardiac rate. The pulse rate is a more sensitive measure of hypovolemia than is the blood pressure, but it is affected by other factors commonly associated with the trauma situation, such as the patient's pain, excitement, and emotional response, resulting in tachycardia without underlying hypovolemia. However, in adults with tachycardia greater than 120 beats per minute (bpm), hypovolemia should be expected and investigated further. Older patients generally are unable to exceed rates of 140 bpm in a hypovolemic state, whereas younger patients may present rates of 160 to 180 bpm with severe intravascular loss. In patients who have pacemakers, are taking heart blocking medications such as propranolol or digoxin, or have conduction abnormalities within the heart, hypovolemia may not cause tachycardia.

The most distal palpable pulse may give some indication of the blood pressure and cardiac output. Generally, if the radial pulse is palpable, the patient's systolic blood pressure is greater than 80 mmHg; if the femoral pulse is palpable, the patient's systolic blood pressure is 70 mmHg or higher; and if the carotid pulse is noted, the systolic blood pressure is greater than 60 mmHg. Pulse rhythm and regularity may also provide clues to increasing hypovolemia and cardiac hypoxia. Cardiac dysrhythmias such as premature ventricular contractions or arterial fibrillation produce an irregular rate and rhythm, signaling the potential loss of compensating mechanisms maintaining myocardial oxygenation.

Decreased intravascular volume is immediately reflected in decreased urinary output because the compensatory mechanisms of the body decrease blood flow to the kidneys in favor of blood flow to the heart and brain. Any patient with significant trauma should always have an indwelling urinary catheter inserted to monitor urine volume every 15 minutes.[29] A minimally adequate urine output is 0.5 mL/kg/hr, and fluid therapy should be initiated to maintain at least this level of urinary output. If the patient's injuries include pelvic fractures or blunt trauma to the groin, a urinary catheter should not be placed until the urethra has been evaluated for injury. If urethral injury is unlikely, the urinary catheter may be placed with minimal concern after a rectal examination. Classic signs of urethral injury include blood at the meatus, scrotal hematoma, or a high-riding boggy prostate on rectal examination.

Alterations in the mental status of the trauma patient caused solely by hypovolemia are uncommon, except in the most progressive preterminal stages of intravascular fluid loss. Compensatory mechanisms maintain blood flow to the brain, and hypoperfusion to the brain does not develop until the systolic blood pressure falls below 60 mmHg. The mental changes usually seen are agitation, confusion, uncooperativeness, anxiety, and irrationality. These alterations in mental status can also be seen in a patient with head trauma, spinal injury, drug or alcohol intoxication, hypoxia, or hypoglycemia. In the emergency situation, these other causes of mental status changes should be investigated when hypovolemia is suspected in the agitated patient who has suffered or possibly has substantial blood loss.[29]

Hypovolemia caused by hemorrhage may commonly cause flat neck veins. Distended neck veins, however, suggest either tension pneumothorax or cardiac dysfunction. As discussed earlier, with tension pneumothorax, an examination of the chest may reveal absent breath sounds and a hyperresonant chest. Cardiac dysfunction results from cardiac tamponade, myocardial contusion or infarction, or an air embolus.

Cardiac tamponade presents a clinical picture similar to that of tension pneumothorax—distended neck veins, decreased cardiac output, and hypotension. Blunt or penetrating trauma may cause blood to accumulate in the pericardial sac. The blood in the pericardial sac results in inadequate cardiac filling during diastole, diminished cardiac output, and circulatory failure. Cardiac tamponade usually is associated with penetrating wounds to the chest that have injured the tissues of the heart. The classic Beck's triad of decreased systolic blood pressure levels, distended neck veins, and muffled heart sounds may be observed, although all three are rarely seen together. The expected distended neck veins caused by increased central venous pressure may be absent because of hypovolemia. The neck veins, if distended, may become distended further during inspiration (Kussmaul's sign), and pulsus paradoxus (lowering of the systolic pressure by > 10 mmHg on normal inspiration) may be accentuated or absent. Tension pneumothorax may mimic cardiac tamponade or, because of the nature of the penetrating injury, may develop at the same time as cardiac tamponade, thus presenting a confusing clinical presentation.

Cardiac tamponade is initially managed by prompt pericardial aspiration through the subxiphoid route (Figure 14-9). Radiographs and physical examination may be not

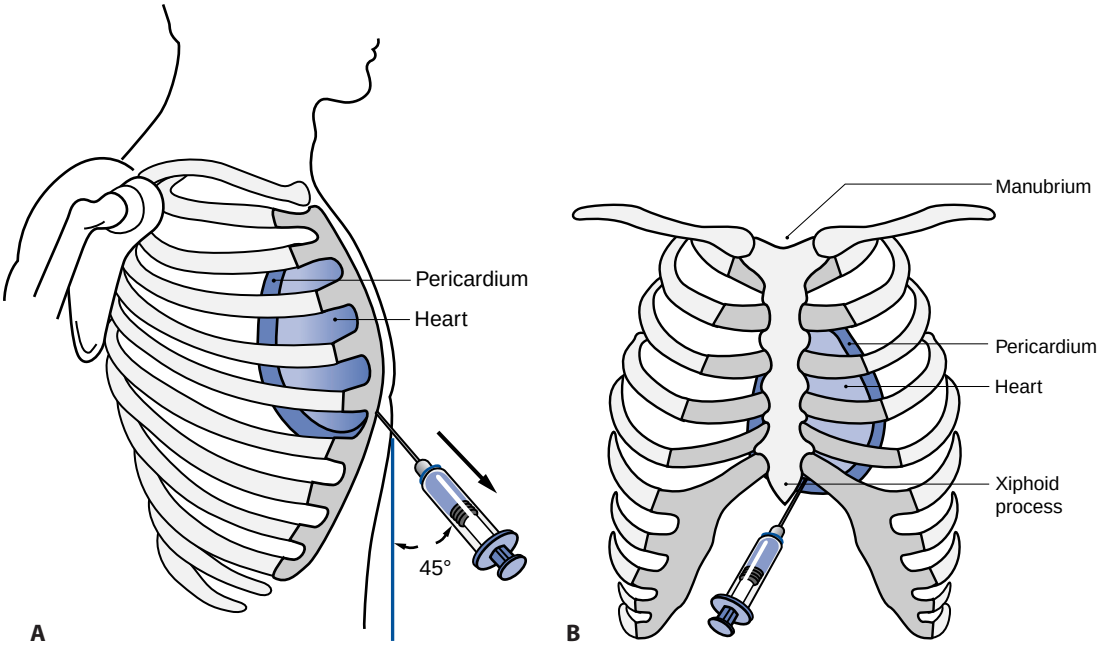


FIGURE 14-9. Pericardiocentesis can be transiently lifesaving when a significant cardiac tamponade develops. **A** and **B,** The patient is placed in a supine position, and a 16- or 18-gauge needle on a 60-mL syringe is introduced just to the left side of the xiphoid process. The needle should be introduced at a 45-degree angle to the chest wall, 45 degrees off the midline and directed toward the posterior aspect of the left shoulder. A popping sensation may be felt as the pericardium is entered. If the blood within the pericardial sac is slightly clotted, it may interfere with the effectiveness of the procedure. Relief of a depressed systolic blood pressure level should be immediate, resulting from an increased stroke volume. The procedure may be required several times until definitive treatment can be initiated. (**A** and **B,** Adapted from Powers M. Initial assessment and management of the trauma patient. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 1. Philadelphia: JB Lippincott; 1992; pp. 269–310.)

helpful in the diagnosis of a cardiac tamponade. A fast scan ultrasound may provide evidence of pericardial fluid, but a high index of suspicion may be the best asset in the diagnosis of a developing cardiac tamponade. A positive pericardial aspiration along with a history of chest trauma is frequently the only method of making a correct diagnosis. Because of the self-sealing qualities of the myocardium, aspiration of pericardial blood alone may temporarily relieve symptoms. All trauma patients with a positive pericardial aspiration require open thoracotomy and inspection of the heart. Pericardial aspiration may not be diagnostic or therapeutic if the blood in the pericardial sac has clotted, as occurs in 10% of patients with cardiac tamponade.[29] If aspiration does not lead to improvement of the patient's condition, only emergent thoracotomy can solve the problem unless another injury to the patient has been overlooked.

Pericardial aspiration through the subxiphoid route involves the insertion of a needle, preferably covered by a plastic catheter (angiocatheter), at 90 degrees slightly to the left of the xiphoid process. The needle is inserted until it clears the sternal border and is then directed at 45 degrees toward the left scapular tip to directly enter the pericardium. Suction is placed on the needle hub to identify by blood return when the needle has entered the pericardial sac. If the needle is properly placed, as little as 50 mL of blood from the pericardial sac should result in a marked improvement in the patient's condition.

Control of Bleeding

Hemorrhage is defined as an acute loss of circulating blood. Normally, the blood volume is approximately 7% of the adult ideal body weight. A 70-kg male has approximately 5 L of circulating blood. The blood volume does not increase significantly in obese patients, and in children, the blood volume is usually between 8% and 9% of body weight (80–90 mL/kg).[7] Bleeding may be external or internal into body cavities. Most external hemorrhage can be controlled with direct pressure to the wound. If an extremity is involved, it should be elevated. Firm pressure should be continuous, and if the dressings become soaked, they should not be removed but, rather, covered with additional dressings. Removal of a dressing may disrupt clot formation and promote further bleeding. Firm pressure on the major artery in the axilla, antecubital fossa, wrist, groin, popliteal space, or ankle may assist in control of hemorrhage distal to the site. Pressure points should be used only if direct wound pressure alone is not effective. Pressure bandages include the use of air-pillow splints and blood pressure cuffs. Pneumatic antishock garments (PASGs) and medical (military) antishock trousers (MASTs) previously used to increase blood pressure in cases of hypotension have been found to be detrimental in some situations such as instances of vascular injuries.[31] The PASG/MAST garments are still used by some to stabilize pelvic fractures. Scalp or skin wounds may best be managed with immediate closure with large monofilament sutures (without

cosmetic closure considerations) and direct pressure until the hemorrhage is controlled.

Because of the rich blood supply to the head and neck, significant hemorrhage may be associated with large scalp wounds, nasal or midface fractures, and penetrating neck wounds. In a short period of time, the scalp may lose a large amount of blood, which oozes from the galea and loose connective tissue layers. The wound can be approximated rapidly with 2-0 nonresorbable sutures without regard to cosmetic closure. Direct pressure should then be placed over the wound to control the hemorrhage and minimize hematoma formation. After the patient has been stabilized, the sutures may be removed and a more cosmetic approach with resorbable sutures may be used to close the galeal layer and to achieve good approximation and orientation of the hair-bearing dermal and skin layers.

Nasal or midface fractures may hemorrhage from tears of the ethmoidal arteries that arise from the internal carotid system or from branches of the maxillary artery system (Figure 14-10). Most hemorrhages from facial injuries can be controlled with direct pressure or packing (Figure 14-11). Internal maxillary artery bleeding from posterior maxillary wall fractures associated with Le Fort I or II level fractures usually can be controlled by pressure with gauze packing for extended periods. Liquid thrombin or epinephrine may be added to the gauze packing, and the patient's head may be elevated to assist with hemostasis. If direct control is necessary, good visualization of the damaged vessel is required. Blind clamping may cause further bleeding from vessels and soft tissues, as well as nerve damage. Ligation of the external carotid artery may be required only in extreme cases; usually, it is ineffective when used alone and without direct control of hemorrhage because of the collateral circulation of the face.

The potential internal sites of hemorrhage are the thoracic cavity, abdomen, retroperitoneum, and extremities. A complete physical examination with radiography and computed tomography (CT) is useful to identify hemorrhages into these areas (Figures 14-12 and 14-13). When there is no evidence of external or intrathoracic bleeding, continued severe hypovolemia is usually the result of bleeding into the abdomen or at fracture sites. Blood loss with fractures should be considered to be at least 1000 to 2000 mL for pelvic fractures, 500 to 1000 mL for femur fractures, 250 to 500 mL for tibia or humerus fractures, and 125 to 250 mL for fractures of smaller bones. A hematoma the size of an apple usually contains at least 500 mL of blood. Control of hemorrhage into internal spaces is not done in the primary survey unless the hemorrhage may have damaging effects on the cardiovascular or pulmonary system. Slow internal hemorrhage may be controlled by splinting, casting, or fixation of fractures; by the defense mechanisms of vascular occlusion, retraction, and clot formation; or by open exploratory surgery.

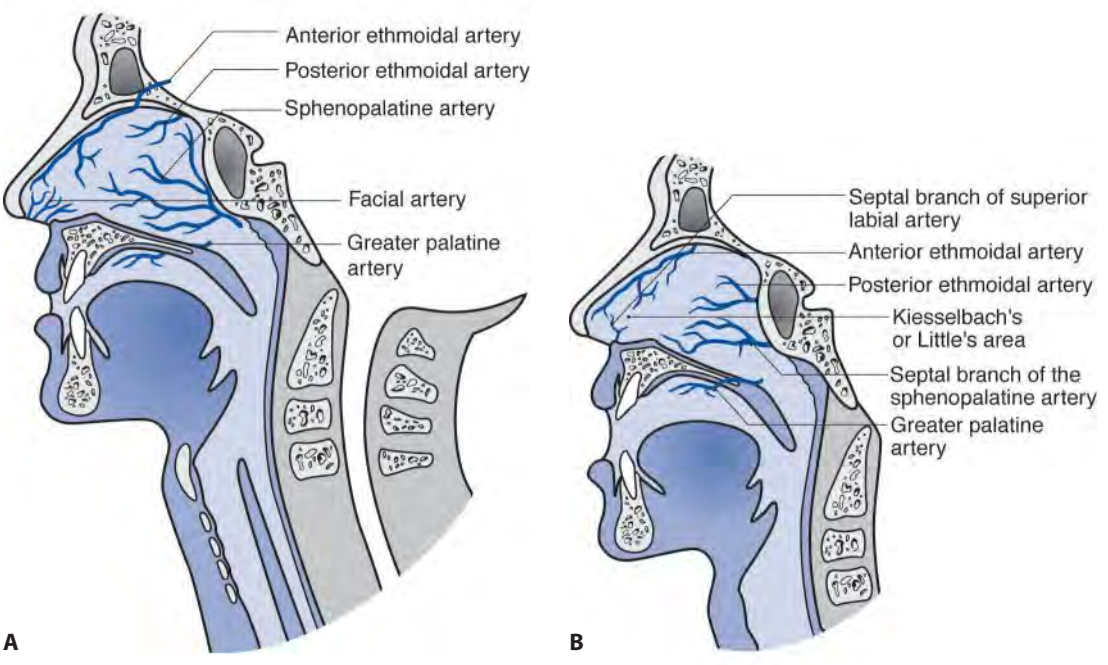


FIGURE 14-10. The lateral wall of the nasal cavity **(A)** and the nasal septum **(B)** receive a rich blood supply from both the internal and the external carotid artery system. The superior aspect of these structures receives a blood supply through the internal carotid system from the anterior and posterior ethmoidal arteries. The middle and inferior aspects are supplied by vessels from the external carotid artery: the facial artery and the nasopalatine, greater palatine, and sphenopalatine arteries from the maxillary artery. The region commonly referred to as *Kiesselbach's* or *Little's area,* in the anterior inferior portion of the nasal septum, receives an abundant blood supply from all the vessels and is the region where most epistaxis originates. (Adapted from Powers M. Initial assessment and management of the trauma patient. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 1. Philadelphia: JB Lippincott; 1992; pp. 269–310.)

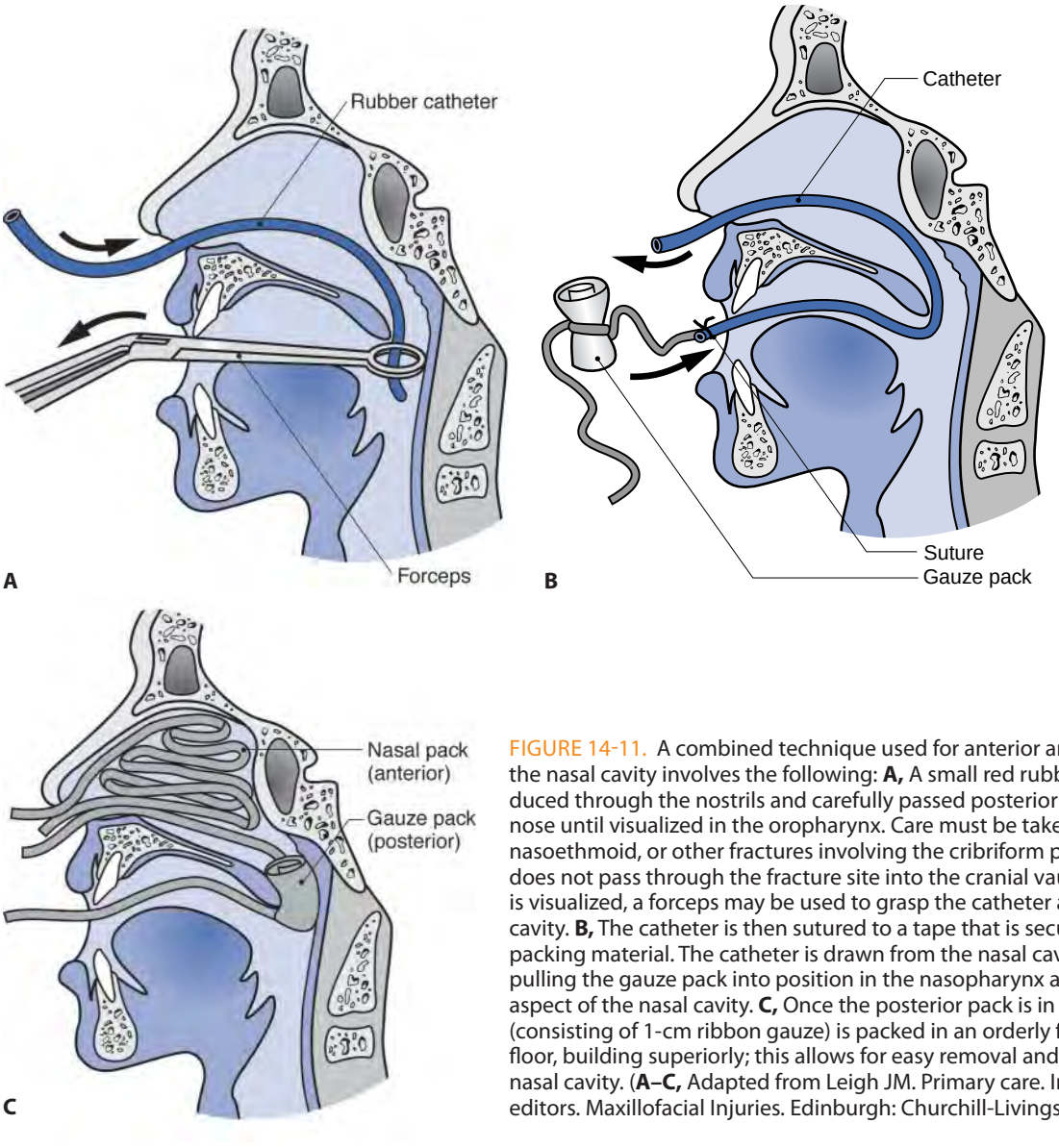


FIGURE 14-11. A combined technique used for anterior and posterior packing of the nasal cavity involves the following: **A,** A small red rubber catheter is introduced through the nostrils and carefully passed posteriorly along the floor of the nose until visualized in the oropharynx. Care must be taken with Le Fort II level, nasoethmoid, or other fractures involving the cribriform plate that the catheter does not pass through the fracture site into the cranial vault. Once the catheter is visualized, a forceps may be used to grasp the catheter and pull it into the oral cavity. **B,** The catheter is then sutured to a tape that is secured to a wad of gauze packing material. The catheter is drawn from the nasal cavity through the nostril, pulling the gauze pack into position in the nasopharynx against the posterior aspect of the nasal cavity. **C,** Once the posterior pack is in place, the anterior pack (consisting of 1-cm ribbon gauze) is packed in an orderly fashion along the nasal floor, building superiorly; this allows for easy removal and efficient packing of the nasal cavity. (**A–C,** Adapted from Leigh JM. Primary care. In Rowe NC, Williams JC, editors. Maxillofacial Injuries. Edinburgh: Churchill-Livingstone; 1985; pp. 54–74.)



FIGURE 14-12. Pelvic fracture. Pelvic fractures, fractures of the femur, and multiple fractures of other long bones may cause hypovolemic shock and life-threatening blood loss, the primary site of which may be difficult to determine. Typical closed fractures of the pelvis may lose 1 to 5 L of blood, femur fractures 1 to 4 L, and arm fractures 0.5 to 1 L from the vasculature.

Hypovolemic Shock in the Patient with Multisystem Injuries

The most common cause of shock seen in the patient with multisystem injuries is hypovolemia caused by hemorrhage. Virtually all multisystemic injuries are accompanied by a degree of hypovolemic shock that presents as a graded physiologic response to hemorrhage. This response can be classified based on the percentage of acute blood loss (Table 14-5).

Class I Hemorrhage: Blood Loss of up to 15%

The clinical symptoms of blood loss of up to 750 mL in the 70-kg adult male are minimal. A mild tachycardia is noted, but the compensatory mechanisms of the body retain normal blood pressure levels, pulse pressure, respiratory rate, and tissue perfusion.

Class II Hemorrhage: Blood Loss of 15 to 30%

Blood loss of 15% to 30% represents a 750- to 1500-mL loss in the 70-kg adult male. Clinical symptoms commonly



FIGURE 14-13. Femur fracture. Fat embolism syndrome is usually associated with major fractures of long bones, especially of the femur. The patient typically does well for 24 to 48 hours and then develops progressive respiratory and central nervous system deterioration. Concomitant laboratory value changes include hypoxemia, thrombocytopenia, fat in the urine, and a slight drop in hemoglobin. Fat enters the venous sinusoids at the fractured site and becomes lodged in the lung alveoli.

expected with this level of blood loss are tachycardia, tachypnea, and a decrease in the difference between systolic and diastolic blood pressure or pulse pressure. The decrease in pulse pressure level is due to the elevation of catecholamines and increased peripheral vascular resistance in response to the decreased intravascular components. The increase in diastolic pressure suggests hypovolemia because there is no noticeable decrease in the systolic pressure in the early stages of blood loss. The peripheral vasoconstriction may show an elongated capillary refill time, and the skin may feel cold and moist.

Class III Hemorrhage: Blood Loss of 30 to 40%

In the 70-kg adult male, a 30% to 40% blood volume loss represents a 1500- to 2000-mL loss, which is fairly detrimental to the survival of vital organ tissues. Patients present with the classic signs of inadequate tissue perfusion, including marked tachycardia (120–140 bpm), tachypnea, marked vasoconstriction, a decreased systolic pressure level, diaphoresis, anxiety, restlessness, and decreased urinary output.

Class IV Hemorrhage: Blood Loss of Greater than 40%

Blood losses approaching half of the intravascular volume produce an immediately life-threatening situation. Symptoms include marked tachycardia, a significant decrease in the systolic blood pressure level to less than 60 mmHg, marked vasoconstriction with a very narrow pulse pressure, marked diaphoresis, obtunded mental state, and no urinary output.

Management

In managing the trauma patient in shock, the speed with which resuscitation is initiated and the time required to reverse shock are the factors crucial to the patient's outcome.[32] The focus should again always be on controlling the hemorrhage, whether it be through basic measures such as pressure and elevation or through rapid imaging/surgical intervention. Aggressive and continued volume resuscitation is not a substitute for definitive control of hemorrhage. Two large-bore (≥16 gauge) short angiocatheters are a minimum for beginning fluid therapy. Initial attempts should be made to percutaneously place the catheters in the basilic or cephalic veins in the antecubital fossa of both arms. Percutaneous placement of femoral, jugular, or subclavian vein catheters may also be used if there are no abdominal injuries or pelvic or femur fractures. When the patient is in an

TABLE 14-5. **Estimated Fluid and Blood Losses***

	Class I	Class II	Class III	Class IV
Blood loss (mL)	≤ 750	750–1,500	1,500–2,000	> 2,000
Blood loss (% vol)	≤ 15	15–30	30–40	> 40
Pulse rate	< 100	> 100	> 120	> 140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate	14–20	20–30	30–40	> 35
Urine output (mL/hr)	> 30	20–30	5–15	Negligible
Mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic
Fluid replacement†	Crystalloid	Crystalloid and blood	Crystalloid and blood	Crystalloid

* Based on the initial presentation of a 70-kg man.

† The guidelines in the table are based on the "3-for-1" rule. This rule is derived from the empirical observation that most patients in hemorrhagic shock require as much as 300 mL of electrolyte solution for each 100 mL of blood loss. Applied blindly, these guidelines can result in excessive or inadequate fluid administration. For example, a patient with a crush injury to the extremity may have hypotension out of proportion with his or her blood loss and requires fluids in excess of the 3:1 guidelines. In contrast, a patient whose ongoing blood loss is being replaced by blood transfusion requires less than 3:1. The use of bolus therapy with careful monitoring of the patient's response can moderate these extremes.

Adapted from American College of Surgeons Committee on Trauma. Advanced Trauma Life Support for Doctors: Student Course Manual. 6th ed. Chicago: American College of Surgeons; 1997; p. 98.

extreme hypovolemic state, placement of percutaneous catheters may be difficult; venous cut-down procedures to expose the saphenous vein provide venous access for fluid resuscitation. Flow is directly dependent on the catheter's internal diameter and is inversely dependent on its length. Therefore, two catheters of the same length and diameter, whether inserted peripherally or centrally, give the identical flow rate, but a longer central catheter delivers a lower possible maximum flow rate than does a shorter peripherally placed catheter. A central line through the subclavian or internal jugular vein routes usually takes longer to place than does a peripheral line and may require disruption of other resuscitation measures such as chest compressions during placement. Furthermore, a central line may complicate resuscitation of the trauma victim by causing or aggravating a developing pneumothorax or hemothorax or other potential complications associated with its placement. Therefore, peripheral intravenous lines are the access of choice in the primary management of the trauma patient.

Circulatory support and proper oxygenation of tissues are indicated by adequate systolic and diastolic blood pressure levels, pulse pressure levels, pulse rate characteristics, and capillary refill times. The clinical observations of these parameters are difficult to quantitate, as is measuring improvement of stabilization of the circulatory system.

Adequate urine production is a predictable sign of renal function, except in cases in which urine production may be enhanced by the use of diuretics as decreased in chronic renal failure. For this reason, urinary output is a prime indication of resuscitation and patient response. A Foley catheter should be placed in the bladder as soon as possible to measure urinary flow. There are three contraindications for the insertion of a Foley catheter, and the catheter should not be placed until all have been ruled out. These contraindications in the traumatized patient are the presence of blood at the urethral meatus, hemorrhage into the scrotum, and a high-riding prostate (Figure 14-14A).[33–35] Attempts to pass a catheter up an injured urethra can convert an incomplete laceration into a complete laceration and can introduce infection into the perineal and retropubic hematoma. A rectal examination should be performed in all trauma patients with suspected pelvic trauma before placement of a catheter. If a pelvic fracture is present, one should consider a urethrogram prior to placing a Foley catheter. With posterior urethral disruption, the prostate may be forced superiorly by a hematoma; if the prostate cannot be palpated, a urethral injury should be suspected (see Figure 14-14B).[36]

The initial intravenous resuscitation fluid used in most hospitals is a balanced electrolyte solution such as lactated Ringer's solution or 0.9% normal saline. During prolonged shock, isotonic fluid is lost from the intravascular and interstitial spaces to the extracellular space. Initially, the patient should be given 2 L of intravenous fluid (20 mL/kg for a pediatric patient) rapidly over 10 to 15 minutes and then observed. If this maneuver does not raise the systolic blood pressure to at least 80 to 100 mmHg, the patient requires additional fluid, blood, and control of blood loss. There is still controversy about the use of colloids (albumin, plasma protein fractions) and artificial plasma expanders (dextran, hetastarch) to treat hypovolemia secondary to trauma. The cost of these materials does not appear to be justified by clinical data.[37,38] Extensive meta-analysis shows a trend toward increased mortality with the use of colloids over crystalloids.[39] However, there is still support for their use,

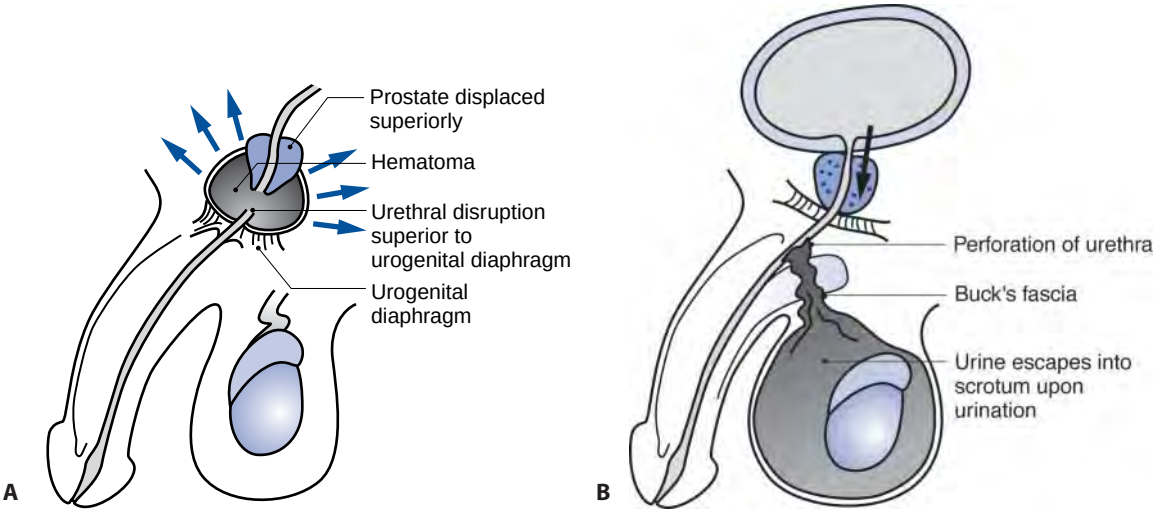


FIGURE 14-14. **A,** The contraindications for placement of a Foley catheter in the trauma patient are the presence of blood at the urethral meatus, hemorrhage into the scrotum, and a high-riding prostate. Blood at the urethral meatus may represent a significant enough disruption of the urethra to prohibit passage of a catheter safely. **B,** The development of a hematoma or urine collection within the scrotum typically results from an anterior urethral disruption from perineal blunt trauma with a perforation of Buck's fascia. With a posterior urethral disruption, the prostate may be forced superiorly by the developing hematoma. (**A** and **B,** Adapted from Powers M. Initial assessment and management of the trauma patient. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 1. Philadelphia: JB Lippincott; 1992; pp. 269–310.)

particularly if blood replacement is delayed or inadequate or in patients with severe head injuries that require fluid restriction therapy to control rising intracranial pressure (ICP) levels.

Most patients respond to initial fluid administration, but this improvement may be transient—especially in patients who have lost greater than 20% of their blood volume.[7]

With excess hemorrhage, red blood cells must be replaced in the intravascular circulation to maintain an optimum oxygen-carrying capacity. The safest type of blood to administer is blood that has been fully cross-matched. Obtaining fully cross-matched blood may require 30 minutes or more and is usually not immediately possible in the trauma situation. Type-specific blood is a safe alternative and can usually be ready within 5 to 15 minutes. With whole blood loss and requirements for early blood replacement, O-negative blood may also be given in patients with excessive hemorrhage.[5] The O blood group is the most common and contains no cellular antigens. Theoretically, O-negative blood can be given to persons regardless of the individual's blood group with minimal risk of antigen-antibody hemolytic reaction. However, no more that 4 U of O-negative blood should be given.[40]

Fresh frozen plasma (FFP) is frequently used as a volume expander and provides all of the clotting factors except platelets. It also provides opsonins and some complement factors, which may be deficient in patients with severe trauma or shock. During massive transfusions, FFP is often given after an elevation of International Normalized Ratio (INR), especially if packed red blood cells are administered in an attempt to prevent coagulation abnormalities. In addition, platelet levels below 100,000/mm^3 may be an indication for a platelet transfusion.[41]

The restoration and maintenance of body temperature are also important in the trauma patient. Appropriate body temperature increases the response to resuscitative measures and decreases the risk of worsening coagulopathy with massive transfusion. The use of body warmers and fluid warmers is strongly recommended.

If the patient initially responds to therapy, blood may not be required immediately, but the patient will require blood if hypovolemic shock continues to develop. A blood sample should be sent to the blood bank as soon as possible for full cross-matching. The patient who is resuscitated initially with O-negative unmatched blood or type-matched blood should be switched to fully cross-matched blood as soon as is reasonably possible to limit the risks of hemolytic reactions.[42] Such blood is compatible within the AB-positive and Rh blood groups but may contain minor antigenic incompatibilities. Ideally, the amount of blood given should be equal to the amount lost by the patient, but this is difficult to assess in the trauma patient. In critically ill or injured patients, the ideal hemoglobin is 10 g/dL (hematocrit ≥ 30%).

If the patient does not respond to initial fluid resuscitation and blood transfusions, either surgical intervention is required to control continued hemorrhage or the initial diagnosis of hypovolemia is incorrect. Measurement of the central venous pressure with a catheter or evaluation of the neck veins may assist with the assessment of hypovolemic shock. Those patients with exsanguinating hemorrhage should have a low central venous pressure, and those with other causes of shock should have a normal or elevated central venous pressure.[7] The ultimate hemodynamic criterion in the treatment of hypovolemic shock is the patient's response. Adequate resuscitation is achieved when adequate oxygenation, circulation, and urine output are restored.

A patient being treated for hypovolemic shock is usually placed in a head-down or Trendelenburg position to empty the venous side of the peripheral circulation back to the heart. Frequently, the patient with multisystem trauma has injuries to the abdomen or chest that may interfere with the respiratory capacity if the patient is in the Trendelenburg position. Alternatively, both of the patient's legs can be elevated while the patient's trunk is maintained in a supine position.[43]

Neurologic Examination

Upon completion of the assessment of the cardiovascular system and control of any external hemorrhage, a brief neurologic evaluation is performed to establish the patient's level of consciousness and pupillary size and reaction. This brief neurologic examination quickly identifies any severe CNS problems that require immediate intervention or additional diagnostic evaluation. A lack of consciousness with altered pupillary reaction to light requires an immediate CT scan of the head and possible management with mannitol or fluid restriction. Be aware of any medications that the patient may have received or drugs she or he may have taken that may affect the pupils. Some individuals will have anisocoria, a condition with an unequal size of the pupils, and provide a confusing examination for neurologic trauma.

The Committee on Trauma of the American College of Surgeons recommends the use of the mnemonic AVPU.[7,8] In this system, each letter describes a level of consciousness in relation to the patient's response to external stimuli: *a*lert, responds to *v*ocal stimuli, responds to *p*ainful stimuli, and *u*nresponsive.

A more detailed quantitative neurologic examination is part of the secondary survey of the trauma patient. The primary survey establishes a baseline; if the patient's neurologic condition varies from the primary to the secondary survey, a change in intracranial status may be present. A decrease in the level of consciousness may indicate decreased cerebral oxygenation or perfusion.

The reactivity of the pupils to light provides a quick assessment of cerebral function. The pupils should react equally. Changes represent cerebral or optic nerve damage or changes in ICP. Further changes in pupil reactivity or levels of consciousness may be due to alterations in ventilation or oxygenation status. The most common causes of coma or depressed levels of consciousness are hypoxia, hypercarbia, and hypoperfusion of the brain.[42] Depressed levels of consciousness and narrow pinpoint pupils may result after an

opiate overdose. Treatment requires the narcotic antagonist naloxone hydrochloride, 0.4 mg initially. Care should be taken to avoid a quick violent withdrawal phase in the opiate abuser; this is accompanied by profound distress, nausea, agitation, and muscle cramps.

Both hypoglycemia and hyperglycemia can cause depressed levels of consciousness. If a quick blood glucose level cannot be obtained (and depending on other injuries), the patient can be given an immediate bolus of 25 g of glucose to manage critical hypoglycemia. A benefit of the glucose load is the hyperosmolar status that may, for a short time, reduce cerebral edema, if present.[44]

Exposure of the Patient

The patient should be completely disrobed so that all of the body can be visualized, palpated, and examined for injuries or bleeding sites. The clothing must be completely removed, even if the patient is secured to a spinal backboard. The easiest method is to cut the clothing down the midline of the torso, arms, and legs to facilitate the examination and assessment. The patient must be quickly covered with warming blankets, use of a patient warming system, use of fluid warmers, and/or increase in the room ambient temperature to avoid cooling of the injured patient. Frequent careful reevaluation of the injured patient's vital signs is important to monitor the patient's ability to maintain an adequate airway, breathing, and circulation (Figure 14-15).

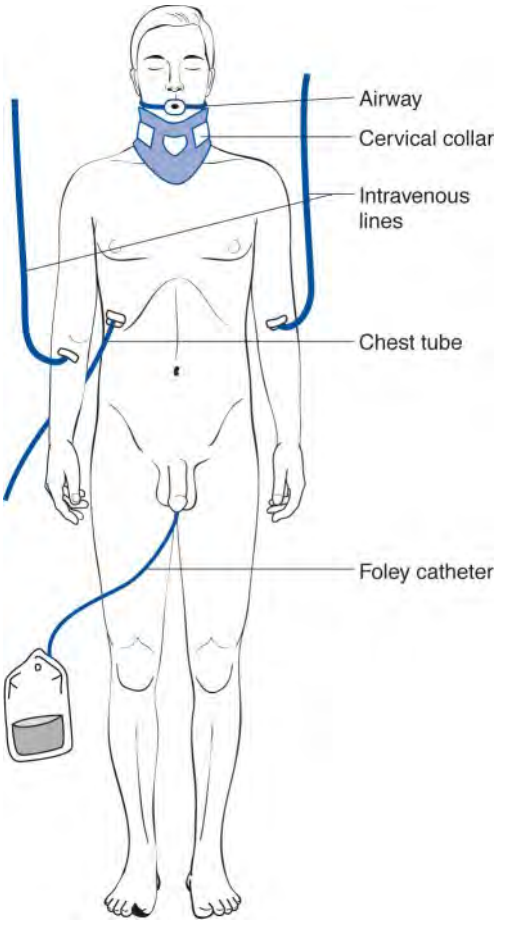


FIGURE 14-15. The primary assessment of the patient with multiple injuries requires evaluation and maintenance of an adequate airway with cervical protection, adequate breathing (including the placement of chest tubes to correct alterations in normal lung and chest wall physiologic conditions), and adequate circulation and hemodynamics, with the placement of two large-bore intravenous lines peripherally and the insertion of a Foley catheter after possible urethral damage is ruled out. The patient should be totally exposed so that the entire body can be examined for injuries. (Adapted from Powers M. Initial assessment and management of the trauma patient. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 1. Philadelphia: JB Lippincott; 1992; pp. 269–310.)

SECONDARY ASSESSMENT

The secondary assessment does not begin until the primary assessment has been completed and management of life-threatening conditions has begun. During the secondary assessment, the patient's vital signs and condition should be constantly monitored to evaluate the therapeutic interventions initiated during the primary assessment and to further assess the patient for any other life-threatening problems not evident during the primary survey. Changes in the patient's vital signs, respiratory and circulatory status, and neurologic functions are expected in the first 12 hours.[7] The secondary assessment includes a subjective and objective evaluation of the injured patient. If at any time during the secondary survey the patient has a significant change in status, a return to the primary survey and management is indicated.

A subjective assessment should include a brief interview with the patient, if possible. A brief health history can be useful, including medications; allergies; previous surgery; a history of the injury; and the location, duration, time frame, and intensity of the chief complaint. Obviously, the comatose patient cannot provide useful subjective information, but family members, bystanders, or other victims may provide some details. It is important to discuss with the emergency medical services (EMS) personnel and review their reports for pertinent details of their findings at the scene and during the patient transport.

The objective assessment should involve inspection, palpation, percussion, and auscultation of the patient from head to toe. Each segment of the body (head and skull, chest, maxillofacial area and neck, spinal cord, abdomen, extremities, and neurologic condition) is evaluated to provide a baseline of the patient's present condition. Special procedures such as peritoneal lavage, radiographic studies, and further blood studies may be done at this time.

Head and Skull

Primary injuries to the head and skull may involve lacerations, abrasions, avulsions, and contusions of the scalp; frac-

tures of the cranium and cerebral contusions; and intracranial bleeding from lacerations or shearing injuries. The brain may also suffer secondary insults from intracranial bleeding, hypoxia, and ischemia. Hypoxia is due to an impaired delivery of oxygen to the brain, whereas ischemia can result from arterial hypotension, elevated ICP, or pressure on intracranial vessels from expanding hematomas. Increased ICP can result in herniation of the brain from the cranial vault (Figure 14-16). The secondary insults of hypoxia and various forms of ischemia are usually preventable. About one half of patients with head injuries have some degree of reversible injury caused by increased ICP that can be controlled with aggressive management. Failure to prevent increased ICP is the most frequent cause of death in hospitalized patients with a severe head injury. Hypertension with concomitant bradycardia may indicate increasing ICP (Cushing's phenomenon). Hypotension with tachycardia usually indicates blood loss. Shock is rarely associated with the primary neurologic injury, and systemic sources of blood loss should be investigated. The classic findings of Cushing's phenomenon are usually present less than 25% of the time, even when the ICP is found to be greater than 30 mmHg. A value greater than 15 mmHg is considered abnormal.

Accurate continual neurologic assessment and examination for mass lesions with CT scans are rapid noninvasive techniques that are not life-threatening for the patient with a head injury and that establish a baseline examination for future studies. When an intracranial injury is suspected, CT scans can quickly and easily be used to diagnose localized intracranial hemorrhage (Figure 14-17), contusion, foreign bodies, and skull fractures. In addition, secondary effects of trauma such as edema, ischemia, infarction, brain shift, and

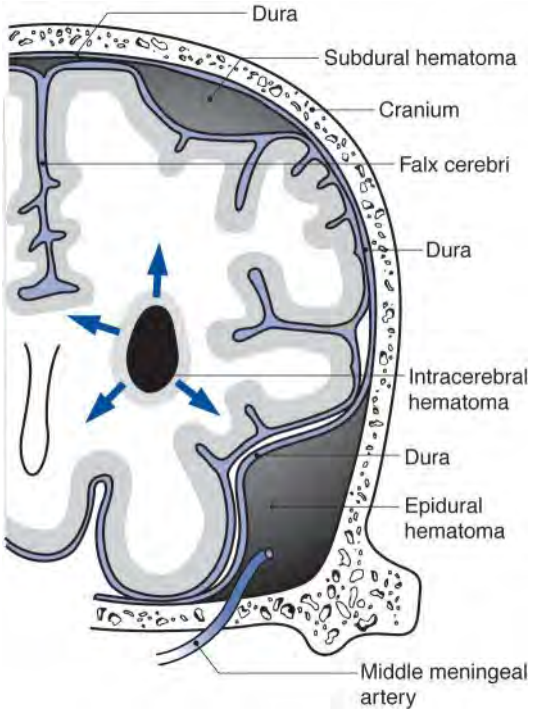


FIGURE 14-16. Mass lesions commonly associated with head trauma include epidural hemorrhage, subdural hemorrhage, and intracerebral hemorrhage. A subdural hematoma is usually caused by venous bleeding with progressive loss of neurologic function. The epidural hematoma is usually associated with skull fractures near the temporoparietal region, with tearing of the middle meningeal artery. (Adapted from Powers M. Initial assessment and management of the trauma patient. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 1. Philadelphia: JB Lippincott; 1992; pp. 269–310.)

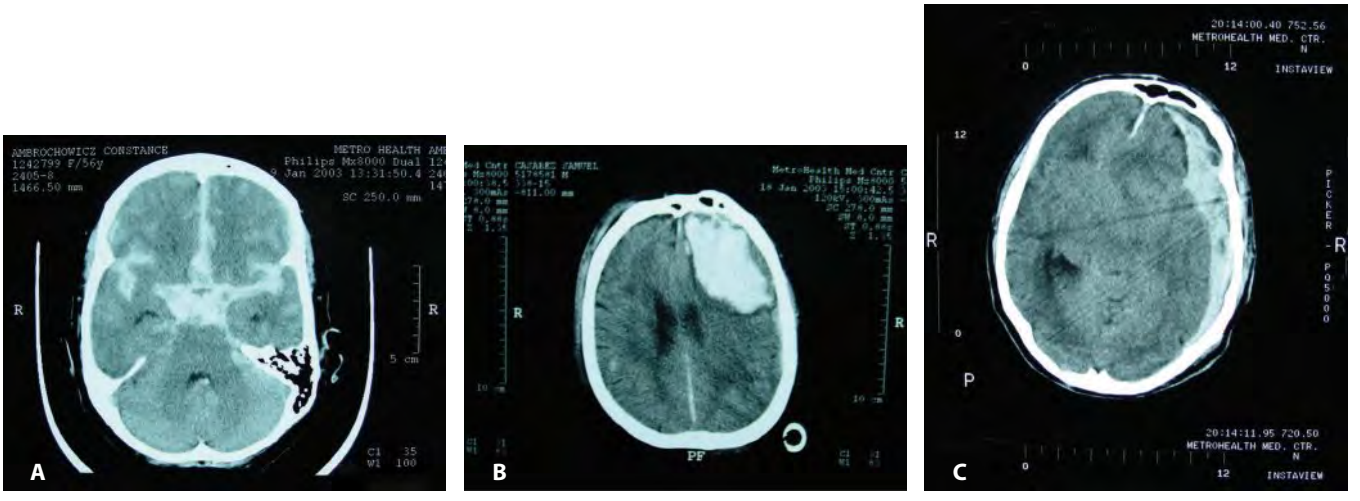


FIGURE 14-17. Computed tomography scans demonstrate anatomic variances associated with intracranial bleeding. **A,** Subarachnoid hemorrhage is defined as blood within the cerebral spinal fluid and meningeal intima and probably results from tears of small subarachnoid vessels. Blood is spread diffusely through the arachnoid matter and usually does not cause mass effect, but may predispose a patient to cerebral vasospasm. **B,** Intracerebral hemorrhage is formed deep within the brain tissue and is usually caused by shearing or tensile forces that mechanically stretch and tear deep small-caliber arterioles as the brain is propelled against irregular surfaces in the cranial vault. Note the surrounding edema and mass effect. **C,** Subdural hematomas are blood clots that form between the dura and the brain. They are usually caused by the movement of the brain relative to the skull, as is seen in acceleration-deceleration injuries. Note the considerable shift of midline to the right.

hydrocephalus can be seen on CT scans. In the acutely traumatized patient, CT scans can be used to diagnose intracerebral and extracerebral blood collections with nearly 100% accuracy. A significant mass lesion can cause cerebral ischemia by elevating ICP or by compressing vascular structures. A CT scan should be done immediately after stabilization of the injured patient, rather than waiting for signs of an expanding intracranial hematoma. Indications for a CT scan include seizure activity, unconsciousness lasting for more than a few minutes, abnormal mental status, abnormal neurologic evaluation, evidence of a skull fracture found on physical examination, and a history of head trauma. A CT of the head may be obtained in all patients with blunt head trauma who have experienced a loss of consciousness or mild amnesia, even those with normal neurologic findings.[45]

Extreme care should always be taken when moving a patient with head trauma to the CT machine because of the high incidence of associated cervical spine fractures in patients with head and facial trauma.[46] If trauma to the spine is suspected, the cervical spine should be immobilized before the patient is moved and the CT examination should be extended to study the cervical spine as well. In addition, any suspected facial injuries should be examined by extending the CT examination inferiorly—as low as the inferior border of the mandible. Unfortunately, in many cases, evaluation and treatment of facial injuries must be delayed for a significant time, which means that the patient is needlessly transported back to the radiology department for further studies because of failure to initially extend the CT examination.

As ICP increases above normal, a fairly standard progression of neurologic abnormalities ensues, involving sections of the brain sequentially: the cerebral cortex, producing an altered state of consciousness; the midbrain, producing dilation and then fixation of the pupils, initially on the side of the lesion, with varying degrees of bilateral hemiparesis; the pons, resulting in a loss of the corneal reflex and the occurrence of the doll's eye reflex (Figure 14-18); and the medulla, producing, in sequence, apnea, hypotension, and death.

The physical examination of the head should include an examination of the scalp for lacerations and foreign bodies. Because of the rich vascular supply of the scalp, especially in children, scalp injuries may result in significant blood loss. Lacerations may overlie an injury to the cranium or intracranial hemorrhage may be present. An untreated scalp wound with a cranial injury may eventually act as a port for bacteria to enter the injured area, causing meningitis or a brain abscess. The head should be examined for signs of a basilar skull fracture: hematoma over the mastoid process behind the ears (Battle's sign); hemotympanum; cerebrospinal fluid (CSF) rhinorrhea, or otorrhea; and subscleral hemorrhage. Whenever a basilar skull fracture is suspected, a nasogastric tube should not be used because the tube may inadvertently pass into the cranial vault.

The neurologic examination should be brief and should evaluate the level of consciousness, motor and cranial nerve function (suggestive of developing mass lesions), brainstem findings, and trends in the neurologic status. Alcohol and drug intoxication are frequently associated with injured patients in the trauma situation and may complicate the neurologic examination. A decreased level of consciousness should not be attributed to alcohol or other drugs until intracranial pathologic conditions have been ruled out.

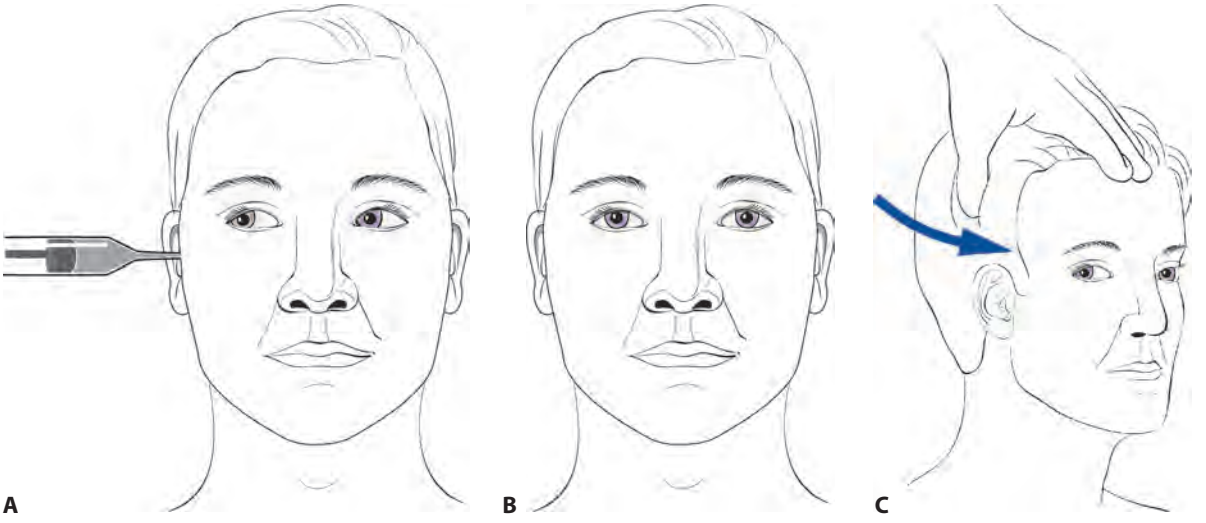


FIGURE 14-18. Responses that test the third, sixth, and eighth cranial nerves, as well as ascending brainstem pathways from the pontomedullary junction to the mesencephalon. **A,** The caloric response (oculovestibular maneuver) involves the placement of cold water into the ear. In a comatose patient, the eyes should tonically deviate toward the irrigated ear. **B,** Patient at rest. **C,** In the oculocephalic response (doll's eye reflex) in comatose patients, the head is turned from the midline and there is a reflex movement of the eyes in the opposite direction of head rotation. (**A–C,** Adapted from Powers M. Initial assessment and management of the trauma patient. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 1. Philadelphia: JB Lippincott; 1992; pp. 269–310.)

The GCS (discussed previously) provides a simple method of grading consciousness and functional capacity of the cerebral cortex (see Table 14-1). It can be used both in the field and as a reassessment tool to assess brain function, brain damage, and patient progress, based on the three behavioral responses: eye opening, best verbal response, and best motor response. Two regions of the brain, if injured, can produce unconsciousness: the cerebral cortices bilaterally and the brainstem reticular activation system, regardless of the cause of injury, can also depress the level of consciousness.[7]

Examination of the motor function is part of the GCS, which gives information about any asymmetry of function. The conscious patient should be asked to move the extremities in response to commands. An inability to do so may represent damage to the limb or spinal cord. In the unconscious patient, deep tendon reflex and plantar response testing can assess both sensory input and motor output. Of special concern is abnormal posturing and nonpurposeful movement to stimulus. Abnormal flexor activity (decorticate) involves flexion of the forearms on the chest with flexion of the wrists and fingers; in abnormal extensor posturing, the arms, hands, and fingers are extended with the hands abducted. In both cases, the lower extremities are extended and no attempt is made to localize the point of stimulation. Although bilateral extensor plantar responses are nonspecific, a unilateral Babinski sign points to corticospinal tract damage.

Pupillary function, eye movements, and eye opening can provide information about the level of consciousness as well as about brainstem function. The size, shape, and reactivity of the pupil to light provide information about second and third nerve function and midbrain activity. A sluggish reactive or a dilated nonreactive (blown) pupil on one side indicates compression of the third cranial nerve by brain herniation in the unconscious patient. The pupillary light reflex can be used to evaluate cranial nerve function and possible elevated ICP with brain herniation. In normal activity, when light is shone in one eye, both pupils constrict equally. The optic or second cranial nerve carries both visual and pupillary fibers. The optic nerves connect shortly after they leave the retina to form the optic chiasm. At the optic chiasm, the nasal fibers cross to join the temporal fibers from the other eye and the visual fibers cross to the visual occipital cortex. The pupillary fibers are relayed bilaterally to the Edinger-Westphal nucleus of the oculomotor or third cranial nerve. The third cranial nerve supplies the sphincter muscle of the iris, allowing it to contract. There is also autonomic innervation of the eyes. The iris is supplied by both sympathetic and parasympathetic fibers. Stimulation of the sympathetic fibers causes the pupil to dilate and the upper eyelid to elevate.

Thus, significant information about the trauma patient can be obtained by looking into the eyes. If a light is shone into the right eye and the left eye does not respond, there may be a disruption of the right optic or left oculomotor nerves. If the light is then shone into the left eye and it does not respond, a disruption of the third cranial nerve should be suspected. Pupillary dilation of one eye may be due to a developing brain herniation on the ipsilateral side, with bilateral pupillary dilation suggestive of significant midbrain injury or loss of parasympathetic function. Conversely, pinpoint pupils after head trauma may indicate drug overdose or loss of sympathetic tone as seen in Horner's syndrome.

The function of the brainstem may also be assessed with evaluation of the corneal reflex, which involves sensory input from the trigeminal (fifth) nerve. The oculocephalic maneuver, or test of the doll's eye reflex, requires an intact vestibular or acoustic (seventh) nerve to permit head rotation to evaluate reflexive movement of the eyes (see Figure 14-18). Obviously, this maneuver is not to be used with patients who have a suspected cervical spine injury. The oculovestibular response test evaluates the third, fourth, sixth, and eighth cranial nerves as well as brainstem activity. In this test, the external auditory canal is irrigated with cold water; there should be full eye movement toward the ear canal lavaged with cold water. If not, there may be a disruption along any of the neural tracts or of the tympanic membrane (see Figure 14-18).

A lumbar puncture should not be performed in patients with acute head injuries. The change in pressure associated with the removal of CSF from the lumbar region may precipitate cerebral herniation in the patient with an elevated ICP.

CSF emerging from the nose or ear is commonly associated with a basilar skull fracture. Clear or red-tinged fluid that drains from the nose or ear should be considered to be CSF. There is no reliable method available in the emergency department for distinguishing CSF from nasal mucous drainage. The use of glucose indicator sticks is associated with a high incidence of false-positive results. A useful aid may be a "ring sign." A drop of the fluid from the nose or ear is placed on a piece of filter paper. If the fluid is CSF, the blood components of the fluid remain in the center and rings of clear fluid form around them.[7]

A CT scan should be performed to determine whether there is a fracture site. The head of the bed should be elevated to 90 degrees. If indicated, the fracture should be reduced. The leakage should cease after 7 days; if it does not, neurosurgical procedures may be indicated to repair the dural tear.

A rectal examination is an essential part of the examination of the patient with a head injury. Rectal sphincter tone is present if the injury is intracranial only; if there is no rectal tone, a coexisting spinal cord injury is present. Coexisting head and spine injuries should be suspected until proved otherwise.

A head injury is initially classified as mild (GCS 13–15), moderate (GCS 9–12), or severe (GCS ≤ 8). Patients with head injuries who experience no loss of consciousness, no amnesia, no palpable fractures, and a GCS score of 15 can be discharged home to a reliable caretaker. Brain imaging is unnecessary, although it is generally recommended that CT imaging be performed owing to its low cost and its convenience. Patients who experience a loss of consciousness or amnesia or have a GCS score of 13 or 14 must undergo an immediate head CT. If this noncontrast study finding is

negative, the patient can be discharged to a reliable caretaker. If there is a focal neurologic finding on examination, a GCS score less than 13, or an intracranial lesion seen on the head CT, the patient should be admitted to an intensive care unit or neurologic observation unit for continuing care. The administration of prophylactic phenytoin at a loading dose of 18 mg/kg intravenously is used by some for control of possible seizure activity. Ongoing seizures may be controlled with a benzodiazepine. Neurosurgical consultation should be obtained early in the management of any significant head trauma. Patients with severe head injuries (GCS < 8) should undergo rapid-sequence intubation technique for airway protection and better control of ICP. The patient's ICP is controlled using various techniques, including reverse Trendelenburg's position, osmotic diuresis (mannitol), hyperventilation of the intubated patient (although there is little or no documented benefit to this procedure), sedation, pharmacologic paralysis, and phenobarbital coma (last resort). Judicious use of resuscitative fluids and control of systemic hypertension also help to control ICP.

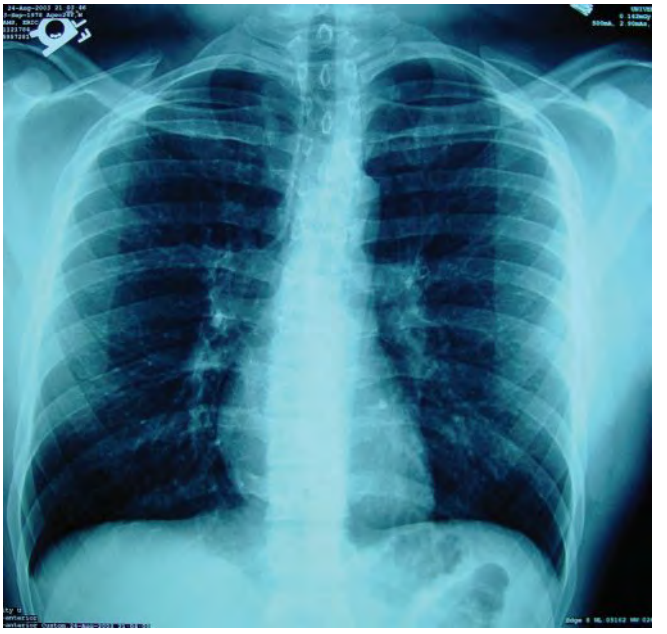


FIGURE 14-19. Normal upright chest radiograph.

Chest

Throughout the secondary assessment of the multiply injured patient, the primary evaluation of airway, breathing, and circulation must be monitored for development of difficulties or overlooked problems. Pneumothorax, open pneumothorax, hemothorax, flail chest, and cardiac tamponade may develop after the primary assessment and must be treated accordingly. It is estimated that chest injuries are responsible for 20% to 25% of all trauma deaths per year in the United States.[26]

The secondary assessment of chest trauma involves the evaluation of an upright chest radiograph for the presence of air in the mediastinum or under the diaphragm, widening of the mediastinum or a shift from the midline, thoracic injuries and fractures that alter lung expansion, and the presence of fluid. Figure 14-19 shows a chest radiograph of a patient without chest trauma. In most instances, the trauma patient needs to be immobilized on a backboard (Figure 14-20), and a supine film is substituted for an upright one. If a chest injury is suspected, a CT scan should also be obtained. An electrocardiogram, arterial blood gas analysis, hematocrit, and urinalysis should be obtained. Potentially lethal injuries to consider in the secondary assessment are pulmonary contusion, aortic disruption, tracheobronchial disruption, esophageal disruption, traumatic diaphragmatic hernia, myocardial contusion, and simple pneumothorax.[7]

Pulmonary contusions are treated in the same manner regardless of whether there is an accompanying flail chest injury. Pulmonary contusions are common in blunt chest trauma because the capillary damage within the lungs results in interstitial and intra-alveolar edema and shunting. Pulmonary contusions and acute respiratory distress syndrome (ARDS) are the most common potentially lethal chest injuries seen in the United States because the resulting respiratory failure does not occur instantaneously but develops in 24 to 72 hours.[24] The patient may complain of pain and dyspnea, and blood gas levels tend to deteriorate progressively over the initial 48 to 72 hours as increasing edema develops in the alveoli. Chest radiographs reveal a developing opacification of the involved areas. Treatment involves adequate ventilation of the lungs, including chest physiotherapy, supplemental oxygen, coughing with deep breathing, and nasotracheal suction. If ventilatory assistance is required, spontaneous ventilation with IMV provides much better ventilation-perfusion matching, better hemodynamics, and quicker weaning than does assisted ventilation. The use of steroids is controversial.[47]

Injury to large intrathoracic arteries or veins may develop with blunt or penetrating trauma; this is the most common cause of sudden death after an automobile accident or a fall from a great height.[7] Common sites of injury are the aortic root and the descending aorta at the origin of the ductus arteriosus and at the diaphragm. These injuries are fatal within a few minutes—only 15% of patients with thoracic aortic injuries are still alive on arrival at a hospital. It is not uncommon for the aortic intima and media to be fractured circumferentially, with only the adventitia and surrounding mediastinal tissues preventing fatal hemorrhage. The patient may appear clinically stable; yet failure to recognize this vascular injury may lead to eventual death. Adjunctive signs on chest radiographs that are suggestive of thoracic vascular injury include a widened mediastinum, fractures of the first and second ribs, obliteration of the aortic knob, deviation of the trachea to the right, the presence of a pleural cap, deviation of the esophagus to the right, and a downward displacement of the left mainstream bronchus.[7] If an aortic rupture is suspected on clinical or radiographic examination, a CT angiogram or aortography should be performed. While waiting for the

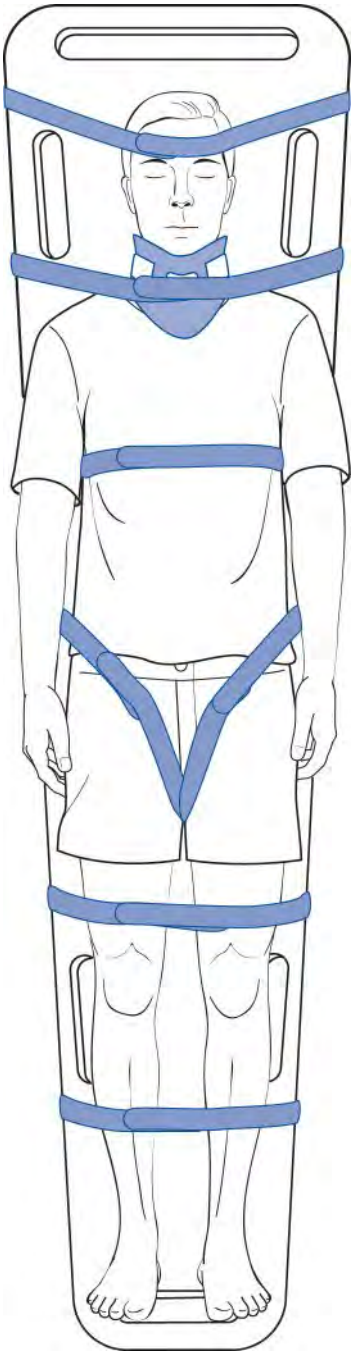


FIGURE 14-20. Stabilization of the trauma victim for transportation is best achieved with the use of a long backboard with bindings and sandbags to control the head in a neutral position. (Adapted from Powers M. Initial assessment and management of the trauma patient. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 1. Philadelphia: JB Lippincott; 1992; pp. 269–310.)

examination, it is important not to let the patient become hypertensive or cough or gag excessively (e.g., as may occur with the placement of a nasogastric tube).

Pain in the mediastinum needs to be investigated and usually requires a surgical consultation. Pain may be secondary to a developing pneumothorax, tracheobronchial or esophageal disruption, or free air arising from the abdomen with disruption of the diaphram. CT scans can be useful in diagnosis and differentiation of the level of injury. A simple traumatic pneumothorax is usually best treated with a chest tube. Small pneuomothoracies may be observed with serial physical examination and chest radiographs in qualified centers. If the patient's condition declines, placement of a chest tube is required. Traumatic diaphramatic hernia is suspected when bowel is seen in the chest or the nasogastric tube lies above the diaphragm in the chest radiograph or CT. Small herniations may be missed on initial examination and present as clinical problems years later. Myocardial contusion most commonly presents with sinus tachycardia but other arrythmias may also arise. An electrocardiogram with cardiology consultation is recommended if there is a strong suspicion that a mycardial contusion is possible.

Maxillofacial Area and Neck

Maxillofacial injuries may cause airway compromise from blood and secretions, from a mandibular fracture that allows the tongue to fall against the posterior wall of the pharynx, from a midface injury that causes the maxilla to fall down and back into the nasopharynx, and from foreign debris such as avulsed teeth or dentures. A large tonsillar suction tip should be used to clear the oral cavity and pharynx. An oral airway assists with tongue position; however, care must always be taken to avoid manipulation of the neck and to provide for access to the oral cavity and dentition for reduction and fixation of any fractures requiring some period of intermaxillary fixation. Neither midface fractures nor cerebrospinal rhinorrhea is a contraindication to nasal intubation or nasogastric tube placement. Care must be taken to pass the tube along the floor of the nose into the pharynx, and the tube should be visualized before intubation of the trachea with an endotracheal tube or of the esophagus with the nasogastric tube. A chest CT or radiograph must be obtained to verify proper position of the tube before use.

The physical examination should begin with an evaluation for soft tissue injuries. Lacerations should be débrided and examined for disruption of vital structures such as the facial nerve or parotid duct. The eyelids should be elevated so that the eyes can be evaluated for neurologic and possible ocular damage. The face should be symmetrical without discolorations or swelling suggestive of bony or soft tissue injury. The bony landmarks should be palpated, beginning with the supraorbital and lateral orbital rims, infraorbital rims, malar eminences, zygomatic arches, and nasal bones. Any step-off or irregularities along the bony margin are suggestive of a fracture. Numbness over the area of distribution of the trigeminal nerve is usually noted with fractures of the facial skeleton.

The oral cavity should be inspected and evaluated for lost teeth, lacerations, and alterations in the occlusion. Any teeth lost at the time of injury must be accounted for because the tooth may have been aspirated or swallowed.

The neck should also be examined for injury. Subcutaneous air may be visualized if massive injury is present; if subtle, it may be detected only by palpation. The presence of air in the soft tissues may be the result of tracheal damage or pneumothorax. Any externally expanding edema or hematoma of the neck must be observed closely for continued expansion and airway compromise. Carotid pulses should be assessed. Palpation for abnormalities in the contour of the thyroid cartilage and for the midline position of the trachea in the suprasternal notch should be performed.

Spinal Cord

There are more than 10,000 spinal cord injuries per year in the United States, usually caused by motor vehicle accidents. Multiple studies have reported a 10% to 20% association of cervical spine injuries with maxillofacial injuries in the multiply traumatized patient, although data suggest no increase in cervical spine injury when facial trauma is present.[48,49] Approximately 55% of spinal injuries occur in the cervical region, 15% in the thoracic region, 15% in the thoracolumbar junction, and 15% in the lumbosacral area.[8] Identification of cervical spine injury is essential in the management of blunt trauma because a missed injury can result in catastrophic spinal cord damage. Tetraplegia as a result of cervical spine injury is not only a tragedy for the patient; it also represents a tremendous financial burden to society.[50] According to the National Spinal Cord Injury Center Databank, in July 1996, the average medical cost of the first year of a cord injury involving C1 through C4 was $417,000.[50] Patients can be expected to have medical costs of $1,350,000 over the course of their lifetime as well as lost wages and productivity. Patients can then expect a greatly shortened lifespan, which varies according to the age of the patient at the time of injury.[48]

A description of the mechanism of injury, especially high-velocity accident, may give clues to a possible injury of the spine such as a whiplash injury. The patient may experience little discomfort from major injury to the chest, abdomen, and extremities as a result of sensory loss from a spinal injury. Because of the loss of sympathetic tone with cervical injuries, the patient may present with a systolic blood pressure level of 70 to 80 mmHg without the tachycardia, cool extremities, poor perfusion, and decreased urinary output noted in the patient with hypovolemic shock. Neurologic shock is due to dilation of the arterial system, loss of muscle tone, and loss of reflexes. The absence of neurologic deficit does not exclude injury to the cervical spine. A complete series of cervical radiographs should be obtained and read before the removal of stabilization. If a helmet is worn by the victim, the helmet should be secured to the long spine board with 8 cm cloth tape, and cervical spine radiographs should be taken and cleared for cervical spine injury before the attempted removal of the helmet.

Physical examination of the patient with a suspected spinal injury should be done carefully, with the patient in a neutral position and with minimal movement of the spine and head (see Figure 14-20). The presence of an unstable cervical spine injury must be considered in the evaluation and resuscitation of every patient with injuries associated with blunt trauma. The catastrophic physical consequences of irreversible quadriplegia, as well as the huge economic costs required to care for this lifelong disability, require that great care must be taken to rule out unstable cervical spine injury. The patient should be treated as if there has been an unstable injury to the nerves, bone, muscles, and other structures of the neck until there is positive clinical and radiographic evidence that there is no injury. The neck and spine should be carefully examined for deformity, edema, ecchymosis, muscle spasm, and tenderness while being carefully supported to avoid further damage associated with an unstable cervical neck injury.

The neurologic examination of the patient with a spinal injury is similar to that of the patient with closed head trauma. The mental status, motor function, sensation over dermatomes, brainstem reflex, and spinal reflexes should all be evaluated and charted. The patient should be carefully examined for rectal tone and bladder control as evidence of autonomic function. Hypoventilation caused by paralysis of the intercostal muscles results from injury to the lower cervical or upper thoracic spinal cord. If the upper or middle cervical spine is injured, the diaphragm will also be paralyzed as a result of involvement of the C3 though C5 spinal cord segments. Abdominal breathing and the use of the respiratory accessory muscles will be evident.[7]

Bachulis and associates[46] evaluated 4941 trauma victims between February 1981 and July 1985 and found that 1923 (39%) had radiographs taken of their cervical spines. Injuries to the cervical spine were detected in 94 patients (5%). Ninety of these patients had cervical spine fractures; 4 had a disruption of the cervical longitudinal ligaments without bony injury and were quadriplegic. In the study, the overall incidence of cervical spine injury in the trauma patient was 2%. Neurologic deficit did not develop in any patient with a neurologically intact spinal cord at the time of admission. The researchers found that of the 94 patients, there were 65 alert patients with no neurologic deficits who had unstable cervical spine injuries. Without exception, these patients complained of either neck pain or pain on palpation of the neck. Other studies have reported that no alert patient without neck pain was found to have any cervical injury.[51] Fischer[51] concluded that a screening radiographic examination of the cervical spine is not indicated in the alert, sober, and cooperative patient with no complaints of neck pain and no tenderness to palpation of the neck, even when significant injury is present. However, the author does recommend screening for all patients with decreased levels of consciousness and a history of an injury that could have conceivably injured the cervical spine, for all patients with neurologic deficits compatible with cervical origin, and for all patients with neck pain or tenderness.[51] Cervical spine stabilization and radiographic examination are required for all patients with severe injuries that might distract and mask neck pain and tenderness. Cervical

spine injuries may result from axial loading, flexion, extension, rotation, lateral bending, and distraction or combinations of these mechanisms of injury (Figure 14-21).

In the study by Bachulis and associates,[46] lateral cross-table cervical spine radiographs were obtained in all injured patients and demonstrated cervical spine injury in 70 patients but not in the other 24, for an unacceptable false-negative rate of 26%. The authors recommended that all patients at risk for cervical spine injury must have a complete initial radiographic examination, including lateral, anteroposterior, odontoid, and right and left oblique views of the cervical spine. CT scanning was found to be the most useful modality to confirm a cervical spine injury in those patients with a suspected injury to the cervical spine not confirmed on plain film radiographs. They recommend the use of CT scans of the neck for patients with a possible neck injury and associated head injury that requires a CT scan of the brain, for patients in whom radiographic visualization of C6 or C7 is difficult, and for patients with a suspected cervical injury that is not detected in screening radiographs.[46] A study by Griffen and colleagues[52] concluded that CT scanning of the cervical spine should completely replace plain film studies in blunt trauma patients. Up to 50% of cervical spine injuries seen on CT radiographs are missed on plain neck radiographic cervical spine series owing to the difficulty in obtaining a clear view of the cervical spine with plain films.

Visualization of all seven cervical vertebrae is important (see Figure 14-21). The shoulders must be distracted inferiorly by pulling down on the arms to provide a clear view of the spinal anatomy from C6 through T1. It is important that a clear view of the spine at the C6 and C7 level be obtained without obstruction by the shoulders to obtain a proper diagnostic study. If visualization of C6 and T1 cannot be obtained, the radiographic view may be improved by placing the arms in a "swimmer's position," with downward traction on one arm and upward traction on the other and the radiograph beam aimed through the axilla of the upward arm. Radiographs should be examined for fractures and fracture dislocations of the spine by evaluation of the anteroposterior diameter of the spinal canal; the contour and alignment of the vertebral bodies; displacement of bony fractures of the laminae, pedicles, or neural fascicles; and soft tissue swelling.[18] Three-way cervical views (anteroposterior, oblique cervical, and lateral cervical) plus an open-mouth odontoid view or a CT scan of the neck coupled with adequate cervical spine immobilization during evaluation and resuscitation should allow the cervical spine to be viewed safely.

On a lateral cervical spine radiograph, the soft tissue thickness between the pharynx and osseous C3 should be less than 5 mm. An increase in this area suggests a fracture. The distance may vary with inspiration or expiration.[7] On the lateral view, the features to be examined are the general contour of the spine, the vertical alignment of the anterior and posterior margins of the vertebral bodies, the midlaminar line, the width of the spinal column, and evidence of compression or fracture of individual vertebrae. On anteroposterior views, the height and alignment of the spinous processes and the interspinous distances are examined. The discovery of any findings suggesting the presence of a cervical spinal injury mandates the use of protective measures. It has been demonstrated that a stabilization device such a cervical collar allows significant movement of the cervical spine.[53] The recommended stabilization for patients with cervical fractures is a cervical collar in combination with a long spinal board. Appropriate head holders or sandbags should be used bilaterally to support the neck laterally, and the head should be secured with an 8-cm cloth tape across the forehead and around the board (see Figure 14-20). Obviously, maintaining a stable airway is critical in patients who have suffered significant head and neck trauma. Cervical neck



FIGURE 14-21. Normal cervical radiographs: lateral **(A)** and anteroposterior **(B).** Radiographs should be examined for prevertebral edema, subluxation, widening of the interspinous distance, widening of the atlantodental interval, bony fractures, malalignment, or jumped facets.

stabilization and protection as well as a nasal trumpet or similar airway protection device may be indicated to maintain a patent airway. If the airway becomes unstable, endotracheal intubation, nasotracheal intubation, tracheotomy, or cricothyroidotomy should be performed, ensuring that the cervical spine continues to be stabilized.

CT should be used for further evaluation of detected or suspected fractures and evaluation of questionable plain films and to complete radiographic examination of areas not well visualized by plain films. The lower cervical spine often is not well visualized on radiographs, even with use of the swimmer's position, and a CT scan is frequently required.

Abdomen

With abdominal trauma, the physical examination is an informative portion of the diagnostic evaluation. Penetrating wounds must be identified, and many surgeons believe that the safest management of penetrating wounds is a laparotomy, although surgical management of penetrating wounds continues to evolve.[7] The abdominal girth should be measured at the umbilicus soon after admission to establish a baseline against which to evaluate possible intra-abdominal bleeding. Abdominal rigidity and tenderness are important signs of peritoneal irritation by blood or internal contents, and they may be the main indications for a laparotomy of a patient injured by blunt trauma. Rectal and pelvic examinations are essential if there is a question of pelvic or perineal injury. A nasogastric tube should be passed, if possible, into the stomach to remove gastric contents.

Plain films have limited value in abdominal trauma. They can be useful in localizing foreign bodies, bony structures, and free air with the use of anteroposterior and cross-table views.

The use of diagnostic peritoneal lavage (DPL), once a standard diagnostic test used in blunt and occasionally penetrating abdominal traumas, has decreased significantly with the advancement in CT and ultrasonography. DPL is indicated in patients with a history of blunt abdominal trauma and increasing pain, with unexplained hypovolemia after multiple trauma, who are candidates for laparotomy but who have questionable findings, and who have experienced severe trauma and may require an extended period under general anesthesia.[7] Absolute contraindications to DPL are a history of multiple abdominal operations and obvious indications for an exploratory laparotomy—free air, evisceration, and/or penetrating trauma. A DPL is usually performed with a sterile intravenous catheter inserted percutaneously through a small midline incision approximately 2.5 to 4 cm below the umbilicus. The catheter is advanced into the pelvis after the bladder has been emptied. If no blood, bile, or intestinal fluid is aspirated, the abdominal cavity is irrigated with 1 L of saline. The fluid is then drained from the abdomen through the intravenous tubing. It is generally felt that the presence of 100,000 red blood cells/mm^3 or 500 white blood cells/mm^3 after blunt trauma is sufficient to make a laparotomy mandatory (Table 14-6).

TABLE 14-6. **Parameters for Evaluation of Peritoneal Lavage Fluid**

Positive	20 mL gross blood on free aspiration (10 mL in children) ≥100,000 RBCs/mm^3 ≥500 WBCs/mm^3 (if obtained ≥ 1 hr after the injury) ≥175 U amylase/100 mL Bacteria (determined with Gram's stain) Bile (by inspection of chemical determination of bilirubin content) Food particles (microscopic analysis of strained or spun specimen)
Intermediate	Pink fluid on free aspiration 50,000–100,000 RBCs/mm^3 100–500 WBCs/mm^3 75–175 U amylase/100 mL
Negative	Clear aspirate ≤50,000 RBCs/mm^3 ≤100 WBCs/mm^3 < 75 U amylase/100 mL

RBCs = red blood cells; WBCs = white blood cells.
Adapted from Powers M. Initial assessment and management of the trauma patient. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 1. Philadelphia: JB Lippincott; 1992; pp. 269–310.

CT scanning of the abdomen is also acceptable if the patient is stable and emergent laparotomy is not indicated. The advantages to CT include that it is noninvasive; is capable of discerning the presence, source, and approximate quantity of intraperitoneal hemorrhage; and occasionally can demonstrate active bleeding. CT scanning coincidentally evaluates the retroperitoneum—an area not sampled by DPL—as well as the vertebral column and can be readily extended above or below the abdomen to visualize the thorax or pelvis. It is helpful in the evaluation of hematuria and, if used early enough, in determining renal artery injury. Disadvantages include suboptimal sensitivity for injuries of the pancreas, diaphragm, small bowel, and mesentery. Injuries of the small bowel and mesentery can have profound morbidity and even mortality if not diagnosed early. In the absence of hepatic or splenic injuries, the presence of free fluid in the abdominal cavity suggests an injury to the gastrointestinal tract and/or its mesentery and mandates early surgical evaluation and possible intervention. Complications also can result from intravenous contrast administration. The cost can also be significant, especially if established indications are not followed.

Ultrasonography or focused assessment with sonography for trauma is rapidly becoming an integral diagnostic component in trauma centers. Ultrasonography has undergone a large number of clinical evaluations in Europe, Asia, and the United States. Its primary role is detecting free intraperitoneal blood after blunt trauma. This is accomplished by a focused examination of specific anatomic areas where blood or fluid is most likely to accumulate. Ultrasonography can also evaluate the pericardial space and intraperitoneal spaces. Ultrasonography carries a host of advantages:

- It is a portable instrument that can be brought to the bedside in the trauma resuscitation area.
- Studies of the pericardial and intraperitoneal spaces can be accomplished in less than 5 minutes.
- Sensitivity in detecting as little as 100 mL to, more typically, 500 mL of intraperitoneal fluid ranges from 60% to 95% in some studies, and specificity for hemoperitoneum is excellent.[54]
- Unlike DPL, ultrasonography can rapidly gauge the mediastinum, is noninvasive, and can be performed serially and by multiple technicians.
- Unlike CT scanning, ultrasonography does not pose a potential radiation hazard and does not require administration of contrast agents.
- Performing focused ultrasonographic examinations with an abdominal trauma patient does not require the skill of a board-certified radiologist, which allows ultrasonography to be more readily accessible to injured patients. Accuracy correlates with length of training and experience, but expertise can be readily accomplished in emergency medicine and surgical training programs.[55]
- Overall, ultrasonography can serve as an accurate and rapid test and is a less expensive diagnostic screening tool than are DPL and CT.

However, there are disadvantages to the use of ultrasonography, including

- It does not image solid organ parenchymal damage, the retroperitoneum, or diaphragmatic defects very well.
- It is technically compromised by the uncooperative agitated patient as well as by obesity, substantial bowel gas, and subcutaneous air.
- Indeterminate studies require follow-up.
- Ultrasonography is less sensitive and more operator dependent than is DPL in revealing hemoperitoneum and cannot distinguish blood from ascites.
- Ultrasonography (as well as DPL) may not detect the presence of solid organ parenchymal damage if free intraperitoneal blood is absent, as in subcapsular splenic injury.[56]
- Finally, ultrasonography is poor for detecting a bowel injury in which hemorrhage tends to be inconsequential, and failure to diagnose hollow viscous perforation in a timely manner can have catastrophic results.

Table 14-7 presents indications, advantages, and disadvantages of ultrasonography, DPL, and CT in blunt abdominal trauma.

Genitourinary Tract

The perineum should be examined for contusions, hematomas, lacerations, and urethral bleeding. A rectal examination should assess for the presence of blood within the bowel lumen, a high-riding prostate, presence of blood or pelvic fractures, and quality of sphincter tone. A vaginal examination should be performed in patients if injury is suspected. Pregnancy tests should be performed on all females of childbearing age.

When an injury to the genitourinary tract is suspected, urologic consultation is required to further evaluate and diagnose the extent of injury. The major cause of urethral ruptures is blunt trauma. Approximately 10% of patients with a pelvic fracture have an associated posterior urethral rupture. The force of the injury causes a shearing effect between the urethra and the urogenital diaphragm.[34] Anterior urethral ruptures are also commonly associated with blunt trauma. Most of these injuries occur in men.[57]

Blood at the urethral meatus is the single best indicator of urethral trauma.[35] The meatus must be carefully inspected for even the slightest amount of blood before inserting a urethral catheter. As discussed previously, attempts to introduce a Foley catheter up an injured urethra can convert an incomplete laceration into a complete laceration with a subsequent retropubic or perineal hematoma.[33] A rectal examination must be performed on all patients with a suspected pelvic injury. With posterior urethral disruption, the prostate may be forced superiorly by a hematoma. If the prostate is not palpable, a genitourinary injury should be suspected.[33]

Absence of blood at the meatus and palpability of the prostate on rectal examination are sufficient evidence to allow the passage of a urethral catheter. If resistance is noted, the catheter should be removed. Retrograde urethrography is the best method to establish continuity of or damage to the urethra.[33]

Urine should be obtained and evaluated for the presence of blood. A urinalysis with 10 or more red blood cells on a high-power field is suggestive of a urinary system injury.

SECTION 3

TABLE 14-7. **Indications, Advantages, and Disadvantages of Diagnostic Peritoneal Lavage, Ultrasonography, and Computed Tomography in Blunt Abdominal Trauma**

	DPL	Ultrasonography	CT
Indication	Document bleeding if ↓ BP	Document fluid if ↓ BP	Document organ injury if BP normal
Advantages	Early diagnosis and sensitive; 98% accurate	Early diagnosis; noninvasive and repeatable; 86–97% accurate	Most specific for injury; 92–98% accurate
Disadvantages	Invasive; misses injury to diaphragm or retroperitoneum	Operator dependent; bowel gas and subcutaneous air distortion; misses diaphragm, bowel, and some pancreatic injuries	Cost and time; misses diaphragm, bowel tract, and some pancreatic pancreatic injuries

BP = blood pressure; CT = computed tomography; DPL = diagnostic peritoneal lavage.
Adapted from American College of Surgeons Committee on Trauma. Advanced Trauma Life Support for Doctors: Student Course Manual. 6th ed. Chicago: American College of Surgeons; 1997; p. 166.

Hematuria is the best indicator of renal injury, and the degree of hematuria may not correlate with the degree of injury. If the patient with a blunt injury is stable but has hematuria, a CT scan can be used to accurately visualize the genitourinary system and abdominal and retroperitoneal contents.

Sexual assault is reported to be the fastest-growing violent crime in the United States. Approximately 40% of rape victims report having sustained physical injury; of those, 54% receive medical care in a hospital emergency department.[58] The responsibility of the medical personnel is not to determine whether a rape has occurred—"rape" is a legal term, not a medical term—but to obtain a thorough history, careful examination, and prompt treatment of injuries. Rape kits are available for collection of materials and maintain a "chain of evidence." Ideally, a rape crisis counselor or specially trained social worker should stay with the patient and arrange follow-up counseling.[59]

Extremities

Pelvic fractures, fractures of the femur, and multiple fractures of other long bones may cause hypovolemic shock and life-threatening blood loss, the primary site of which may be difficult to determine. Typical closed fractures of the pelvis may lose 1 to 5 L of blood, femur fractures 1 to 4 L, and arm fractures 0.5 to 1 L from the vasculature.[60] Certain extremity injuries are considered life-threatening because of associated complications—massive open fractures with ragged dirty wounds; bilateral femoral shaft fractures (open or closed); vascular injuries, with or without fractures, proximal to the knee or elbow; crush injuries of the abdomen and pelvis; major pelvic fractures; and traumatic amputations of the arm or leg.[7]

Physical examination should consist of inspection and palpation of the chest, abdomen, pelvis, and all four extremities. Areas of tenderness, discoloration, swelling, and deformity should be inspected, and proper radiographs should be obtained. All peripheral pulses should be examined for evidence of vascular injury. Pulse rates should be equal; any abnormality of distal pulse rate suggests a vascular injury and must be explained. Doppler examination of the extremity is useful, but angiography is the best test for definitively evaluating a suspected vascular injury when the diagnosis is in doubt.[7]

Direct pressure should be used to control hemorrhage, and fractures should be splinted as quickly as possible. Splints should generally include joints above and below the site of injury. Prompt orthopedic consultation should be obtained.

Fat embolism syndrome is usually associated with major fractures of long bones, especially of the femur. The patient typically does well for 24 to 48 hours and then develops progressive respiratory and CNS deterioration. Concomitant laboratory value changes include hypoxemia, tachypenia, tachycardia, thrombocytopenia, fat in the urine, and a slight drop in hemoglobin. A petechial patch may be present. Fat enters the venous sinusoids at the fractured site and becomes lodged in the lung alveoli. Fat embolism syndrome has been reported to occur with 30% to 50% of major long bone and pelvis fractures.[61] However, with the current coordinated management of multiply injured patients, the incidence of both fat embolisms and ARDS is decreased by expeditious femoral shaft and pelvic fracture treatment.[56] The primary treatment is ventilatory assistance. Therapy with steroids and acetylsalicylic acid has been shown to be helpful, possibly because of a reduction of platelet aggregation.

With a better understanding of fluid and electrolyte therapy, early aggressive management of hemorrhagic shock and prompt surgical treatment are now possible. However, in the interest of acute resuscitation, orthopedic injuries are often overlooked initially and are treated at a later time. When these injuries involve the spine, pelvis, or femur, immobilization of the patient is necessary for the purpose of traction. In immobilized patients with unstable fractures, there is an increased morbidity caused by respiratory failure or sepsis with related multiple organ failure. The severely injured patient with orthopedic fractures who survives the acute phase of treatment generally undergoes a prolonged course in the intensive care unit. This leads to morbidity secondary to decreased musculoskeletal function (e.g., muscle wasting, stiff joints, loss of limb length) caused by delays in fracture stabilization and subsequent patient mobilization.[62] Studies have shown that early fracture stabilization can significantly decrease mortality, musculoskeletal morbidity, and cardiopulmonary and metabolic consequences commonly associated with multiple trauma.[60]

Long bone fractures are a common cause of fat embolism and ARDS. Operative fixation of long bone fractures in patients with multiple injuries within the first few days of injury can minimize the development of fat embolism.[61] Primary rigid fixation allows the patient to get out of bed and assume an upright position, thus improving pulmonary and musculoskeletal function. Early mobilization, along with the use of mechanical ventilation with PEEP, lowers the incidences of ARDS and remote organ failure.[62]

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CHAPTER 15

Soft Tissue Injuries

Alan S. Herford, DDS, MD, and G.E. Ghali, DDS, MD, FACS

In the United States, over 11 million traumatic wounds are treated in emergency departments each year. Facial lacerations account for approximately 50% of these wounds, with motor vehicle collisions being the most prevalent cause of soft tissue damage to the face.[1] Facial injuries affect both function and aesthetics. There is often a psychological aspect associated with the injury secondary to patient's concern regarding permanent scarring and subsequent facial disfigurement. According to one survey, cosmetic outcome is the single most important aspect of care to the patient.[2]

Goals of treatment are to achieve a repair without infection and to minimize scarring. Timely diagnosis and repair are important to improve outcomes of these injuries.

PRINCIPLES OF MANAGEMENT

The initial examination involves evaluating and stabilizing the trauma patient. Any life-threatening conditions should be identified and managed immediately. The conditions of the airway, breathing, and circulation are examined, followed by a general neurologic assessment with particular attention to cervical spine and cranial injuries.

It is important to achieve hemostasis when stabilizing and evaluating the patient who has sustained trauma. Most bleeding will respond to application of a pressure dressing. Occasionally, surgical exploration and packing of the wound under general anesthesia may be indicated. In rare instances, vessels in the neck may need to be ligated. Indiscriminate clamping inside the wound should be avoided because damage to important structures such as the facial nerve or parotid duct may result. It is unusual for bleeding from soft tissue injuries to the face to result in a shock state. Lacerations involving the scalp can occasionally be difficult to control with pressure and may require clamping, ligation, or electrocautery.

In soft tissue injuries not involving the face, the length of time from initial injury to treatment is important. Secondary risk of infection increases with the lapse of time.[3] Because of the rich vascularity of the face there is no "golden period" for suture repair of facial wounds. In fact, healing of facial wounds is unaffected by the interval between injury and repair.[4]

Facial soft tissue injuries are common in pediatric patients. This patient population heals quickly, but also tends to develop hypertrophic scars. Proper wound management during the healing phase can help to minimize the risk of adverse scar formation.[5]

Patients who are immunized and have received a booster injection within the last 10 years do not require tetanus prophylaxis if the wound is not tetanus-prone. Tetanus-prone wounds are those with heavy contamination from soil or manure, devitalized tissue, or deep puncture wounds. If the wound is tetanus-prone and the patient has not received a booster injection within 5 years before the injury a 0.5-mL tetanus toxoid boost injection should be given. If the patient has not received a booster within 10 years before the injury, they should receive a booster injection for any wound. Patients who are not immunized should receive both a booster injection and 250 units of tetanus immunoglobin followed by a full course of immunization.[6]

The primary goal in managing traumatic wounds is to achieve rapid healing with optimal functional and aesthetic results. Despite good wound care, some infections still occur. Prophylactic antibiotics have a role in management of certain types of wounds.[7]

Treatment of soft tissue injuries involves early reconstructive procedures addressing both the soft tissue and the underlying bony injury in a minimum number of stages.[8,9] In patients with large avulsion of tissue, definitive early reconstruction of the tissue loss with regional or microvascular flaps may be required.[10,11] Various local flaps provide reliable and aesthetic treatment options for early repair of soft tissue defects secondary to trauma.[12]

ANATOMIC EVALUATION

After the initial evaluation and resuscitation, injuries to the soft tissues should be evaluated during the secondary survey. Patients sustaining trauma often have associated soft tissue injuries. Facial injuries can be superficial but may extend to involve adjacent structures including bones, nerves, ducts, muscles, vessels, glands, and/or dentoalveolar structures. Associated injuries, including vascular injury, may develop acutely or days after the injury.[13,14]

A thorough head and neck examination determines the extent of associated facial wounds. Peripheral cranial nerves are commonly involved with lacerations involving the face. The facial nerve divides the parotid gland into deep and superficial portions (Figure 15-1). Any injury to the gland should raise suspicion for associated facial nerve injury.[15] The facial nerve exits the stylomastoid foramen and divides into five branches within the parotid gland (Figure 15-2). Proximal facial nerve injuries posterior to a vertical line drawn from the lateral canthus should be repaired using microsurgical techniques. Because of the significant peripheral anastomoses, repair of facial nerve injuries involving distal branches anterior to the canthal plane is unnecessary (Figure 15-3).

The surgeon should have a true knowledge about the anatomy of the facial nerve branches.[16] The buccal branch has a close relationship with the parotid gland for over 2.5 cm after it emerges from the parotid and is normally inferior to the parotid duct.[17]

Injury to the parotid gland is often associated with penetrating wounds of the face and may lead to leak of saliva into the soft tissue.[18] The parotid duct is approximately 5 cm in length and 5 mm in diameter. It exits the gland and runs along the superficial surface of the masseter muscle and then penetrates the buccinator muscle to enter the oral cavity opposite the upper second molar. Treatment of parotid duct injuries depends on the location of the injury. These injuries should be repaired in the operating room with the aid of magnification. If the injury involves the proximal duct while it is still in the gland, the parotid capsule should be closed and a pressure dressing placed. If the injury is located in the midregion of the duct, the duct should be repaired. Injuries involving the terminal portion of the duct should be drained directly into the mouth. Lacrimal probes are useful in cannulating the duct and identifying injuries.[19] A Silastic catheter is placed to bridge the defect. The severed ends are then sutured over the catheter, which is left in place for 10 to 14 days (Figure 15-4). The parotid capsule should be closed to prevent formation of a parotid duct fistula or sialocele. Lacerations are closed primarily and a pressure dressing is placed to prevent fluid accumulation.

Several protocols are available for evaluation and treatment of penetrating injuries to the neck, face, and temporal bone. If there is suspicion that deep critical structures have been injured, the appropriate protocol should be followed. (Figure 15-5)

SEQUENCE OF REPAIR AND BASIC TECHNIQUE

A decision is made to repair the wound in the emergency department or to perform the repair in the operating room under a general anesthetic. Large, complicated lacerations demand ideal lighting and patient cooperation. In injuries in which there is a concern that deep structures have been damaged, a general anesthetic affords the best opportunity for

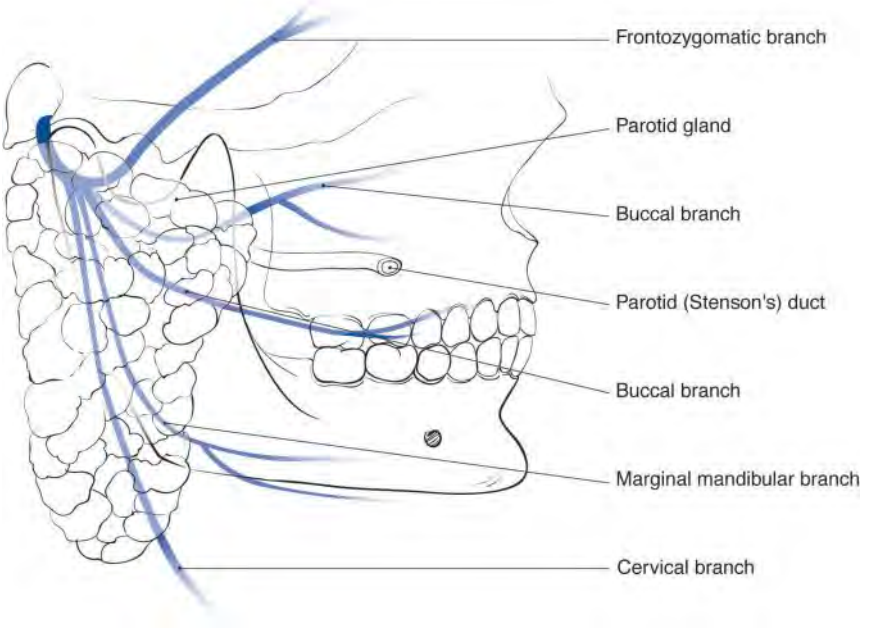


FIGURE 15-1. The facial nerve divides the parotid into a deep and a superficial lobe.

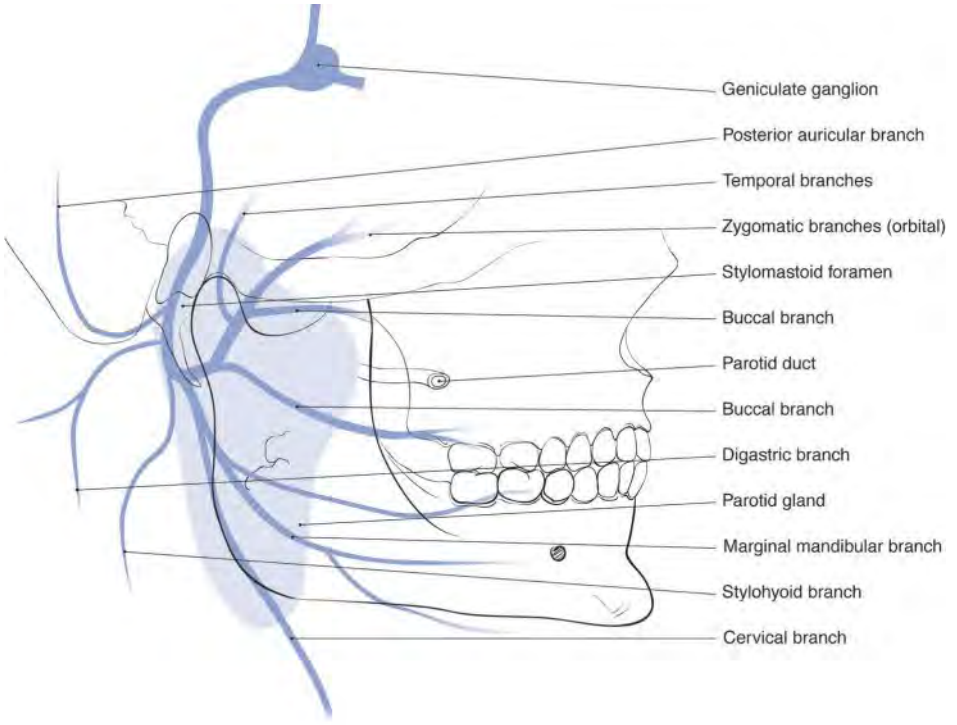


FIGURE 15-2. Anatomy of the facial nerve.

exploration and repair. The patient may require repair of other traumatic injuries in the operating room, and often, definitive repair of associated facial soft tissue injuries can be performed at the same time.

A thorough evaluation of the seventh cranial nerve should be undertaken before injection of anesthetic or administration of a general anesthetic. Injecting local anesthetic before cleaning the wound will allow more effective preparation. Local anesthetics containing epinephrine have been used successfully in all areas of the face but may not be optimal in areas where tissue monitoring is critical or where extensive undermining of the soft tissue is necessary.[20] One should avoid injecting directly into the wound when important landmarks could be distorted. Regional nerve blocks are beneficial in minimizing the amount of local anesthesia required and also prevent distortion of the tissues.[21]

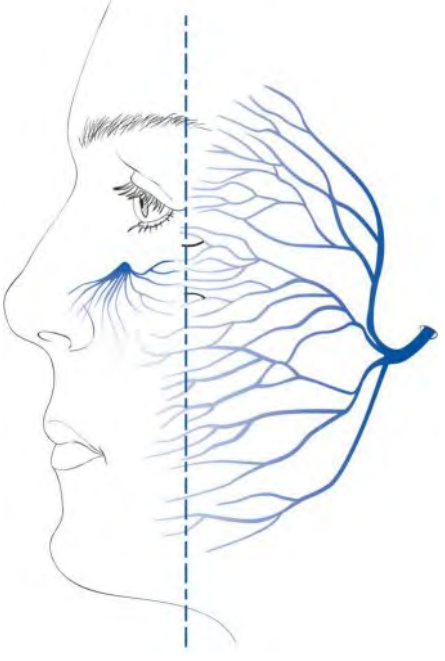


FIGURE 15-3. Zone of arborization of the facial nerve.

After adequate anesthesia has been obtained, the wound is thoroughly débrided. Nonvital tissue is conservatively excised in an attempt to salvage most of the tissue. Devitalized tissue potentiates infection at a wound site and leads to release of inflammatory cytokines from monocytes and macrophages that delay wound healing. An anaerobic environment results and limits leukocyte function.[22] Soft tissue wounds are often contaminated with bacteria and foreign material. Treatment of these injuries involves copious irrigation and is aimed at minimizing the bacterial wound flora and removing any foreign bodies. With respect to infection rates, studies have shown no statistical difference in wounds irrigated with normal saline when compared with other solutions. Pulsatile-type irrigation devices may be helpful to remove debris, necrotic tissue, and loose material. Hydrogen peroxide impedes wound healing and has poor bactericidal activity. A good rule is to avoid irrigating the wound with any solution that would not be suitable for irrigating the eye. Careful and meticulous cleaning of the wounds primarily will avoid unfavorable results such as "tattooing," infection,

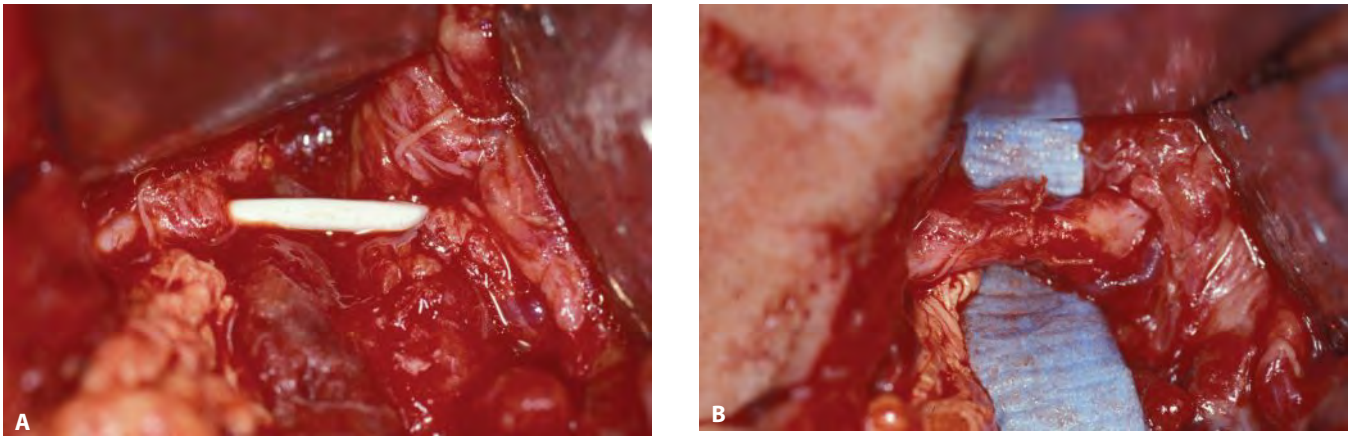


FIGURE 15-4. **A,** Laceration shows the parotid duct severed and cannulated with a polyethylene tube. **B,** The duct is sutured over the tubing.

hypertrophic scarring, and granulomas. A scrub brush and detergent soap may be necessary to remove deeply embedded foreign material.

Proper cleaning and good surgical technique are imperative in minimizing infection. Infections are rare when the wound is closed so that no dead space, devitalized tissue, or foreign bodies remain beneath the sutured skin. Hydrogen peroxide is minimally bactericidal and toxic to fibroblasts even when diluted to 1:100.[23] Diluted hydrogen peroxide is useful in the postoperative period in cleaning crusts away from incision lines in order to minimize scarring.

Common methods for closing wounds include suturing, applying adhesives, and stapling. A layered closure is almost always necessary and eliminates dead space beneath the wound. If the dead space is not obliterated, accumulation of inflammatory exudates may occur. This leads to infection, which in turn, may cause tension across the epidermis. Tension can cause necrosis of the skin edges due to



FIGURE 15-5. **A,** Facial nerve weakness. **B,** Laceration involving distribution of facial nerve. **C,** Temporoparietal facial flap used for facial resuspension. **D,** Gold weight placed in upper eyelid to aid in closure of eye.

impairment of the vascular supply and may cause an increase in scarring.[24]

Injuries involving anatomic borders such as the vermilion of the lip must be reapproximated precisely. Examples of these landmarks include eyebrows, lip margins, and eyelids. Lacerations should be closed by placing a suture in the center of the laceration to avoid creating excessive tissue on the end of the laceration (dog-ear). Deep layers should be reapproximated with 3-0 or 4-0 buried resorbable sutures. The superficial skin is closed with 5-0 or 6-0 suture. It is important to avoid causing puncture marks when grasping the wound edges.

Skin sutures should be removed 4 to 6 days after placement. By this time, the wound has regained only 3% to 7% of its tensile strength and adhesive strips (Steri-Strips) help support the wound margins.[25]

At 7 to 10 days after suture removal, the collagen has begun to cross-link. The wound is now able to tolerate early controlled motion with little risk of disruption[26] (Figure 15-6). As the wound heals, it will contract along its length and width and become inverted due to collagen and fibroblast maturation. Initial management is aimed at producing a slightly everted wound edge. The wound continues to remodel up to a year after injury but never regains greater than 80% of the strength of intact skin.

Tissue adhesives are gaining in popularity. Some studies have suggested similar cosmetic outcomes in wounds treated with octylcyanoacrylate when compared with standard wound closure techniques for non–crush-induced lacerations treated less than 6 hours after injury.[27–29] Closure of lacerations with octylcyanoacrylate is faster than standard wound closure methods. However, its use should be avoided in complex lacerations involving the face where there are aesthetic concerns.

Suture materials and different surgical techniques do not show substantial differences in relation to outcome. General characteristics of the patient (i.e., sex and age) and of the wound (i.e., length and site) seem to be important predictors

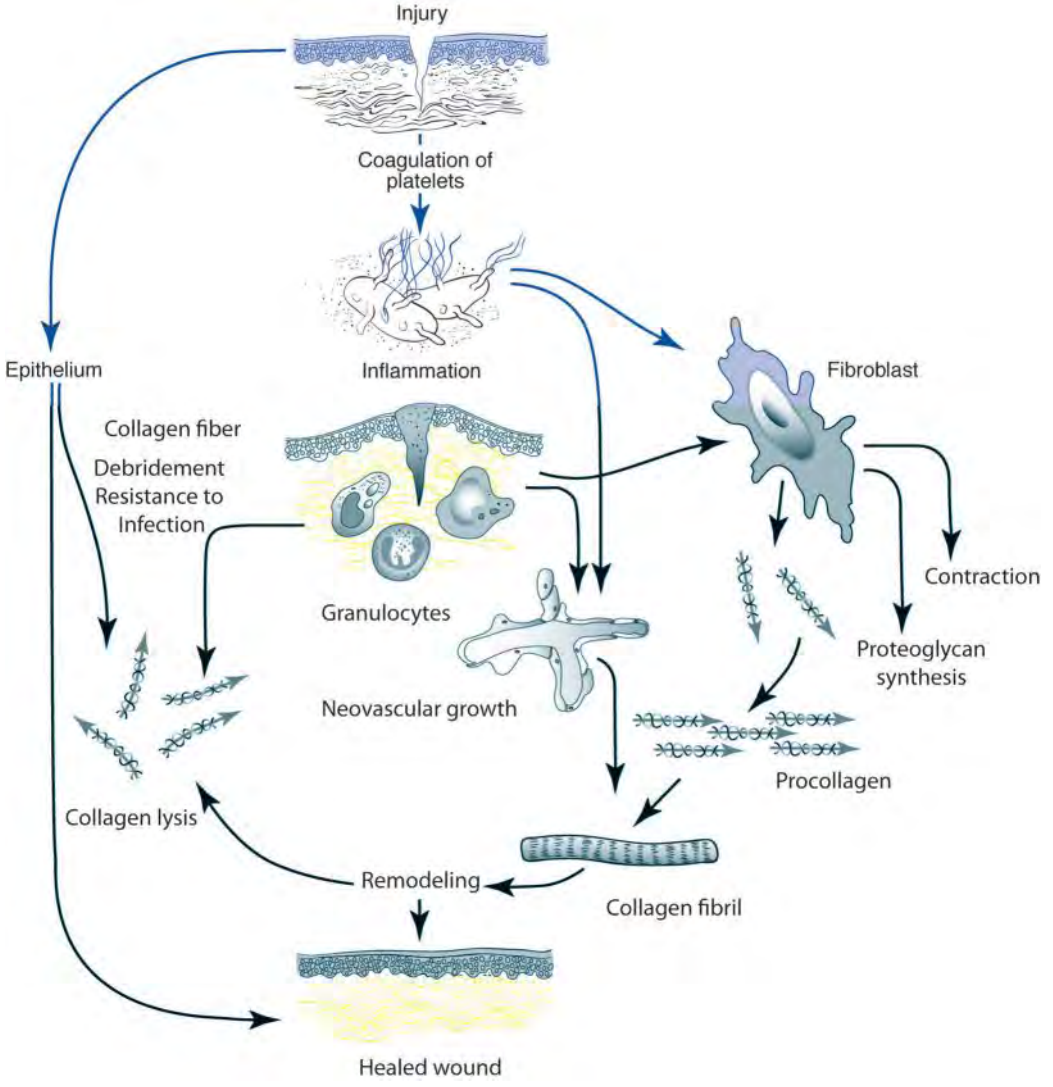


FIGURE 15-6. Stages of wound healing.

of adverse tissue reaction.[30,31] Suboptimal appearance is associated with wounds that are infected, wide, or incompletely approximated or have sustained a crush injury. The total number of bacteria is more important that the species of bacteria contaminating a wound. Greater than 10^5 aerobic organisms per gram of tissue are needed for contamination and crush-type wounds are 100 times more susceptible to infection.[32]

Delayed primary closure may be necessary in some instances. Patients who may benefit from a delayed procedure include those with extensive facial edema, a subcutaneous hematoma, or wounds that are severely contused and contain devitalized tissue. Secondary revision procedures are usually undertaken months later to allow for scar maturation.

Clinical examination and radiographs are used to diagnose fractures of the face. Facial fractures are ideally treated before soft tissue repair. If repair of the facial bones is delayed, it is optimal to close the lacerations initially. The wounds can be reentered and revised if needed to access to the fracture site.

TYPES OF INJURIES

Abrasions

Abrasions are caused by shear forces that remove a superficial layer of skin. The wound should be gently cleansed and irrigated with normal saline. These superficial injuries usually heal with local wound care. It is important to determine whether foreign bodies have been embedded into the wound. Failure to remove all foreign material can lead to permanent "tattooing" of the soft tissue. After cleansing the wound, the abrasion is covered with a thin layer of topical antibiotic ointment to minimize desiccation and secondary crusting of the wound.

Reepithelialization without significant scarring is complete in 7 to 10 days if the epidermal pegs have not been completely removed. If the laceration significantly extends into the reticular dermal layer, significant scarring is likely.

Contusions

Contusions are caused by blunt trauma that cause edema and hematoma formation in the subcutaneous tissues. The associated soft tissue swelling and ecchymosis can be extensive. Small hematomas usually resolve without treatment, but hypopigmentation or hyperpigmentation of the involved can occur but is rarely permanent. Large hematomas should be drained to prevent permanent pigmentary changes and secondary subcutaneous atrophy.

Lacerations

Lacerations are caused by sharp injuries to the soft tissue (Figure 15-7). They can have sharp, contused, ragged, or stellate margins. The depth of penetration should be carefully explored in the acute setting. Closure is performed using a layered technique. If the margins are beveled or ragged, they should be conservatively excised to provide perpendicular skin edges to prevent excessive scar formation. Rarely is there an indication for changing the direction of the wound margins by Z-plasty at the time of primary wound repair. Flaplike lacerations occur when a component of the soft tissue has been elevated secondary to trauma. Eliminating dead space by layered closure and pressure dressings is especially important in these "trap-door" injuries.

Avulsive Injures

Avulsive injures are characterized by the loss of segments of soft tissue. Undermining the adjacent tissue followed by primary closure can close small areas. When primary closure is not possible, other options are considered. These include local flaps or allowing the wound to heal by secondary intention followed by delayed soft tissue techniques. If a significant

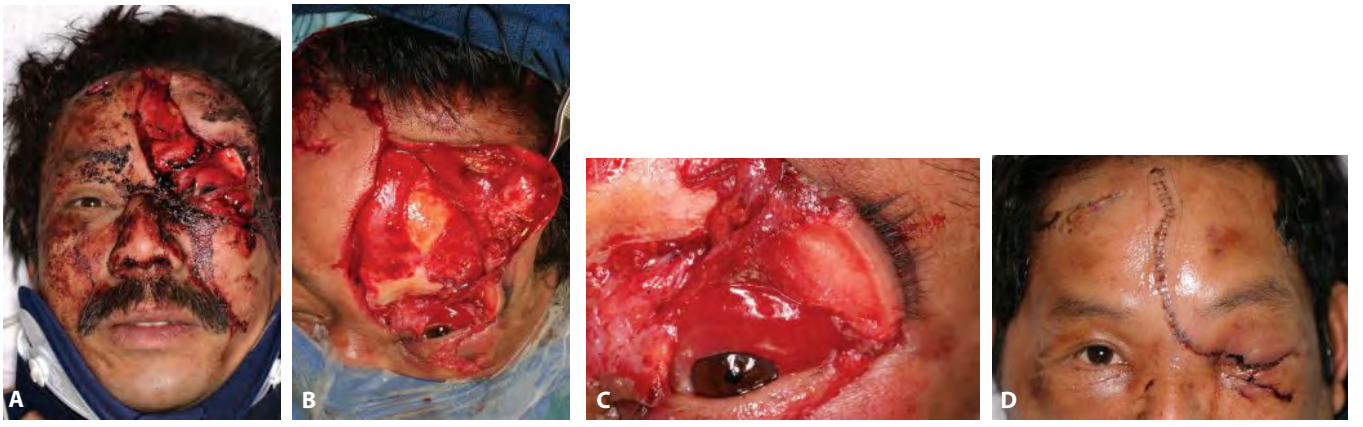


FIGURE 15-7. **A,** Patient with extensive lacerations. **B,** Laceration involving the eyelid. **C,** Partially avulsed upper eyelid. **D,** Closure of lacerations

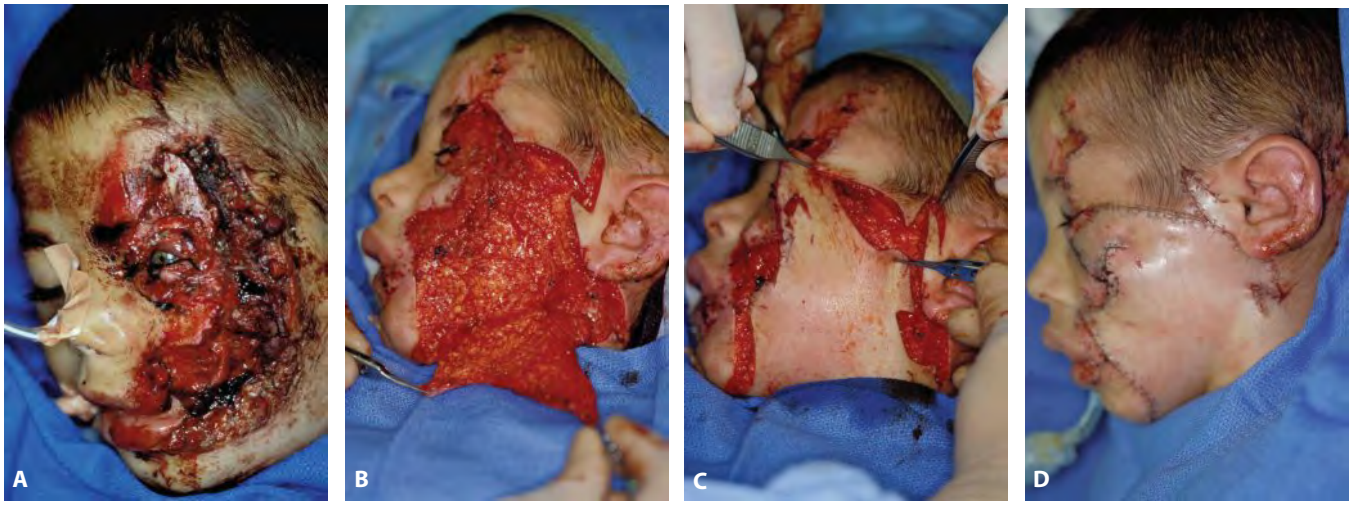


FIGURE 15-8. **A,** Patient with avulsive injuries including the upper and lower eyelids. **B,** Elevation of multiple advancement and rotation flaps to gain coverage. **C,** Securing the flaps into position. **D,** Closure of lacerations and flaps.

amount of soft tissue is missing, then a skin graft, local flaps, or free tissue transfer may be necessary (Figure 15-8).

Animal and Human Bites

Bite wounds are especially prone to infectious complications. Such complications can create more difficulties than the initial tissue damage itself for restoring an aesthetic appearance. Dog bites are most common in children and the midface is frequently involved.[33,34] Canines can generate 200 to 450 psi and examination for fractures should be performed.[35] Management of bite injuries involves liberal amounts of irrigation and meticulous primary closure.[36] Wound irrigation and débridement are important in reducing infection.

Animal and human bites are most often polymicrobial, containing aerobic and anaerobic organisms. Dog bites are often open and lend themselves to vigorous irrigation and débridement. Cats have a large quantity of bacteria in their mouth, with the most frequent and important pathogen being *Pasturella multicida*.[37] Cat bites are associated with a twofold higher risk of infection than the more common dog bite wounds. Because cat bites usually cause puncture wounds, they are difficult to clean. Having the patient follow-up 24 to 48 hours after the initiation of therapy allows the surgeon to monitor the wound for any signs of infection.

Antibiotic prophylaxis for animal bites continues to be debated with few good prospective studies available.[33,38] Amoxicillin clavulanate is the current drug of choice for bite wounds. Antibiotic prophylaxis should be directed at *P. multicida* for infections presenting within 24 hours of injury. For wounds that present after 24 hours of injury, *Streptococcus* and *Staphylococcus* species are more common and antibiotic prophylaxis with a penicillinase-resistant antibiotic should be chosen.[39]

Immediate closure of bite injuries is safe, even with old injuries.[40] There is approximately a 6% rate of infection when bite wounds are sutured primarily and is acceptable in lacerations where there are cosmetic concerns.[41] Extensive animal bite wounds involving the face should be treated according to the criteria of aesthetic reconstructive surgery. Delay in presentation beyond 24 hours is not necessarily a contraindication to immediate repair, but extensive crushing of the tissue or extensive edema usually dictates a more conservative approach, such as delayed closure.[42] Rabies prophylaxis should be given for bite wounds that occur in an unprovoked domestic dog or cat that exhibits bizarre behavior or an attack by a wild animal such as a raccoon, skunk, bat, fox, or coyote.[43]

Gunshot Wounds to the Face

Gunshot wounds require careful attention and evaluation for associated facial fractures. Both entry and exit wounds should be evaluated. Exit wounds often produce marked tissue destruction and require acute débridement. Regional flaps can be useful in treating facial soft tissue defects caused by gunshot wounds[10] (Figure 15-9). The primary phase requires urgent airway management as well as control of any active bleeding. Many of the patients have combined hard and soft tissue injuries and will require staged surgeries for reconstruction.[44]

Ballistic facial injuries are grouped by etiology: gunshot, shotgun, and high-energy avulsive injuries.[45] Blast injuries, such as those caused by improvised explosive devices (IEDs), show varying degrees of facial injures in different patterns with or without flash burns. These patients are subjected to intense thermal inhalation that leads to oronasopharyngeal edema and toxic fume inhalation that may require the management of life-threatening airway compromise.[46] Over



FIGURE 15-9. **A,** Patient who sustained a shotgun wound with avulsion of tissue. **B,** Preoperative radiograph shows associated comminuted facial fractures. **C,** Reapproximation of the bone and soft tissue. **D,** Postoperative radiograph shows the reduction of multiple facial fractures.

the past 20 years, advances in imaging and introduction of craniofacial approaches with rigid fixation has led to an evolution of treating facial injuries. The aesthetic and functional results of facial injury are improved dramatically by the combination of a definitive open reduction of bone with early replacement of soft tissue into its primary position. Immediate definitive reconstructions with rigid fixation of the facial fractures and closure of the lacerations are recommended. Standard incisions often need to be modified because of the soft tissue wounds.

REGIONAL CONSIDERATIONS

Certain anatomic areas deserve special consideration. Reestablishment of anatomic zones with proper orientation is critical in achieving optimal aesthetic results.

Scalp and Forehead

Scalp wounds can occasional cause a large amount of blood loss owing to the rich vascular supply in this region and the inelasticity of the scalp preventing contraction and closure of the vessels. The layers of the scalp (SCALP) include the skin (S), subcutaneous tissue (C), aponeurosis layer (A), loose subepicranial space (L), and pericranial layer (P).

In patients sustaining scalp injuries, it is important to evaluate for associated intracranial injuries. Careful inspection should be performed to look for evidence of skull fractures. Because the scalp has an excellent blood supply in the subcutaneous tissues as well as the pericranial layers, avulsed tissue, skin grafts, and various flaps have a high rate of survival. One study showed no significant difference in rate of infection in scalp lacerations that were irrigated compared with those that were not.[47]

In avulsive defects in which the pericranium is intact and primary closure is not possible, a split-thickness skin graft can be used. A secondary reconstructive procedure involving various rotational and advancement flaps or tissue expansion can be undertaken after healing of the defect.[48] If the cranial bone is exposed with large avulsive defects, then various flap procedures are indicated primarily.

Reconstruction of the eyebrow is difficult secondarily, and efforts to repair lacerations primarily without distortion are important. Eyebrows should never be shaved, because regrowth of the hair is unpredictable. Closure of lacerations should attempt to salvage as much tissue as possible. Care should be given to avoid damage to the remaining hair follicles. Scars can be removed 6 to 12 months later with incisions made parallel to the hair follicles to avoid injury.

Eyelid and Nasolacrimal Apparatus

A thorough ophthalmologic examination is important to assess for injuries to the globe and to evaluate and document visual acuity. Closure of lacerations involving the eyelids is done in a layered fashion (Figure 15-10). Care should be taken to precisely reapproximate the eyelid margins and the tarsus (Figure 15-11). The conjunctiva and tarsus are closed with resorbable sutures with the knot buried to avoid irritating the cornea. The orbicularis muscle is then closed followed by closure of the skin. Injuries involving the upper eyelid may include detachment of the levator aponeurosis and Muller's muscle from the tarsal plate. The muscles should be identified and reattached to the tarsal plate in order to prevent ptosis and restore levator function.

The lacrimal gland produces tears, which flow across the cornea and drain into canaliculi via the puncta of the upper and lower eyelid margins (Figure 15-12). From the canaliculi, the tears enter the nasolacrimal duct and drain into the inferior meatus of the nose. Any lacerations that involve the medial third of the eyelid should be carefully inspected for damage to the canaliculus.[49] Repair is accomplished by introducing a lacrimal duct probe into the puncta and into the wound (Figure 15-13). The ends of the lacerated duct are identified

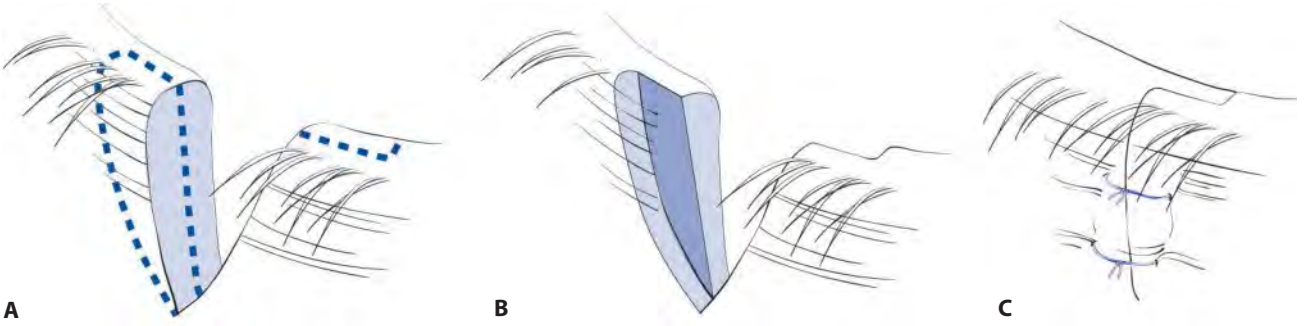


FIGURE 15-10. Surgical repair of the eyelid. **A,** Excision of the outer lamina on one side and the inner lamina on the other. **B,** After excision. **C,** Closure. Inner sutures are buried to avoid suture material irritating the conjunctiva.

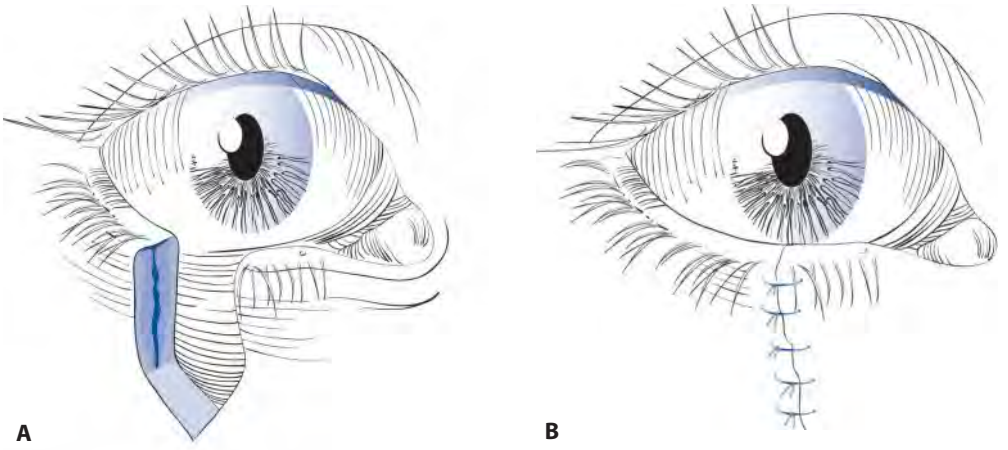


FIGURE 15-11. **A,** Pentagon resection of the lower lid allows straight closure of the tarsal plate. **B,** Closure is made with no suture material through the conjunctiva.

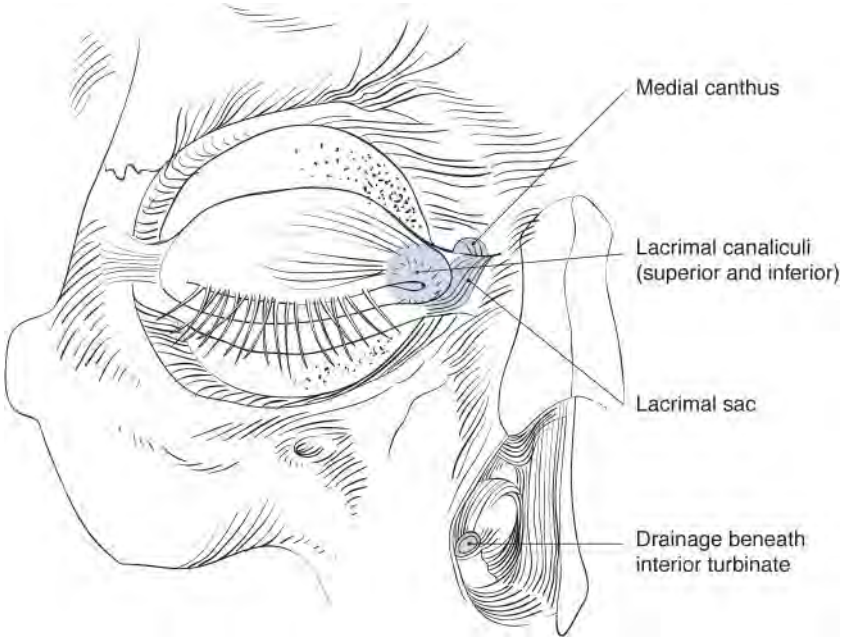


FIGURE 15-12. Nasolacrimal system anatomy.

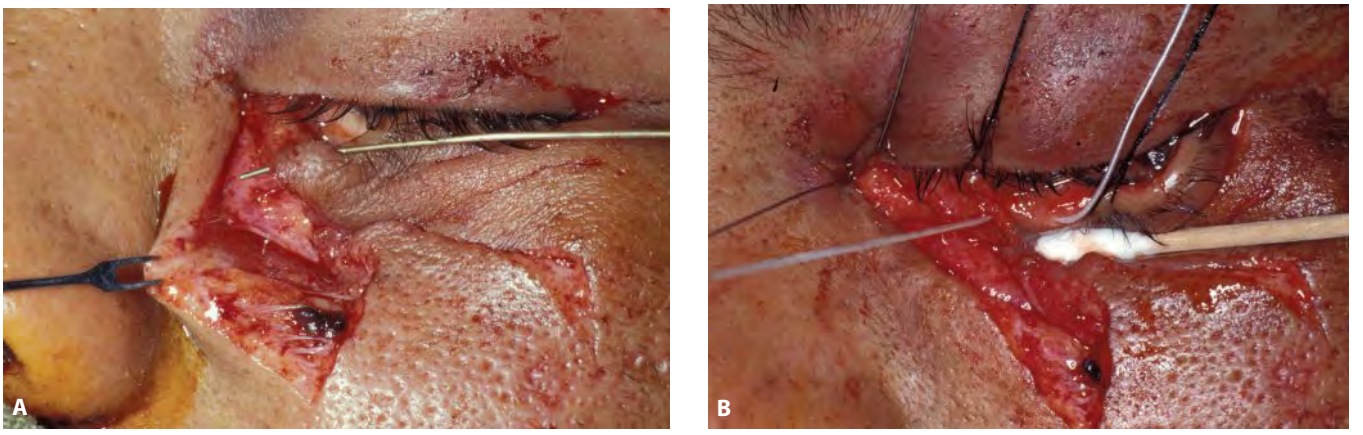


FIGURE 15-13. **A,** Lacrimal probe identifies the disrupted canaliculi. **B,** A Silastic tube is cannulated through the canaliculi.

and approximated over a Silastic tube (Crawford tube). The tube is left in place for 8 to 12 weeks. If only one canaliculus is intact and functioning, the patient most likely will have adequate drainage.[50] If the patient exhibits chronic epiphora postoperatively, then a dacrocystorhinostomy is indicated.

Avulsive injuries to the eyelids are treated with skin grafts and/or local flaps. Defects up to 25% of the eyelid length can be closed primarily. Skin grafts harvested from the opposite eyelid provide excellent texture and color match (Figure 15-14).



FIGURE 15-14. **A,** Laceration involving partial avuslsion of the upper eyelid. **B,** Healing with retraction of the upper eyelid. **C,** Placement of a graft to replace missing soft tissue. **D,** Reconstructed eyelid.

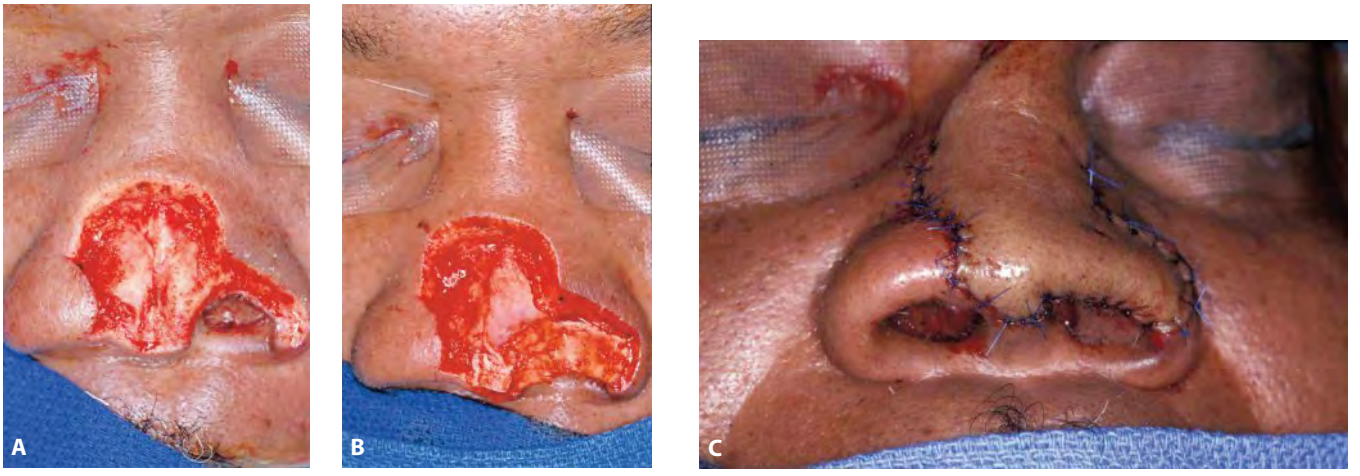


FIGURE 15-15. **A,** Nasal tip defect resulting from a bite. **B,** Grafting of cartilage to provide soft tissue support. **C,** The pedicled flap is sutured into place. The flap is divided 3 weeks later.

Nose

The nose occupies a prominent position on the face and is often injured. Injuries of the internal nose should be evaluated using a nasal speculum. The septum should be evaluated for the presence of a hematoma, which appears as a bluish elevation of the mucosa. Hematomas involving the nasal septum should be evacuated with a small incision or needle aspiration. Nasal packing or Silastic nasal splints can be placed to prevent recurrence of the hematomas and are removed in 7 to 10 days. A running 4-0 chromic gut mattress suture placed through and through the septum can prevent recurrence. Untreated hematomas can lead to infection and necrosis of the cartilage, which may cause collapse of the septum and a resultant "saddle nose."

There is an excellent blood supply to the nose. Lacerations of the external nose should be closed with 6-0 nonabsorbable sutures. Key sutures should be placed to reapproximate anatomic landmarks to ensure proper orientation, especially around the nasal rim. Bone, cartilage, and/or skin grafts may be required to reconstruct avulsive defects of the nose. Skin grafts harvested from the periauricular regions provide excellent color and texture match.[51] Local flaps may be required to restore missing tissue (Figure 15-15).

Ear

Injuries involving the external ear should alert one to the possibility of other injuries. An otoscopic examination of the external auditory canal and tympanic membrane combined with a hearing assessment should be performed before treatment. Injuries to the auricle include ecchymosis, abrasion, laceration, hematoma, and partial or total avulsion.

Hematomas involving the ear usually occur when the ear sustains a glancing blow. These should be drained with a needle or incision. An incision is often preferable to simple aspiration because there is less of a chance of reaccumulation of the hematoma.[52] Evacuation of the hematoma prevents fibrosis and development of a "cauliflower ear" deformity. A bolster dressing should be placed to prevent recurrence of the hematoma. A stent can also be fabricated from polysiloxane impression material and kept in place for 7 days.[53]

The ear has a very good vascular supply and can maintain tissue on a small pedicle. Injuries involving the cartilage often do not require sutures. If sutures are required, a minimal number are used to avoid devitalizing the region of cartilage (Figure 15-16). Avulsive injuries of the ear can involve a portion of the ear or the entire ear (Figure 15-17). If the avulsed segment is 1 cm or less, it can be reattached and allowed to revascularize.[54] For larger avulsive injuries, the ear should be examined for vessels for the possibility of microvascular reattachment. A more predictable method is to use the "pocket principle" described by Mladick and coworkers[55] (Figure 15-18). The detached ear is dermabraded to remove the superficial dermis and reattached to the stump. It is then buried underneath a skin flap elevated in the posterior auricular region to provide vascular supply to the reattached ear. Approximately 2 to 3 weeks later, the revascularized ear is uncovered and allowed to reepithelialize.

If salvage of the ear is not possible, other alternatives include staged reconstruction with rib cartilage, skin flaps, or silicone implants. The introduction of osseointegrated implants has made prosthetic reconstruction an appealing treatment option (Figure 15-19).

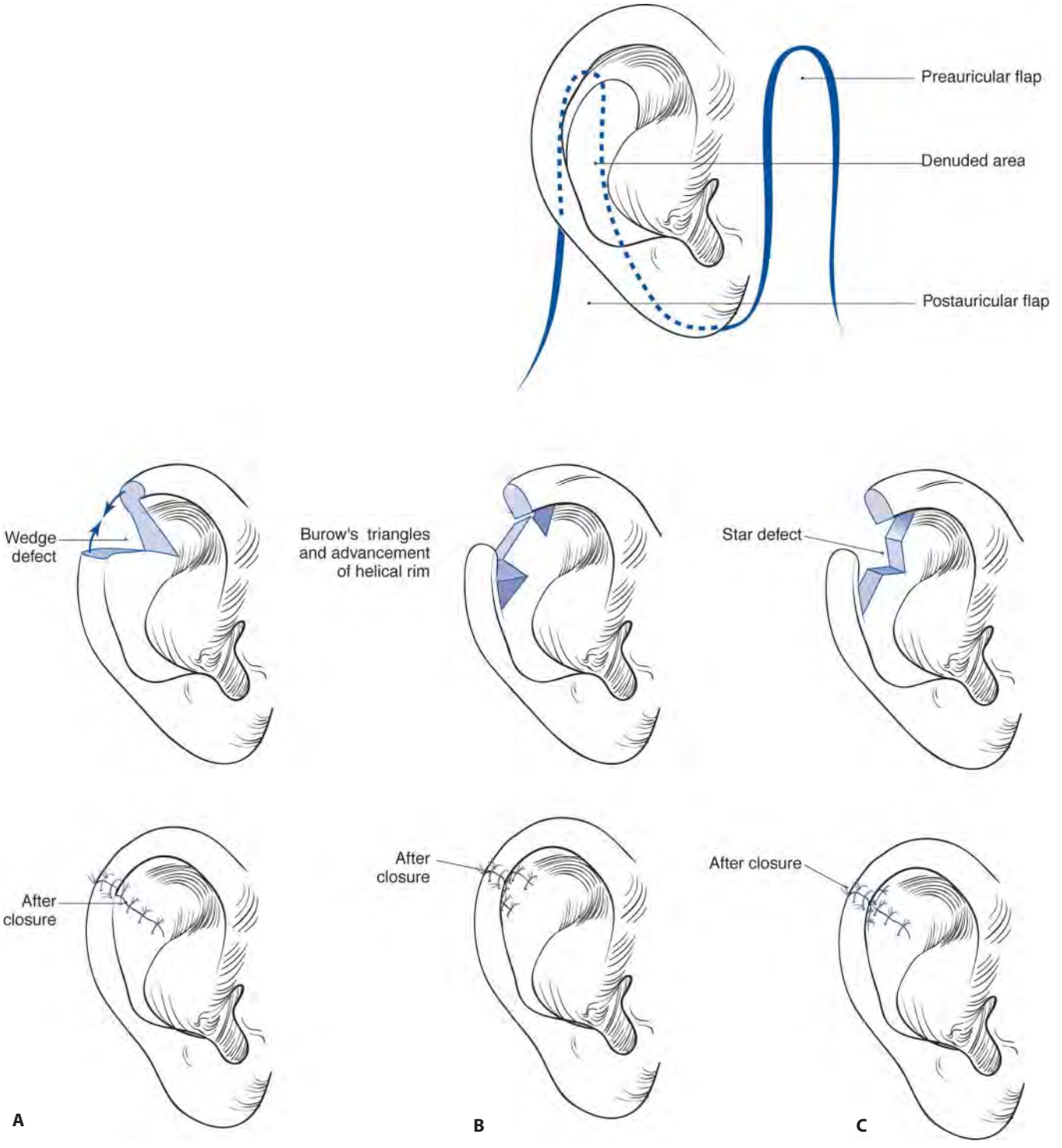


FIGURE 15-16. **A,** Conversion of a defect to a wedge. **B,** The use of Burrow's triangles. **C,** Conversion of a defect to a star.

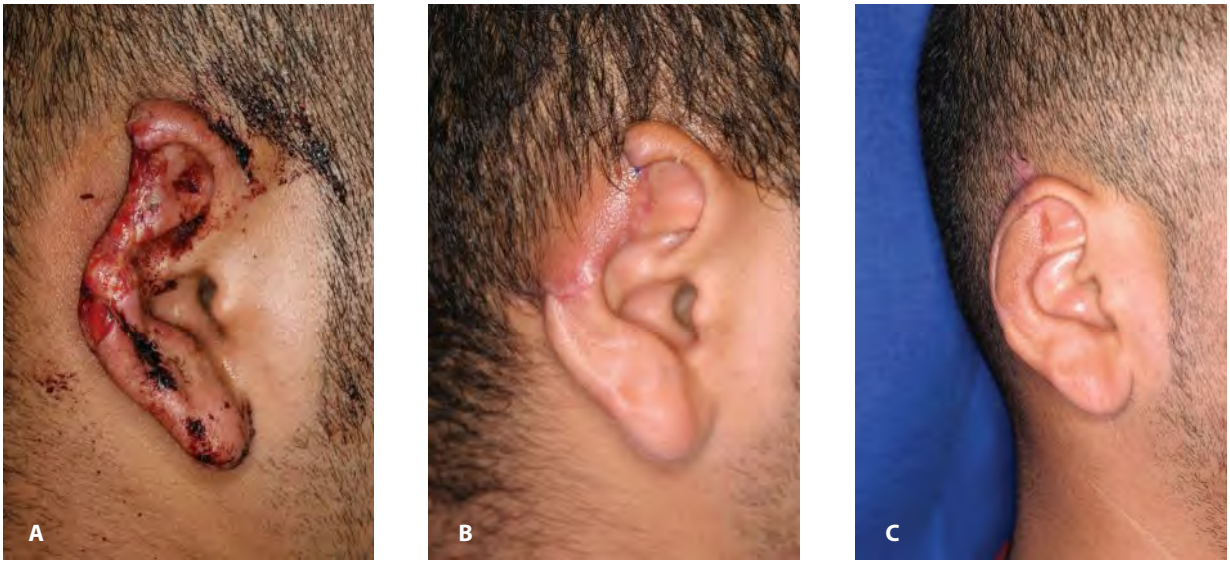


FIGURE 15-17. **A,** Auricle injury with loss of the helix. **B,** Closure of the grafted site with a posterior skin flap. **C,** Postoperative appearance.

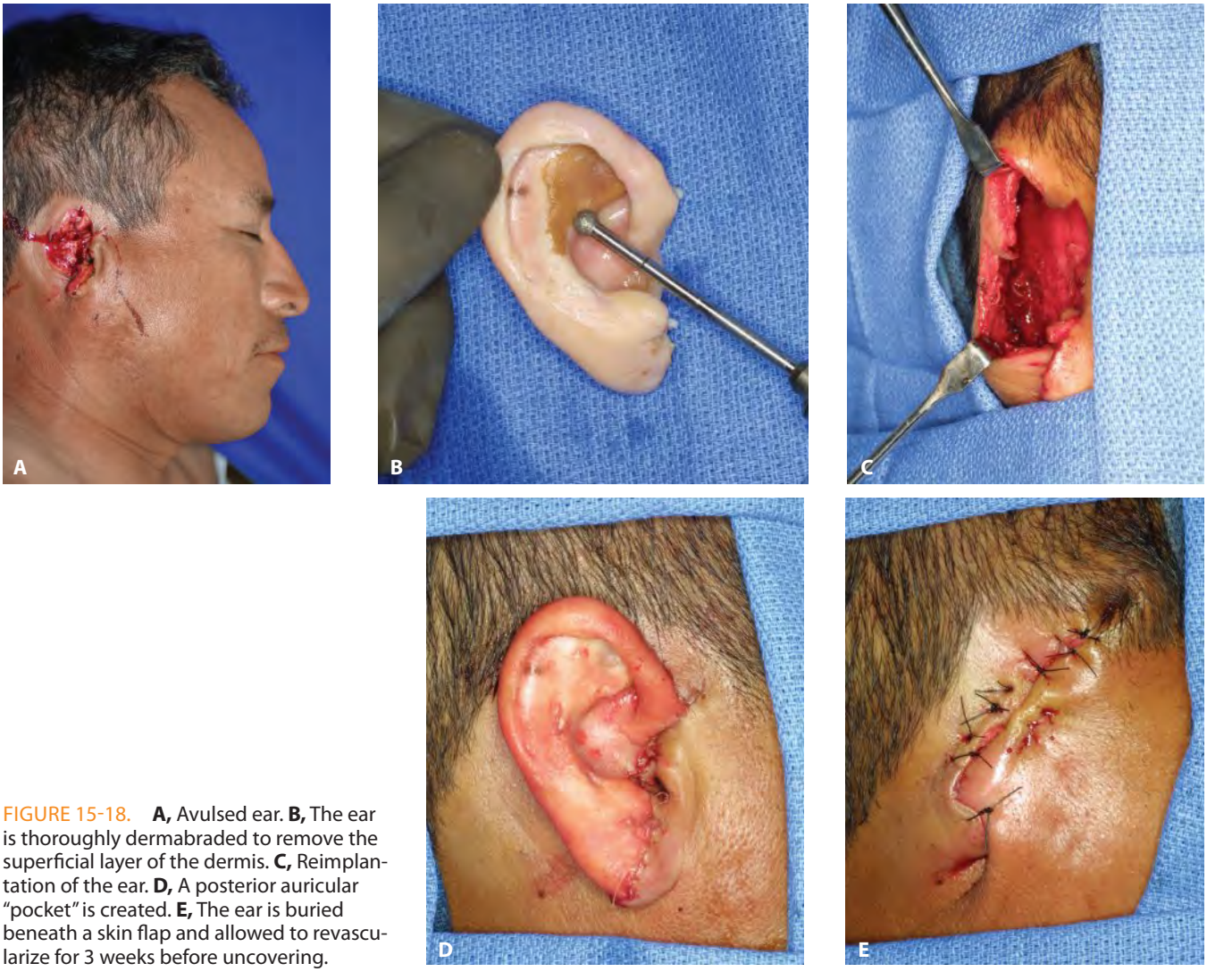


FIGURE 15-18. **A,** Avulsed ear. **B,** The ear is thoroughly dermabraded to remove the superficial layer of the dermis. **C,** Reimplantation of the ear. **D,** A posterior auricular "pocket" is created. **E,** The ear is buried beneath a skin flap and allowed to revascularize for 3 weeks before uncovering.



FIGURE 15-19. **A,** Osseointegrated implants are placed in the area of the defect. **B,** The prosthesis secured in place with magnets.

Lip

The lip anatomy involves a transition of mucosal tissue to skin. Scars that affect the orbicularis oris may result in functional difficulties. Nerve blocks are helpful in wounds involving the lip to prevent distortion caused from injecting directly into the wound. A single suture should be placed initially to reapproximate the vermilion border exactly. Deep tissues are closed in layers followed by closure of the mucosa with 4-0 chromic and skin closure with 6-0 nylon suture.

Avulsive defects of the lips require special attention. Up to one fourth of the lip can be closed primarily with acceptable functional and aesthetic results. Injuries that involve a greater amount of tissue loss can be reconstructed with a variety of flaps (i.e., Abbe-Estlander or Karapanzic) (Figure 15-20).

Neck

Successful management of penetrating injuries of the neck depends on a clear understanding of anatomy of the region. Injuries can involve deep structures affecting the vascular, respiratory, digestive, neurologic, endocrine, and skeletal systems.[56] The neck is divided into three anatomic zones.[57] Zone I extends from the level of the clavicles and sternal notch to the cricoid cartilage. Zone II is from the level of the cricoid cartilage to the angle of the mandible. It is the most surgically accessible and is the easiest to evaluate intraoperatively without the aid of preoperative diagnostic testing. Zone III extends from the angle of the mandible to the base of the skull.

There is controversy regarding which penetrating neck wounds require exploration.[56–60] Serial physical examinations

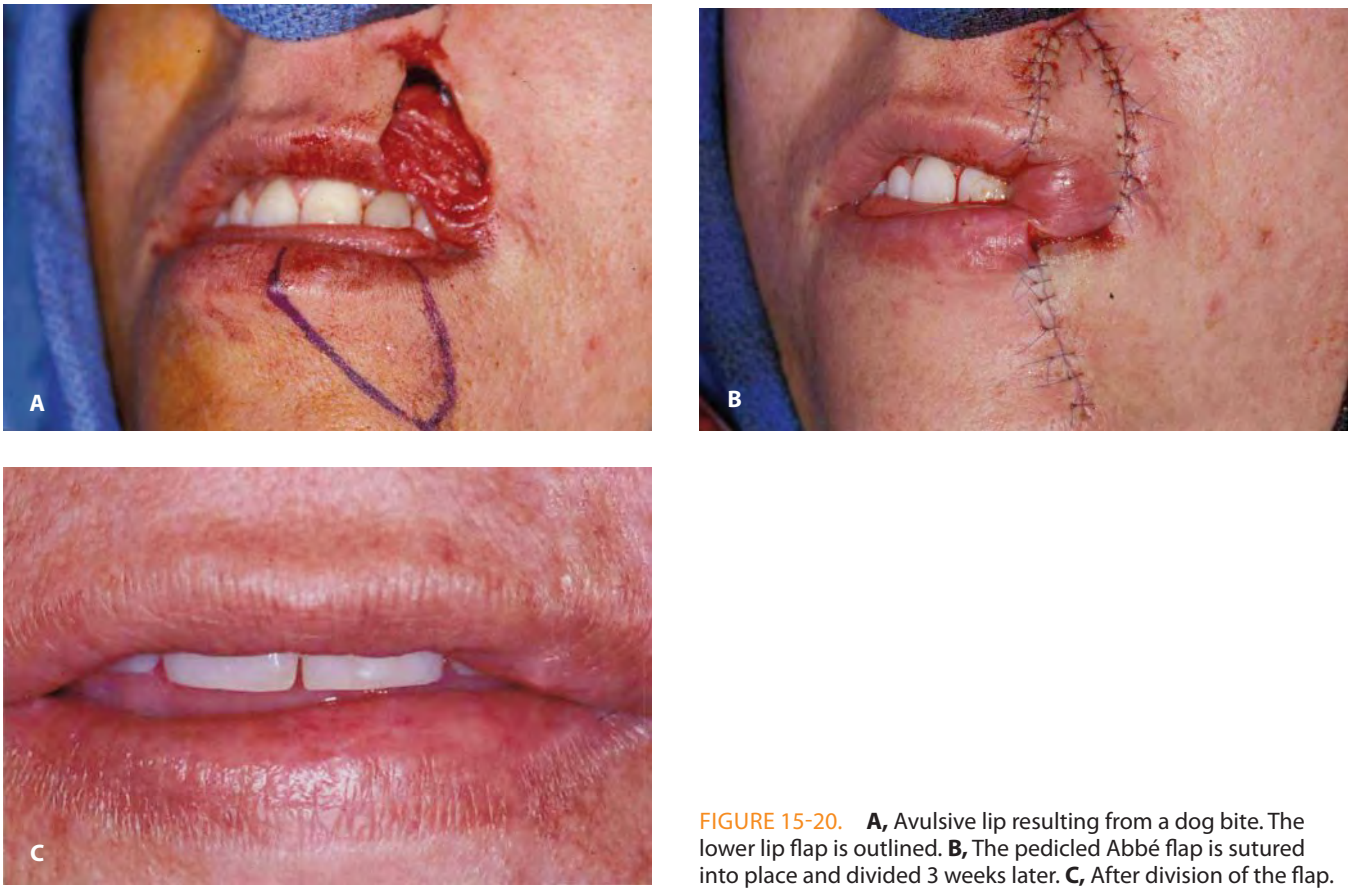


FIGURE 15-20. **A,** Avulsive lip resulting from a dog bite. The lower lip flap is outlined. **B,** The pedicled Abbé flap is sutured into place and divided 3 weeks later. **C,** After division of the flap.

alone have been shown to be effective. In cases in which serial physical examinations are not possible, mandatory exploration of neck wounds may be beneficial. There should be a high index of suspicion for esophageal injuries because complications can be devastating if repair is delayed. Primary repair is most often indicated in tracheal and vascular injuries.

POSTOPERATIVE WOUND CARE

Careful postoperative care and follow-up are important to optimize results. Wounds should be monitored closely to determine whether early intervention is indicated to minimize scar contracture or hypertropic scarring. Local flaps and grafts may be indicated secondarily. Local injection of steroids provides an adjunct in the management of specific types of injuries. Facial scars continue to mature over a period of 12 to 18 months. One study found no difference in outcome of surgical scars treated with pulsed carbon dioxide laser when compared with dermabrasion.[61]

Keeping a wound clean and scab free allows for more rapid reepithelialization.[62] Epithelial cells survive and migrate better in a moist environment. Antibiotic ointment can enhance this migration. It is not epithelialization that provides strength to the wound but rather the collagen fibers supporting the surface. Rebuilding of fibers takes time and suturing a wound splints the skin together until new connective tissue is built.

Cleaning daily with dilute hydrogen peroxide and dressing with polymyxin B sulfate and bacitracin zinc (Polysporin) ointment is standard. Patients should avoid sun exposure for the first 6 months after the injury to avoid hyperpigmentation of the areas.

SUMMARY

Soft tissue injuries involving the face can be devastating to the patient. Primary repair of these wounds is almost always advantageous over delayed secondary procedures. The primary goals of treatment are to restore the patient to his or her preoperative state of function and achieve an aesthetic result.

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16

CHAPTER

Rigid versus Nonrigid Fixation

Edward Ellis III, DDS, MS

Internal fixation simply implies the placement of wires, screws, plates, rods, pins, and other hardware *directly to the bones* to help stabilize a fracture. Internal fixation can be *rigid* or *nonrigid*, depending on the nature of the fracture and the type, strength, size, and location of the hardware placed. Because various degrees and many types of *nonrigid* fixation exist, it is useful to first define *rigid* internal fixation. By default, any technique that does not satisfy this definition can then be considered *nonrigid.*

RIGID INTERNAL FIXATION

The term *rigid internal fixation* has many definitions. For instance, one definition is "any form of bone fixation in which otherwise deforming biomechanical forces are either countered or used to advantage to stabilize the fracture fragments and to permit loading of the bone so far as to permit active motion."[1] This definition, although admittedly long and perhaps confusing, encompasses the essence of the technique as practiced today and includes clues to the methods of applying the appropriate hardware. A more basic definition that includes the same objectives is "any form of fixation applied directly to the bones which is strong enough to prevent interfragmentary motion across the fracture when actively using the skeletal structure."[2] Most of the differences in technique are in the application of the fixation. Inherent in these definitions is the prerequisite for surgical exposure to anatomically align the fragments (open reduction) and secure the fixation hardware. To rigidly stabilize fractures, an operative procedure is necessary.

Examples of rigid fixation in the mandible are the use of two lag screws or bone plates across a fracture, the use of a reconstruction bone plate with at least three screws on each side of the fracture, and the use of a large compression plate across a fracture (Figure 16-1). Properly applied, these fixation schemes are of sufficient rigidity to prevent interfragmentary mobility during the healing period.

An inseparable corollary to the prevention of interfragmentary mobility by rigid fixation is a peculiar type of bone healing in which no callus forms. The bones instead go on to heal by a process of haversian remodeling. Histologically, osteoclasts cross the fracture gap and are followed by blood vessels and osteoblasts (Figure 16-2). New bone is laid down by the osteoblasts, forming osteons that cross the gap and impart microscopic points of bony union to the fracture.[3] A remodeling phase then converts the entire area to morphologically normal bone. This type of bone healing is termed *primary* or *direct* bone union, and it requires absolute immobilization between the osseous fragments, that is, *rigid* fixation, and minimal distance (gap) between them.

NONRIGID INTERNAL FIXATION

Any form of bone fixation that is *not* strong (rigid) enough to prevent interfragmentary motion across the fracture when actively using the skeletal structure is considered *nonrigid.* The basic difference between rigid and nonrigid fixation centers on interfragmentary mobility. If there is mobility of the osseous fragments during active use of the skeletal structure after application of internal fixation devices, internal fixation is nonrigid. An example of nonrigid fixation is a transosseous wire placed across a mandibular fracture. The wire can provide stability only by virtue of its (limited) ability to prevent spreading of the gap, but by itself, the wire cannot neutralize torsion and/or shear forces. Additional fixation measures then become necessary, such as the use of maxillomandibular fixation (MMF) (Figure 16-3).

However, various forms of nonrigid fixation are recognized, and there is a continuum between rigid fixation and no fixation at all. Some forms of nonrigid fixation are strong enough to allow active use of the skeleton during the healing phase but not of sufficient strength to prevent interfragmentary mobility. These types of fixation have been called *functionally stable fixation,* indicating that there is adequate

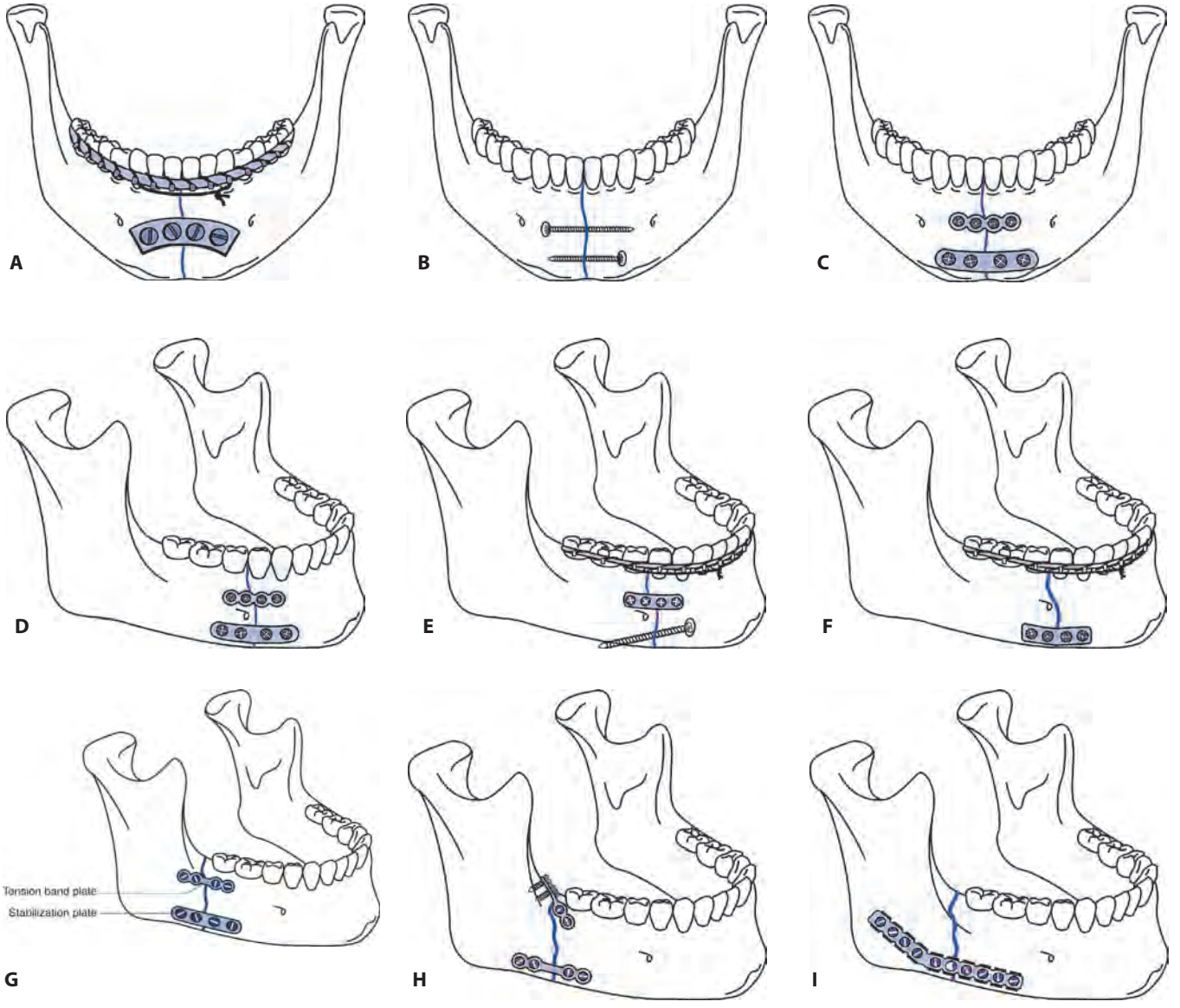


FIGURE 16-1. Examples of rigid fixation schemes for mandibular fracture. **A,** A large compression plate in combination with an arch bar for a symphysis fracture (two-point fixation). **B,** Two lag screws inserted across a symphysis fracture (two-point fixation). **C,** Two bone plates for a symphysis fracture (two-point fixation). These may or may not be compression plates. Typically, the larger one at the inferior border is a compression plate and the one located more superiorly is not. **D,** Two bone plates for a mandibular body fracture (two-point fixation). These may or may not be compression plates. Typically, the larger one at the inferior border is a compression plate and the one located more superiorly is not. **E,** A lag screw placed at the inferior border combined with a smaller bone plate located more superiorly (may or may not be compression plate; two-point fixation). The use of an arch bar offers a third point of fixation. **F,** A large compression plate placed at the inferior border of a body fracture combined with an arch bar (two-point fixation). **G,** A compression plate at the inferior border of an angle fracture combined with a noncompression plate at the superior border (two-point fixation). The upper plate could also be a compression plate. **H,** Two noncompression miniplates applied to an angle fracture (two-point fixation). **I,** Reconstruction bone plate applied to the inferior border of an angle fracture (one-point fixation). Rigidity is provided by virtue of the thickness (strength) of the plate and the use of at least three bone screws on each side of the fracture.

stability to allow function even though there is not adequate stability to allow direct bone union. Many of the fixation schemes that are being used clinically in the maxillofacial area are not truly *rigid* fixation, but *functionally stable* fixation. Functionally stable fixation in maxillofacial surgery is a spectrum that varies from one region of the facial skeleton to another, from one fracture to the next, and from one patient to the next. Examples of functionally stable fixation include the single miniplate technique of treating mandibular angle or body fractures (Figure 16-4).[4] In spite of the interfragmentary motion that these techniques may permit, the clinical outcomes are excellent, indicating that absolute immobility of the fragments is unnecessary for satisfactory recovery.

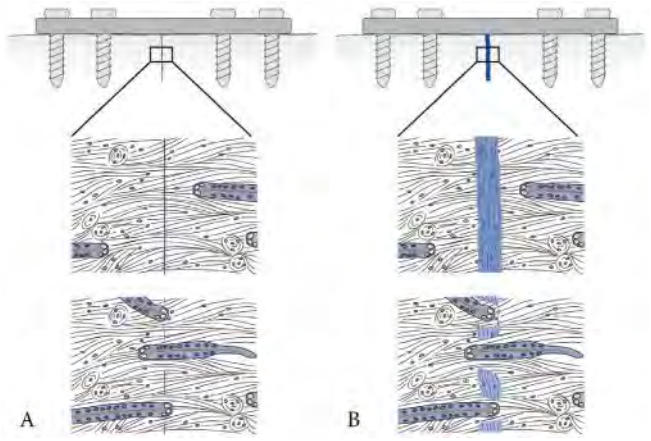


FIGURE 16-2. Types of primary bone healing. **A,** When there is minimal distance between the fragments and the fragments are rigidly immobilized, osteoclasts from one fragment "drill" their way into the fracture gap and into the opposite fragment. Behind them come fibrovascular tissue and osteoblasts, which begin to lay down new bone. With maturation, these become new haversian canals. This process is usually called *contact healing.* **B,** When a small gap exists between the rigidly immobilized fragments, lamellar bone is laid down within the fracture gap. Then the process described in **A** occurs, with new haversian canals crossing the gap. This process is sometimes called *gap healing.* With either of these types of primary bone healing, no external callus would be found along the outside of the fragments if they were rigidly immobilized. (**A** and **B,** Adapted from Schenk R, Willenegger H. Morphological findings in primary fracture healing. Symp Biol Hung 1967;7:75.)

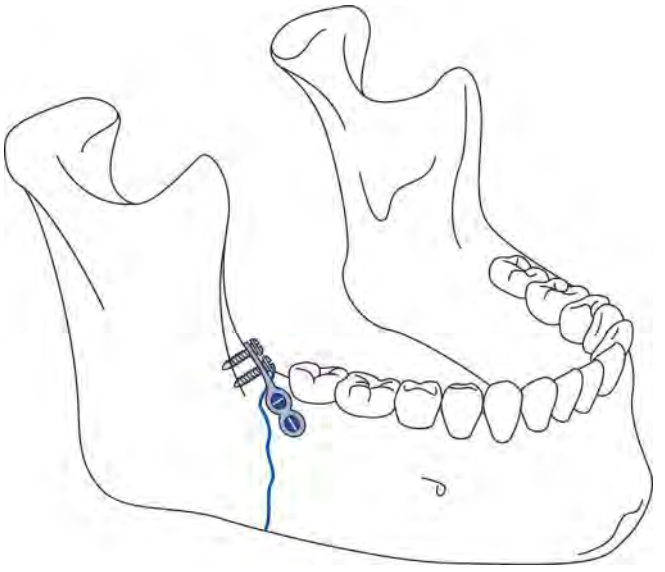


FIGURE 16-4. The Champy method of treating angle fractures using a single, noncompression miniplate attached with 2.0-mm monocortical screws. Because this plate is placed in the most biomechanically advantageous area for this region (superior border), a small plate can neutralize the functional forces and permit active use of the mandible during the healing process. However, although this technique is functionally stable, interfragmentary motion probably occurs to some extent during function. It is therefore not rigid fixation. (Adapted from Champy M, Loddé JP, Schmitt R, et al. Mandibular osteosynthesis by miniature screwed plates via a buccal approach. J Maxillofac Surg 1978;6:14–19.)

In the late 1950s, the Swiss Association for the Study of Internal Fixation (AO/ASIF) promulgated four biomechanical principles in fracture management[5]:

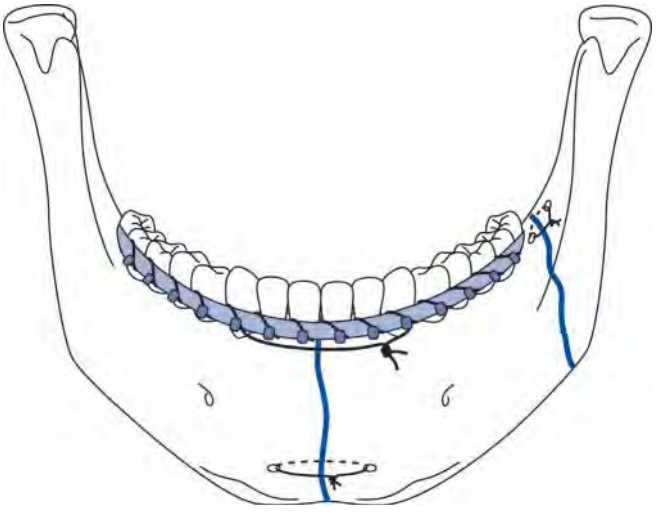


FIGURE 16-3. Internal wire fixation of symphysis and left angle fractures. Note that these wires are not sufficiently stable to allow use of the mandible during the healing process, so maxillomandibular fixation is applied for at least 5 weeks (in an adult) to maintain stability.

1. Accurate anatomic reduction.
2. Atraumatic operative technique preserving the vitality of bone and soft tissues.
3. Rigid internal fixation that produces a mechanically stable skeletal unit.
4. Avoidance of soft tissue damage and "fracture disease" by allowing early, active, pain-free mobilization of the skeletal unit.

These principles had as their aim the rigid fixation of fractures. In recognition of the finding that functionally stable fixation is very effective clinically, in 1994, the AO/ASIF changed its third biomechanical principle from rigid internal fixation to functionally stable fixation.

Bone healing under the condition of mobility between the osseous fragments is termed *indirect* or *secondary* bone healing. In such circumstances, there is deposition of periosteal callus, resorption of the fragment ends, and tissue differentiation through various stages from fibrous to osseous (Figure 16-5). Bone cannot form across a mobile gap. The formation of a callus can be thought of as nature's internal fixation, providing stability to the osseous fragments so that bone union can proceed. The appearance of a callus on a radiograph indicates that there is mobility between the fragments, requiring the deposition of the callus to "immobilize" the fragments to allow ossification to proceed.

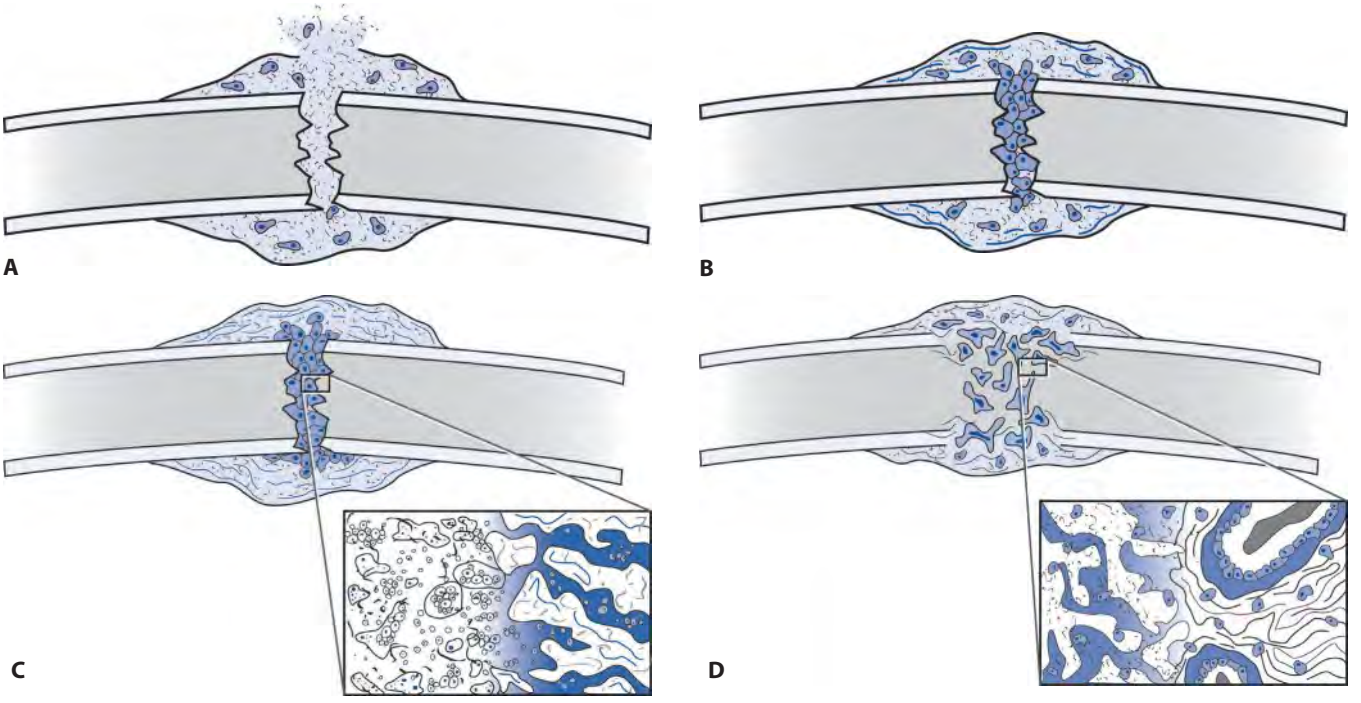


FIGURE 16-5. Secondary or indirect bone healing. **A,** After a fracture occurs, a subperiosteal hematoma is formed followed by initial invasion of granulation tissue. **B,** A thin rim of bone forms under the periosteum by membranous ossification. Hyaline cartilage is formed, progressing toward and eventually penetrating the fracture gap. **C,** The cartilage cells form columns, increase in size, and narrow the intercellular matrix. Small finger-like extensions of vessels penetrate these columns. The cartilage is replaced with woven bone. **D,** As the osseous matrix matures, remodeling and replacement of woven bone continues until a lamellar pattern is present. (**A–D,** Adapted from Müller ME, Algöwer M, Willenegger H, editors. Manual of Internal Fixation. New York: Springer-Verlag; 1970.)

SELECTION OF FIXATION SCHEMES: HOW MUCH FIXATION (RIGIDITY) IS ENOUGH?

With that prelude into definitions of fixation types, the remainder of this chapter discusses some of the variables in the selection of fixation schemes for fractures of the mandible. Because the mandible is the only bone in the face that is mobile and subjected to deforming forces from powerful muscles, not much will be said about the midface. However, whether in trauma or orthognathic surgery, the type of fixation that is required in the midface is functionally stable "adaptation" osteosynthesis. The bones are simply placed into a certain position and the fixation devices are applied to maintain that position. One would, therefore, not use compression plates in the midface (with the possible exception of the frontozygomatic suture area) because of their ability to change the spatial relationship of the bones by applying an active force across the fracture or osteotomy. However, bone plates of sufficient strength must be applied across a fracture or osteotomy gaps to allow the transmission of functional forces across the gap without an alteration in the occlusion. The application of very thin bone plating systems seems to be able to provide such stability in most fractures or osteotomies when placed in multiple locations. For instance, at the Le Fort I level, four thin bone plates (1.3- or 1.5-mm systems) provide functionally stable fixation under most circumstances. However, when there has been a large movement of the maxilla such as in a maxillary advancement or inferior repositioning procedure, thicker and stronger bone plates would usually be required (Figure 16-6).

BIOMECHANIC STUDIES VERSUS CLINICAL OUTCOMES

When selecting a fixation scheme for a given fracture, one has to consider many things, such as the size and number of fixation devices, their location, the surgical approach, and the amount of soft tissue disruption necessary to expose the fracture and place the fixation devices. In choosing the fixation scheme for the fracture, one might intuitively believe that more rigid fixation is better than less rigid fixation. However, it is clear from the literature on outcomes of mandibular fractures that the stability of the fracture construct is only one variable in determining a successful outcome for the patient. The results from every study ever performed in the laboratory or in computer modeling have shown that two bone plates applied to a fracture are more stable than one.[6–8] However, there has never been any statistically significant evidence from clinical studies that two plates perform better than one. In fact, the results of the author's own studies for fractures of the angle of the mandible show that two plates perform much more poorly than does one plate in that location.[9–13] One must, therefore, be very careful in applying

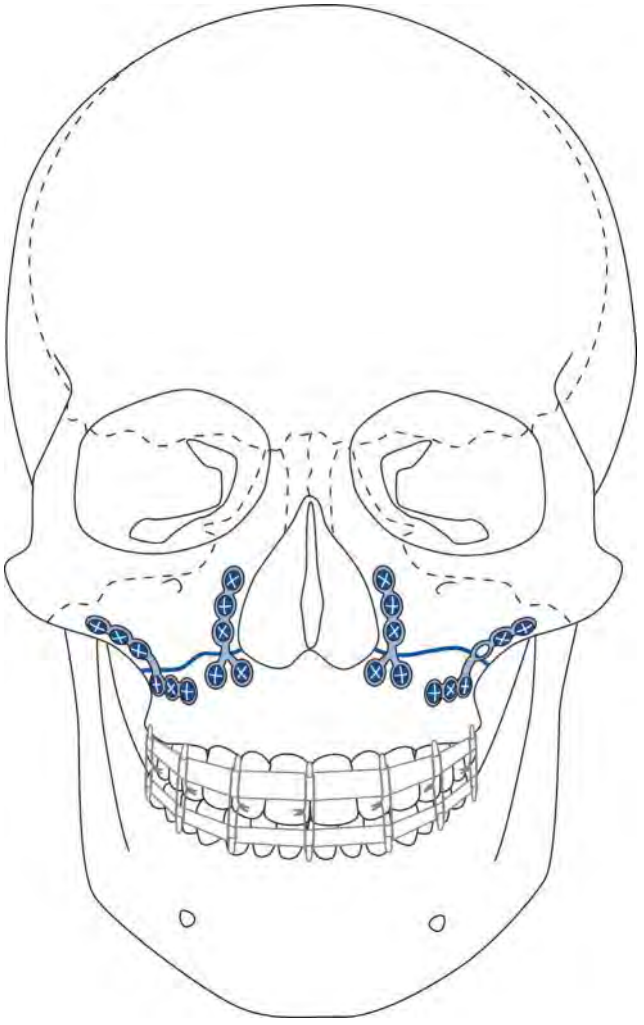


FIGURE 16-6. Use of four bone plates across the Le Fort I level of a fracture. These plates can be quite thin (e.g., 1.3 or 1.5 mm) if four are used. However, stronger plates (e.g., 2.0 mm) should be used where bone interfaces are less favorable or when the maxilla is placed into a position that is resisted by soft tissue forces (e.g., maxillary advancement or inferior repositioning).

treatment recommendations from laboratory studies to the patient. Fracture stability is only one factor in the treatment equation. There are many others, such as maintenance of blood supply, that must also be considered when determining treatment recommendations.

LOAD-BEARING VERSUS LOAD-SHARING FIXATION

The most simplistic way to discuss fixation schemes for fractures is to break them down into those fixation devices that are load-bearing and those that share the loads with the bone on each side of the fracture (load-sharing). *Load-bearing fixation* is a device that is of sufficient strength and rigidity that it can bear the entire load applied to the mandible during functional activities. Injuries that require load-bearing fixation are comminuted fractures of the mandible, those fractures in which there is very little bony interface because of atrophy, or those injuries that have resulted in a loss of a portion of the mandible (defect fractures). In such cases, the fixation device must bridge the area of comminution, minimal bone contact, or bone loss and bear all of the forces transmitted across the injured area that are generated by the masticatory system. Load-bearing fixation is sometimes called *bridging fixation* because it bridges areas of comminution or bone loss. The most commonly used load-bearing device is a mandibular reconstruction bone plate (Figure 16-7). Such plates are relatively large, thick, and stiff. They use screws that are generally greater than 2.0 mm in diameter (most commonly 2.3, 2.4, or 2.7 mm). When secured to the fragments on each side of the injured area by a minimum of three bone screws, reconstruction bone plates can provide temporary stability to the bone fragments. The bone plates are not prosthetic devices and will usually fail in time (several months to years later) by either loosening of the screws or fracture of the plate, but they can provide stability until the comminuted fragments have consolidated and/or the missing bone has been replaced with grafts.

Load-sharing fixation is any form of internal fixation that is of *in*sufficient stability to bear all of the functional loads applied across the fracture by the masticatory system. Such a fixation device(s) requires solid bony fragments on each side of the fracture that can bear some of the functional loads. Fractures that can be stabilized adequately with load-sharing fixation devices are simple linear fractures, and these constitute the majority of mandibular fractures. Fixation devices that are considered load-sharing include the variety of 2.0-mm miniplating systems that are available from a number of

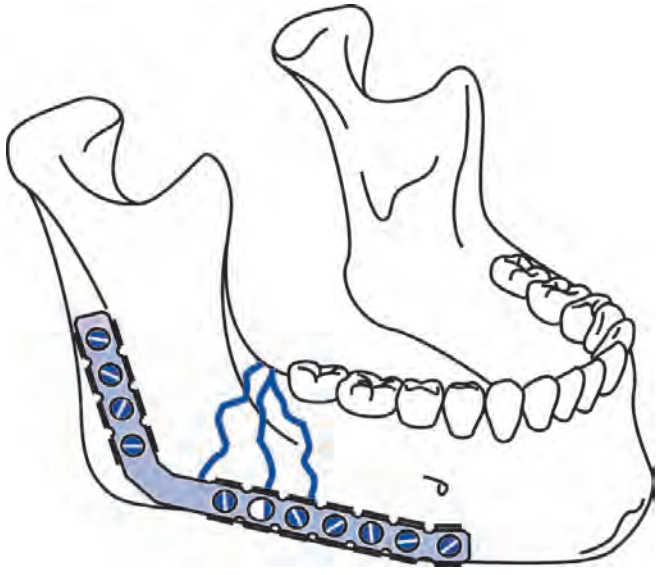


FIGURE 16-7. Example of load-bearing fixation. The application of a large reconstruction bone plate across an area of comminution or bone loss allows active use of the mandible. The plate bears all of the loads applied across the fracture. It, therefore, is unnecessary for the intervening bony fragments to share any of the load.

manufacturers. Examples of load-sharing fixation for angle fractures are demonstrated in Figure 16-1A–H. Lag screw techniques are also load-sharing in that the bone that is compressed is sharing the functional loads with the screws. Simple linear fractures can also be treated by load-bearing fixation. Comminuted or defect fractures, or those in which a minimum of bone contact is present, cannot be treated by load-sharing fixation because there is insufficient bone stock adjacent to the fracture to resist displacement by functional forces.

REGIONAL DYNAMIC FORCES

Different regions of the mandible undergo different magnitudes and direction of forces. In simplistic terms, fractures of the angle under most functional situations tend to "open" at the superior border (Figure 16-8A and B). Therefore, the application of fixation devices at the superior border is more effective in preventing this separation of fragments under function than applying them at the inferior border (see Figure 16-8C and D). There is little tendency for isolated fractures of the angle to have medial or lateral displacement during function, so the fixation requirement is mainly to prevent separation of the superior border. Relatively small plates can, therefore, adequately control this fracture. The Champy miniplate technique functions extremely well for this fracture and consists of a 2.0-mm miniplate applied with monocortical screws along the superior border (see Figure 16-4).[4] Because metallic plates have high tensile strength, even thin plates work adequately at the angle to prevent the tendency for a gap to form at the superior border under function.[13]

Isolated fractures of the mandibular body behave similarly under function, with a tendency for a gap to form at the superior surface, but the more anterior the fracture, the more

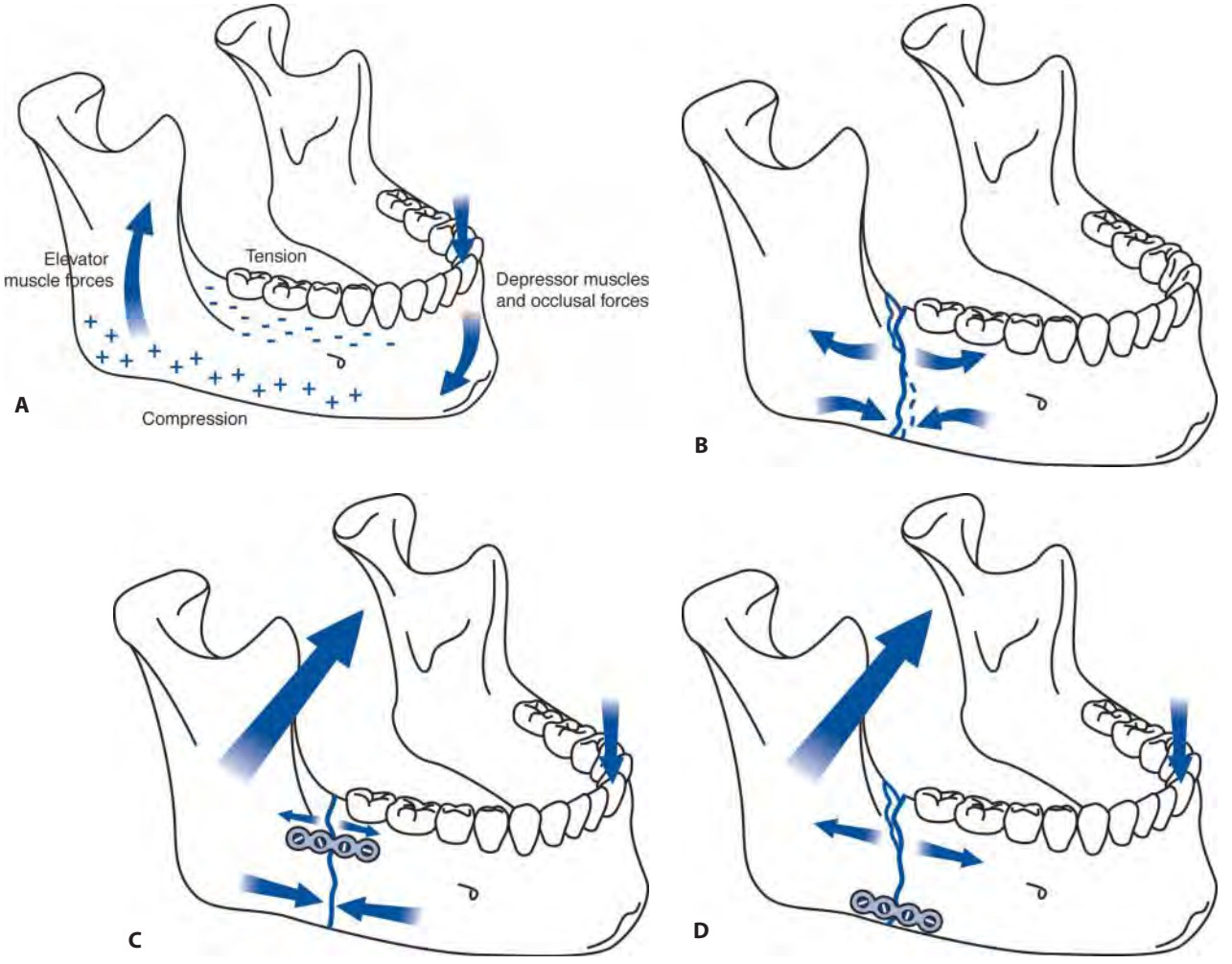


FIGURE 16-8. Functional forces acting across the intact mandibular angle or body region **(A)** and after a fracture **(B).** Note that a gap tends to form at the superior border of a fractured mandibular angle secondary to muscle and occlusal forces. The superior border is, therefore, called the *zone of tension (separation),* whereas the inferior border is under compressive force during function (compression zone). **C,** A small bone plate applied along the zone of tension (separation) is very effective in countering the forces of mastication and effectively neutralizes the forces, maintaining closure of the fracture gap. **D,** A small plate applied in the zone of compression (inferior border) is very ineffective in neutralizing the muscle forces, and a gap will easily form superiorly in the zone of tension.

tendency for torquing of the fragments to occur, causing mediolateral misalignment of the inferior border. Whereas the arch bar may provide sufficient resistance to the tendency for a gap to form between the teeth under function, a plate or lag screws somewhere else on the body of the mandible is necessary to prevent the mediolateral displacement that accompanies the torquing motion under function. For isolated body fractures, this can be a relatively small plate, such as a 2.0-mm miniplate or even a single lag screw combined with a solid arch bar (Figure 16-9).

The directions of forces that are distributed through the anterior mandible vary with the activity of the mandible. This means that the classic zones of tension on the superior and compression on the inferior surfaces of the mandible are not absolute.[7,8] Instead, the anterior mandible undergoes shearing and torsional (twisting) forces during functional activities.[4,14] Application of fixation devices must, therefore, take these factors into consideration. This is why most surgeons advocate two points of fixation in the symphysis: either two bone plates, two lag screws, or possibly one plate or lag screw combined with an arch bar (see Figure 16-1A–H).[14]

ONE-POINT VERSUS TWO-POINT FIXATION

Mandibular fractures can be treated by the application of fixation devices at one place along the fracture or at more than one point, generally two. There is no doubt that the addition of a second point of fixation provides more stability to the fracture. However, to take mechanical advantage of more than one point of fixation, the fixation devices should be placed as far apart as possible. Because fixation devices are applied to the lateral surface of the mandible, the ability to use two-point fixation requires that there be sufficient height of bone so that the fixation devices can be placed far apart. For instance, an atrophic mandibular fracture, in which there is a vertical height of only 15 mm, would not gain much mechanical advantage from placing two bone plates on the lateral surface (Figure 16-10). In such instances, a single stronger bone plate should be applied below the inferior alveolar canal (Figure 16-11). For the majority of fractures in the dentulous mandibular body and symphysis, there is sufficient height of bone to place one load-sharing plate along the inferior and one along the superior aspect of the lateral cortex. However, the ability to do so will depend on the local anatomy. If one chooses to use two load-sharing bone plates to provide rigid fixation, one must be cognizant of the position of the tooth roots and the inferior alveolar/mental nerves. If there is insufficient room between the roots of the teeth and the inferior alveolar/mental nerves, one might choose to use a single bone plate along the inferior border rather than to risk injury to the tooth roots or inferior alveolar/mental nerves when placing the second bone plate (see Figure 16-1F). Depending on the size of the plate and whether or not an arch bar will also be used to provide another point of fixation, the fixation could be rigid or functionally stable.



FIGURE 16-9. Example of a simple isolated mandibular body fracture treated by the application of arch bars and a single 2.0 miniplate.

COMPRESSION VERSUS NONCOMPRESSION PLATE OSTEOSYNTHESIS

Many types of bone plates are available for clinical use. In their most simplistic forms, plates are either compression plates or noncompression plates. Compression plates have the ability to compress the fractured bony margins, helping to bring them closer together, and imparting additional stability by increasing the frictional interlocking between them (Figure 16-12). Although these properties might be advantageous, the application of compression by a plate creates a dynamic force that can work to one's disadvantage if the plate is not perfectly applied. Compression plates are safest to use in fractures in which there is minimal obliquity and in which there are sound bony buttresses on each side of the fracture that can be compressed by the plate.

One should only use compression plates if one desires absolute rigidity across the fracture. If micromotion across the fracture occurs, compression plate osteosynthesis will often fail by becoming loose. Therefore, if compression plate osteosynthesis is desired, rigid fixation must also be desired. If this means that two plates are necessary to achieve absolute rigidity, they should be used. If it means that a larger compression plate need be applied, that should also be done.

LOCKING PLATE–SCREW SYSTEMS

Over the past 10 years, there has been an introduction of locking plate–screw systems into maxillofacial surgery. These plates function as internal fixators, achieving stability by locking the screw to the plate. There are several potential advantages to such fixation devices. Conventional bone plate–screw systems require precise adaptation of the plate to the underlying bone. Without this intimate contact, tightening of the screws will draw the bone segments toward the plate, resulting in alterations in the position of the osseous segments and the occlusal relationship. Locking plate–screw systems offer certain advantages over other plates in this regard. The most significant advantage may be that it becomes unnecessary for the plate to intimately contact the underlying bone in all areas. As the screws are tightened, they

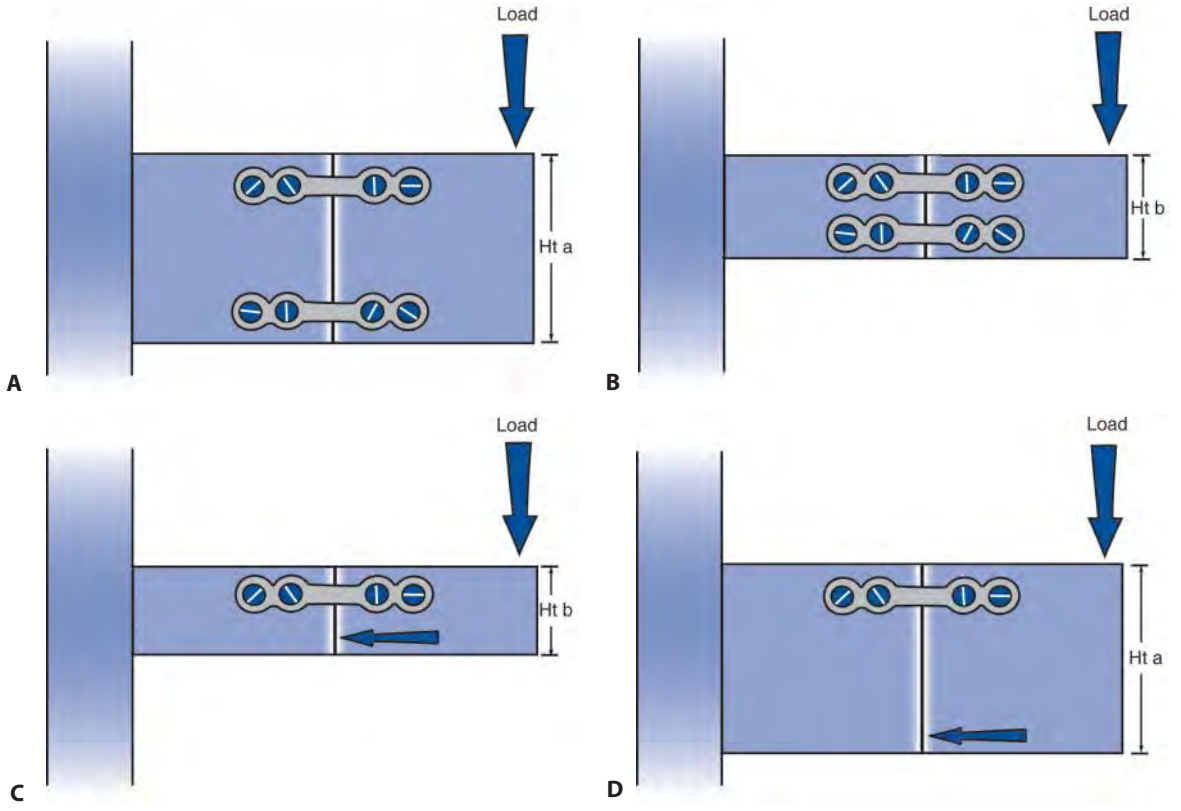


FIGURE 16-10. Biomechanical effectiveness of different constructs. **A** and **B** demonstrate biomechanical effectiveness of two plates when placed at different distances from one another. **A,** The load is applied to a fracture construct where there is a large fragment (Ht a) and a great separation between the two bone plates. This is a very stable construct. **B,** The load is applied to a fracture construct where the bone fragment is small (Ht b) and there is little distance between the two bone plates. This construct is much less stable than the one in **A** because of the limited space between the two plates, in spite of the fact that the same two bone plates are applied. **C** and **D** demonstrate biomechanical effectiveness of two constructs when only one plate is applied. **C,** A single plate is applied to a construct with little vertical height (Ht a). **D,** A single plate is applied to a construct with a greater vertical height (Ht a). The construct with a greater vertical dimension **(D)** is much more stable because of the greater buttressing effect provided by the longer moment arm of the increased vertical dimension of bone.

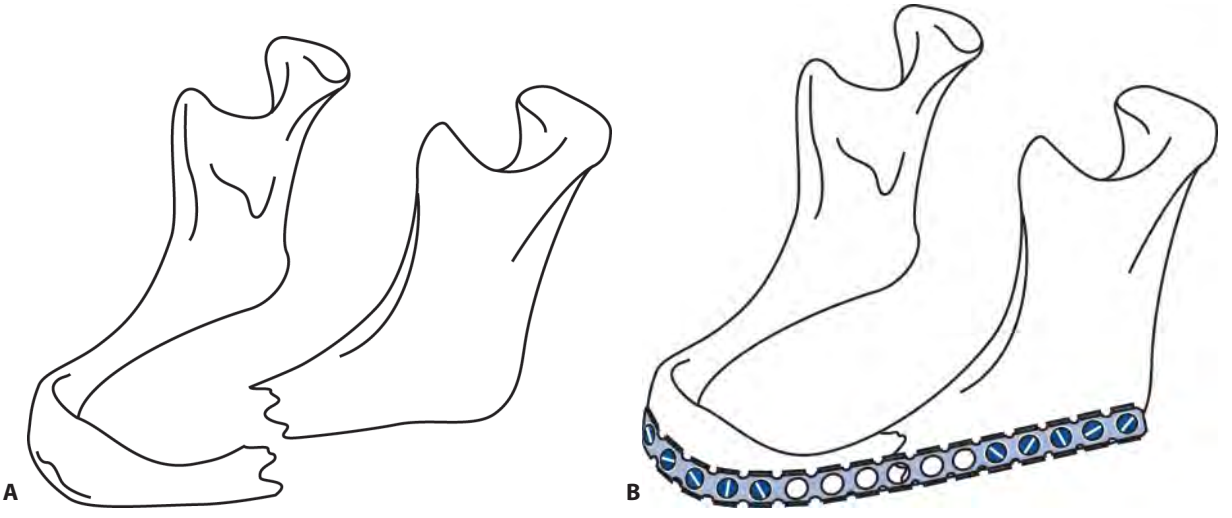


FIGURE 16-11. Use of a single strong bone plate (reconstruction plate) when the vertical height of the mandible is small. **A,** Atrophic mandible fractured through the body region. **B,** Reconstruction bone plate applied to the fracture to provide rigid fixation. Even if there were room to place two smaller bone plates on the lateral cortex, they would be so close to one another that their mechanical effectiveness would be minimal.

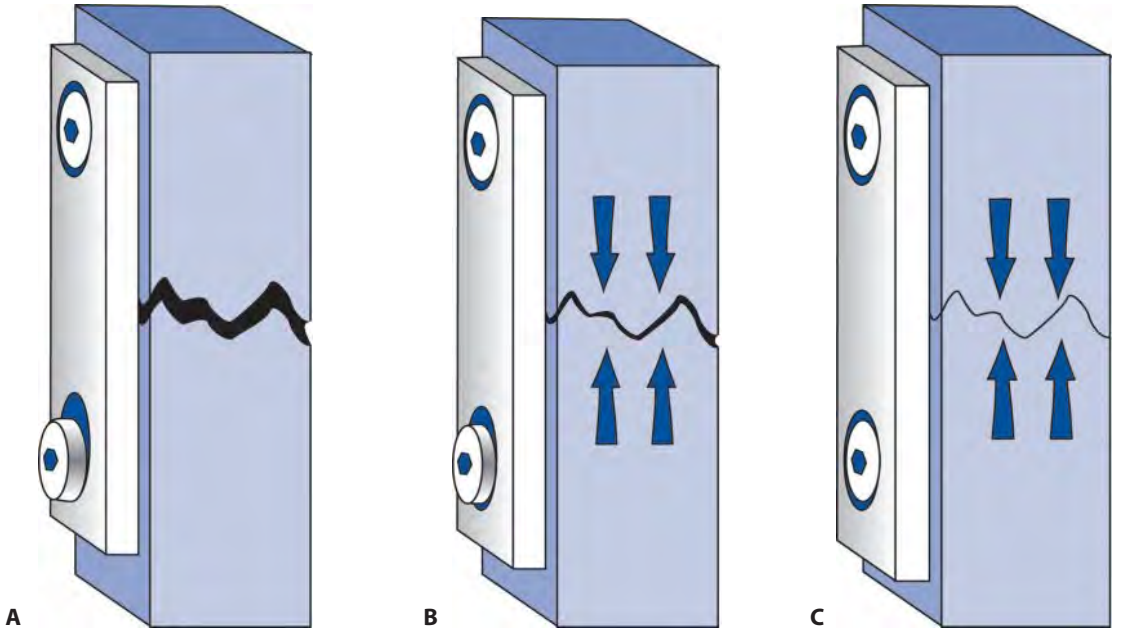


FIGURE 16-12. Compression plates help to minimize the fracture gap and to impart stability by the frictional interlock they induce. The screw holes in at least one of the oval bone plate holes is/are drilled eccentrically, that is, away from the fracture, so that as the screw(s) is/are tightened **(A),** the V-shaped undersurface of the screw head contacts the plate and forces the plate away from the fracture **(B),** imparting compression to the bone fragments and closing the gap **(C).**

"lock" to the plate, thus stabilizing the segments without the need to compress the bone to the plate (Figure 16-13). This makes it impossible for the screw insertion to alter the reduction. This theoretical advantage is certainly more important when using large bone plates, such as reconstruction plates, which can be very difficult to perfectly adapt to the contours of the bone. Another theoretical advantage to the use of locking bone plate–screw systems is that the screws are unlikely to loosen from the bone. This means that even if a screw is inserted into a fracture gap, loosening of the screw will not occur. The possible advantage to this property of a locking plate–screw system is a decreased incidence of inflammatory complications from loosening of the hardware. It is known that loose hardware propagates an inflammatory response and promotes infection. For the hardware or a locking plate–screw system to loosen, loosening of a screw from the plate or loosening of all of the screws from their bony insertions would have to occur. Both of these are unlikely. A third advantage to a locking screw–plate system is that the amount of stability provided across the fracture gap is greater than when standard nonlocking screws are used.[15,16]



FIGURE 16-13. A locking plate-screw system. Note the second set of threads just under the head of the screw that will lock into receptacle threads inside the hole of the bone plate.

Whereas the possible advantages to a locking plate–screw fixation system are theoretical, whether clinical results can be improved is not clear from the literature. However, given the potential advantages that locking plate–screw systems provide, such systems should be considered whenever noncompression plates are chosen for a fracture.

LAG SCREW FIXATION

The lag screw fixation technique consists of using screws to compress fracture fragments without the use of bone plates. To apply the lag screw technique, two sound bony cortices are required because this technique shares the loads with the bone. The hole in the cortex under the head of the screw is called the *gliding hole.* It is the same diameter as the external diameter of the screw threads, so the threads will not engage this cortex. The screw threads on the terminal end of the screw engage the opposite cortex. By tightening the screw, a tensile force is created within the screw that compresses the bony cortices together, tightly reducing the fracture (Figure 16-14).

As with using compression bone plates, lag screw fixation is a technique that should be used only to provide absolute rigid fixation. Micromotion across a fracture secured with

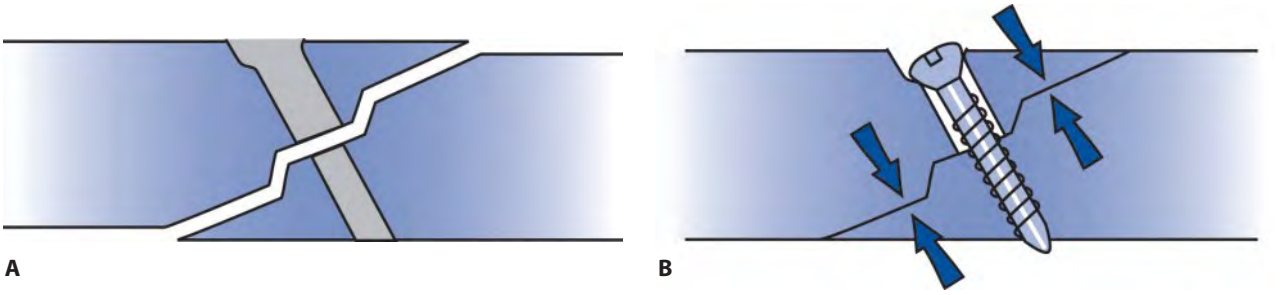


FIGURE 16-14. Technique of lag screw placement. **A,** The outer cortex is drilled to the external diameter of the screw threads and is countersunk to receive the head of the screw. The inner cortex is drilled to the internal diameter of the screw. **B,** Screw tightening creates compression of the bony interfaces because the head of the screw compresses the outer cortex against the inner cortex that is engaged by the screw threads.

lag screws will likely result in dissolution of the bone around the screws, with loss of stability. Therefore, lag screws should be selected only when there is sufficient bone available to place at least two screws into sound bone that can, in all likelihood, create rigidity across the fracture.

The use of lag screws has several advantages over the use of bone plates. It uses less hardware when compared with the use of plates, thus making it more cost-effective. When properly applied, lag screws are a very rigid method of internal fixation. Because there is no plate to be bent, the insertion of a lag screw is quicker and easier and the reduction more accurate than when bone plates are used. One must understand completely that the lag screw technique of fixation is one that relies on compression of bone fragments. If the intervening bone is unstable due to comminution or is missing, compressing across this area will cause displacement of the bone fragments, overriding of segments, and/or shortening of the fracture gap, resulting in problems with the occlusion. One should always place the lag screw in a direction that is perpendicular to the line of fracture to prevent overriding and displacement during tightening of the screws (Figure 16-15).

PLATE FATIGUE

Bone plates may break under function, resulting in possible loss of fixation, infection, nonunion, and/or malunion. Plates break for a number of reasons, but most fracture in vivo because of fatigue. Plates used in maxillofacial surgery today are usually made of titanium. Titanium is a relatively biocompatible material and has material properties that are considered adequate for internal fixation when appropriate

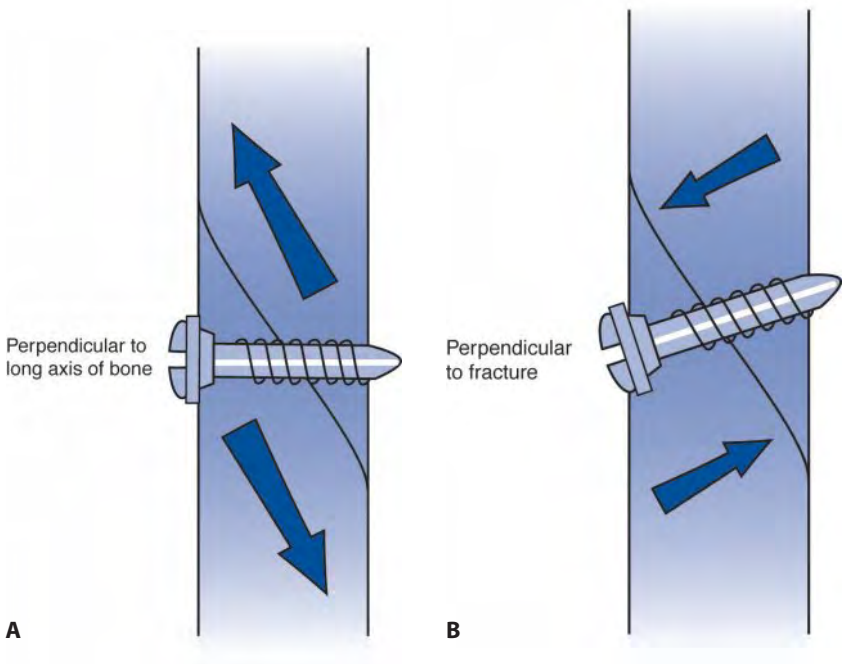


FIGURE 16-15. Improper **(A)** and proper **(B)** methods of placing lag screws. The screw should always be drilled perpendicular to the line of fracture to prevent sliding of the fragments during tightening of the screws.

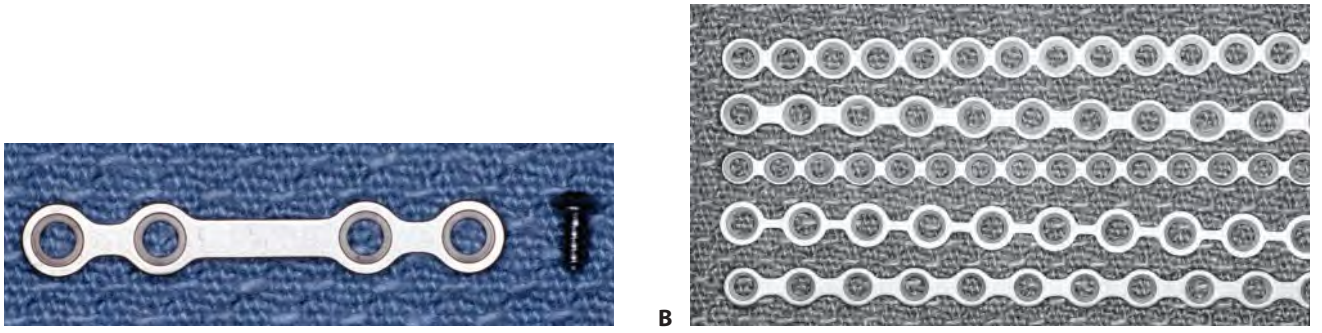


FIGURE 16-16. A standard 2.0-mm miniplate **(A)** and adaptation miniplates **(B)** (the 2.0 mm refers to the size of the screw that this plate accommodates, not the size of the plate). These plates have very good tensile strength but readily fracture under cyclic loading because of their thin cross-section.

plates are selected. One of the undesirable properties of titanium is its brittleness (or lack of ductility) when compared with bone. One only has to bend a miniplate back and forth a couple of times to see how readily it will fracture. Placement of bone plates on areas of the mandible that are constantly and repeatedly deformed under function can result in fatigue fracture of the plates. Examples are 2.0-mm miniplates or 2.0-mm adaptation plates applied to the condylar process or similar plates applied to the atrophic mandible (Figure 16-16). The condylar process is constantly undergoing mediolateral tilting during opening and closing movements of the mandible. The atrophic mandible similarly undergoes "wishboning" during function (Figure 16-17).[17] The less the amount of bone stock present, the higher the magnitude of these movements. Thus, atrophic mandibles undergo much more wishboning than do large dentulous mandibles. Because of the small cross-sectional area of the condylar process, this area of the mandible similarly flexes during function.

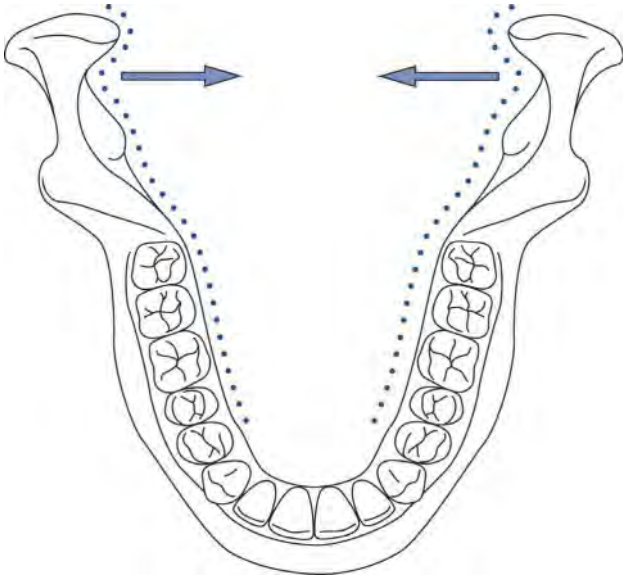


FIGURE 16-17. Under function, the mandible "wishbones" in and out.

Bone plates applied to such areas of the fractured mandible have to be able to not only acutely withstand the deforming forces applied but also must withstand the chronically applied cyclic loading until such time that the bone has healed. This is why several authors have recommended thicker, stronger 2.0-mm plates (minidynamic compression plates) (Figure 16-18) or *two* 2.0-mm miniplates for condylar process fractures and reconstruction bone plates for atrophic mandibular fractures.[18–21] This problem with the atrophic mandible is the reason the AO/ASIF has recommended, "The weaker the bone, the stronger the plate must be."[21]

SINGLE VERSUS MULTIPLE MANDIBULAR FRACTURES

Because of the shape of the mandible, fractures of the mandible are often multiple. Most surveys show that just under 50% are isolated, the same amount are doubly fractured, and a small percentage have more than two fractures. Fixation requirements for double (or multiple) fractures differ from isolated fractures. One can use less rigid forms of fixation on isolated fractures, because the forces generated during function are less complex than when a second or third fracture is present. For instance, there is minimal tendency for fractures of the symphysis, body, or angle to result in widening of the mandible unless fixation devices are incorrectly applied.

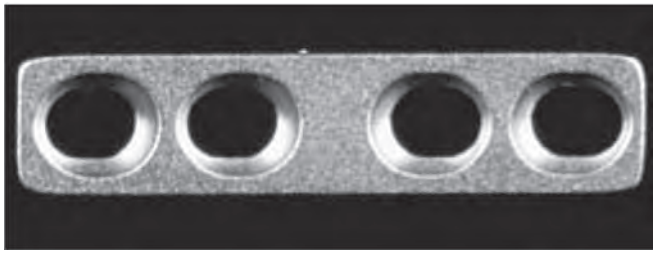


FIGURE 16-18. Example of a stronger 2.0-mm bone plate than the miniplate shown in Figure 16-16. The minidynamic compression plate shown has a thicker cross-sectional area and a broader strap between the holes. This plate is useful for fractures of the mandibular condylar process and rarely fractures for that application.

The application of a single 2.0-mm miniplate along the lower border of the mandible combined with an arch bar is usually adequate fixation for isolated simple linear fractures of the symphysis and body regions (two-point fixation). If an arch bar is not used or the teeth are not sound, one should either use a stronger plate at the inferior border or add another 2.0-mm miniplate more superiorly along the lateral cortex. The application of a single 2.0-mm miniplate along the superior border is also adequate fixation for most isolated simple linear fractures of the angle region.[4] Lag screws can also be used instead of or in addition to plates, where appropriate.

When two fractures are present, there is a greater tendency for the segments to displace because of the bilateral loss of support that occurs. Widening of the mandible must be prevented by applying adequate internal fixation to resist that tendency. With bilateral simple linear fractures, one should always consider using a more rigid form of fixation on at least one of the fractures. For instance, when an angle fracture is combined with a contralateral body or symphysis fracture, one should consider treating the body or symphysis fracture with either two 2.0-mm miniplates or a stronger bone plate at the inferior border, as well as using the arch bar as another point of fixation (Figure 16-19). The angle fracture can then be treated with a single superior border 2.0-mm miniplate. Similarly, if an angle fracture is combined with a contralateral condylar process fracture, one should consider the application of more stable fixation at the angle if the condylar process is going to be treated closed using no MMF and functional therapy (Figure 16-20). In that case, two 2.0-mm miniplates (or an alternative rigid treatment) should be considered. If the condylar process were going to undergo open reduction and internal fixation, or if several weeks of MMF were going to be used, the angle fracture could be treated with a single superior border 2.0-mm miniplate (functionally stable but not rigid fixation).[4]

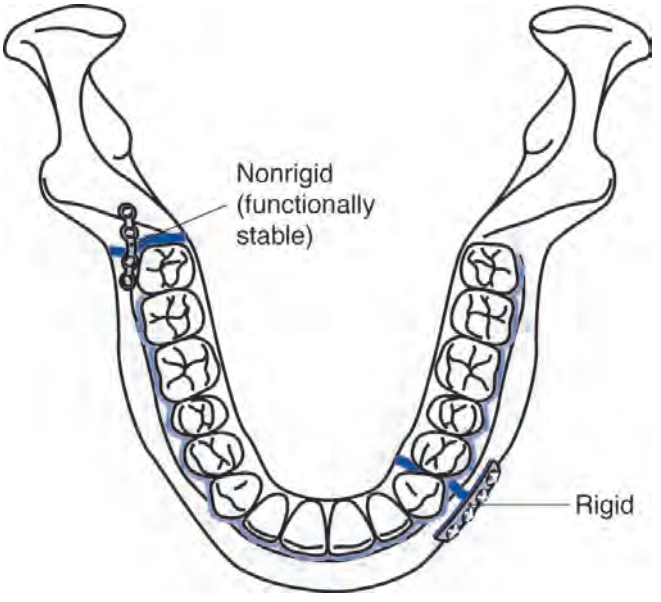


FIGURE 16-19. Possible fixation scheme for right-angle and left body fractures of the mandible. The more accessible body fracture is treated with a more rigid form of fixation (e.g., a thicker bone plate at the inferior border or two miniplates). The angle fracture can then be treated with a functionally stable form of fixation, which is easier to apply than would be a rigid technique at the angle. The angle fracture is thus treated as if it were an isolated fracture, with a single four-hole 2.0-mm miniplate.

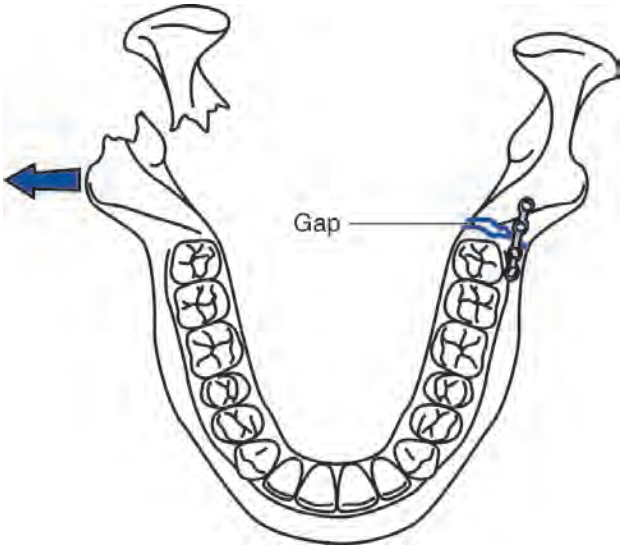


FIGURE 16-20. Demonstration of how widening of the mandible can occur after an angle fracture treated without rigid fixation is combined with closed treatment of a contralateral condylar process fracture. The single four-hole 2.0-mm miniplate that works very well in this location for isolated fractures of the mandibular angle may not be able to prevent the tendency for widening. With the loss of the articulation at the temporomandibular joint on the right side, the entire right side of the mandible can also cause torquing at the left angle fracture under function, leading to displacement and malocclusion.

The fracture pattern that has the most tendency for widening is the midsymphysis fracture combined with condylar process fractures, especially when both condyles are fractured. In such cases, the musculature attached to the lingual surface of the mandible pulls the mandible posteriorly, and because there is no posterior support via the temporomandibular joints, the lateral mandibular fragments open like a book. Such fractures must be carefully managed to first restore the mandibular width and then to maintain it. A short thin bone plate, such as a 2.0-mm miniplate, or even two 2.0-mm miniplates, may not offer sufficient resistance to the tendency to widen (Figure 16-21A). If one chooses to treat the condylar process fracture(s) closed, very stable fixation must be applied across the reduced mandibular symphysis to retain the normal width of the mandible. This can be achieved by several techniques, but the most stable is to use either a reconstruction plate applied across the symphysis (see Figure 16-21B) or, if the fracture is linear, two well-placed lag screws (see Figure 16-1B). The application of two thicker 2.0-mm bone plates (thicker than miniplates) would also

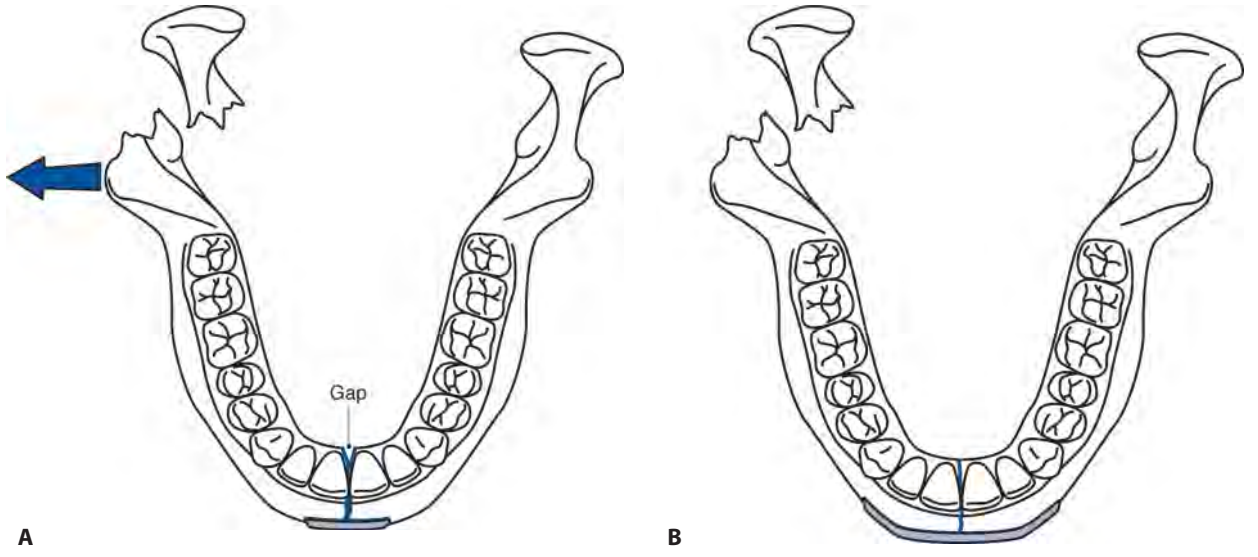


FIGURE 16-21. **A,** Combination of a symphysis fracture treated with a single short bone plate and concomitant closed treatment of a condylar process fracture can result in widening of the mandible. Because the bone plate is applied along the buccal cortex, it has a mechanical disadvantage in preventing widening of the mandible. **B,** To prevent this, a longer, thicker, stronger plate should be applied that "yolks" the mandible.

suffice (see Figure 16-1C). If one chose to open the condylar process fractures, the symphysis fracture can be treated as an isolated symphysis fracture, with whatever technique the surgeon usually chooses.

SUMMARY

Although the number of plating sets and fixation schemes are numerous, one can usually treat most fractures with very few instrument sets. It is possible to treat the majority of fractures of the mandible either with lag screws, 2.0-mm miniplates, or reconstruction bone plates. There are, however, fractures in which one may wish to use 2.0-mm screws but thicker plates than miniplates, for instance, condylar process fractures or fractures of the atrophic mandible. In those cases, one can use thicker and stronger bone plates that accommodate 2.0-mm screws. For these situations, a locking 2.0-mm bone plating set that has plates of varying lengths and thicknesses allows one to choose the appropriate bone plate for almost any location.

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CHAPTER 17

Management of Alveolar and Dental Fractures

Richard D. Leathers, DDS, and Reginald E. Gowans, DDS

HISTORY

Although there is speculation about whom the first dental surgeons were, dentoalveolar trauma has existed since humans began to walk the earth. Altercations with humans and animals, accidents, as well as dental treatment misadventures each have had a part in the development of today's dentoalveolar treatment protocols.

Arguably, Hippocrates of Cos, who lived during the Greco-Roman period (350 BC–AD 750), was the first to document treatment regimens for dentoalveolar trauma in his writings. He discussed binding teeth together in mandible fractures. Gold wire or linen thread was used as "bridle wire." He alluded to various splinting techniques that involved teeth that were distant to the fractured or subluxed area (Figure 17-1). In the same way, to expedite the healing process, he stressed recapturing proper occlusion, a concept still practiced today.

We could theoretically think of Hippocrates as one of the first investigators to see the value in "evidenced-based" treatment protocols; he is credited with separating the obscure religious beliefs from true medical observation.[1,2]

Archigenes (~59 BC–AD 17), a Roman physician and dentist, believed that a broken tooth should initially be treated with a medieval endodontic procedure by intrapulpal cautery with a hot iron instrument.[3]

Claudius Galen (~AD 130–200), a Greek physician, also subscribed to the belief that reestablishing occlusion was essential in treating dentoalveolar fractures (see Figure 17-1).[3]

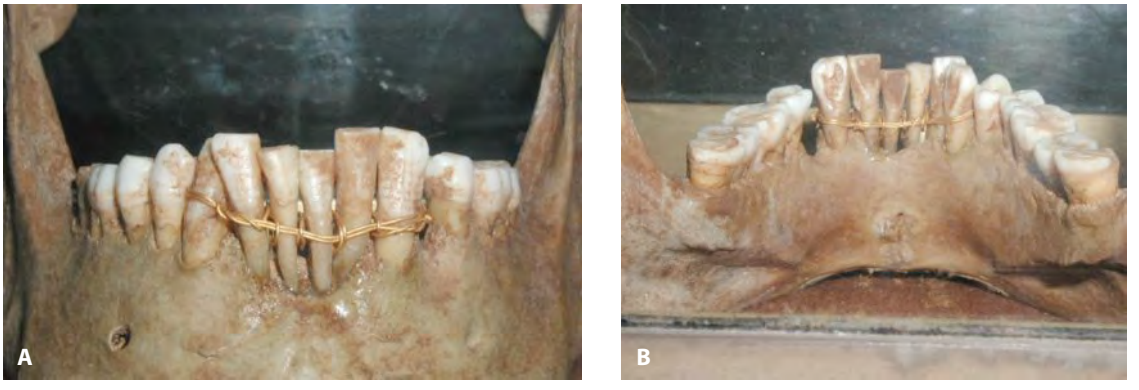


FIGURE 17-1. Mandible found at the ancient site of Sidon in Lebanon (dated 500 BC). Gold wire was used to splint periodontally involved anterior incisors. **A,** Frontal view. **B,** Lingual view. (**A** and **B,** Reproduced with permission from The Archaeological Museum, American University, Beirut, Lebanon.)

ETIOLOGY AND INCIDENCE

Dentoalveolar injuries commonly occur in the pediatric, teenage, and adult populations. Each group has specific etiologies that pertain to age, sex, and demographics.

In the pediatric group, the primary cause of these injuries is falls. Possibly during the first years of life, the early anatomic development and skeletal weight distribution cause the poor coordination that leads to falls. In the larger surveys, the pediatric population accounts for 5% of all facial fractures.[4] Andreasen[5] reported a bimodal trend in the peak incidence of dentoalveolar trauma in children aged 2 to 4 years and 8 to 10 years. Likewise, there was an overall prevalence of 11% to 30% in the children with primary dentition. Those with permanent or mixed dentition ranged from 5% to 20%. The ratio of men to women was 2:1.

Children and adolescents overlap with respect to the etiology of dentoalveolar injury. Contact sports and playground activities lead to most injuries. In fact, approximately one third of all dental trauma is secondary to sporting accidents.[6]

The use of mouthguards and appropriate head gear, however, has helped to decrease sport-related injuries.[7]

Child abuse appears to be another significant cause of dentoalveolar and facial injury. An alarming census of child abuse is documented in the literature. In the year 2000, an estimated 879,000 children were abused. Of these, 19.3% were physically abused.[8] In the United States, over 50% of physical trauma in child abuse occurs in the head and neck region. Internationally, about 7% of all physical injuries involve the oral cavity, with 9% between ages 0 and 19 years.[9,10]

Generally, adult injuries are caused by motor vehicle collisions, contact sports, altercations or assaults, industrial accidents, and iatrogenic medical or dental misadventures.

Demographic and behavioral research has increased the profession's understanding of psychosocial issues that relate to facial trauma.

Leathers and colleagues[11,12] reported on orofacial injury profiles in an inner-city hospital. They found that most orofacial injuries resulted from intentional violence, and the victims were primarily socially and economically disadvantaged groups in the minority populations.

Black and coworkers[13] related substance abuse—specifically alcohol and "street drugs"—with orofacial injuries. They found that a significantly greater proportion of patients who screened positive for drug and alcohol abuse at the time of injury had a previous history of head injury and/or orofacial injury. Further, we should consider the high rate of recidivism in this population as another behavioral factor.

Other groups at increased risk of dentoaveolar trauma are those with seizure disorders, mental disorders, and congenital maxillofacial abnormalities.

Lockhart and associates[14] reported findings, by the Risk Management Foundation, indicated that damage to the teeth was the most frequent anesthesia-related claim, often resulting in litigation. Poor laryngoscopy technique and the unmonitored biting force of the comatose patient also potentially caused dentoalveolar injury.[15,16]



FIGURE 17-2. Blunt facial trauma resulting in soft tissue lacerations and dental and alveolar compromise.

With direct trauma, maxillary incisors are the most frequently traumatized teeth, especially if they are associated with a class II division 1 malocclusion. Trauma to the primary dentition usually results in various luxations (~75%), whereas in permanent dentition, crown, or crown-root fractures are the norm (39%).[17] Indirect trauma to the dentition usually results from the forceful impact of the mandible with the maxilla, following a blow to the chin region. These traumas will often result in injury to the posterior teeth (Figure 17-2).[5]

HISTORY AND PHYSICAL EXAMINATION

Obtain a thorough history of the patient and the traumatic incident. Preinjury data—such as biographic, demographic, past medical history, time of incident, occlusion, location of incident, loss of consciousness, and nature of the incident—could potentially expedite the treatment process.[18,19]

The potential for aspiration, airway compromise, and neurosensory deficit dictates that the clinician should thoroughly evaluate all dentoalveolar-injured patients before managing dental injuries. The initial examination should be systematic, methodical, and comprehensive (see Figure 17-2). Equally, an injury that could involve tooth or alveolar fracture may be substantial enough to cause a brief loss of consciousness. The clinical presentation of closed head injuries, such as basal skull fractures and epidural hematomas, may be occult. Hence, if these are not recognized early, they may have devastating consequences. Davidoff and colleagues[20] reported that it was not uncommon for a closed head injury to result when a loss of consciousness of less than 1 hour occurred, along with facial trauma. Signs of confusion followed by "lucid intervals" may require further radiographic and/or computed tomography scan studies.[21]

Unaccounted-for avulsed teeth, free tooth fragments, or dislodged restorations raise the suspicion of aspiration. For this reason, auscultation of the chest to rule out wheezing or labored breathing is essential. Owing to its anatomic position, the right mainstem bronchus is often the site of foreign body dislodgment. Support any positive finding with proper neck, chest, and abdominal radiographs.[22] If foreign bodies

exist in the abdomen, arrange follow-up for the patient with radiographs, and monitor for the risk of gastrointestinal obstruction until the foreign body is cleared.

MAXILLOFACIAL EXAMINATION

For medicolegal purposes, consider preoperative photographs before invasive treatment.

Include the following in the patient examination[23]:

- Extraoral soft tissue
- Intraoral soft tissue
- Jaws and alveolar bone
- Teeth (displacement and mobility)
- Percussion and pulp testing

Ensure that the patient is cleaned extraorally with a mild antiseptic soap, while taking care not to further inoculate injury sites with debris or foreign bodies. Consider tetanus prophylaxis, depending on previous immunization compliance and wound presentation (Table 17-1).[24]

Thoroughly inspect superficial and deep lacerations, abrasions, or any soft tissue compromise. The mechanism of injury elicited in the history and the soft tissue defect alert the surgeon to suspect underlying hard tissue damage, such as to the maxilla, the mandible, the temporomandibular joint (TMJ), and alveolar fractures. Success rates are time-dependent with dentoalveolar trauma, and generally perioral soft tissue lacerations (lips) should be repaired after intraoral treatment, except in cases of poor hemorrhage control. In children, women, and the elderly, if the injury observed fails to correlate well with the history given, suspect and subsequently rule out abuse. Authorities, such as social services representatives, initiate proper legal protocols, if necessary.

Before any intraoral manipulations, obtain initial radiographic studies (e.g., in the pediatric patient, knowledge of the errant deciduous tooth root to the permanent tooth bud position). The chance of further damage could be exponentially disastrous to both the future eruption and the morphology of the developing permanent tooth.[25–27]

Approach intraoral soft tissue examination with caution. Carefully manipulate and handle traumatized tissues to avoid further compromise. Depending on the mechanism of injury, bone or tooth fragments may have penetrated these delicate areas. Closely inspect hematoma formation or ecchymotic areas. Buccal mucosal lacerations should raise the suspicion for Stensen's duct or orifice injuries. The lips, the floor of the mouth, and the tongue regions are all areas at risk for penetrating or secondary injury and, thus, should be inspected accordingly. Account for all fractured or missing teeth and restorations or assume they were swallowed, aspirated, or lodged within adjacent structures. Similarly, arrange for radiographic evaluation of the maxillary and nasal sinuses before further treatment.[28–30]

While examining for jaw and alveolar bone fractures, the presence of gross mobility or pericoronal bleeding of the involved teeth may be noted. Sublingual ecchymosis at the floor of the mouth is pathognomonic for an underlying mandible fracture. Step defects, crepitation, malocclusion, and gingival lacerations all raise suspicion of possible underlying bony defects.

Assess all fractured teeth for enamel, dentin, and pulpal involvement. Complete mobility of the crown may indicate crown-root fracture. Superficial crazing or infractions may be identified with a direct light source, transilluminating perpendicular to the long axis of the tooth from the incisal edge. Inspect and consider each tooth at risk, even at sites distal to the initial traumatic impact. Indirect trauma of the chin may cause posterior dentition defects, such as vertical or cusp fractures. Check occlusion and note any displacements, intrusions, or luxations. The direction of force is most commonly in a buccal-lingual direction.

Test percussion sensitivity and pulp vitality to rule out periodontal ligament (PDL) injury or one of the many forms of fractures. Gentle tapping of the injured and noninjured control teeth is the technique of choice. Use the handle of a mouth mirror or a specially designed calibrated percussion instrument. Tactile, auditory, and visual senses are used. Dullness may alert the surgeon to the possibility of a luxation injury or alveolar fracture. The quality of this sound indicates that the teeth are not in optimal contact with the adjacent bony structure. If the enamel is fractured or infraction has occurred, the sound is reminiscent of a "cracked tea cup."[31] The typical sound of the uninjured tooth is that of solid metallic resonance. Percussion testing, in and of itself, can add insult to injury; thus, control and caution are warranted.

Evaluate tooth vitality via various pulp testing modalities. Mechanical, thermal, and electrical noxious stimuli are used. These tests use various stimuli to check for conduction disturbances at the sensory receptors of the pulp. The pulp comprises both nonmyelinated and myelinated nerve

TABLE 17-1. **Summary of Tetanus Prophylaxis**

	NON–TETANUS-PRONE WOUNDS		TETANUS-PRONE WOUNDS	
History of adsorbed tetanus	Td*	TIG	Td*	TIG (250 U IM)
Unknown or ≤ 3 doses	Yes	No	Yes	Yes
≥ 3 doses†	No‡	No	No§	No

*For children < 7 yr old: DTP (DT, if pertussis vaccine is contraindicated) is preferred to tetanus toxoid alone. For persons ≥ 7 yr old, Td is preferred to tetanus toxoid alone.
†If only three doses of fluid toxoid have been received, a fourth dose of toxoid, preferably an adsorbed toxoid, should be given.
‡Yes, if > 10 yr since last dose.
§Yes, if > 5 yr since last dose. (More frequent boosters are not needed and can accentuate side effects.)
DTP = diphtheria-tetanus-pertussis; Td = tetanus and diphtheria toxoids adsorbed—for adult use; TIG = tetanus immune globulin—human.
Adapted from Alexander RH, Proctor HJ. Advance Trauma Life Support.(ATLS) Course for Physicians. 5th ed. Chicago: American College of Surgeons; 1993. p. 357.

fibers, which regulate vascular changes and respond to pain stimuli, respectively. As the tooth develops, the pain fibers (i.e., myelinated) increase, while simultaneously lowering the electrometric pulp stimulation.[32,33] This concept sheds light on some of the treatment differences in open and closed apices of the permanent dentition.

Pulp testing in the acute phase of dentoalveolar fracture is controversial and heavily based on the cooperation and communication of the patient as well as the repair process of the injured pulp tissue. The fear of possibly experiencing increased pain during testing, especially in children, limits verbal objectivity and may render pulp testing too unreliable. Also, acutely injured teeth may revascularize in approximately 1 month, thus increasing the risk of false-negative results during pulp testing. The developmental stage of the involved teeth also plays a significant role in the repair process. Incomplete apical development increases the chances of pulp repair and revascularization. As the tooth matures and apical width constriction starts, the chances of pulp repair decrease. Bacterial invasion in the pulp injury zone increases the risk of total pulp necrosis. Paradoxically, occasionally, uninjured teeth may not respond as expected. Even with this controversy in mind, pulp testing continues. Some of the testing paraphernalia are listed as follows[34]:

- Mechanical stimulation
  - Dental probe
  - Cavity prepping with drills
  - Saline-laden cotton pledget (fractured teeth)
- Thermal test
  - Heated gutta-percha
  - Ice
  - Ethyl chloride
- Carbon dioxide snow
  - Dichlorodifluoromethane
- Electrometric test
  - Electric pulp testers

Laser Doppler flowmetry (LDF), a relatively new pulp testing apparatus, has shown promise. A laser beam, directed at the coronal-labial aspect of the pulp, is scattered by pulp blood cells that, in turn, produce a Doppler frequency shift. The fraction of light scattered back is detected and processed to elicit a signal. The basic theory is that the pulp revascularization process can be monitored. Studies have shown that, in cases in which electrometric tests were negative and LDF displayed vascular perfusion, the LDF accuracy of pulp vitality reached 100%. The drawbacks to this form of testing are poor light transmission when blood pigments from discolored teeth are encountered, complexity of equipment use, and poor price containment.[35,36]

To ensure completeness, generate a standardized treatment record during the evaluation process, which systematically culminates in a diagnosis, treatment plan, and prognosis. Figure 17-3 provides the dentoalveolar trauma record, which should include, but is not limited to, these entities.

RADIOGRAPHIC EXAMINATION

Radiographic examination is essential to determine whether any underlying structures are damaged and should include periapical, occlusal, and panoramic radiographs. The periapical radiograph provides the most detailed information about root fractures and the dislocation of teeth. After treatment, periapical films can confirm the proper positioning of an avulsed or luxated tooth into the alveolus.

Occlusal radiographs, however, provide a larger field of view, and the detail is almost as sharp as a periapical radiograph. When occlusal radiographs or periapical films are used to examine soft tissues for the presence of foreign bodies, reduce the radiographic exposure time.

The panoramic radiograph is a useful screening view and can demonstrate fractures of the mandible and maxilla as well as fractures of the alveolar ridges and teeth. In the hospital setting, dental radiographs may not be available. Although not ideal, plain films, such as the mandibular series and the Caldwell views, may reveal tooth and alveolar injuries.

In the trauma patient whose tooth has not been accounted for at the accident scene, arrange for chest films to rule out the possibility of aspiration. Abdominal radiographic films can determine whether displaced teeth or prosthetic appliances have been ingested.

CLASSIFICATION OF DENTOALVEOLAR INJURIES

Once the diagnosis of dentoalveolar injury is made, the injury is classified for ease of communication and treatment planning. Many classification systems have been proposed over the years based on the anatomic site of injury, the cause, the treatment alternatives, or a combination of these. The two most common systems are those developed by Ellis and Davey (Figure 17-4) and Andreasen (Figures 17-5 to 17-7). The most commonly used simple and comprehensive classification of dentoalveolar injuries is one that was developed by Andreasen and originally adopted by the World Health Organization system for disease classification, using the International Classification of Diseases codes. The classification can be applied to both permanent and primary dentition. It includes descriptions of injuries to teeth, supporting structures, and gingival and oral mucosa. Injuries to the teeth and supporting structures are divided into dental tissues, pulp, periodontal tissues, and supporting bone as follows:

- Dental tissues and pulp.
  - Crown infraction (i.e., a craze line or crack in the tooth without loss of tooth substance).
  - Crown fracture confined to enamel, or enamel and dentin, with no root exposure (uncomplicated).
  - Crown fracture producing a pulp exposure (complicated).
  - Fracture involving the enamel, dentin, and cementum without pulp exposure (uncomplicated crown root fracture).
  - Fracture involving the enamel, dentin, and cementum with pulp exposure (complicated crown-root fracture).

Dentoalveolar Traum a Record

Name: _____ Date: _____

Age:
Sex:
Incident:
  Cause
  Location
  Time
Neurologic status:
  Loss of consciousness
  Consciousness
  Headache
  Nausea, vomiting
Extraoral findings:
Intraoral findings:
Radiographic findings:
  Posteroanterior
  Occlusal
  Panoramic
  Other
Tooth vitality findings (pulp testing):
Tooth mobility (+1, +2, +3):
Ellis classification (I, II, III, IV):
Luxation: Yes _____ No _____ Type _____
Avulsion: Yes _____ No _____ Storage medium _____ Time _____
Supporting structure trauma:

Diagnosis:

Treatment plan:

Prognosis: Good _____ Fair _____ Guarded _____
Examined by:

FIGURE 17-3. Dentoalveolar trauma record.

- Root fracture involving the dentin and cementum and producing a pulp exposure (root fracture).

- Injuries to periodontal tissues are divided into six categories and encompass what are commonly referred to as *subluxations* and *avulsions.*
  - Concussion: defined as an injury to the periodontium producing sensitivity to percussion without loosening or displacement of the tooth.
  - Subluxation: the tooth is loosened but not displaced.
  - Luxation (i.e., lateral, intrusion, and extrusion) dislocation, or partial avulsion: the tooth is displaced without an accompanying comminution or fracture of the alveolar socket.
- Injuries to the supporting bone.
  - Comminution of the alveolar housing, often occurring with an intrusive or lateral luxation.
  - Fracture of a single wall of an alveolus.
  - Fracture of the alveolar process, en bloc, in a patient having teeth but without the fracture line necessarily extending through a tooth socket.
  - Fracture involving the main body of the mandible or maxilla.

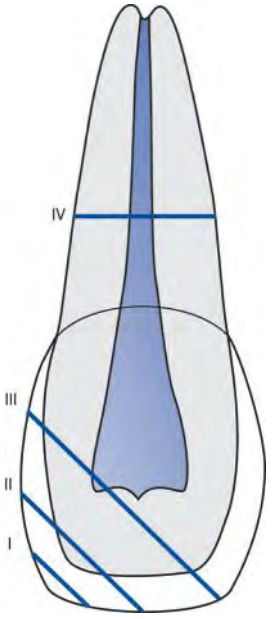


FIGURE 17-4. Ellis classification: I = fracture within enamel; II = fracture of enamel-dentin; III = fracture involving pulp; IV = fractures involving the roots.

Categories of injuries to the gingival or oral mucosa area include the following:

- Abrasion
- Contusion
- Laceration

TREATMENT OF INJURIES TO THE HARD TISSUES AND PULP

Enamel Fractures (Crown Infraction)

These injuries include fractures, chips, and cracks confined to enamel, not crossing the enamel-dentin border but terminating at the border. The cracks or fractures can be seen by indirect light or transillumination.

Treatment involves smoothing the rough edges or repairing with composite resin. It is difficult to predict future pulpal vitality; for this reason, perform pulp testing immediately after the injury and again in 6 to 8 weeks.

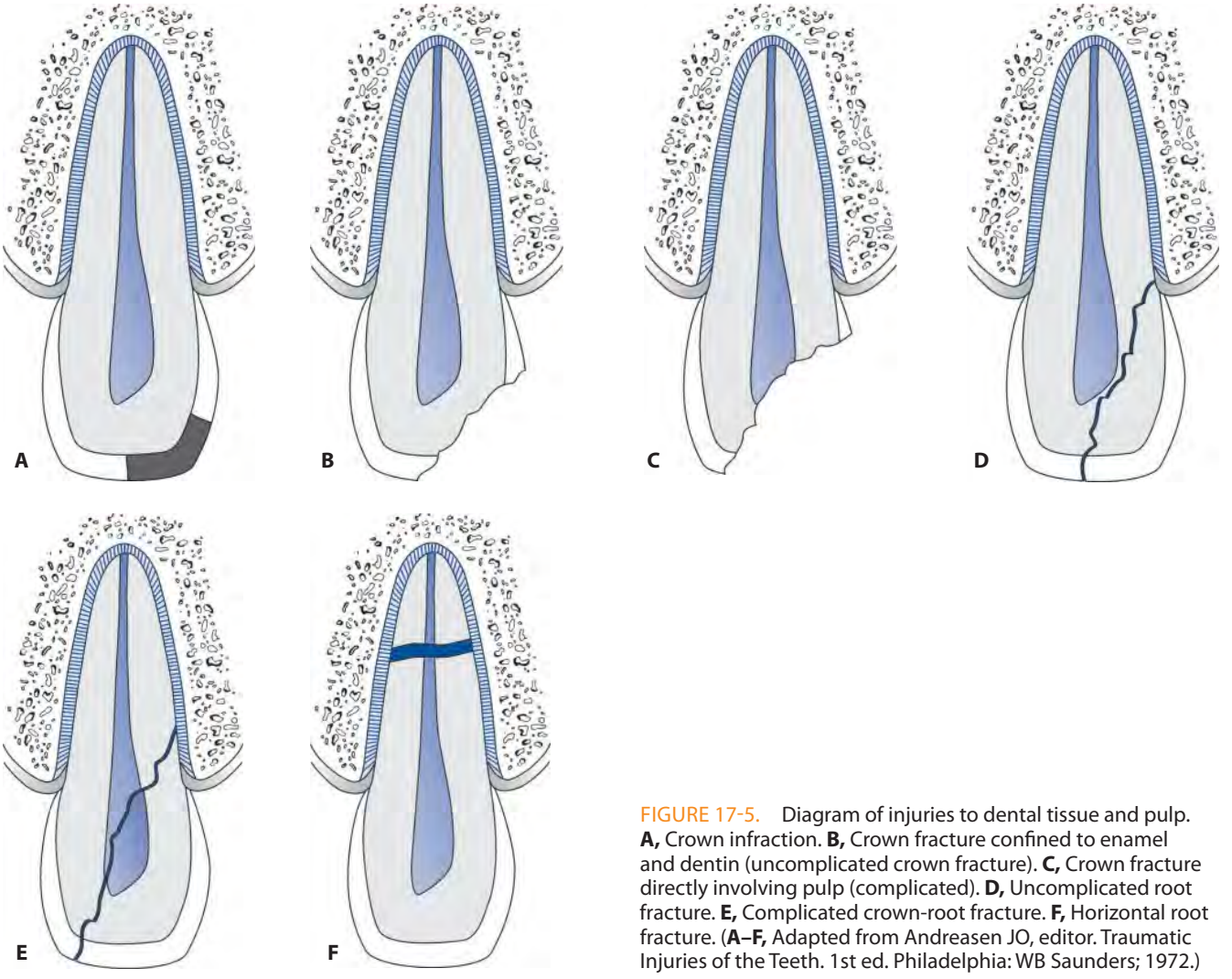


FIGURE 17-5. Diagram of injuries to dental tissue and pulp. **A,** Crown infraction. **B,** Crown fracture confined to enamel and dentin (uncomplicated crown fracture). **C,** Crown fracture directly involving pulp (complicated). **D,** Uncomplicated root fracture. **E,** Complicated crown-root fracture. **F,** Horizontal root fracture. (**A–F,** Adapted from Andreasen JO, editor. Traumatic Injuries of the Teeth. 1st ed. Philadelphia: WB Saunders; 1972.)

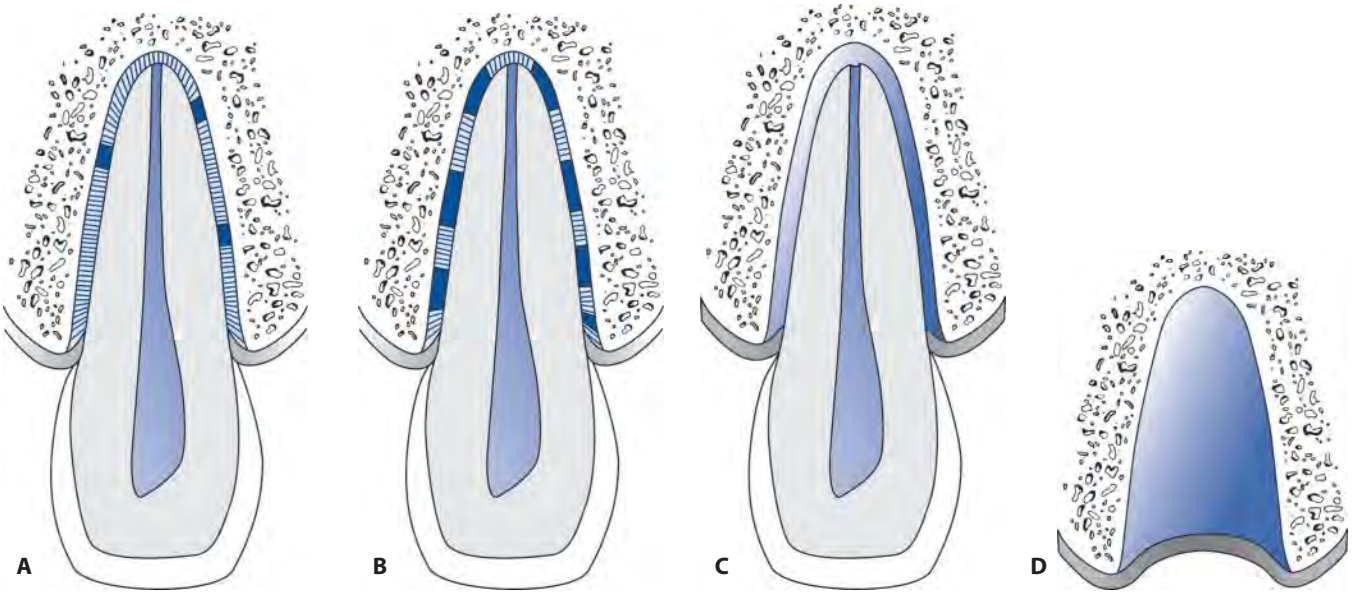


FIGURE 17-6. Diagram of injuries to periodontal tissues. **A,** Periodontal concussion. **B,** Subluxation. **C,** Luxation, dislocation, or partial avulsion. **D,** Exarticulation or avulsion. (**A–D,** Adapted from Andreasen JO, editor. Traumatic Injuries of the Teeth. 1st ed. Philadelphia: WB Saunders; 1972.)

Crown Fracture without Pulp Involvement

Crown fractures are the most frequent injuries in the permanent dentition. Crown fractures that expose dentinal tubules potentially may lead to contamination and inflammation of the pulp, eventually resulting in pulpal necrosis if untreated. Luxation injury concomitant to crown fractures, with or without pulp exposure, is the primary source of pulpal complications after injury. Prognosis is better if the enamel-dentin fracture involves a tooth that has not been luxated because the blood supply to the pulp has not been disturbed; the immunologic defense systems in the pulp will combat bacterial invasion (Figure 17-8).

Treatment is directed at protecting the pulp by sealing the dentinal tubules. Although zinc oxide–eugenol cement has been one of the best agents for producing a hermetic antibacterial seal, it is generally not recommended at the site where a composite resin restoration is placed because the eugenol component may interfere with polymerization, at least with some composites. A similar effect has been seen with a hard-setting calcium hydroxide paste, resulting in bond strength reduction in certain dental-bonding agents. In fractures with dentin exposure only, we recommend a dental bonding agent, followed by a composite restoration. With pulp exposure, the preferred treatment is calcium hydroxide placed directly over the exposure and sealed in place with a glass ionomer cement followed by a dentin-bonding agent and composite.[37]

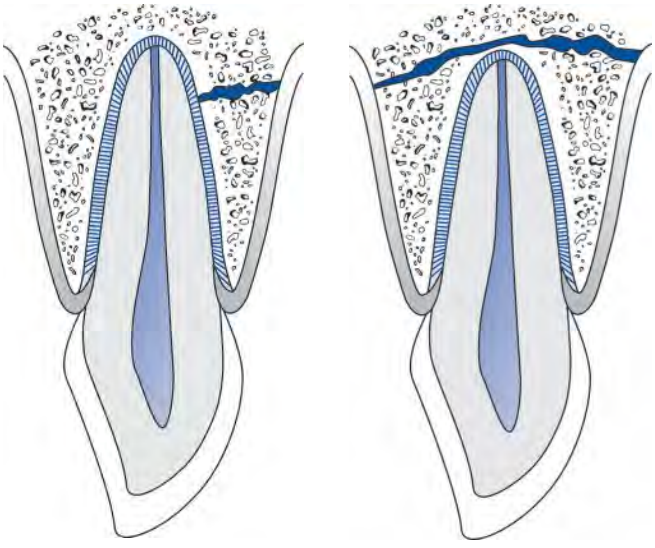


FIGURE 17-7. Diagram of injuries to supporting alveolar bone. **A,** Fracture of single wall of the alveolus. **B,** Fracture of the alveolar process. (**A** and **B,** Adapted from Andreasen JO, editor. Traumatic Injuries of the Teeth. 1st ed. Philadelphia: W.B. Saunders; 1972.)



FIGURE 17-8. Crown fracture without pulp involvement. The fracture of the central incisor involved both enamel and dentin. Treatment involved sealing the dentinal tubules with a dentinal bonding agent followed by an esthetic composite restoration.

Crown Fracture with Pulp Involvement

Crown fractures involving the enamel, dentin, and pulp are called *complicated crown fractures* by Andreasen and *class III fractures* by Ellis. Prognosis depends on the length of time that has elapsed since the injury occurred, the size of the pulp exposure, the condition of the pulp (vital or nonvital), and the stage of root development. Make every effort to preserve the pulp in immature teeth. Conversely, in mature teeth with extensive loss of tooth structure, pulp extirpation and root canal therapy are prudent before post, core, and crown restoration. The prognosis is best for teeth with a vital pulp exposure if the fracture is treated within the first 2 hours.

Treatment requires direct pulp capping for small pinpoint exposures. If a patient's tooth has an open apex and a small pulp exposure is seen within 24 hours, it should be directly pulp-capped with calcium hydroxide. Perform calcium hydroxide pulpotomies for larger exposures and for small exposures in teeth with open apices over 24 hours old. The direct pulp cap of calcium hydroxide pulpotomy is designed to allow a tooth with an open apex to complete root development. Teeth that have calcium hydroxide pulpotomies usually require root canal therapy along with a post and core and ultimately coronal coverage.

In fractures with a vital pulp and a closed apex, perform a direct pulp cap if there is a small pulp exposure and if the patient is seen within 24 hours. If the pulp exposure is larger than 1.5 mm or if it has been present for over 24 hours, carry out root canal therapy.

Crown-Root Fracture

With a fracture that is longitudinal and follows the long axis of the tooth or if the coronal fragment constitutes more than one third of the clinical root, extraction is generally recommended. However, with a fracture line that is above or slightly below the cervical margin, appropriate forms of conservative therapy can usually be used to restore the tooth. Crown lengthening or orthodontic elevation of the involved tooth may be necessary.

Root Fracture

Root fracture is limited to fractures involving the roots only (Ellis IV). Most root fractures occur in the apical and middle one thirds and rarely in the cervical one third. Root fractures are not always horizontal; in fact, they are often diagonal in angulation. Radiographs taken immediately after an injury may not show a horizontal or diagonal root fracture. After 1 or 2 weeks when inflammation, hemorrhage, and resorption have caused the fragments to separate, the radiograph will show the damage more conclusively.

Root fractures in the apical or middle one third are usually not splinted unless there is excessive mobility (Figure 17-9). Treatment of mobile root fractures consists of apposition of the fractured segments with rigid splinting for 12 weeks.

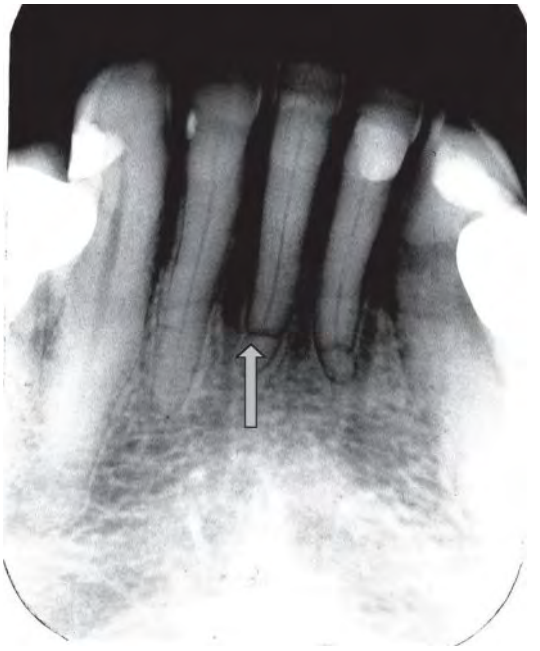


FIGURE 17-9. Mandibular central incisors with fractures of the apical one third. No stabilization was used. Vital pulp testing was noted after 8 weeks. Note the interposition of connective tissue at the fracture site *(arrow)*. (Courtesy of Dr. Thomas G. Dwyer and Dr. James R. Dow, Roseville, CA.)

Treatment for cervical one third root fractures usually involves extraction of the tooth or orthodontic extrusion of the root.

Periodontal Tissue Injury and Treatment

Injury to the periodontal tissue presents itself in many ways. Radiographically, this injury usually involves an evident dislocation or a movement of the tooth, and narrowing or loss of periodontal space may be seen. The fate of the tooth that has sustained a periodontal injury is twofold. Primarily, we see the injury from the localized impact and the late complication of the secondary resorptive process. The likely result of displacement injuries is the development of some type and degree of resorption. Thus, to better treat these types of injuries, it would behoove the surgeon to understand this process, both clinically and conceptually. This process affects both primary and permanent dentition. The etiology and pathogenesis are essentially identical to that seen in avulsion injuries, which we discuss later in this chapter in "Exarticulations (Avulsions)."

Classification of Root Resorption

Root resorption is classified as either root surface resorption or root canal resorption. Root surface resorption, also known as external root resorption, is most commonly seen after intrusive injuries and less in subluxation injuries. It is classified into three types: (1) surface resorption, (2) replacement resorption, and (3) inflammatory resorption.

Surface Resorption

Surface resorption indicates that the luxated or avulsed tooth root displays superficial resorption lacunae, which are repaired with newly formed cementum. Although not usually seen on radiographs, these may appear as vague excavations or cavities on the lateral root surface. A normal lamina dura is usually present. This development is a response to localized PDL and/or cementum injury. The process is less aggressive and self-limiting compared with the other resorption processes.

Replacement Resorption

Replacement resorption, also known as *ankylosis*, presents as an indistinguishable merging of bone and root substance. The root substance is being ultimately replaced by bone, and radiographically, a loss of the periodontal space and progressive root resorption is seen.

Inflammatory Resorption

Inflammatory resorption appears as well-circumscribed areas of cementum and dentin resorption. The localized adjacent periodontal tissue is markedly inflamed. The onset of inflammation is a result of the infected and necrotic pulp tissue within the root canal. The radiograph shows an appearance of root resorption with lines of adjacent bone radiolucency.

Root Canal Resorption

Root canal resorption, also known as *internal root resorption*, presents less often than root surface resorption. Studies found that it appears in both permanent and primary teeth. Radiographic imaging may be equivocal; labial or lingual presentations of surface resorption may be erroneously superimposed over the root canal. To avoid a misdiagnosis, supplemental radiographic views are warranted. Root canal resorption is classified as two types: (1) internal replacement resorption and (2) internal inflammatory resorption.

Internal Replacement Resorption

Internal replacement resorption shows metaplastic replacement of normal pulp tissue into cancellous bone, resulting in a widened pulp chamber. This is a characteristic process seen in root fractures and, to a lesser extent, in luxation injuries.

Inflammatory Resorption

Internal inflammatory resorption, often located at the cervical region of the pulp, presents radiographically as an irregular or oval-shaped radiolucent enlargement within the pulp chamber. This condition relates to the ingression of bacteria via dentinal tubules within a necrotic pulp delineated as the *necrotic pulp zone*. Possibly, this zone is responsible for the progression of the process. Normal pulp tissue is altered and transformed into granulation tissue with giant cells that resorb the dentinal walls of the root canal, giving the chamber an enlarged appearance. The cessation of this process will require root canal therapy (Figure 17-10).

The potential devastating effects of the resorptive process require immediate and proper treatment of periodontal injuries.

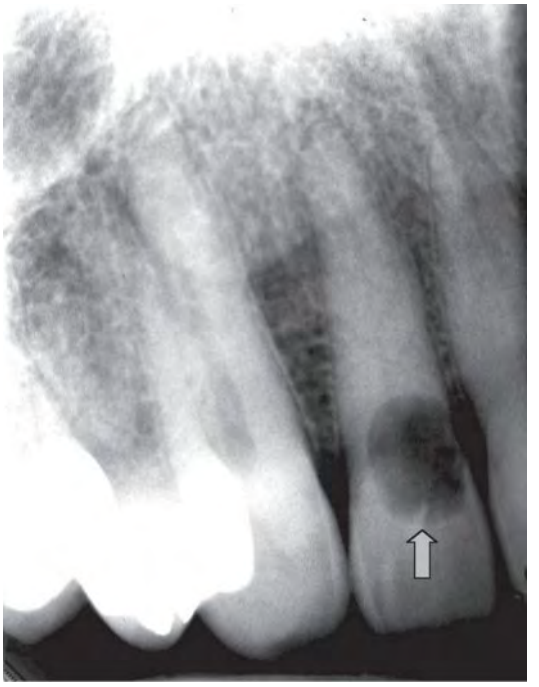


FIGURE 17-10. Maxillary lateral incisor with a history of periodontal injury (subluxation). Evidence of internal root resorption—specifically, internal inflammatory resorption *(arrow)*—is seen.

Classification of Periodontal Injuries

Periodontal injuries are classified as concussions and displacements. Displacements include subluxations, intrusive luxations, extrusive luxations, and lateral luxations.

Concussion

Often, this injury is overlooked because no acute clinical or radiographic evidence of trauma is seen. No abnormal mobility, displacement, or bleeding is apparent; only minimal injury to the tissues was acquired. Frequently, the history of the insult guides the surgeon to the suspected tooth or teeth. The hallmark to diagnosis is a marked reaction to percussion in both the horizontal and the vertical directions. The discomfort is similar to that of a "hot tooth," hyperemic quality. Because a concussed tooth may take on a chronic course or exhibit progressive problematic sequelae, it warrants close monitoring.

Treatment includes taking the suspected tooth out of occlusion to avoid function. If at all plausible, consider occlusal adjustments on the opposing dentition, thereby limiting further trauma to the involved tooth.

Displacements

Displacement injuries, or luxations, principally involve the primary and permanent maxillary central incisors. The mandibular teeth are less at risk, unless a class III malocclusion exists. Generally, displacement injuries are more prevalent in primary dentition owing to the increased elasticity and resilience of the bony supporting structures. Conversely,



FIGURE 17-11. Patient with subluxed left maxillary central incisor. Bleeding and ecchymosis at the gingival margin crevice denotes rupture of the periodontal tissues. Treatment involved stabilization with a custom-fabricated nonrigid splint for 2 weeks.

permanent teeth will have an increased risk of tooth fracture.[38,39] The specific luxation classification depends on the force and direction of traumatic impact. Fifteen percent to 61% of luxation injuries occur in the permanent dentition and 62% to73% in the primary dentition. Multiple teeth are usually involved in luxation injuries.[40]

Subluxation

Subluxation injuries occur when there is an injury to the tooth-supporting structures that causes abnormal loosening; however, there is no clinical or radiographic displacement of the involved tooth. The tooth is sensitive to percussion testing and occlusal forces. Rupture of the periodontal tissues is usually evident as bleeding at the gingival margin crevice (Figure 17-11).

Treatment is similar to that for concussion injuries with occlusal adjustments and vitality testing. Excessive mobility may necessitate nonrigid stabilization. Continue follow-up evaluation and vitality testing for 6 to 8 weeks.

Approximately 26% of injuries with this classification result in pulpal necrosis, and endodontic treatment is indicated. Studies show that external resorption will occur in 4% of these injuries.[41] Subluxation has the lowest frequency of periodontal tissue injury resorption.

Intrusive Luxation

Intrusive luxations may cause marked displacement of the tooth into the alveolar bone, with possible comminution or fracture of the alveolar socket. Percussion sensitivity is limited, and decreased mobility is noted because the tooth is essentially locked in. A high-pitched metallic sound is elicited on percussion, reminiscent of an anklyosed tooth. The intrusive injury is more commonly seen in the maxilla because of its less dense anatomy and irregular premaxillary configuration. The superiorly placed hollow cavities and thin floors of the nasal and maxillary sinuses create a formula for relative ease of dislodgment of teeth to these sites when intrusive forces are encountered. Intrusive injuries are the

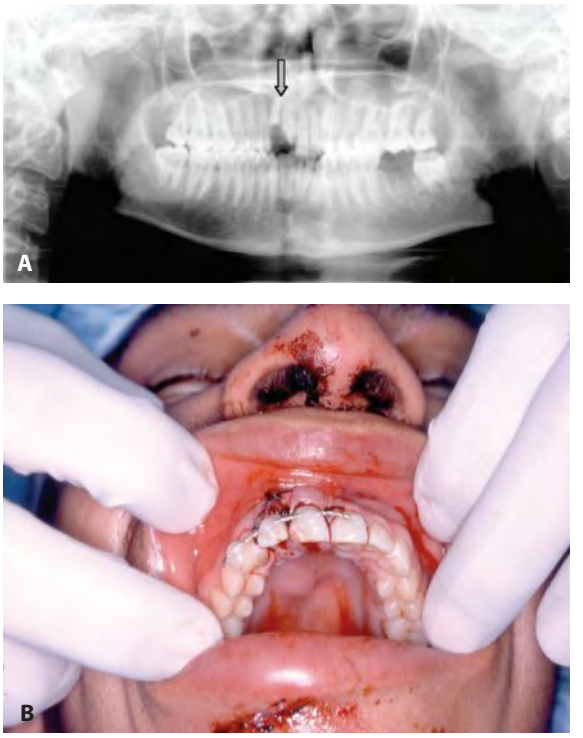


FIGURE 17-12. **A,** Panorex view depicts extent of intrusion. **B,** Teeth extruded, aligned, and stabilized with nonrigid splint. Soft tissue gingival wound repaired.

most severe of the luxation injuries that involve the pediatric patient. The intruded primary tooth may be impinging on the tooth bud of the permanent successors in a buccal-occlusal position.[26–28] The incidence of pulpal necrosis is relatively high (96%). Inflammatory resorption incidence may reach 52% as a result of the necrotic pulp (Figure 17-12).

Treating intrusive injuries depends on root development. If incomplete root development exists, allow the intruded tooth to re-erupt. Continue this process for approximately 3 months. If re-eruption does not occur, to facilitate this process, place an orthodontic extruding appliance. If pulp necrosis occurs, seek endodontic therapy. In cases of complete root development with closed apices, reposition the tooth atraumatically, and stabilize with a nonrigid splint. Then, initiate endodontic therapy in approximately 10 to 14 days after injury. Use CaOH as a canal filler in this therapy to retard or inhibit the inflammatory or replacement resorption process.[42–44] In fact, use CaOH in any intrusive luxation injuries that result in the displacement of the tooth in excess of 3 to 5 mm, and initiate within 2 weeks. This, along with instrumentation of the canal, will eradicate the bacterial contamination and allow for the repair of the PDL.

Replace the CaOH filler if it resorbs during the healing process. Arrange for frequent radiographic follow-up at 3-month intervals, and continue for 6 to 12 months. Perform conventional root canal therapy with gutta-percha obturation when signs of resorption have ceased.

Extrusive Luxation

Extrusive luxations are the partial displacement of the tooth out of the socket in a coronal or incisal direction with lingual deviation of the crown. This results in the rupture and severance of the neurovascular and periodontal ligament (PDL) tissues, respectively. There is gross mobility and bleeding at the gingival margin. Further, radiographically, the PDL space is widened. A dull sound is heard on percussion testing. Pulp necrosis occurs approximately 64% of the time, and a relatively low frequency of external resorption is seen at 7%.[41]

It is treated by delicately placing the extruded tooth back into the proper position in the socket. Check and recheck occlusion to ensure no rotation has occurred. Then, stabilize the tooth with a nonrigid splint for approximately 2 to 3 weeks. If signs of pulp necrosis occur, employ endodontic therapy.

Lateral Luxations

Lateral luxations may result from traumatic forces that displace the tooth, or teeth, in many directions; however, the lingual direction appears to be the most prevalent. These luxations often involve the bony alveolar socket. The radiographic appearance is similar to the extruded tooth on occlusal views, with the PDL space widening in the apical direction. Linear or comminuted fractures are the norm. Lingual and buccal plate expansion may render the tooth mobile. Localized soft tissue compromise is often apparent. When bony defects exist beneath the gingiva, it is common to see complex lacerations and step defects. Because the tooth is often locked in an errant position, the percussion resonance and mobility resemble the intruded tooth.

The key to treatment is to reestablish preinjury occlusion. Delay soft tissue repair until this is completed. Manipulate the tooth or teeth back into the socket. If an alveolar segment is involved, reposition it. Digitally apply buccal and lingual pressure in cases of traumatic bony expansion to ensure early PDL repair. Apply a nonrigid splint that is extended to and supported by the presumably uninjured adjacent teeth. Leave the splint in place for 2 to 8 weeks, depending on bony healing, which may require longer stabilization time. Avoid the use of disimpaction devices, such as forceps or hemostats, while attempting to reestablish proper alignment of teeth or segments. Excessive fulcruming forces may further compromise the tooth and/or supporting structure.

In persons who may have experienced delayed treatment in excess of 48 hours, reestablishing occlusion may be difficult and traumatic. Consider spontaneous or orthodontic realignment. Continue frequent radiographic follow-up and vitality testing for several months. Adjacent teeth that may have become devitalized warrant vitality testing. Any signs of pulp necrosis should be met with immediate endodontic therapy.

Another complication to consider is the loss of marginal bone support in both lateral and intrusive luxation injuries, which can occur as a temporary or permanent condition. It is seen clinically as an ingrowth of granulation tissue at the gingival crevice, resulting in a loss of attachment. This is the normal process of periodontium healing and takes up to 6 to 8 weeks. When this process occurs, continue maintenance of the splint and pay close attention to oral hygiene compliance to prevent further bone loss.

The frequency of this bony loss reaches 5% for lateral luxations and 31% in intruded luxations.[45]

Exarticulations (Avulsions)

Seemingly, avulsion injuries are the worst of the dentoalveolar injuries. By definition, these injuries involve tooth, or teeth, that are completely dislodged from the socket for a period of time. Owing to the higher risk of aspiration, supporting structure damage, or actual physical loss of the tooth, these injuries require special attention. Old ideology and myths still plague the use of newer proven protocols.

Avulsion injuries occur from 0.5% to about 16% in the permanent dentition and occur less in the primary dentition (7%–13%), with children ages 7 to 9 years being most associated with this injury. These injuries usually involve a single tooth, with the maxillary central incisor most often at risk, which is due to the relative instability of the PDL during the progressive eruption of these teeth.[46]

The treatment of such injuries must be geared toward early reestablishment of PDL cellular physiology. The fate of the avulsed tooth depends on the cellular viability of the periodontal fibers that remain attached to the root surface before re-implantation. Although extraoral time is a factor, newer physiologically compatible solutions are available that can maintain and/or replenish PDL cell metabolites. Two such solutions are Hank's balanced salt solution and ViaSpan (Figures 17-13 and 17-14).[47–49]

Both Hank's solution and ViaSpan are physiologic with compatible pH and osmolality (Table 17-2). ViaSpan is the solution of choice for organ storage during transport for transplantation. The relative availability and cost effectiveness of Hank's solution make it the medium of choice in storage of avulsed teeth. Commercially available by Phoenix Lazarus, Inc., Save-A-Tooth, an emergency tooth preserving system that contains Hank's solution as its active ingredient, is a mainstay in many athletic first aid kits.



FIGURE 17-13. Hank's balanced salt solution, commercially available as Save-A-Tooth (Phoenix Lazarus, Inc.).



FIGURE 17-14. ViaSpan, cold storage solution currently available as an organ transport solution.

Other methods for temporarily storing an avulsed tooth are milk, saliva, and saline; however, their ability to replenish cellular metabolites has not been documented. Milk is a readily available medium for the lay person, and because time is of the essence, it is the medium of choice in the absence of Hank's solution or ViaSpan. Milk will only prevent further cellular demise; thus, it is used specifically when teeth have been extraoral for less than 20 minutes. Any periodontal ligament extraoral exposure greater than 15 minutes will deplete most of the cell metabolites; for this reason, a longer period of extraoral time limits milk's effectiveness to maintain cellular viability. Unlike Hank's solution and ViaSpan, which can store avulsed teeth and replenish cellular metabolites for 24 hours and 1 week, respectively, milk as a storage medium becomes ineffective after approximately 6 hours.[50,51]

Treatment

Considering the root maturation, the extraoral time, and the general health of the tooth preinjury determines the route of treatment. The idea of early or immediate replantation should be adopted.

TABLE 17-2. **Solutions to Replenish Periodontal Ligament Cell Metabolites**

Solution	Characteristics
Hank's balanced salt solution	pH = 7.2 Osmolality = 320 mOsm
ViaSpan	pH = 7.4 Osmolality = 320 mOsm
Cow's milk	pH = 6.5–6.7 Osmolality = 225 mOsm

Teeth that are in poor condition from a hygiene standpoint are generally not replanted. Those that present with moderate to severe periodontal disease, gross caries involving the pulp, apical abscess formations, infection at the replanting site, and bony defects and/or alveolar injuries, in which supporting bone is lost, are less likely to be considered for replantation.

To optimize success of treatment, replant and stabilize avulsed teeth within 2 hours (120 min); PDL cells become irreversibly necrotic after this time frame. Attempt to salvage avulsed teeth, even if the critical 2-hour period has passed, but the prognosis becomes progressively worse.

Teeth with open apices greater than 1 mm diameter have a prognosis much better than that of the more mature or closed-root apex. Treat the tooth with an open root within the 2-hour timeframe by placing it in Hank's solution for about 30 minutes. Next, place the tooth in a 1 mg/20 mL doxycycline bath for 5 minutes, followed by immediate replantation and splint stabilization.[47,52] If radiographic or clinical evidence of pathology is noted, perform an endodontic apexification procedure with a CaOH filling. The CaOH should be periodically replaced until the apex is closed, followed by conventional root canal therapy.

Newer materials for apexification procedures are on the market that decrease the need for multiple CaOH replacements—one of which is ProRoot MTA (Mineral Trioxide Aggregate), marketed by Densply Tulsa Dental. Contrary to CaOH, MTA provides a hard-setting nonresorbable surface with cavity adaptation. It provides excellent tissue biocompatibility and allows for immediate apical seal.[53,54]

The increased potential for reestablishment of pulpal circulation in teeth with open apices has been shown to improve prognosis of survival of the pulp and PDL in the avulsed tooth (Figures 17-15 and 17-16). This revascularization process is optimized by the topical application of doxycycline.

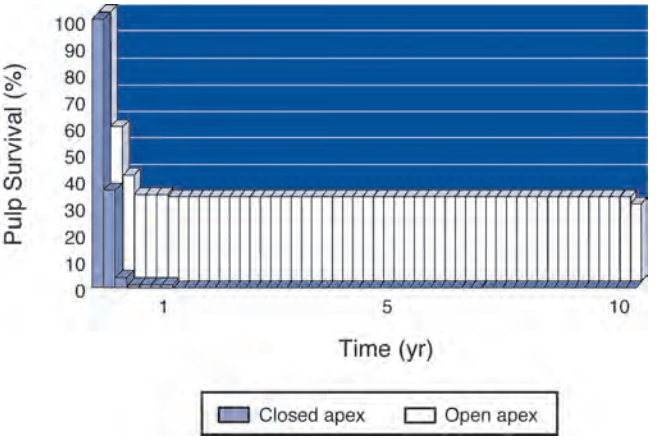


FIGURE 17-15. Studies by Andreasen and colleagues support the increased potential for pulpal healing after replantation related to stage of root development (closed vs open apex). (Adapted from Andreasen JO, Andreasen FM. Textbook and Color Atlas of Traumatic Injuries to the Teeth. 3rd ed. Copenhagen: Munksgaard; 1994; pp. 202–210.)

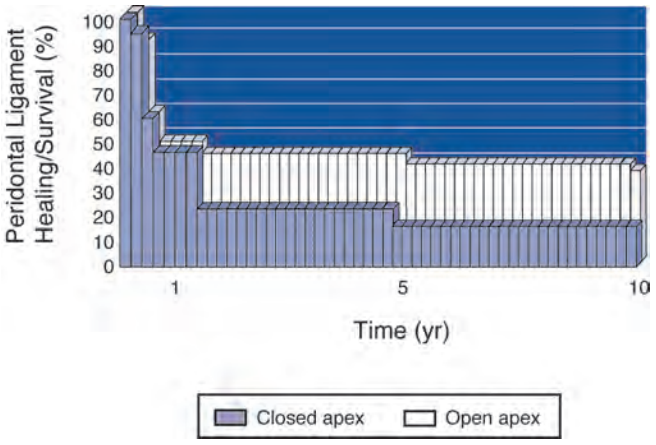


FIGURE 17-16. Periodontal healing/survival after replantation related to stage of root development (closed vs. open). (Adapted from Andreasen JO, Andreasen FM. Textbook and Color Atlas of Traumatic Injuries to the Teeth. 3rd ed. Copenhagen: Munksgaard; 1994; pp. 202–210.)

Individuals who have avulsed teeth with mature or closed apices and who present within the 2-hour timeframe are treated by placing the tooth in Hank's solution for about 30 minutes, followed by replantation and splinting for 7 to 10 days. Carry out endodontic cleansing and shaping of the canal, and place a CaOH filling just before splint removal. Final gutta-percha obturation is contingent on resolving canal and/or root pathology (6–12 mo). Late failure of the replantation process is manifested as either inflammatory or replacement resorption owing to a necrotic pulp or compromised PDL, respectively.

In individuals who experience an extraoral period that exceeds 2 hours, apical root morphology plays little role in the success rate. Eliminate the necrotic PDL strands manually or chemically in a sodium hypochlorite wash for approximately 30 minutes. Perform root canal therapy extraorally with conventional cleansing and shaping of the canal. Withhold final obturation until the canal, dentinal tubules, and root surface have been treated with various chemicals in a stepwise fashion. First, a citric acid bath for 3 minutes, followed by rinsing with 0.9% NaCl, will open and débride the dentinal tubules, thus allowing unimpeded ingrowth of connective tissue to the root surface. Second, the tooth should be moved to a 1% stannous fluoride solution for 5 minutes. This will decrease the risk of the resorption process.

Finally, set up a 5-minute bath of 1 mg/20 mL doxycycline, which will rid the root surface of residual bacterial remnants and facilitate pulpal revascularization. Complete the final obturation with gutta-percha. The tooth is then replanted into preinjury alignment and splinted for 7 to 10 days (Tables 17-3 and 17-4).[50,52,55]

Splinting Protocol and Technique

Splinting after avulsion and displacement injuries immobilizes the tooth or segment into proper preinjury alignment and allows for the initial pulpal revasculature and PDL healing course. Several techniques have been advocated in the past; however, the acid-etch/resin splint (or variants of this technique) is the treatment of choice.[56,57] This technique fulfills the requirements of acceptable splint utilization in a maxillofacial traumatic injury (Table 17-5).

TABLE 17-3. **Treatment Summary for Avulsed Teeth**

≤2 Hr Open Apex
1. Replant immediately if possible.
2. Transport in Hank's solution or milk.
3. Present to nearest qualified facility (decrease time, call first).
4. Check ABCs; evaluate for associated injuries (history and physical examination).
5. Store in Hank's solution for about 30 min.
6. Transfer to a 1-mg/20 mL doxycyline bath for about 5 min.
7. Perform radiography (posteroanterior, occlusal, panoramic, chest).
8. Initiate local anesthesia.
9. Irrigate socket with saline solution.
10. Perform tetanus prophylaxis as needed.
11. Initiate antibiotic coverage.
12. Replant tooth.
13. Splint for 7–10 days
14. Perform apexification with CaOH in cases of pathosis.

Closed Apex
1. Store in Hank's solution for about 30 min.
2. Replant.
3. Splint for 7–10 days.
4. Perform endodontic cleansing and shaping of canal at time of splint removal.
5. Fill canal with CaOH (6–12 mo).
6. Perform final gutta-percha obturation (~6–12 mo).

ABC = airway, breathing, and circulation.

TABLE 17-4. **Treatment Summary for Teeth Avulsed > 2 Hours***

1. Replant immediately, if possible.
2. Transport in Hank's solution or milk.
3. Present to nearest qualified facility (decrease time, call first).
4. Check ABCs; evaluate for associated injuries (history and physical examination).
5. Bathe tooth in sodium hypochlorite for ~30 min vs. manual débridement of the periodontal ligament.
6. Perform extraoral RCT.
7. Bathe tooth in citric acid (~3 min).
8. Bathe tooth in 1% stannous fluoride (~5 min).
9. Transfer to a 1-mg/20 mL doxycyline bath for ~5 min.
10. Perform radiography (posteroanterior, occlusal, panoramic, chest).
11. Initiate local anesthesia.
12. Perform tetanus prophylaxis as needed.
13. Initiate antibiotic coverage.
14. Replant tooth.
15. Splint for 7–10 days.

*Open or closed apex.
ABC = airway, breathing, circulation; RCT = root canal therapy.

SECTION 3

TABLE 17-5. **Splint Requirements**

The splint should
1. Be able to be applied directly in the mouth without delay due to laboratory procedures.
2. Stabilize the injured tooth in a normal position.
3. Provide adequate fixation throughout the entire period of immobilization.
4. Neither damage the gingiva nor predispose to caries and should allow for a basic oral hygiene regimen.
5. Not interfere with occlusion or articulation.
6. Not interfere with any required endodontic therapy.
7. Preferably fulfill esthetic demands.
8. Allow a certain mobility (nonrigid) to aid periodontal ligament healing in cases of fixation after luxation injuries and replacement of avulsed teeth; however, after root fracture, the splint should be rigid to permit optimal formation of a dentin callus to unite the root fragments.
9. Be easily removed without re-injury to tooth.

Adapted from Andreasen JO, Andreasen FM. Textbook and Color Atlas of Traumatic Injuries to the Teeth. 3rd ed. Copenhagen: Munksgaard; 1994; pp. 347–348.

TABLE 17-6. **Sequence of Acid-Etch Splinting Technique***

1. Perform alveolar bony reduction and/or replantation.
2. Perform localized cleansing and débridement.
3. Isolate and dry area.
4. Custom fabricate wire (~26 Ga), double-stranded monofilament nylon line, or paper clip. Extend wire to at least one or two teeth on either side of the involved tooth or teeth.
5. Etch the incisal half of the labial surface of the involved and adjacent teeth with gelled phosphoric acid for 30–60 sec.
6. Remove etchant with water stream for ~20 sec
7. Air-dry etched surface; surface should appear chalky white.
8. Passively place prefabricated wire to involved teeth.
9. Stabilize splint with fast-setting autocure or light-cure composite resin.
10. After resin is set, smooth rough edges with a fine acrylic or diamond finishing bur (check occlusion).
11. Perform soft tissue and gingival repair as needed.
12. Remove splint in 7–10 days.

*It may be prudent to use a composite shade that differs from the natural color of the involved teeth because this will facilitate ease of removal and prevent trauma to enamel.

The acid-etch technique is the only system that most closely adheres to these recommendations (Figure 17-17). The arch bar, self-curing, Essig, intracoronal, and circumferential splints may rarely present with an indication but are not routinely recommended. Each has been demonstrated to violate one or many of the basic splint requirements. The arch bar, in particular, produces an eruptive or extrusive force because of the placement of the wire beneath the height of contour of the tooth. Also, the rigid nature of these techniques will facilitate the external resorption process (Table 17-6).

TREATMENT OF FRACTURES OF THE ALVEOLAR PROCESS

Owing to the exposed anatomy, alveolar fractures usually occur at the incisor and premolar regions. Treatment involves early reduction and stabilization of the involved segments. Depending on the fracture's severity, use either an open or a closed technique. Digital manipulation and pressure, along with rigid splint stabilization, will usually be sufficient in the closed technique. Leave the splint in place for approximately 4 weeks.

A gross displacement and/or impedance to reduction may necessitate the open technique. Inability to freely reduce fracture segments may be due to root or bony interferences or impaction (apical lock) (Figure 17-18). Access to the area involves an incision that provides adequate exposure and is located apical to the fracture lines. The segment is then disimpacted or freed up. Proper alignment and occlusion are then attained, and the segments are stabilized with suitable transosseous wire or a small (2.0-mm) monocortical plate. Ensure that the closure of the wound is meticulous

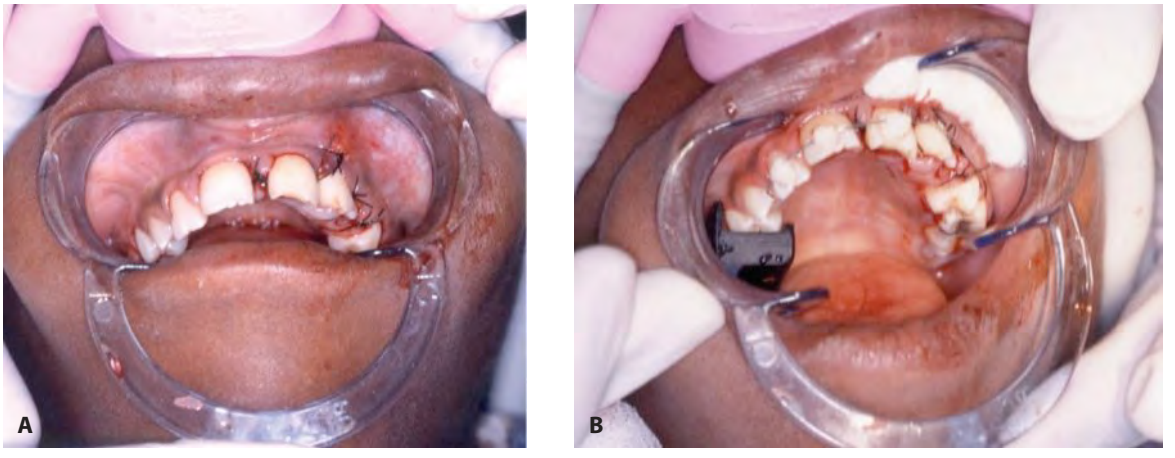


FIGURE 17-17. Acid-etch splinting technique. **A,** Subluxed tooth and alveolar fracture associated with maxillary left central incisors. **B,** Nonrigid passive placement of splint. Traumatized teeth are placed into preinjury alignment, acid-etched, and stabilized with composite resin. Splint is free of occlusion and soft tissue trauma.

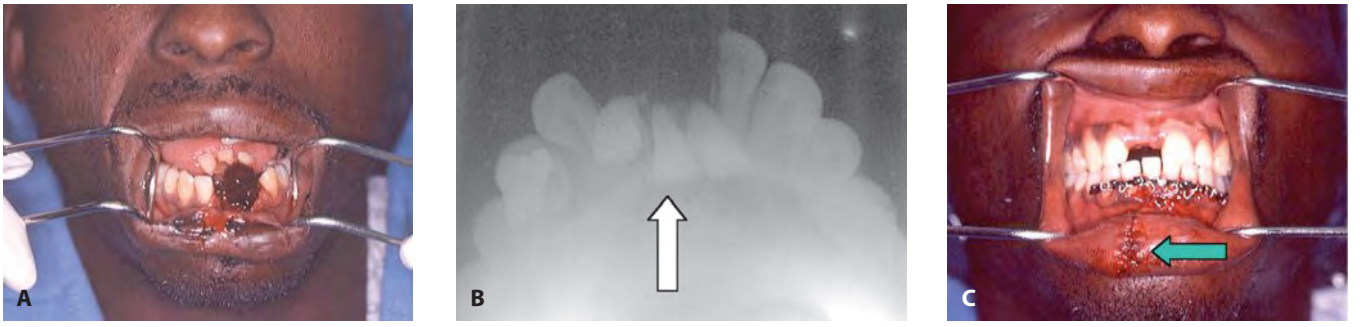


FIGURE 17-18. **A,** Blunt facial trauma resulting in alveolar fracture and perioral soft tissue lacerations. **B,** Occlusal radiograph confirms alveolar fracture with lingual displacement ("apical lock") of mandibular central incisors and left lateral incisors *(arrow).* **C,** Alveolar fracture disimpaction, reduction, and stabilization with arch wire. Débridement and repair of perioral soft tissues *(arrow).*

to prevent exposure of bone and/or hardware to the ingress of bacteria.

Stabilize teeth that may be mobile in the fractured segment with an appropriate secondary splint after bony stabilization. Likewise, avoid removing teeth that are considered nonsalvageable and that are within the bony segment until the bony healing phase is completed (~4 wk). Obvious infection and inadequate bony envelopment indicate early removal.

Successful treatment of alveolar fractures is associated with the pulpal healing after the injury. When the fracture level is apical to the root tips, the vascular supply to the pulp is less at risk; however, if the line of the fracture and root apices are in contact, the teeth in the alveolar segment are at a higher risk for internal or external resorption.

In concomitant injuries, such as maxillary or mandibular fractures, early maxillomandibular fixation is accomplished with a technique that will allow for dual treatment of the dental and/or alveolar injury and the jaw injury (e.g., arch bars and maxillomandibular fixation). Perform the more invasive open reduction if indicated.

Avulsive injuries will often expose bone and jeopardize tooth support. Aim treatment at soft tissue coverage in the form of judicious mucosal advancement flaps. Consider early removal for teeth without bony support.

Treatment of Trauma to the Gingiva and Alveolar Mucosa

Traumatic injury to the oral soft tissue mainly consists of abrasion, contusion, and laceration. If these injuries are not addressed, they can place the underlying bony tissue at risk for devitalization. Frequently, these injuries may alert the surgeon to underlying trauma. The ultimate goal of treatment is to reestablish vital soft tissue bony coverage.

Abrasion

An abrasion is a superficial wound wherein the epithelial or gingival tissue is rubbed, worn, or scratched. Treatment consists of local cleansing with a mild disinfectant soap for the skin and saline rinsing and/or irrigation of the gingiva. Antibiotic coverage is seldom necessary. Inspect the wound for possible foreign body (asphalt) accumulation, which could lead to unsightly accidental tattooing. If present, carry out meticulous removal within 12 hours, with care not to further inoculate the patient.[58] The removal process includes a technique that aligns the surgical blade perpendicular to the direction of the abrasion.

Contusion

A contusion, a hemorrhage of subcutaneous tissue without laceration or break of overlying soft tissue, is similar to a bruising injury caused by blunt trauma. Treating gingival contusion includes local cleansing and observation. This injury may be associated with an underlying hematoma or ecchymotic formation, which is generally self-limiting. Antibiotic coverage is usually unnecessary.

Laceration

Lacerations are the most common form of facial injury. Gingival lacerations may involve an underlying bony defect. Treatment involves early cleansing and reapproximation. Remove devitalized tissue in a conservative manner, and suture in a manner that limits wound tension. Consider antibiotic and tetanus prophylaxis. More serious avulsive gingival wounds warrant close inspection of remaining tissue and underlying bony integrity. Exposure of any underlying bony defect may indicate localized keratinized sliding or advancement flaps. If nonkeratinized tissue is used for coverage, future grafting may be indicated.

Pediatric Dentoalveolar Trauma Treatment

The poor coordination of pediatric patients who are learning to walk, as well as their relatively large pulp chamber–to–tooth ratio, accounts for most pediatric dentoalveolar injuries. Managing the patient may require sedation and restraint; thus, additional factors must be dealt with during the treatment regimen.

Displacement injuries are more prevalent than are tooth fractures in the primary dentition secondary to the relative resilience of the surrounding bone. Similarly, these injuries

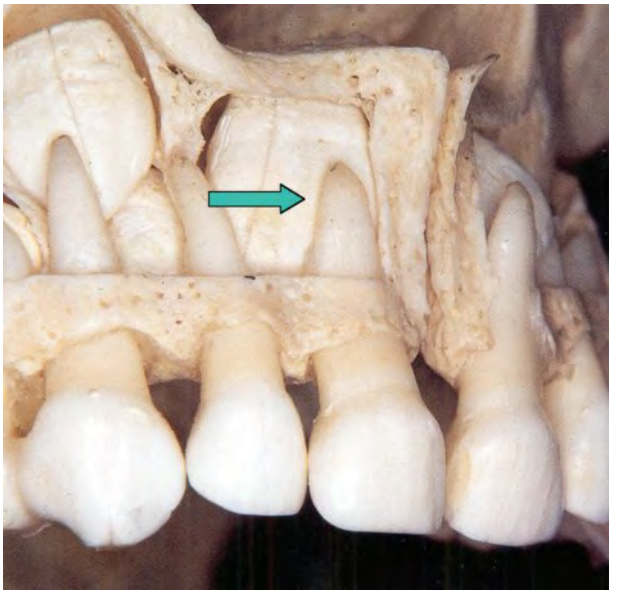


FIGURE 17-19. Anatomic position of the primary dentition to the developing permanent tooth bud. Note the "buccal-occlusal" and "buccal-incisal" position of the primary roots *(arrow)*.

are more common in the pediatric dentition than in the permanent dentition.

Treating the primary dentition is dictated by the likelihood that the permanent tooth bud may be compromised, secondary to the buccal-occlusal position of the primary teeth to the permanent tooth bud (Figure 17-19). Transmission of force to the developing tooth is possible in displacement injuries, which may cause interference with odontogenesis, ultimately resulting in enamel discoloration and/or hyploplasia (Figure 17-20). Table 17-7 provides a summary of the treatment regimen.

TABLE 17-7. **Treatment of Pediatric Injuries**

Type of Injury	Treatment
Crown Fractures	
Class I (enamel only)	Smooth rough edges.
Class II (enamel and dentin)	1. CaOH or glass ionomer liner over dentin. 2. Composite resin restoration.
Class III (Pulpal Involvement)	
Vital pulp	1. Formocresol pulpotomy. 2. Coronal coverage.
Nonvital pulp	1. ZnOH-eugenol pulpectomy. 2. Coronal coverage.
Class IV (Root Fracture)	
Apical third	No treatment; follow-up.
Cervical third	1. Remove tooth fragments. 2. Allow apical third to resorb if compromise to permanent tooth bud is expected.
Luxations	
Subluxation	Monitor/follow-up.
Lateral luxations	Realign/remove prn.
Extrusion	Realign/remove prn.
Intrusion	1. Allow to re-erupt 4–6 wk. 2. Remove if in contact with permanent successor. 3. Remove if infection presents.

prn = as needed.

Andreasen and Ravn[59] reported on the general prognosis of the traumatized permanent successors, secondary to forces applied by the primary dentition. They found that the individual's age at the time of injury and the type of luxation play a major role in the errant development of the permanent dentition (Figure 17-21).[21,22,59]

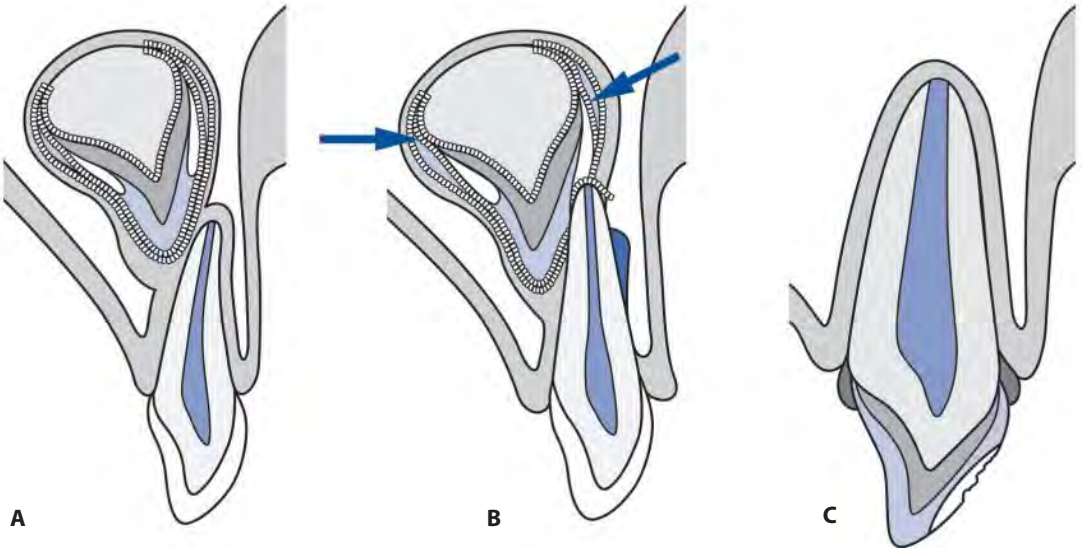


FIGURE 17-20. **A,** Normal position of primary tooth to permanent tooth bud. **B,** Apical intrusion of primary root impinging on permanent tooth bud. *Blue arrows* denote permanent tooth bud. **C,** Hypoplasia of permanent tooth secondary to apical intrusion.

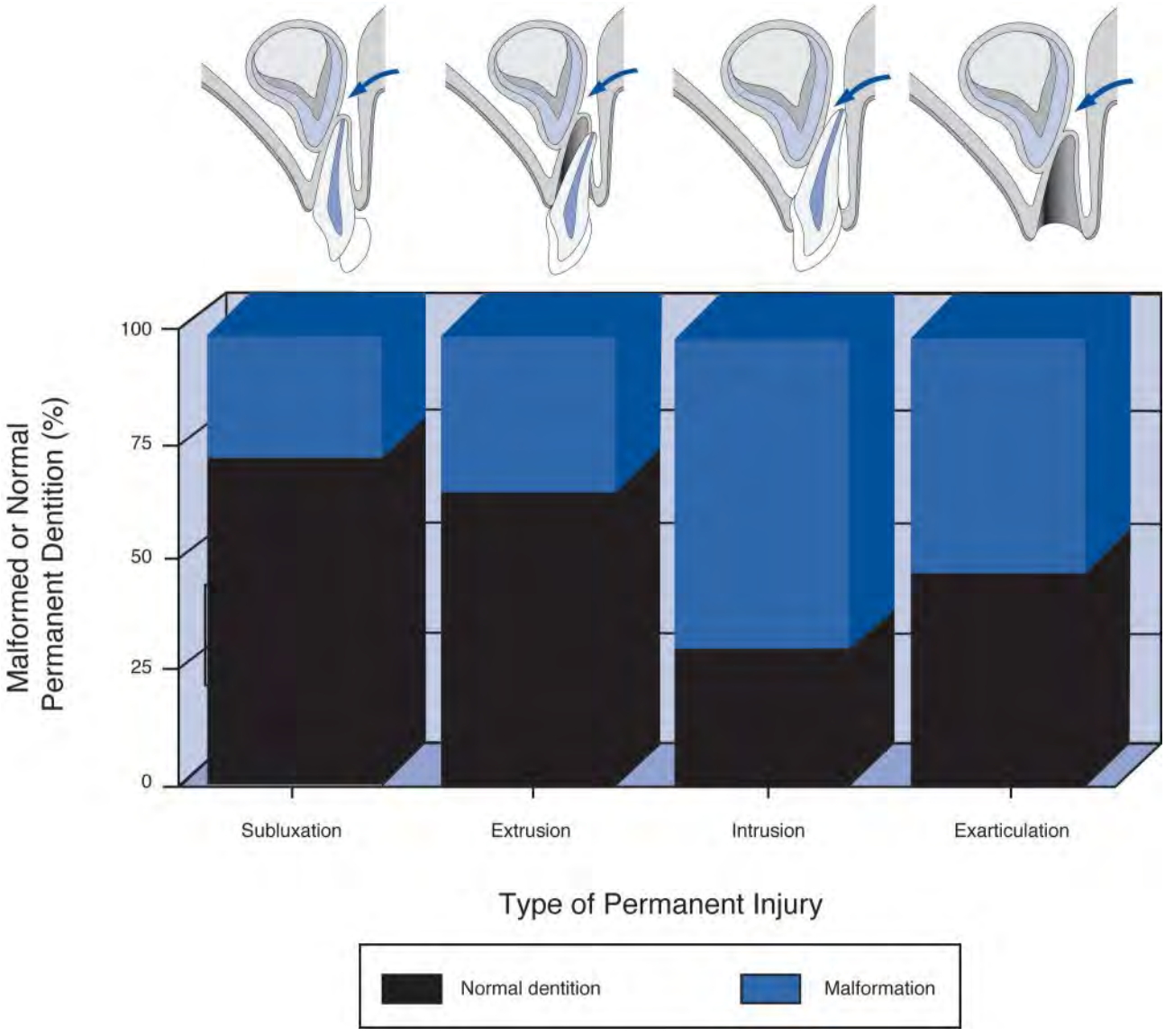


FIGURE 17-21. Association of the type of luxation injury with respect to the malformation of the permanent dentition. (Adapted from Andreasen JO, Ravn JJ. Enamel changes in permanent teeth after trauma to their primary predecessors. Scand J Dent Res 1973;81:203.)

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CHAPTER 18

Contemporary Management of Mandibular Fractures

R. Bryan Bell, DDS, MD, FACS

For better or worse, there may be no single injury that is associated more with the contemporary oral and maxillofacial surgeon than the mandibular fracture. Although varying in their location and severity, they invariably involve the teeth and the occlusion. Therefore, formal training in dentistry is extremely helpful for achieving favorable treatment outcomes. Although closed treatment of less complex mandibular injuries continues to play an important role in the treatment paradigm, technologic advances in the reduction, stabilization, and fixation of even the most complex of mandibular fractures have resulted in unparalleled predictability with which patients are returned to form and function. This chapter focuses on the contemporary management of patients with mandibular trauma, provides the reader with a literature-based guide to clinical decision-making, and when possible, illustrates an organized approach to the trauma patient with facial injuries.

HISTORICAL PERSPECTIVES

At the time of this writing, a Medline query for reconstruction of the mandible yielded more than 3000 scientific articles. Indeed, the medical literature has collected articles detailing advances in mandibular reconstruction for centuries, with the earliest known writings on mandibular fractures appearing in the Edwin Smith Papyrus in 1650 BC.[1] Hippocrates later described manual reduction of mandibular fractures using dental fixation and bandaging.[2] Salicetti[3] is the first surgeon to recognize the importance of maxillomandibular fixation (MMF) in his textbook, *Cyrurgia*, published in 1476. Barton[4] described a systemic method of bandaging mandibular fractures in 1819, a technique that was utilized extensively through the 19th century. It was not until Gilmer[5] reintroduced the concept of interdental wire fixation in the United States in 1887, however, that the basic principles of reduction, stabilization, and fixation that we know of today as the foundation for mandibular fracture repair were introduced.

Perhaps during no other time period has the treatment of maxillofacial injuries been so advanced than World Wars I and II. [6–13] In Germany, as early as August 1914, a hospital of 225 beds was opened at Dusseldorf for the specific treatment of wounds of the face and jaws, and by April 1915, similar institutions had been established in other parts of the German empire.[14] August Lidemann in Dusseldorf, Martin Wassmund in Berlin, and Hans Pichler in Vienna led three important German schools that grew out of the war experience and influenced maxillofacial surgery to this day. On the allied side, Charles Valadier, a French American dentist, was stationed in Abbeville and later at a field hospital in Boulogne. Valadier established a jaw fracture unit at the 83rd general hospital at Wimereux. Valadier was joined in July 1916 by Captain H. Lawson-Whale, an otolaryngologist, and later by Captain Frederick John Clemenison, another otolaryngologist who assisted Valadier in many of the surgical aspects of the facial fracture management.[14] Varazad Kazanjian, an Armenian American dentist who came to the Western Front with the Harvard-based American Expeditionary Forces in 1917, made numerous contributions to mandibular fracture repair. By combining dental splints with transosseous wires and plaster head caps, he was able to achieve bony union in cases that previously were thought to be untreatable and brought a new level of predictability to mandibular fracture management. His innovative approach, skill, and dedication to the treatment of soldiers with maxillofacial injuries lead to his nickname the “miracle man of the Western front.”[14]

In 1915, Harold Gillies, a New Zealand-born otolaryngologist, established the first comprehensive maxillofacial unit at Cambridge Hospital in Aldershot, England. Valadier's unit in France became a staging ground for Gillies' unit in England. The Aldershot Center soon became inadequate and Gillies was transferred to Queens Hospital in Sidcup, England. Gillies was joined by Dr. Kelsey Fry, an oral surgeon, who proposed to Gillies, "I'll take the hard tissues, and you take the soft,"[14] This arrangement of shared responsibility lead to countless innovations, which were chronicled in Gillies' classic textbook published in 1920, *Plastic Surgery of the Face*,[6] and Fry and coworkers' *The Dental Treatment of Maxillofacial Injuries*.[13] A generation of surgeons subsequently made pilgrimage to Great Britain to learn from Gillies, arguably the most influential maxillofacial surgeon of the 20th century.[15–18]

EPIDEMIOLOGY

It is difficult to obtain a true snapshot of the epidemiology of mandibular trauma, because the literature is filled with institutional reviews that vary in their demographics, geography, economy, and setting (civilian vs. military).[19–32] There are clearly trends in the incidence of facial injuries, depending upon whether a country is developing or already developed. There has been an increase in injuries from motor vehicle collisions in developing countries related to the increases in volume of traffic, rural to urban drift of population, poor road conditions, increased import of second-hand vehicles, driving under the influence of alcohol, noncompliance with seat belt or crash helmet use, and the lack of air bags.[33] Conversely, the incidence of facial injuries has greatly decreased since the early 1990s in most developed countries.[26,27] The presence of safe roads, strict safety laws for vehicles (e.g., construction, air bags), and strict safety laws for driving (e.g., speed limits, seat belt laws, stiff penalties for driving under the influence of alcohol) are primarily responsible for the decrease in injuries in developed countries.[34]

Data from the U.S. Agency of Healthcare Research and Quality also confirms that the incidence of mandibular fractures is trending downward, presumably due to public safety legislation (Figure 18-1).[35] According to the Healthcare Cost and Utilization Project (H-CUP), the number of mandibular fractures diagnosed in patients who were discharged from U.S. hospitals in 2008 was approximately 30,826. Sixty percent of these patients were aged 18 to 44, 76% were male, and a vast majority presented in urban areas (96%).

Despite these improvements, mandibular fractures remain a common occurrence at urban trauma centers. At a representative level I trauma center, mandibular fractures are present in more than 13% of all severely injured patients admitted by the trauma service (not including isolated facial injuries) and entered into the trauma registry.[30] They are the fourth most common facial fracture, occurring in 37% of

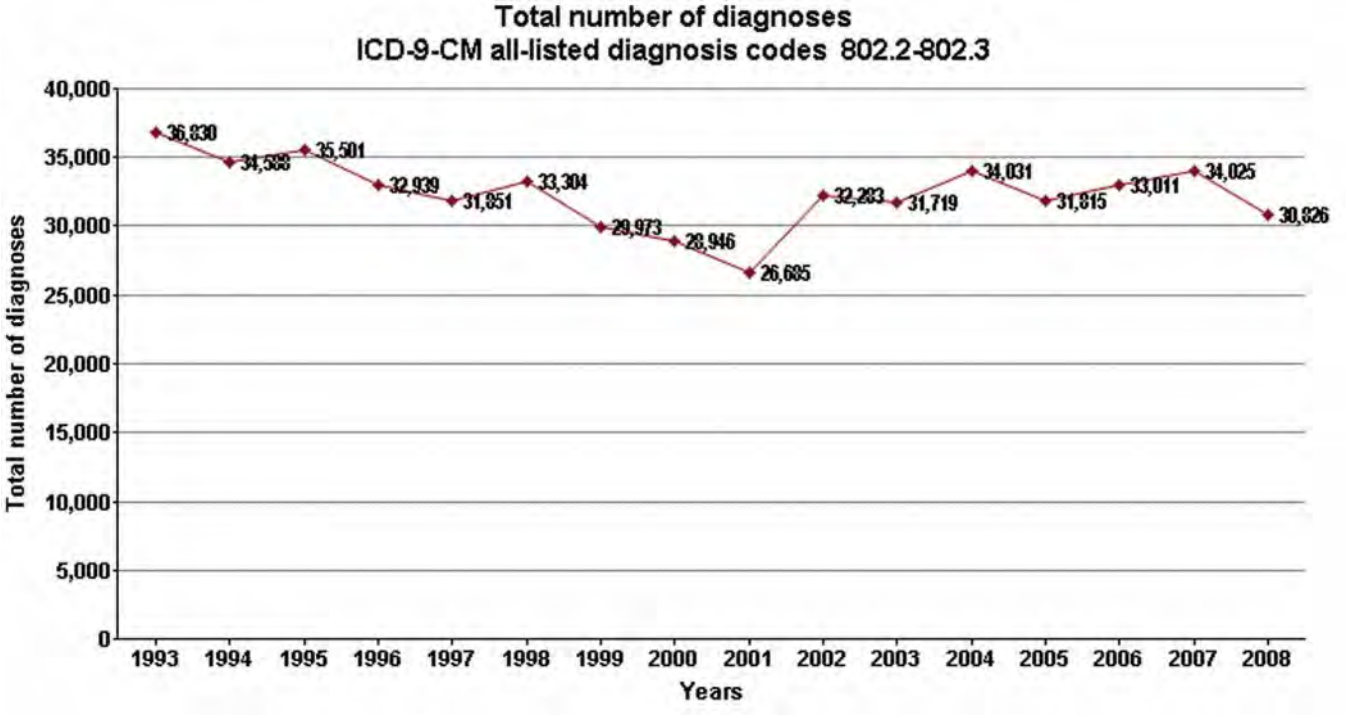


FIGURE 18-1. United States Trend Information from 1993 to 2008 (Healthcare Cost and Utilization Project [H-CUP]). Total number of mandibular fractures, International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM) diagnosis codes 802.2–802.3. Weighted national estimates from H-CUP Nationwide Inpatient Sample (NIS), Agency for Healthcare Research and Quality (AHRQ), based on data collected by individual states and provided to AHRQ by the states. Statistics based on 10 or fewer weighted cases in the nationwide statistics are not reliable. These statistics are suppressed and are designated with an asterisk (*).

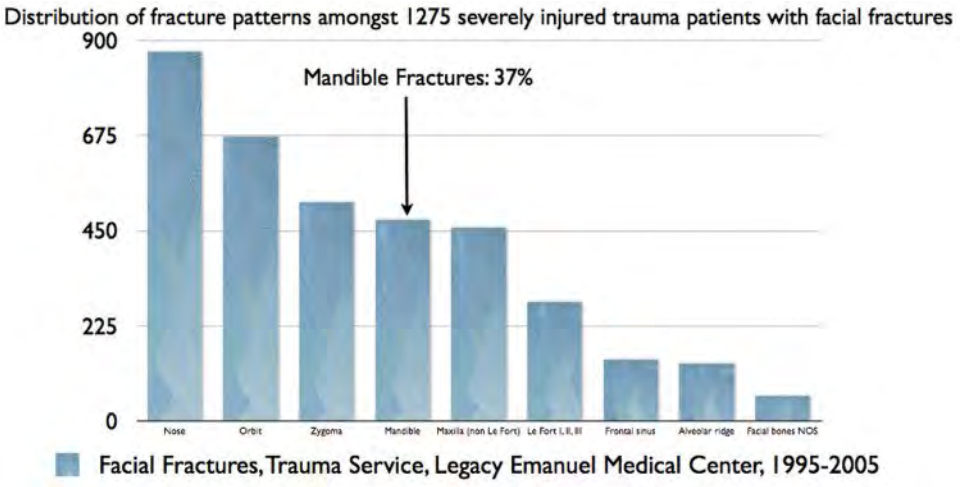


FIGURE 18-2. Distribution of fracture patterns amongst 1275 severely injured trauma patients with facial fractures. Trauma Registry Data, Legacy Emanuel Medical Center, 1995–2005.

patients, and are the most commonly operated upon (Figure 18-2). Among patients with facial injuries, mandibular fracture occurs in between 37% and 67% of the cases, depending upon the setting (urban or rural, military or civilian).[19–33] There is great variability in the distribution of etiologies based upon numerous factors, but motor vehicle collisions and assaults are, by far, the most common causes of facial injury at urban U.S. trauma centers. Other relatively common causes include sports-related injuries, industrial or work place accidents, and falls. Mandibular fractures associated with midfacial fractures are more frequently caused by motor vehicle collisions, whereas isolated mandibular fractures may be seen more often in assaults.[31]

CLASSIFICATION

Ivy and Curtis[36] provided a practical classification for mandibular injury in 1926 that describes pattern of fractures as occurring in the symphysis, parasymphysis, body, angle, ramus, neck of the condyle, and coronoid process. In their review of mandibular fractures, Haug and Greenburg[28] reported that males with mandibular fractures outnumbered females by a ratio of 3:1, and right- and left-sided fractures had equal distribution. The anatomic order of frequency of mandibular fractures was the body (29.5%), angle (27.3%), condyle (21.1%), symphysis (19.5%), ramus (2.4%), and coronoid (0.2%) (Figure 18-3).

Whereas this simple description of fractures is useful for communication, it is not helpful in either therapeutic decision-making or scientific evaluation owing to the differences in fracture pattern and severity caused by variables such as kinetic energy and etiology. A number of authors have attempted to subclassify patterns of mandibular injury while accounting for such variables.[37–48]

Existing classification systems have a number of shortcomings including a lack of scoring for a combined fracture, an inability to classify multiple separate osseous regions with isolated fractures, and an inability to classify severe fracture patterns resulting from high-velocity or gunshot injuries. In an effort to overcome some of these limitations, Buitrago-Tellez and colleagues[49] proposed a comprehensive classification system based upon imaging analysis (Figure 18-4). This Arbeitsgemeinschfat fur Osteosynthesefragen/Association to the Study of Internal Fixation (AO/ASIF) scheme defined three fracture types (A, B, C), three groups within each type (e.g., A1, A2, A3), and three subgroups within each group (e.g., A1.1, A1.2, A1.3) with increasing severity from A1.1 (lowest) to C3.3 (highest). The mandible was divided into two vertical units (I and V), two lateral horizontal units (II and IV), and one central unit (III) composing the symphyseal and parasymphyseal region. Type A fractures are nondisplaced, type B are displaced, and type C are multifragmentary/defect injuries.

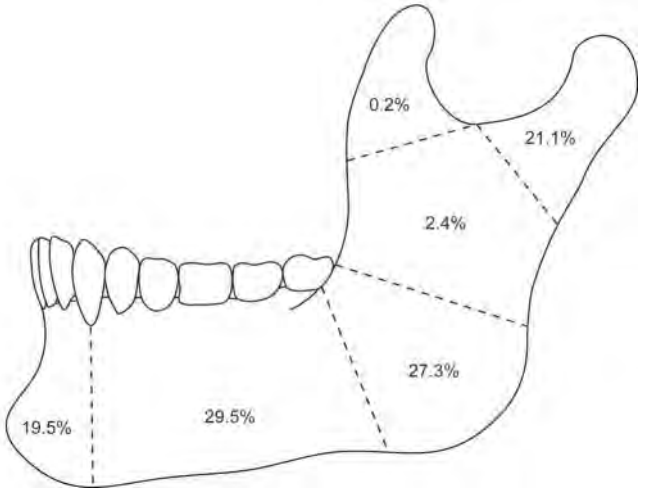


FIGURE 18-3. Anatomic distribution of mandibular fractures. (From Haug RH, Prather J, Indresano AT. An epidemiologic survey of facial fractures and concomitant injuries. J Oral Maxillofac Surg 1990;48:926–932.)



FIGURE 18-4. Arbeitsgemeinschfat fur Osteosynthesefragen/Association to the Study of Internal Fixation (AO/ASIF) Comprehensive classification of mandibular fractures with division lines for vertical, lateral horizontal, and central units. **A,** Vertical mandibular units *(green),* lateral horizontal units *(orange),* and central mandibular units *(red)*. The vertical manidbular units include the subcondylar/condylar region, the ascending ramus, and the mandibular angle. The lateral horizontal units comprise the lateral mandibular bone/body and the dentoalveolar components. The central unit includes symphyseal and parasymphyseal regions. **B,** Lateral view shows the vertical mandibular unit *(green),* the lateral horizontal unit *(orange),* and central unit *(red)*. **(A** and **B,** From Buitrago-Tellez CH, Audige L, Strong B, et al. A comprehensive classification of mandibular fractures: a preliminary agreement validation study. Int J Oral Maxillofac Surg 2008;37:1080–1088.)

BIOMECHANICAL CONSIDERATIONS

The mandible develops zones of tensile and compressive force during normal function. The location of these zones is dependent upon the direction, location and magnitude of force, stress, or load applied. An understanding of biomechanics is necessary not only for predicting patterns of injury but also for evaluating the effectiveness of fixation techniques.

Current theories regarding mandibular biomechanics were largely derived from a series of experimental studies performed in the 1960s by Huelke and associates.[50–55] They applied forces of varying magnitudes to dried skulls and, using a stress and strain model, observed the resultant tensile and compressive forces. They found that, with the exception of the mandibular condylar region, experimentally produced fractures were in areas of tensile strain rather than in areas of compression. Condylar head injuries, conversely, tended to be produced by a load parallel to the ramus, due to compression. For example, when a force is applied to the parasymphysis region of the mandible, a compressive strain develops along the buccal aspect, and tensile strain occurs along the lingual surface. A fracture will occur on the lingual side of the mandible, which then propagates to the buccal side (Figure 18-5). The contralateral condyle is pushed into the glenoid fossa and fractured by compressive forces.

Traditionally, muscle attachments and their counteracting forces have been thought to play the most important role in determining the pattern, direction, and degree of displacement for mandibular fractures. The concept of a patient having a "favorable" or "unfavorable" fracture was developed, based upon the direction of a fracture line as viewed on radiographs in the horizontal or vertical plane (Figure 18-6). A *horizontally favorable fracture line* resists the upward displacing forces, such as the pull of the masseter and temporalis muscles on the proximal fragment when viewed in the horizontal plane. A *vertically favorable fracture line* resists the medial pull of the medial pterygoid on the proximal fragment when viewed in the vertical plane. Anteriorly, the combined action of the suprahyoid musculature on bilateral fractures can result in downward rotation of the distal segment (e.g., "bucket handle fracture"), cause displacement of the tongue musculature posteriorly, and cause potential airway obstruction.

Despite long-term acceptance, it is clear that the concept of "favorableness" is oversimplified. For example, it does not incorporate the effect of varying locations of occlusal load. While tension occurs along the upper margin (and compression at the lower margin) of a body fracture when the incisors are loaded, conditions may change dynamically as the bite target moves posteriorly approaching the fracture location.[56] Thus, in some instances, compression can occur at the superior border and tension at the inferior border of the fracture (Figure 18-7A). Similarly, in an isolated fracture at the mandibular symphysis, an incisor load acts as a pivot point around which the mandible rotates. The masseteric sling will produce rotation around the anteroposterior axis causing separation at the lower border of the fractured mandible (tension) that is greater than that of the superior border (compression) (see Figure 18-7B).

The presence or absence of posterior teeth was, for many years, thought to contribute further to fracture patterns.[57–59] It turns out, however, that this may be true only for the impacted third molar. Some evidence suggests that there is no correlation between posterior occlusion and the incidence or pattern of mandibular fracture, except when impacted third molars were present, when the incidence fractures of the angle were significantly more common.[41,60]

TREATMENT: HISTORICAL PERSPECTIVES

The Splint Age, 1866–1918

Closed treatment of mandibular fractures using dental splints without intermaxillary fixation (IMF) was the mainstay of treatment at the beginning of the 20th century. The Gunning

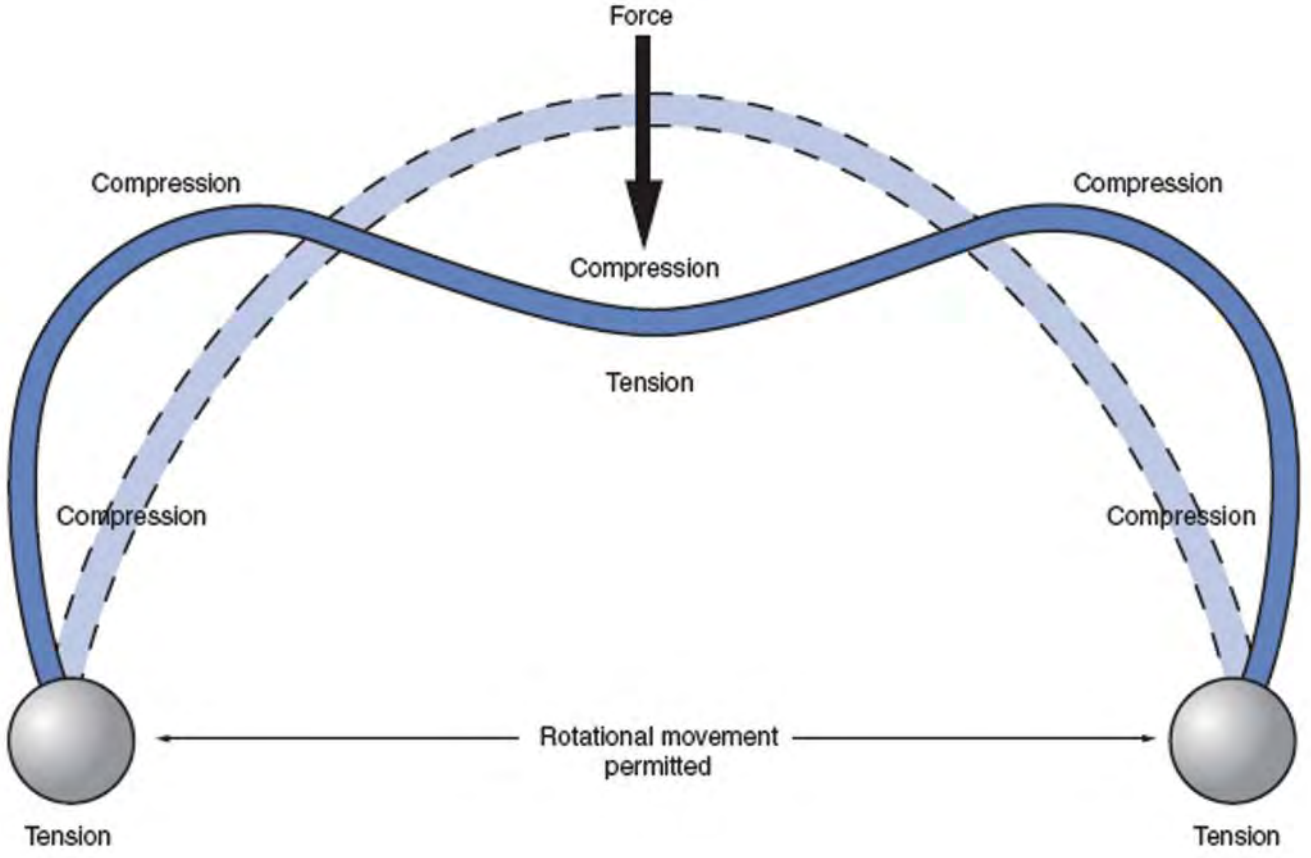


FIGURE 18-5. Force applied to the symphysis of the mandible, resulting in a compressive strain along the buccal aspect and tensile strain along the lingual surface. A fracture will occur on the lingual side of the mandible that then propagates to the buccal side. The contralateral condyle is pushed into the glenoid fossa and fractured by compressive forces. (From Chacon GE, Larsen PE. Principles of management of mandibular fractures. In Miloro M, Ghali GE, Larsen PE, Waite P, editors. Peterson's Principles of Oral and Maxillofacial Surgery. Hamilton, Ontario, Canada: BC Decker; 2004.)

Vulcanite dental splint was commonly in use since its introduction during the American Civil War; however, these were contraindicated in fractures posterior to the teeth and often resulted in open bite malocclusions.[61] Simon P. Hullihen, considered by some the father of American oral and maxillofacial surgery, described in the late 19th century a continuous dental splint that he had constructed for a case of resection of the alveolus, which he then used for treatment of interdental fractures in the body of the mandible. This could be made of metal, vulcanite, celluloid, or hard rubber and

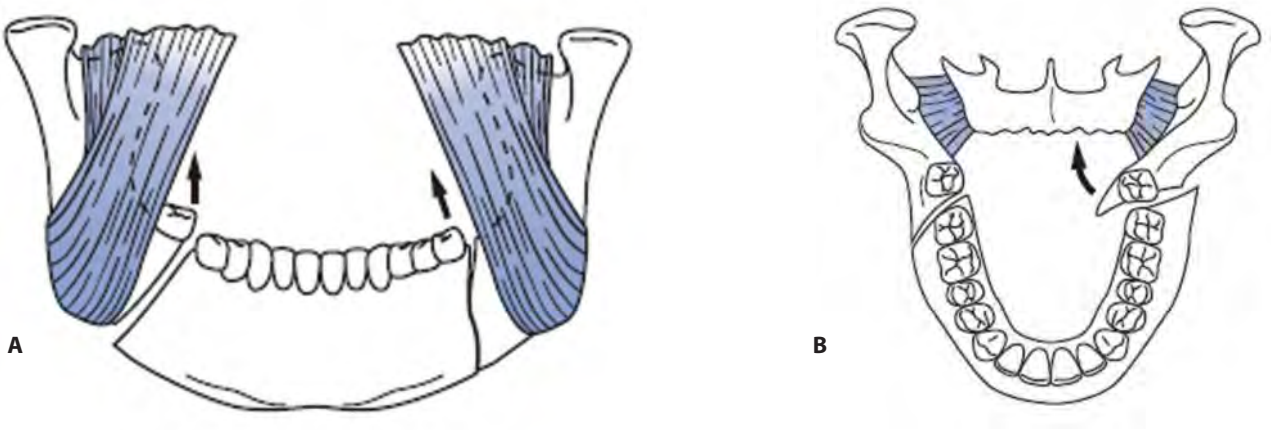


FIGURE 18-6. Muscle action and fracture pattern resulting in "favorable" and "unfavorable" mandibular fracture patterns. **A,** Horizontally unfavorable **(left)** and horizontally favorable **(right). B,** Vertically favorable **(left)** and vertically unfavorable **(right). (A** and **B,** From Chacon GE, Larsen PE. Principles of management of mandibular fractures. In Miloro M, Ghali GE, Larsen PE, Waite P, editors. Peterson's Principles of Oral and Maxillofacial Surgery. Hamilton, Ontario, Canada: BC Decker; 2004.)

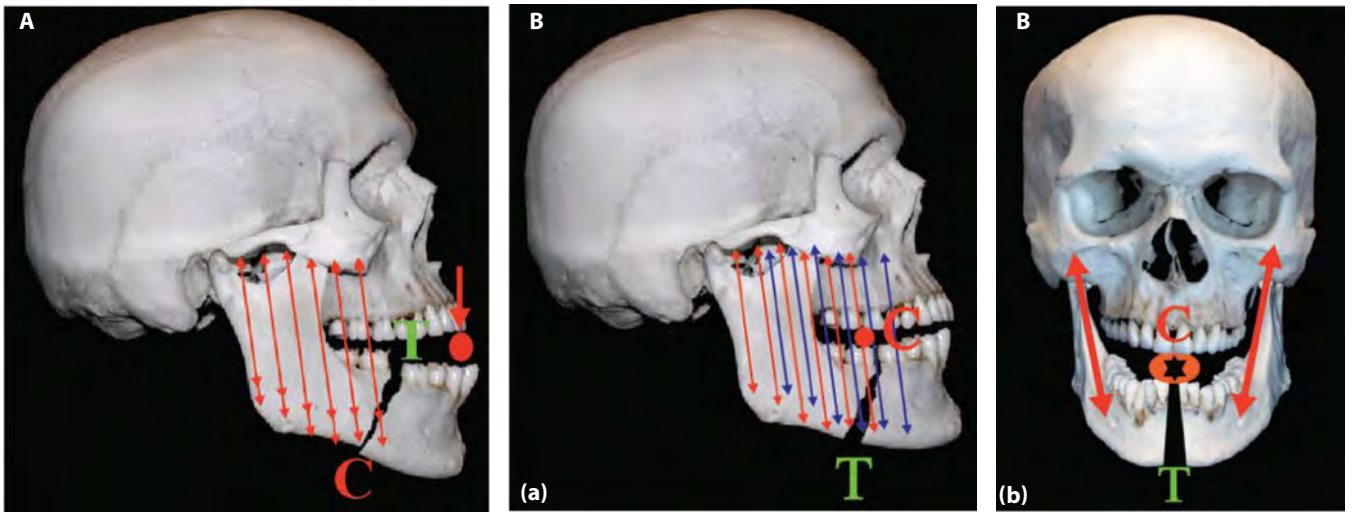


FIGURE 18-7. **A,** The combined action of the suprahyoid musculature and an incisal load on bilateral mandibular fractures that results in downward and backward rotation of the distal segment, displacement of tongue musculature posteriorly, and potential airway obstruction. Note the resultant zone of compression (C) at the inferior border and zone of tension (T) at the superior border, the knowledge of which is critical for understanding appropriate areas of osteosynthesis in the stabilization and fixation of mandibular angle/body fractures. **B,** Alternative forces that are dependent upon occlusal load. **(a)** Mandibular body/angle fracture with posterior occlusal load that may cause tensile stresses at the inferior border and compressive forces at the superior border. **(b)** Symphyseal fracture with incisal loading causing tension (T) at the inferior border and compression (C) at the superior border. This action may be heightened in cases of symphyseal fracture with bilateral condylar fractures, because the action of the lateral and medial pterygoid pulls the ramus segments in and down, causing splay at the inferior border. **(A** and **B,** From Rudderman RH, Mullen RL, Phillips JH. The biophysics of mandibular fracture: an evolution toward understanding. Plast Reconstr Surg 2008;121:596–607.)

was molded over a plaster or metal reproduction of the dentures. G.V. Black, the father of modern dentistry, is credited with using a vulcanite splint, combined with circumferential wiring of the bone (transmandibular wiring) to immobilize an edentulous mandible.[62] Other splints used for the treatment of maxillofacial injuries were described that were constructed over dies made from the casts of lower teeth, using metals such as silver or aluminum. The making of splints, however, generally took several days and often resulted in unfavorable outcomes for patients, such as nonunion or malunion. This resulted in a re-evaluation of dental splint fixation as a way of managing mandibular fractures.

The Wire Age, 1918–1968

Dr. Thomas Gilmer of Chicago was, in 1881,[5] the first modern surgeon to advocate what we now consider IMF for the treatment of complex maxillomandibular fractures. He did this through fixation using dental bands or wires fastened directly to the necks of teeth. The German surgeon Sauer[63] shortly afterward described the use of heavy round arch wire that was applied to the mandibular teeth only without absolute fixation in 1889. These same principles were embodied in the development of orthodontic clamp bands that were devised by Loher in Germany and Angle in America and later used by Schroder, Hauptmeyer, and others in orthodontic treatment.[64]

Following Gilmer's early description, Robert Ivy, an American Army oral and maxillofacial surgeon, modified a technique described by Colonel Robert Oliver[65] as well as Eby[66] in 1918. Ivy employed a 24-gauge wire inserted between the premolars of the teeth and connecting upper and lower eyelets with a third tie wire twisted tightly to allow for complete immobilization of the lower jaw (Figure 18-8).[64] This technique of using "Ivy loops" was often augmented with an alternative method based upon German innovation during the same period. The German technique involved a silver half-round arch wire that was molded to conform to the vestibular surfaces of teeth and then attached to the latter with wire ligatures; this was a precursor to the contemporary method of IMF popularized by Erich and Austin[67] (Figure 18-9). A multiple loop wiring technique was later devised by Colonel Roy A. Stout of the dental core in the U.S. Army (Figure 18-10).[68] A heavier form of continuous wiring was developed by Risdon of Toronto, Canada.[69]

By the end of World War II, more than 90% of all fractures of the mandible, regardless of their position, were successfully

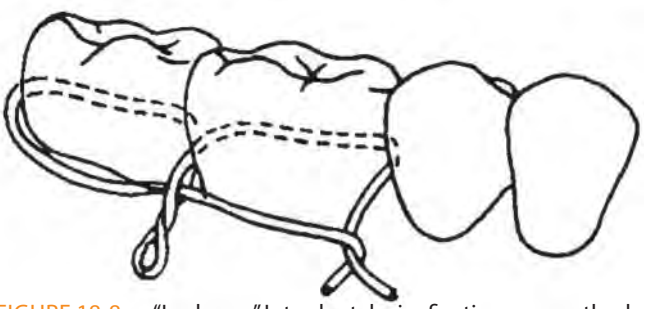


FIGURE 18-8. "Ivy loops." Interdental wire fixation as a method of providing intermaxillary fixation as described by Ivy.[64]

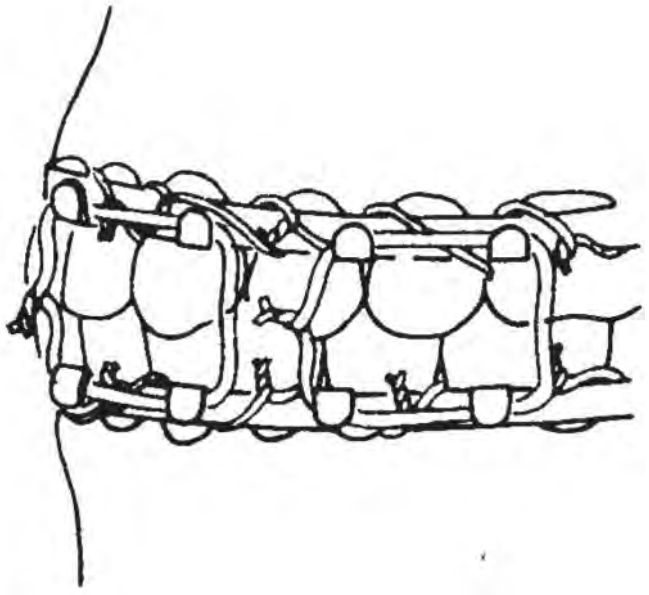


FIGURE 18-9. "Erich arch bars." Interdental wire fixation with a continuous arch wire and ligatures as described by Erich.[67]

treated utilizing closed methods and interdental wire fixation. In certain cases, however, Ivy and many others recommended additional methods for the treatment of what were known as "special cases." These included a fracture in the molar or premolar region with a long edentulous posterior fragment; fractures in the molar or premolar region with teeth in the posterior fragment but no opposing teeth in the upper jaw; fractures of the edentulous or almost edentulous mandible; fractures of the mandible with an edentulous upper jaw; comminuted fractures at the symphysis with loss of incisor teeth and bone; and fractures of the mandible complicated by fractures of the maxilla.[64] In these instances, wire was occasionally passed through a hole in the angle of the mandible via a transcervical approach and fixated to a plaster of Paris head cap in order to stabilize the proximal mandibular segment. This was often combined with vulcanite splints and bite splints placed in the posterior mandibular arch as advocated by Kazanjian and Converse.[70]

The control of edentulous fragments was always a difficult clinical scenario. Eventually, the splints, wires, and head caps used in World War I were replaced by external pin fixation. This method, introduced by Roger Anderson, an orthopedic surgeon, for fractures of the long bones, applied the use of Kirshner pins and a semiadjustable traction bar attached to locking units, as a precursor to the well-known Joe Hall Morris fixation appliance. By the middle of the 20th century, external pin fixation became commonly used for comminuted mandibular fractures (Figure 18-11).

Open reduction and internal fixation (ORIF) was considered hazardous during the first half of the 20th century. Even Robert Ivy, in his classic textbook on fractures of the jaws published in 1945, stated, "Sometimes, in a case of difficult reduction, the surgeon may be tempted to wire or plate the fragments, forgetting or ignoring the fact that nearly all mandibular fractures are compound into the mouth and are constantly bathed in the oral secretions. Infection occurs around

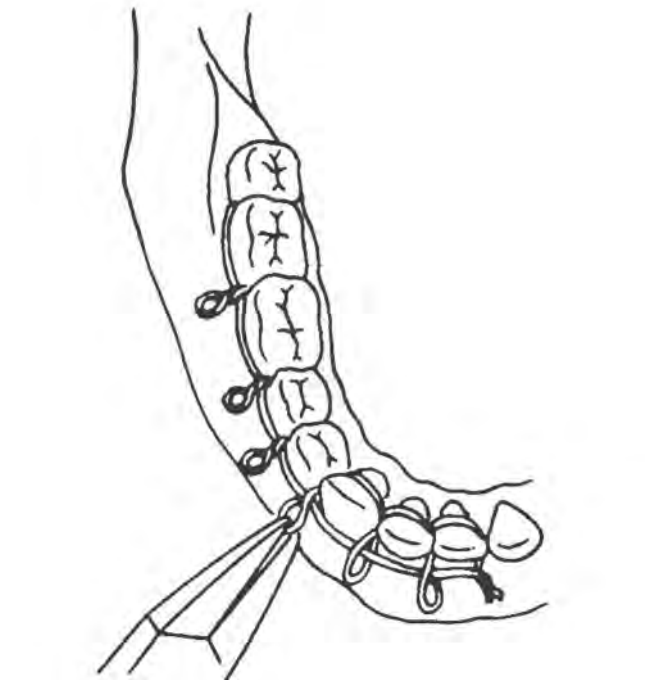


FIGURE 18-10. "Stoudt wires". Interdental wire fixation as a method of providing a continuous arch wire and intermaxillary fixation as described by Stoudt.[68]

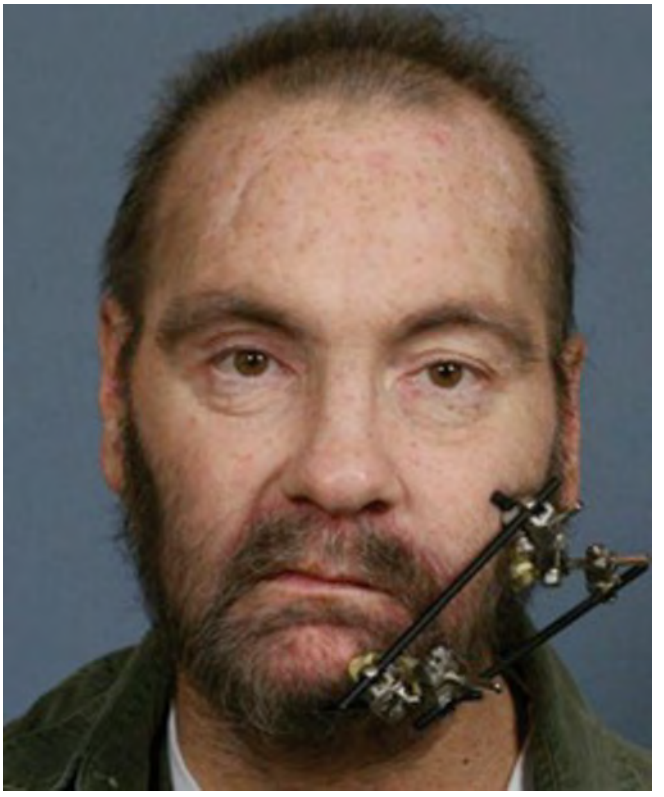


FIGURE 18-11. Contemporary "Joe Hall Morris" external fixator for the mandible. (From Synthes, Paoli, PA.)

the wire or the metal plate, inviting osteomyelitis with sequestrum formation and great delay in union. *The only justification for wiring or plating of the fragments is one of those rare cases of fractures in an edentulous person where the oral mucous membranes have not been broken and where there is consequently a chance of maintaining asepsis.* Even here a wire or plate usually gives insufficient stability."[64] Thus, dogmatic beliefs such as these were held through the majority of the 20th century, and it would take a group of European orthopedic surgeons to change them.

The Metal Plate Age, 1968 to Present and the Evolution of Modern Systems of Internal Fixation

Rigid Internal Fixation and the AO/ASIF: "The German School"

Modern systems of fixation that are today routinely used for the management of mandibular fractures have their origins in orthopedic surgery.[71] Hansmann, in 1886,[72] was the first to develop and present a procedure for the open reduction and fixation of long bone fracture using a plate and screw system. Whereas various surgeons in Europe utilized the principles of plate and screw fixation for fractures of the long bones beginning in the late 19th century, it was not until the mid-20th century that Martin Allgower, Maurice Muller, Robert Schneider, and Hans Willenegger founded the Arbeitsgemeinschfat fur Osteosynthesefragen/Association to the Study of Internal Fixation (AO/ASIF) in Switzerland and introduced the idea of axial compression into surgical routine for limb fractures.[73,74]

Luhr[75–80] applied these principles of ORIF using "compression osteosynthesis" to the mandible in 1968. These early plates were made of vitallium and contained eccentric holes and self-cutting screws with a conical head. Originally, these heavy plates were shaped to fit around the inferior border of the mandible and were placed in bicortical fashion to avoid the roots of teeth and the inferior alveolar nerve. When plates were placed at the band of compression alone, a diastasis was often created, leading surgeons to combine the use of "compression band plates" placed at the inferior border with arch bars fixed to the teeth along the line of tension. The concept of tension banding, which was adapted from Pauwels' work[81,82] on extremity fractures, was later applied to the mandible along the alveolar ridge where tensile stresses could be neutralized.

Vitallium was difficult to bend and was eventually replaced by stainless steel, which was in turn replaced with titanium. In 1969, the AO/ASIF introduced a dynamic compression plate (DCP,) for limb surgery (Figure 18-12). The concept of dynamic compression was then applied to the craniomaxillofacial skeleton by Spiessl in a series of publications beginning in 1969.[83–86] These plates were fixated to the lower border of the mandible using bicortical screws, and a superior border plate was secured to the area of the alveolar ridge using monocortical screws to neutralize the tensile stresses (Figure 18-13).

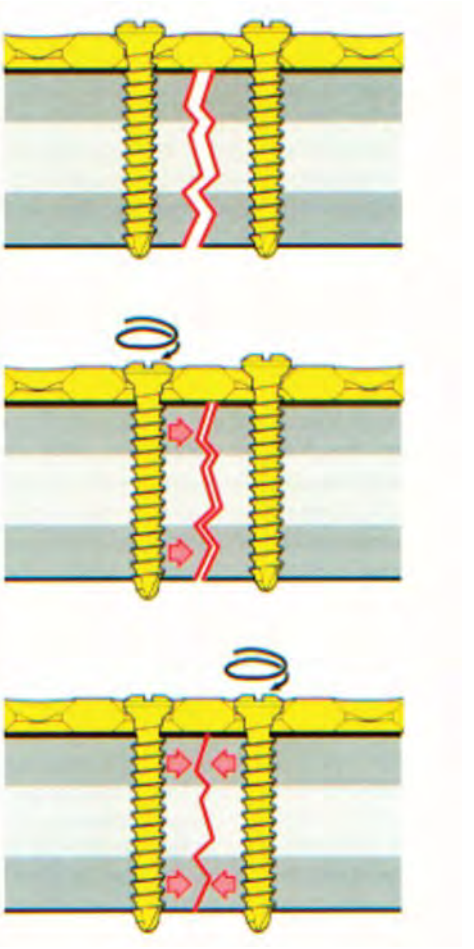


FIGURE 18-12. AO/ASIF dynamic compression plating. The screws are placed on the outside of the plate hole, which then "ramps down" as it is turned, thus causing compression of the fractured segments as the screws are tightened.[184]

The DCPs were later modified by Niederdellmann and Schilli[87,88] by creating two additional holes that were angled at 45 degrees to exert compression along the band of tension.

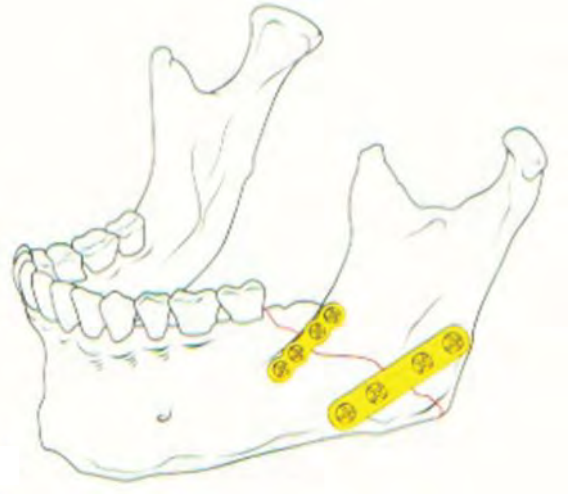


FIGURE 18-13. AO/ASIF technique for compression plating of the mandibular angle: a dynamic compression plate is placed at inferior border and second plate at superior border.[184]

These so called "eccentric dynamic compression plates" (EDCPs) led to compression at the alveolar ridge, the site at which the tensile stresses occur.

Compression osteosynthesis was examined in a histologic animal study by Reuther[89] in 1980, in which he found ossification patterns similar to primary fracture healing. By the 1970s, however, it was apparent that compression was not always advisable for patients with comminuted fractures, infected fractures, or segmental mandibular defects. This led to the development of large "reconstruction plates" that allowed more rigidity between the plate and the bone fragments. Referred to as "load-bearing plates," Bowerman and Conroy[901] first applied these reconstruction plates to bridge the gaps of complex or comminuted fractures in 1976.

In 1980, the first commercially available mandibular reconstruction plate system made of titanium was introduced by Raveh and coworkers.[92–96] Termed the *THORP* (titanium-coated hollow screw and reconstruction plates) *system*, the head of the actual screw was hollow and contained threads into which a smaller screw was inserted. This allowed the screw to be compressed into the plate, leading to stability independent of contact between the plate and the screw. By providing rigid, load-bearing fixation, the concept of an "internal external fixator" was born and the era of large bridging plates used to span segmental defects of the mandible was founded.

Although these large reconstruction plates were effective for bridging continuity defects of the mandible, their size and rigidity were again impediments to their use for routine or even comminuted fractures of the mandible. Thus, a similar but smaller reconstruction plate was developed in the mid-1990s by the AO/ASIF, called the *UniLOCK 2.4* (universal locking plate). In this system, the locking mechanism between the plate and the screws was obtained by placing threads on the screw itself, which then locked into the inner thread of the plate (Figure 18-14). Contemporary mandibular reconstruction plates use variations of the UniLOCK system over the older THORP system and are widely accepted for providing load-bearing fixation of the mandible.[97,98] The concept of utilizing threaded screws to lock into plate mechanisms has also been applied to miniplate fixation.

The technique of utilizing DCPs in the mandible has, however, undergone a period of re-evaluation. There appears to be no significant advantage of compression plating in the mandible, and in fact, it may contribute to the development of diastasis if not utilized properly. Many maxillofacial

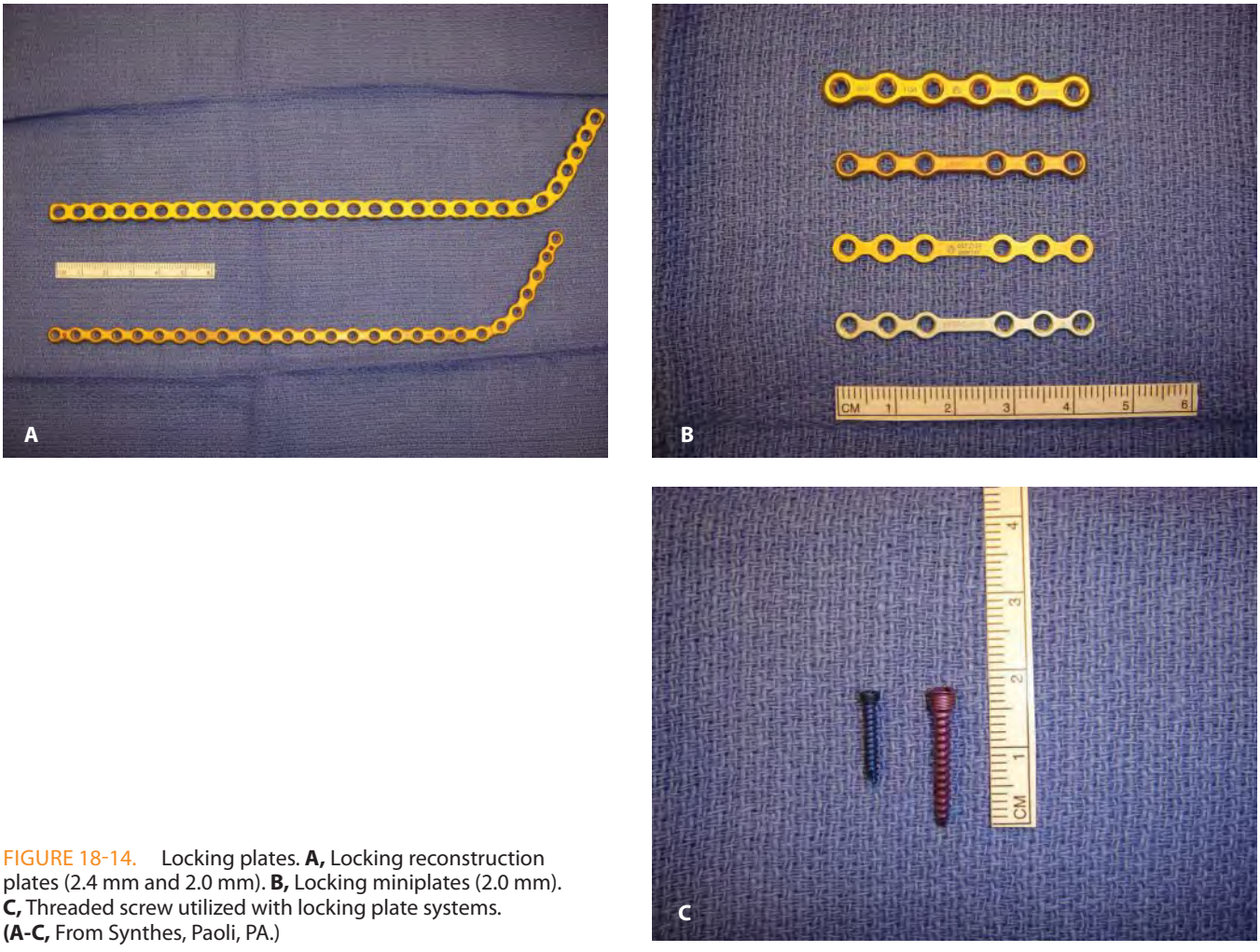


FIGURE 18-14. Locking plates. **A,** Locking reconstruction plates (2.4 mm and 2.0 mm). **B,** Locking miniplates (2.0 mm). **C,** Threaded screw utilized with locking plate systems. **(A-C,** From Synthes, Paoli, PA.)

surgeons have abandoned the use of compression plating in favor of miniplate fixation and/or the use of locking plates and screws.

The AO/ASIF principles using rigid internal fixation as applied to the bands of compression and tension have withstood the test of time. They have, however, been complemented by a parallel school of mandibular fracture fixation based on miniplate osteosynthesis.

Functional Fixation, Michelet and Champy: "The French School"

Shortly after Luhr and Spiessl developed and popularized the concept of compression osteosynthesis in German-speaking European countries, French surgeons began to develop their own techniques using miniplates applied along ideal lines of osteosynthesis, applying monocortical screws to avoid injury to tooth roots (Figure 18-15). Michelet and colleagues[99] first applied vitallium miniplates to a series of mandibular fractures in the early 1970s and reported favorable clinical outcomes.

Champy and associates[100–106] later modified Michelet's technique and went a step further by providing a biomechanical basis for treatment. A series of experimental studies, performed in the mid-1970s, defined the ideal lines of osteosynthesis as the line of maximum tensile stress from the oblique ridge along the base of the alveolus to the mental foramen. Champy's group hypothesized that a single miniplate, placed at the ideal line of osteosynthesis, designed to cope with the tensile stresses, should be adequate to stabilize mandibular fractures and achieve predictable union. Early clinical studies were performed using stainless steel miniplates, but these were later changed to titanium.

DIAGNOSIS OF MANDIBLE FRACTURES

Diagnosis of the patient with facial fractures consists of assessment of the occlusion, palpation of the facial contours, bimanual manipulation of the mandible to assess for fracture mobility, assessment of symmetry and deviation upon mouth opening, maximal interincisal opening, and evaluation of the dentition for avulsion of teeth and/or dentoalveolar fractures. In addition, a comprehensive cranial nerve evaluation should be performed, with particular emphasis placed on evaluation of neurosensory disturbance in the distribution of the inferior alveolar nerve/mental nerve. The presence or absence of altered sensation in the distribution of the inferior alveolar/mental nerve should be documented.

The radiographic evaluation of a patient with suspected facial injury/mandibular fracture should consist of imaging in at least two planes. In the past, evaluation of the mandible



FIGURE 18-15. Miniplate fixation. **A,** Champy's ideal lines of osteosynthesis. **B,** Contemporary commercially available mandibular plating set with 2.0-mm nonlocking and locking plates and screws. **C,** 2.0-mm miniplates. (**A-C,** From Stryker, Kalamazoo, MI.)

with plain films in four views (right and left oblique, reverse Townes and posteroanterior [PA] mandible view) was advocated. Today, a panoramic radiograph, which allows accurate assessment of the involvement and condition of teeth and their relationship to the adjacent fractures, should be obtained whenever possible. A panoramic radiograph alone, however, is not adequate and must be combined with a reverse Townes and/or PA view to achieve a second dimensional image to allow a more complete evaluation of the mandibular condyles. Alternatively, or in addition to these films, a computed tomography (CT) scan will provide the most complete injury assessment and three-dimensional view of the fractures, particularly in cases of high-energy or severe comminution. In current oral and maxillofacial surgical practice, all patients will usually obtain at least a panoramic radiograph, with or without a CT scan. If the CT scan is not available or not obtained, a reverse Townes view is also added to the panoramic radiograph. In general, a CT scan is routinely obtained in most cases of facial injuries for a thorough and comprehensive assessment of the extent of the head and neck injuries.

PERIOPERATIVE MANAGEMENT

Displaced fractures that extend through the tooth-bearing region of the mandibular arch often benefit from temporary stabilization of the fracture before ORIF. So-called bridle wires can be placed in the emergency department at the time of admission and will aid in hemorrhage control and airway stabilization (see Figure 18-22A). If utilized, bridle wires should be placed through the second tooth on either side of the fracture to prevent avulsion of a mobile tooth or teeth fragments. The patient is placed on a full liquid diet and scheduled for treatment expeditiously.

The administration of antibiotics should begin as soon as possible for all mandibular fractures that extend through the tooth-bearing regions. Preoperative antibiotics have been shown to significantly reduce the incidence of postoperative infections.[107–109] There is conflicting evidence for the use of postoperative antibiotics. Neither of the two most recent prospective, randomized, clinical trials could prove a benefit to the postoperative administration of antibiotics in patients who underwent ORIF of mandibular fractures.[110,111] Penicillin is the antibiotic of choice, if prescribed, unless an allergy is present. Many surgeons also prescribe chlorhexidine gluconate oral rinse before and after surgery until all wounds/incisions are closed.

OPERATIVE MANAGEMENT

The primary goals of treating mandibular fractures are (1) to restore the patient to form and function by returning the patient to the preinjury occlusion and achieving anatomic reduction when possible and (2) to achieve osseous union predictably. The appropriate technique to achieve these goals varies based upon location of the fracture, the energy of the fracture, and whether or not there is load-sharing potential. The use of interdental fixation (arch bars, interdental wires, or IMF screws), the approach (open or closed; transoral or transcervical), hardware selection (load-bearing or load-sharing), the management of teeth in the line of fracture (to retain or remove), and postoperative rehabilitation (period of IMF vs. immediate function) also depends upon the surgeon's preference or training, location of fracture, and the presence or absence of concomitant fractures.

Closed Treatment with MMF versus ORIF

The decision to treat a patient with a mandibular fracture by closed reduction using MMF or by ORIF remains modestly controversial. Few studies have compared the outcomes of the two treatment modalities and some authors have suggested that MMF offers considerable cost savings over ORIF.[112,113] Publication of the first prospective, randomized, controlled clinical trial studying conventional MMF versus ORIF to treat mandibular fractures was published in 2008 and again ushered in a controversy regarding the most predictable and cost-effective method of treatment of mandibular fractures.[114] In this study by Shetty and coworkers,[114] a total of 336 patients were randomly assigned to receive either MMF or ORIF and followed prospectively for 12 months. The study failed to show a clear overall benefit of ORIF of moderately displaced fractures over MMF. A major limitation of the study, however, was that participating clinicians overruled the randomized treatment assignment in a substantial number of cases and many patients were lost to follow-up. Other investigators, such as Hoffman and colleagues,[115] Thaller and associates,[116] and Dodson and Pfeffle,[117] have argued that ORIF may in fact be the more cost-effective approach for treating mandible fractures if the costs of treating potential complications are considered. Experience dictates that MMF is certainly reliable, safe, and cost-effective for some patients. There is no doubt, however, that ORIF remains an important treatment option in complex injuries. Compelling data describing predictors of successful MMF use are lacking; thus, determination of the best treatment method should be made after careful assessment of the various clinical variables and informed consent of the patient.

Interdental Wire Fixation as an Aid to ORIF

The use of interdental wire ligatures or arch bars for intraoperative MMF has been a time-honored and reliable technique to aid in the reduction and stabilization of mandibular fractures before the application of plate fixation. The problem with this technique, however, is that it is time-consuming and there is a significant risk of skin puncture, possibly resulting in disease transmission to the surgeon.[118] IMF screws have been introduced as an alternative to interdental wire fixation in order to decrease the risk of disease transmission (Figure 18-16).[119] Pigadas and coworkers,[120] published the results of a prospective, randomized clinical trial on cross-infection control in maxillofacial trauma surgery and found IMF screws to



FIGURE 18-16. Intermaxillary fixation (IMF) screws. **A,** Preoperative appearance of a patient with displaced mandibular parasymphysis fracture. **B,** IMF screws are applied between the cuspid and the premolar teeth (depends on the location of fracture) with self-drilling screws, IMF is applied with wire or elastic bands. **C,** Erich arch bars are applied to a patient with multiple fractures or concomitant midfacial fractures.

be a safer alternative to wiring methods with significant reduction in glove perforation rates and a decrease in operative time. Other authors have attempted to minimize these risks and improve operating room efficiency even more by omitting wire/arch bar MMF altogether, in favor of manual reduction of the fractures alone.[121–123] Whereas this approach does not appear to have gained much traction among North American oral and maxillofacial surgeons, recent evidence suggests that many experienced surgeons from other countries and other surgical specialties do not, in fact, routinely use arch bars or interdental wire fixation to aid in the reduction, stabilization, and fixation of mandibular fractures.[124] In general, Erich arch bars, interdental "Stout wires," and manual reduction alone all have efficacy for achieving favorable bony union in the ORIF of selected mandibular fractures.[125]

There is no question that Erich arch bars are preferable for the treatment of comminuted fractures and those mandibular fractures with concomitant midfacial fractures. However, there are many isolated mandibular fractures that are minimally displaced or favorably displaced for which the application of arch bars, with their associated risk of skin puncture and added operative time, may not be warranted. In these cases, the use of interdental wire fixation or manual reduction alone may be considered. Most surgeons now use IMF screws in most cases of isolated, noncomminuted, dentate fractures.

Surgical Approach

Most fractures of the mandible today are treated by means of ORIF. The principles of reduction, stabilization, and fixation, in that order, should be followed when considering surgical treatment. The vast majority of mandibular fractures may be approached utilizing intraoral incisions (Figure 18-17A-C) However, comminuted fractures of the ramus of the mandible, gunshot wounds and atrophic mandibular fractures are relatively common indications for the use of transcervical or transfacial incisions (see Figure 18-17D). Mandibular condyle fractures are a separate entity and are reviewed in the next chapter.

Hardware Selection

A fixation system will provide either absolute (rigid) stability or functional stability. *Rigid stability* occurs when no movement whatsoever occurs across the fracture gap. Rigid stability is an ideal therapeutic principle and probably no fixation system is able to provide absolute stability in all dimensions in a system as dynamic as the mandible. *Functional stability* occurs when movement is possible across the fracture gap but is balanced by external forces and remains within the limits that allow for the fracture to progress to union. Excessive mobility at a fracture site will lead to bone resorption and fibrous tissue ingrowth. When mobility is present, any internal device will promote bone resorption and infection.

Modern internal fixation systems provide functional stability in one of two ways: (1) *load-sharing stability* is provided by a fixation system in conjunction with stabilizing forces provided by anatomic abutment of noncomminuted fracture segments (Figure 18-18) and (2) *load-bearing stability* is functional stability provided solely by the fixation system (Figure 18-19). In contemporary fracture plate modules, load-bearing fixation is provided by locking reconstruction plates, generally 2.3, 2.4, and 2.7 mm in diameter, whereas load-sharing fixation is generally 2.0 mm in diameter. These principles of rigid internal fixation, as espoused by the AO/ASIF, and championed in the craniomaxillofacial skeleton by Spiessl, Schilli and Prein, have been the gold standard by which all other methods of craniomaxillofacial fixation have been compared.[86]

In contrast to the concepts of stability popularized by the AO/ASIF, Michelet and Champy advocated "semi-rigid fixation," based upon ideal lines of osteosynthesis (Figure 18-20). These lines are based upon the concept that only tensile stresses are harmful to fracture healing. Thus, fractures located proximal to the first premolar may be safely stabilized with a single miniplate placed in the midbody position (2.0 mm). Fractures anterior to the first premolar should be stabilized with two plates (a tension band and a compression band) separated by 4 to 5 mm and

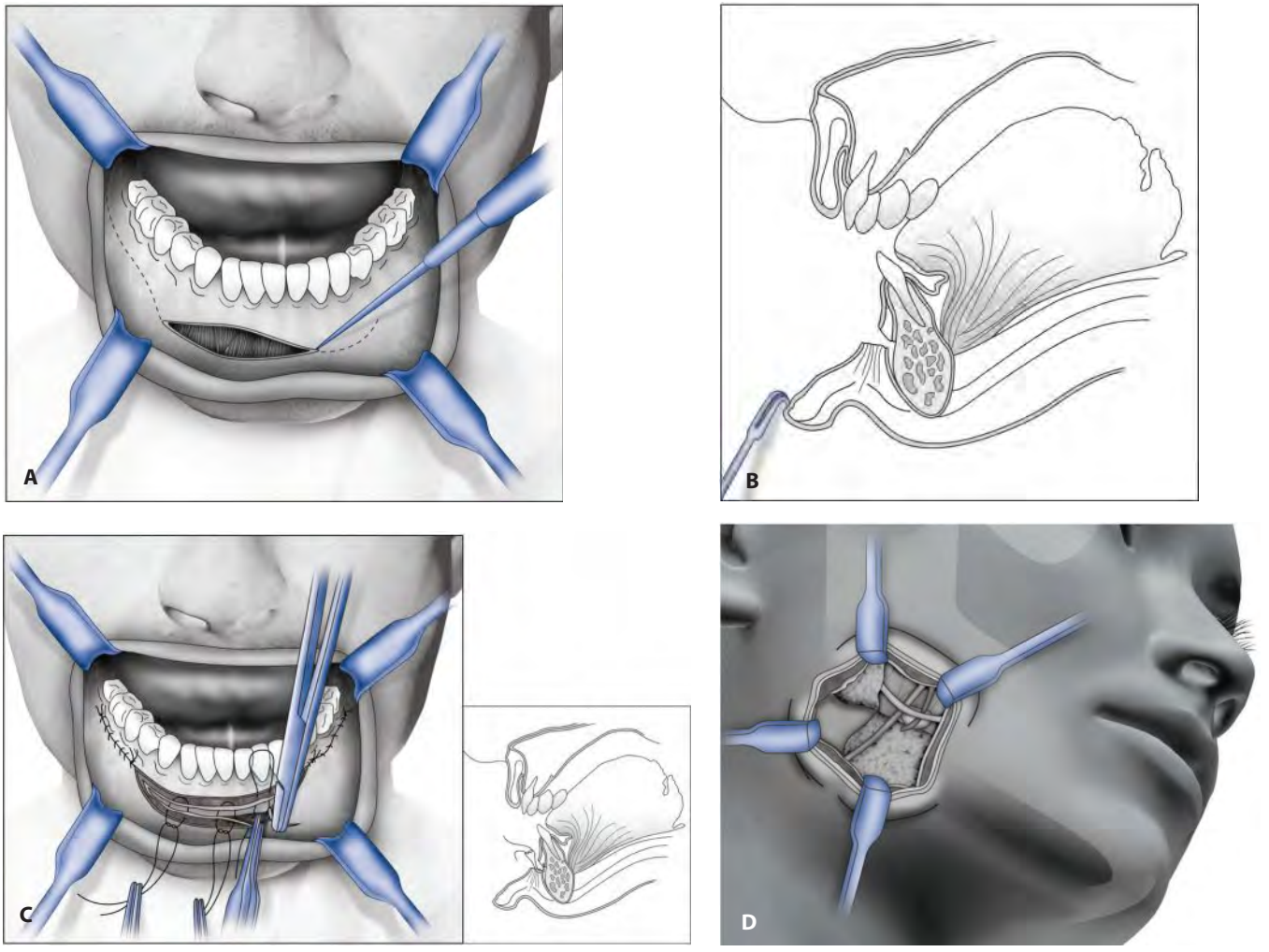


FIGURE 18-17. Approaches for most mandibular fractures. **A,** Transoral approach. Incision is made with a knife or bovie cautery and carried through the mucosa, mentalis muscle, and periosteum. Care is taken to identify, preserve, and protect the mental nerves exiting the mental foramena on both sides. **B,** Sagittal view of transoral approach depicts division of the mentalis muscles and skeletonization of the anterior mandible. **C,** Two-layered closure is achieved with 4-0 Vicryl sutures to reapproximate the mentalis muscle and 3-0 chromic to reapproximate the mucosa. **D,** Submandibular incision, also termed a "Risdon" approach. An incision is made 2 finger breadths beneath the angle of the mandible and extended through the skin, subcutaneous tissue, and platysma. The superficial layer of the deep cervical fascia is incised below the marginal mandibular branch of the facial nerve, and dissection to the pterygomasseteric sling and mandible proceeds beneath this layer. The posterior facial vein may be used as a landmark to ensure preservation of the facial nerve (dissection below the posterior facial vein). **(A-D,** From Ellis E III, Zide MF. Surgical approaches to the facial skeleton. Baltimore, MD Williams & Wilkins; 1995; pp. 113, 114, 120.)

generally placed on either side of the mental nerve (see Figure 18-20).

As noted previously, the application of adequate fixation is important for the successful treatment of mandibular fractures. *Rigid internal fixation* is a term that has been applied to the application of hardware to prevent movement across the fracture line when normal functional forces are applied. Rigid fixation permits primary bone healing without callus formation and immediate return to full function. Examples of this type of fixation include large commercially available locking and nonlocking reconstruction bone plates, the application of multiple plates at a fracture site, or lag screw fixation (Figure 18-21).

It turns out that functionally stable fixation is all that is necessary for successful healing of most fractures. Micromotion occurs in this paradigm that permits healing of the fracture by secondary bone healing, with formation of a callus, and no need for IMF. An example of nonrigid fixation is the use of a single miniplate at the angle of the mandible as described by Champy.[100] Thus, functionally stable fixation may also result in osseous healing and achieve the predictable results required in contemporary centers.

As discussed earlier, fixation requirements are considered by the ability of the host bone to share some of the functional loads. *Load-bearing fixation* is of sufficient strength to resist the functional masticatory forces during the healing phase

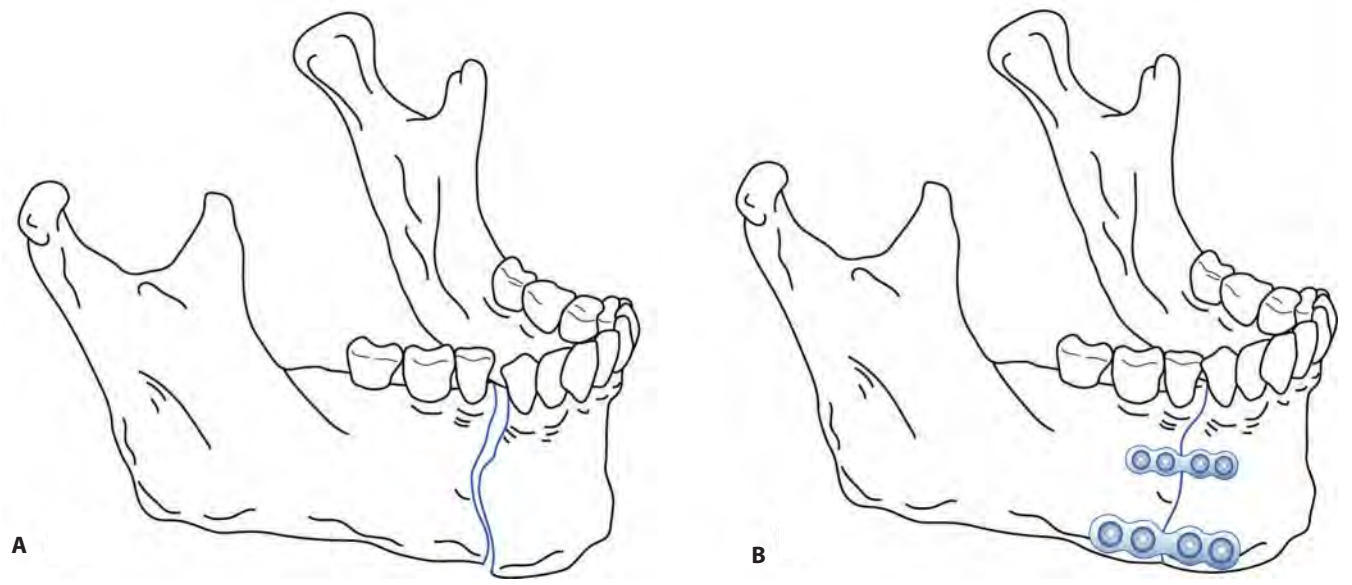


FIGURE 18-18. Load-sharing fixation. **A,** Linear fracture of the right parasymphysis. **B,** Stability is provided by a fixation system in conjunction with stabilizing forces provided by anatomic abutment of noncomminuted fracture segments. Fixation is applied to the inferior border (zone of compression) and superior border (zone of tension).

and the host bone fracture sites share none or little of the functional load. In contrast, *load-sharing fixation* refers to a scheme whereby the functional load is shared between the hardware and the bone along the fracture site. The indications for providing load-bearing fixation are those fractures with comminuted segments, atrophic mandibular fractures, and fractures with avulsed or missing segments. Commercially available plates and screws that provide load-bearing fixation are generally 2.3- or 2.4-mm locking reconstruction plates. Load-sharing fixation is indicated in cases in which no comminution or bone defects are present and when intact bone cortices are opposed to one another after fracture reduction. The majority of mandibular fractures can be adequately treated with load-sharing fixation.

Teeth in the Line of Mandible Fractures

The management of teeth in the line of fracture has been a source of controversy in the literature for decades.[126] Various authors have attempted to use specific criteria such as tooth mobility, interference with fracture reduction, pulpal pathology, and location of the fracture to determine whether or not

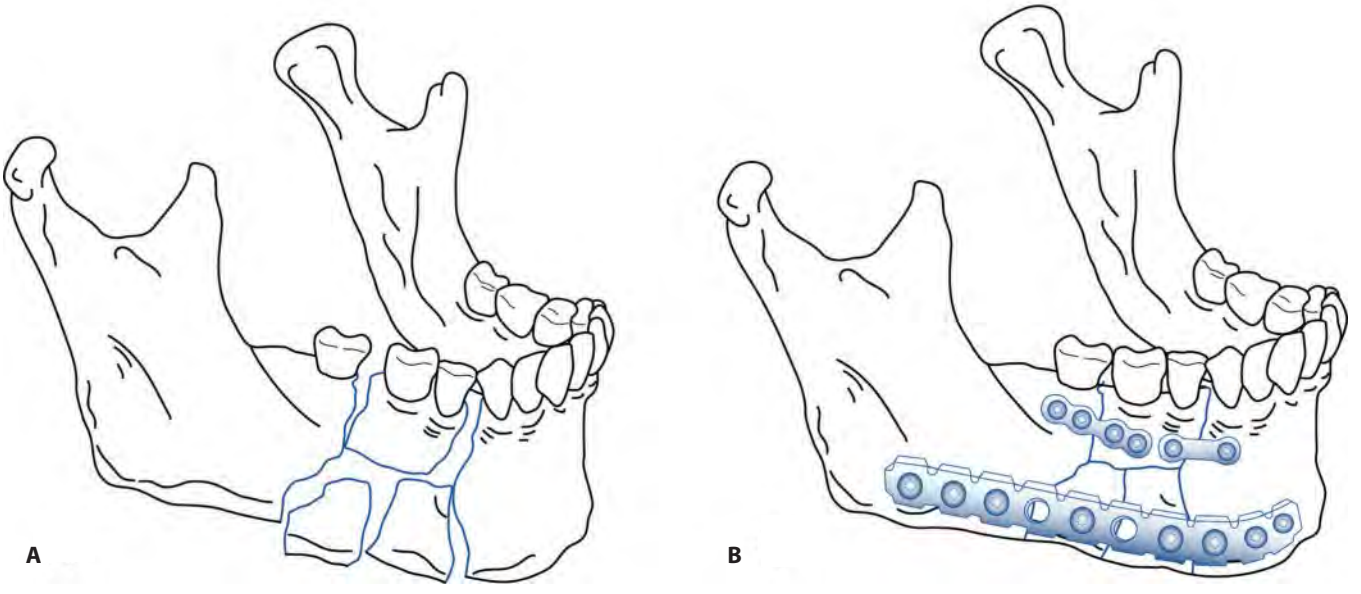


FIGURE 18-19. Load-bearing fixation. **A,** Comminuted fracture of the right mandibular body. **B,** Functional stability is provided solely by the fixation system. Contemporary fixation systems involve a 2.3- or 2.4-mm locking reconstruction plate at the inferior border combined with secondary fixation of comminuted segments at the superior border.

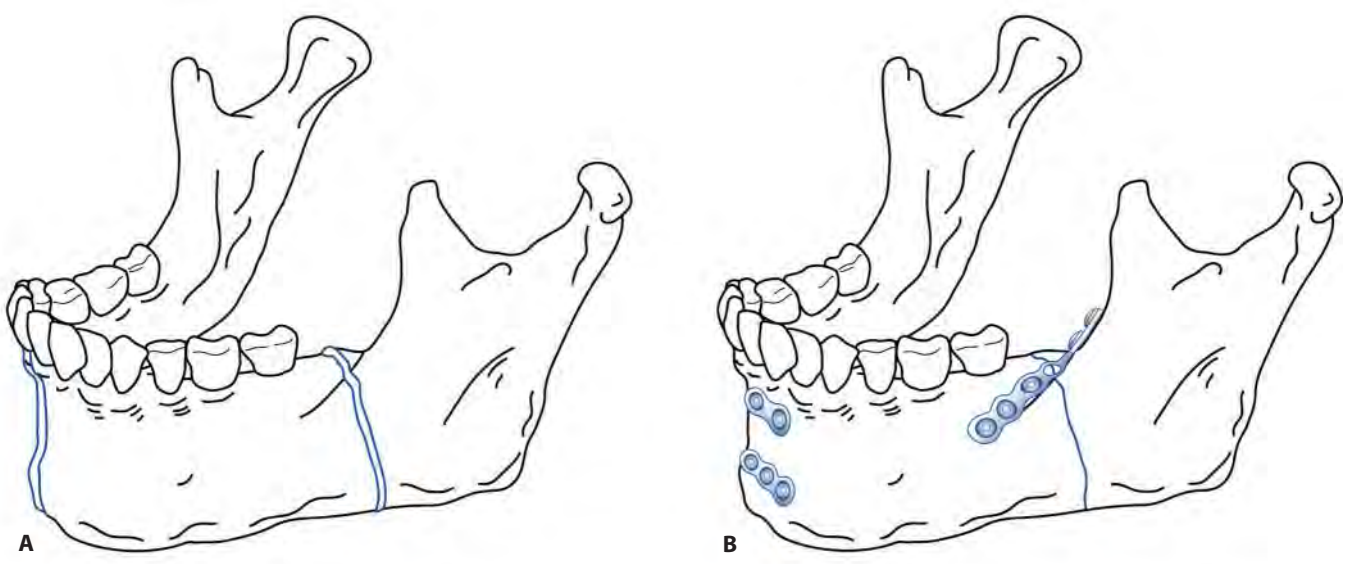


FIGURE 18-20. Functional stabilization along Champy's ideal lines of osteosynthesis. Fractures located proximal to the first premolar may be safely stabilized with a single miniplate placed in the midbody position (2.0 mm). Fractures anterior to the first premolar should be stabilized with two plates (a tension band and compression band) separated by 4 to 5 mm and generally placed on either side of the mental nerve.

the tooth should be removed.[126–133] The preponderance of evidence suggests that teeth in the line of fracture (including third molars) may be retained providing that they do not interfere with favorable reduction, stabilization, and fixation of the fracture and are not grossly mobile or infected. Although there are no universally agreed upon criteria, *many surgeons retain all teeth in the line of fracture unless they are grossly mobile, infected, or inhibit fracture reduction* (Figure 18-22). This approach has yielded a complication rate similar to other reported complication rates, regardless of the presence or absence of teeth in the line of fracture.[125]

Isolated Mandibular Symphysis, Parasymphysis, and Body Fractures

Noncomminuted fractures within the tooth-bearing region of the mandible are readily approached via a transoral, vestibular incision. Care is taken to avoid injury to the branches of the mental nerve during the surgical approach, and typically, the mental nerve is mobilized by incising the periosteal envelope and exposing the fascicles so that the nerve may be retracted and protected during reduction, stabilization, and fixation of the fracture.

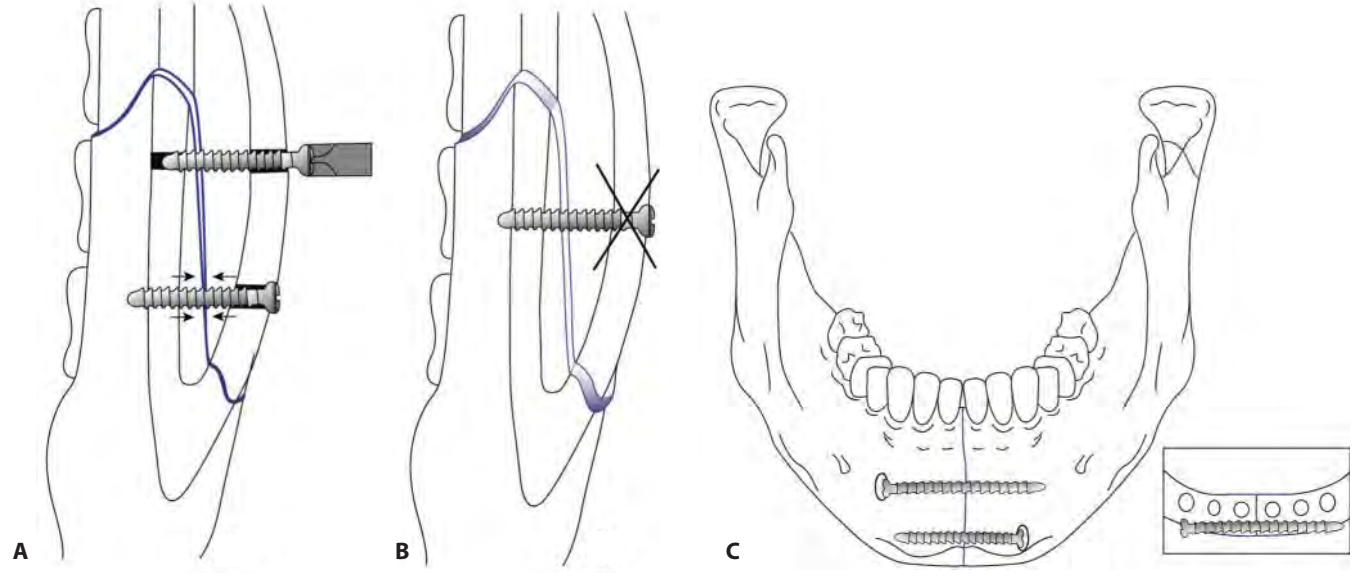


FIGURE 18-21. Lag screw fixation. **A,** A larger bur hole is created in the outer cortex and carried to the inner cortex, at which time a smaller drill size is used on the inner cortex. **B,** As the screw engages the inner cortex, it compresses the outer and inner cortex together, creating a rigid construct. **C,** Lag screw fixation applied to the mandibular symphysis.

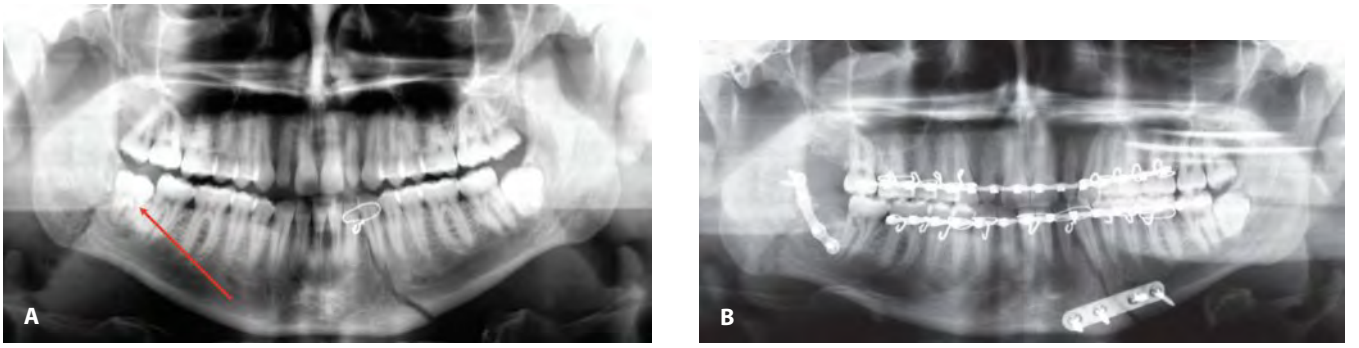


FIGURE 18-22. Teeth in the line of fracture. **A,** Preoperative panoramic radiograph demonstrates a partially erupted third molar in the line of fracture at the right angle. The tooth is loose and interfering with anatomic reduction. **B,** The third molar is removed and the angle fracture stabilized with a superior border plate. The parasymphysis is stabilized with a compression band dynamic compression plate (DCP) and arch bars at the tension band. Teeth in the line of fracture are retained unless they are mobile, infected, or interfering with anatomic reduction.

With the patient in the IMF utilizing arch bars, interdental wires, IMF screws, or stabilized by manual reduction, a bone reduction forceps is applied to reduce the osseous segments in cases of linear fracture. Medial pressure is applied to the angles of the mandible in cases of comminution or when the mandibular condyles are also involved, to prevent facial widening. The plate is slightly overbent and care is taken to ensure that no diastasis exists buccally or lingually (Figure 18-23). Holes are drilled through the outer cortex to engage the reduction forceps and the bone is compressed tightly. Evaluation of the occlusion for the presence of crossbite, mandibular angle flaring, and accurate positioning of

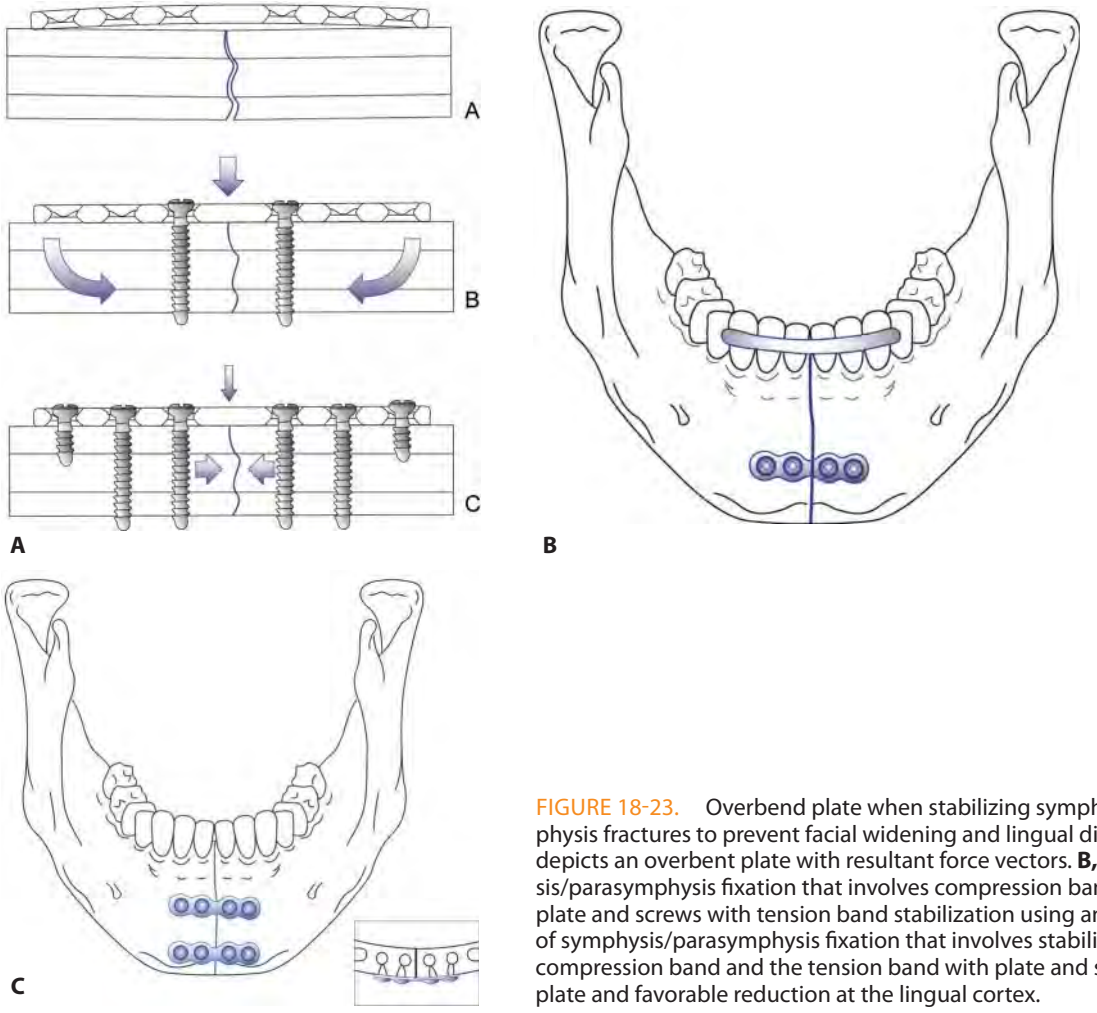


FIGURE 18-23. Overbend plate when stabilizing symphysis and parasymphysis fractures to prevent facial widening and lingual diastasis. **A,** Diagram depicts an overbent plate with resultant force vectors. **B,** Method of symphysis/parasymphysis fixation that involves compression band stabilization with plate and screws with tension band stabilization using an arch bar. **C,** Method of symphysis/parasymphysis fixation that involves stabilization of both the compression band and the tension band with plate and screws. Note overbent plate and favorable reduction at the lingual cortex.

occlusal wear facets is important at this point in the procedure. Preexisting anterior open bites can be challenging to assess, but typically the lack of wear facets and the presence of mamelons in the incisor teeth will indicate that indeed perhaps no anterior occlusion was present. It should not, however, be assumed that patients with few wear facets have an anterior open bite.

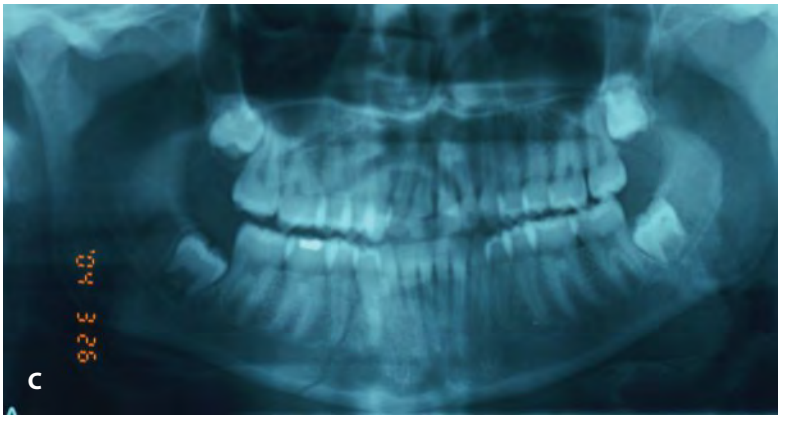
Once reduced and stabilized with a reduction forceps, rigid internal fixation is applied. Both the bands of tension and compression must be stabilized. If an arch bar is utilized, this can act as a tension band and a single 2.0- or 2.4-mm locking or nonlocking titanium plate placed at the inferior border of the mandible will generally adequately stabilize these fractures at the symphysis, parasymphysis, or body (see Figure 18-23B). If arch bars are not utilized, however, additional fixation must be applied to the tension band as well (see Figure 18-23C). The tension band plate should be placed first to prevent splaying of the teeth along the fracture segment and should be placed in monocortical fashion. The inferior, compression band plate should be placed in bicortical fashion using copious irrigation. It is appropriate to provide two points of fixation at both the tension band (2.0-mm plates and screws placed in monocortical fashion) and the inferior border compression band (2.0-mm plates and screws placed in bicortical fashion) (Figure 18-24).

Careful attention should be paid to cases of bilateral mandibular fractures involving the symphysis or parasymphysis. Lateral muscular forces will cause widening of the mandible by splaying the angles outward and creating a defect on the lingual aspect of the anteriormost fracture. Reduction of these fractures involves applying manual medial forces at the angles, stabilizing the anterior fracture with a reduction forcep, and overbending the plates that are used to provide rigid fixation at the symphysis or parasymphysis. The use of a locking reconstruction plate for this purpose is recommended. It may be desirable to also treat one or both condyles with ORIF to avoid lateral displacement and resultant facial widening. Failure to adequately account for the lateral muscular forces will result in a gap along the lingual cortex and facial/mandibular widening (Figure 18-25). This discrepancy will then be cumulative, because the upper facial skeleton is built upon a widened mandible.

The primary anatomic concerns for fractures in the body and symphysis region of the mandible are related to the







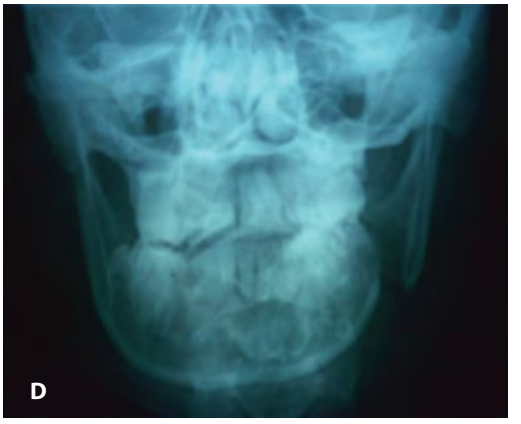
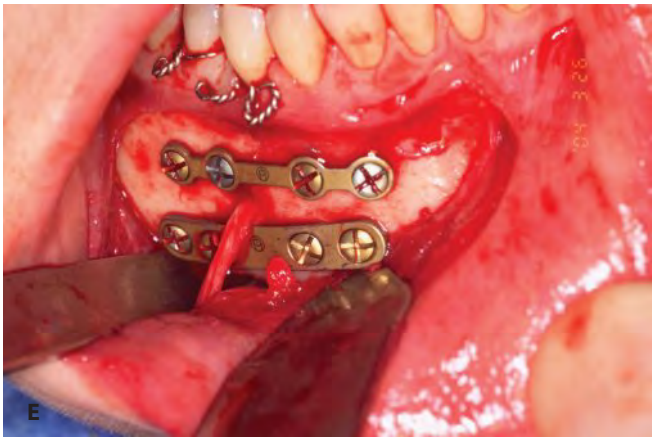


FIGURE 18-24. A 17-year-old female with bilateral mandibular fracture involving the right parasymphysis and left angle. **A,** Preoperative appearance. **B,** Preoperative occlusion. **C,** Preoperative panoramic radiograph. Note that the left angle fracture is poorly visualized. **D,** Preoperative posteroanterior (PA) radiograph. Note that the left angle fracture is now readily apparent. *(continued)*







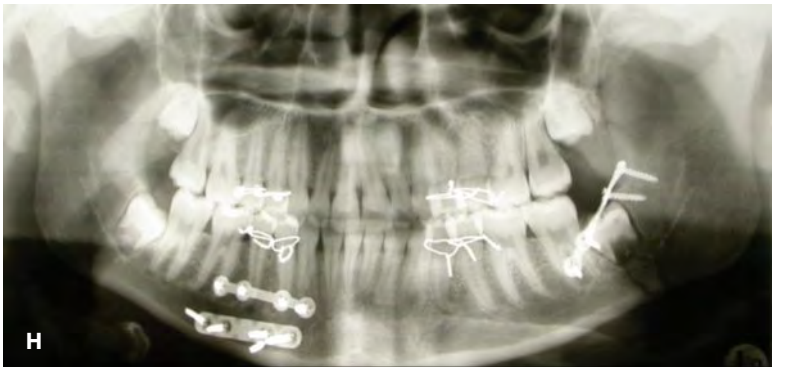






FIGURE 18-24. *(continued)* **E,** Intraoperative view demonstrates stabilization with 2.0-mm plates and screws placed at the compression band (inferior border) and tension band (superior border). Note that tension band stabilization is necessary because Stoudt wires are being used instead of Erich arch bars. **F,** Intraoperative view demonstrates stabilization of the left angle fracture with single 2.0-mm miniplate placed in "Champy fashion." **G,** Immediate postoperative occlusion. **H,** Immediate postoperative panoramic radiograph. **I,** The 6-month postoperative appearance. **J,** The 6-month postoperative occlusion.



FIGURE 18-25. A 40-year-old woman with mandibular fractures involving the symphysis and condyles bilaterally, initially managed elsewhere and referred for revision. **A,** Preoperative appearance. Note facial widening. **B,** Preoperative PA radiograph. Note untreated mandibular subcondylar fractures with significant angular displacement. **C,** Preoperative axial computed tomography (CT) image demonstrates inadequate reduction and fixation of the symphysis fracture. Note diastasis at lingual border of mandible. **D,** Illustration of planned surgical treatment to include load-bearing fixation placed at the symphysis and open reduction and internal fixation (ORIF) of the mandibular condyles bilaterally. **E,** The 5-year postoperative appearance. Note favorable restoration of facial width. **F,** The 5-year postoperative occlusion.

presence of both the inferior alveolar nerve canal and the tooth roots. If bicortical screws are utilized, they must be placed below the inferior alveolar nerve canal when positioned posterior to the mental foramen. At any point along the mandibular arch, care must be taken to avoid screw placement into the roots of teeth; but if this does occur, it may be of little consequence. In addition, there is clinical and experimental evidence to suggest that permanent damage to the pulp and supporting tissues is a rare occurrence when screws abrade or even enter root substance.[134–138] The need for postoperative endodontic therapy is extremely rare.

Mandibular Angle Fractures

The ideal treatment of mandibular angle fractures has been controversial. Fractures of the mandibular angle, often complicated by the presence of a third molar, have been shown to have the highest rate of postoperative complications of all mandibular fractures.[132,139,140]

Numerous techniques have been utilized to treat mandibular angle fractures (Figure 18-26). The options for treatment of mandibular angle fractures include the placement of one miniplate along the superior border, miniplates along the superior and inferior border, a compression plate along the inferior border, a reconstruction plate along the inferior border, and a lag screw.

Edward Ellis and colleagues have thoroughly investigated outcomes for the treatment of mandibular angle fractures (Table 18-1). In a series of clinical papers beginning in 1993, they described complication rates of 0% to 32% depending upon the technique utilized.[142–150] The highest complication rate and most significant type of complications occurred

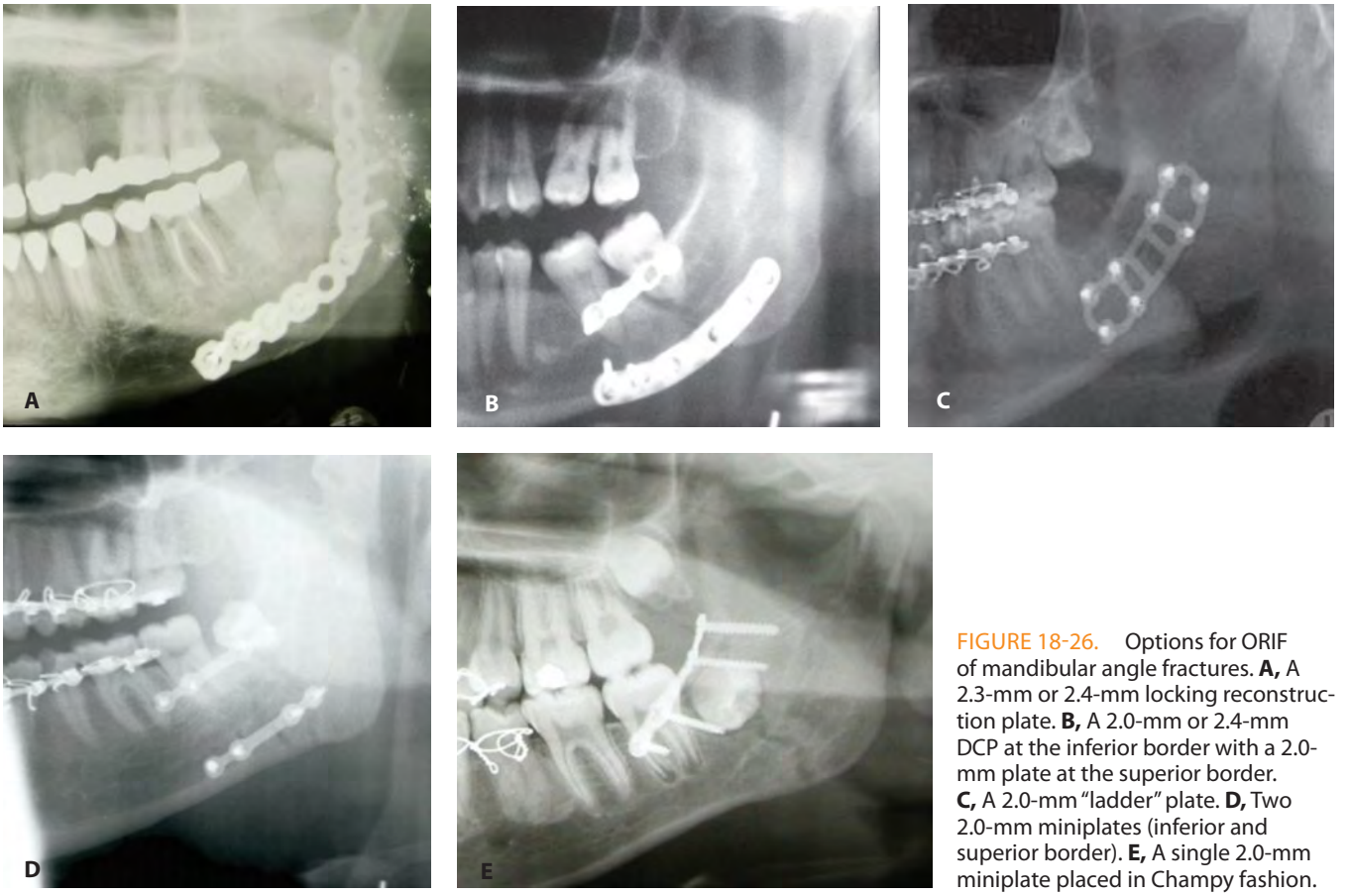


FIGURE 18-26. Options for ORIF of mandibular angle fractures. **A,** A 2.3-mm or 2.4-mm locking reconstruction plate. **B,** A 2.0-mm or 2.4-mm DCP at the inferior border with a 2.0-mm plate at the superior border. **C,** A 2.0-mm "ladder" plate. **D,** Two 2.0-mm miniplates (inferior and superior border). **E,** A single 2.0-mm miniplate placed in Champy fashion.

when two DCPs were utilized (32%).[147] In contrast to the traditional AO/ASIF technique that involves a transbuccal trocar and longer operating times, a single, noncompression miniplate placed in Champy style via a transoral approach yielded a complication rate of only 2.5%.[142,143,149]

Complication rates are often difficult to interpret, because the definition of a complication is variable. Bell and Wilson[125] found a complication rate of 32% in a series of 162 angle fracture patients. However, this number is misleading because virtually all patients had a favorable outcome with successful bony union and a return to premorbid occlusion. All but 2 patients had their complications managed on an outpatient basis, under local anesthesia or intravenous sedation and almost always after bony union had been achieved. The majority of complications consisted of hardware removal, which is a limitation inherent in the Champy technique itself. The severity of complications in this study was actually similar to previous publications by Ellis and colleagues.[142,143,149] Ellis and Walker[142] reviewed 81 patients treated in similar fashion with a Champy style and noted favorable results. Sixteen percent experienced complications requiring a second surgical intervention. However, most of the complications were minor and could be treated in the office.

The AO/ASIF advocated compression plating at the inferior border, occasionally with an additional miniplate at the superior border as a matter of routine in the early years of

TABLE 18-1. **Outcomes for Treatment of Mandibular Angle Fractures**

Treatment	Study Reference	Sample (No. of Angles)	Major Complication*
Nonrigid fixation	Passeri et al., 1993[143]	99	17%
AO reconstruction plate (2.7 mm)	Ellis, 1993[146]	52	7.5%
Solitary lag screw	Ellis & Ghali, 1991[144]	88	13%
Two minidynamic compression plates (2.0 mm)	Ellis & Karas, 1992[145]	30	13%
Two mandibular dynamic compression plates (2.4 mm)	Ellis & Sinn, 1993[147]	65	32%
Two noncompression miniplates (2.0 mm)	Ellis & Walker, 1994[148]	67	23%
One noncompression miniplate (2.0 mm)	Ellis & Walker, 1996[142]	81	2.5%
One malleable noncompression miniplate (1.3 mm)	Potter & Ellis, 1999[149]	51	0%

**Major complication* refers to the necessity of hospitalization to treat the complication.

rigid internal fixation. Over time, however, most experienced surgeons have come to adopt the use of miniplate fixation and abandoned the larger compression plates for noncomminuted fractures. Functionally stable osteosynthesis whereby a single noncompression miniplate is placed along the superior border of the external oblique ridge has gained widespread acceptance. Described by Michelet and colleagues[99] and validated by Champy and associates,[100–106] 3 decades of experience have shown that this method results in predictable clinical outcomes with or without the use of postoperative MMF and that it meets the biomechanical principles set forth by the AO/ASIF.[141]

A recent study by Gear and associates[24] evaluated various treatment modalities for mandibular angle fractures that were used by experienced surgeons. They found that more than half of these experienced surgeons utilized single miniplate fixation as advocated by Champy, or a single miniplate plus arch bars. Only 10% used miniplate fixation and even fewer used locking screw and/or compression plating.

In fact, the usual approach to most mandibular angle fractures has evolved from one that applied AO/ASIF principles of compression plating to the inferior border combined with superior border plating placed via a transbuccal trocar, to that described by Champy of a single superior border plate placed transorally.[125] For the majority of noncomminuted isolated fractures of the mandibular angle, a single 2.0-mm miniplate is placed at the superior border. A second 2.0-mm miniplate may be added to either the inferior border or just below the first using a transbuccal trocar for additional stability.

Bilateral Mandible Fractures

Bilateral mandibular fractures are common and alter the fixation requirements necessary to achieve stability in the postoperative period.[151] The second fracture changes the complexity of the force vectors that act across the fractured fragments; therefore, fixation requirements are altered. In general, load-bearing rigid internal fixation utilizing a locking reconstruction plate or two miniplates should be applied on at least one of the fractures (Figures 18-27 and 18-28). This is typically done on the tooth-bearing fracture rather than the angle or the condyle owing to ease of access.

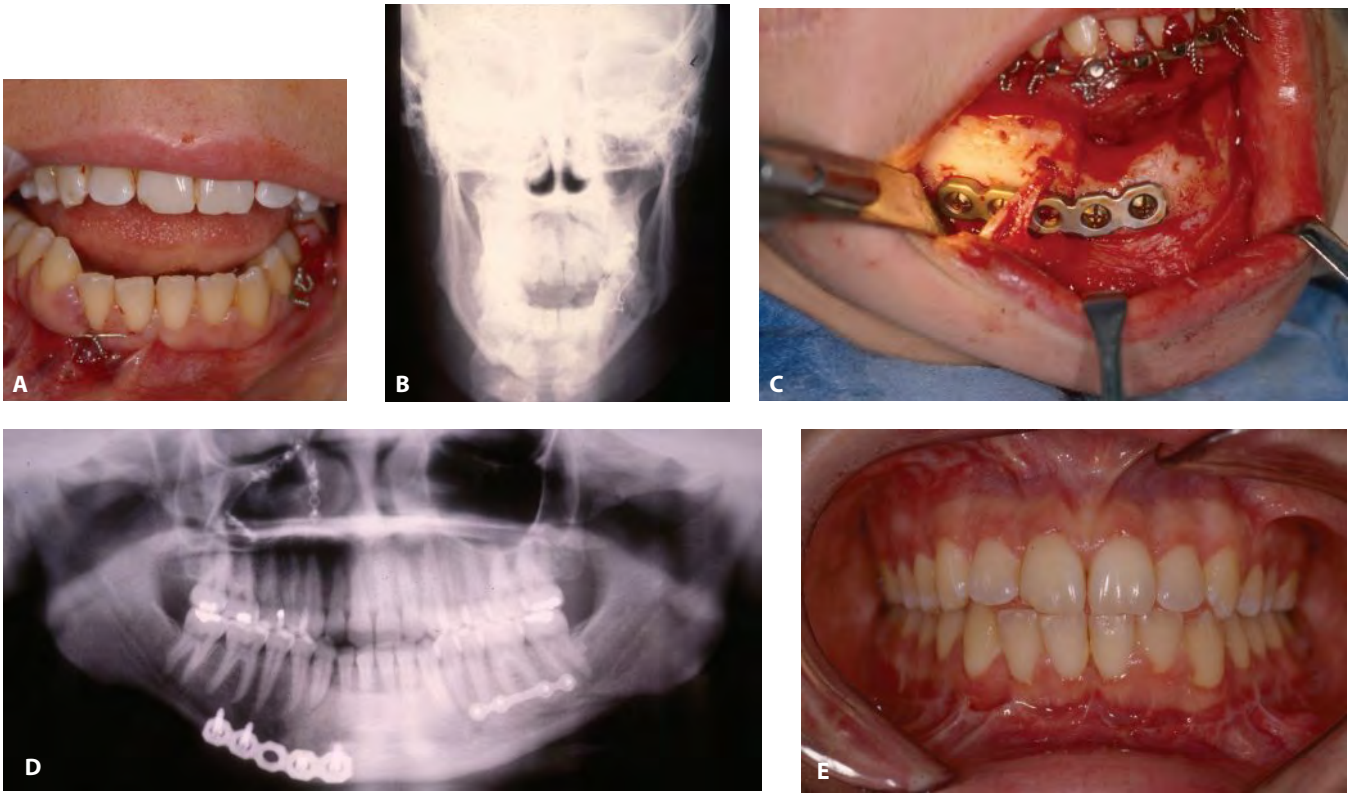


FIGURE 18-27. A 28-year-old woman with bilateral mandibular fracture involving the right parasymphysis and left body. **A,** Preoperative occlusion. Note bridle wire stabilization. **B,** PA radiograph demonstrates displaced fracture of the right parasymphysis and left body. **C,** Fixation of the right parasymphysis fracture with a 2.4-mm locking reconstruction plate placed at the compression band (inferior border) with the Erich arch bar serving as tension band stabilization. Note load-bearing fixation provided in this bilateral fracture. **D,** The 6-week postoperative panoramic radiograph demonstrates favorable reduction, stabilization, and fixation of the right parasymphysis and left body. Note the left body is stabilized with single 2.0-mm tension band plate along the ideal lines of osteosynthesis. **E,** Postoperative occlusion.

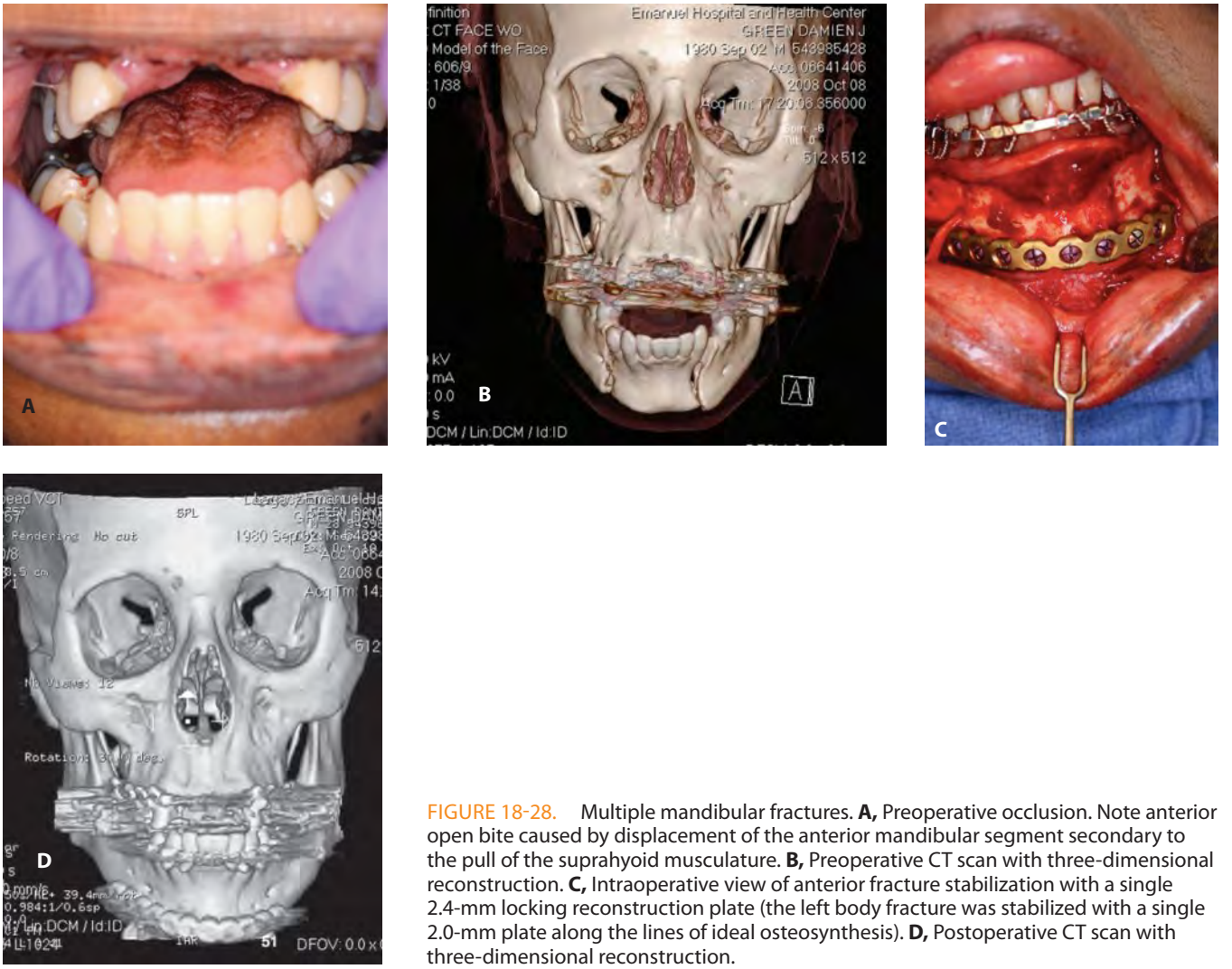


FIGURE 18-28. Multiple mandibular fractures. **A,** Preoperative occlusion. Note anterior open bite caused by displacement of the anterior mandibular segment secondary to the pull of the suprahyoid musculature. **B,** Preoperative CT scan with three-dimensional reconstruction. **C,** Intraoperative view of anterior fracture stabilization with a single 2.4-mm locking reconstruction plate (the left body fracture was stabilized with a single 2.0-mm plate along the lines of ideal osteosynthesis). **D,** Postoperative CT scan with three-dimensional reconstruction.

Comminuted Mandibular Fractures

A comminuted fracture is that which is disrupted and follows multiple lines within the same region of the mandible. They are less common than linear fractures but can occur at any location depending upon the trajectory of the kinetic energy applied.

Comminuted fractures require load-bearing fixation because the surrounding bony fragments are incapable of sharing any of the functional loads transmitted during healing. A 2.3- or 2.4-mm locking reconstruction plate using three or four screws on either side of the comminuted area typically provides load-bearing fixation. Once the major proximal and distal segments are stabilized with a locking reconstruction plate, the remaining segments may be stabilized to one another using multiple miniplates.

In cases of severe comminution, it is often helpful to stabilize the smaller fractures with miniplates first and then apply the locking reconstruction plate to the remaining construct in order to provide load-bearing support.

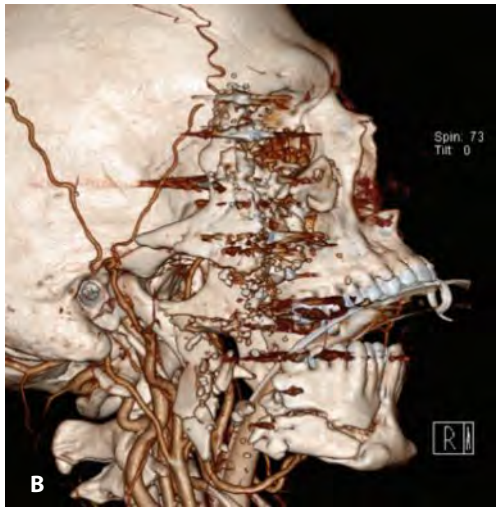
Recently, the use of virtual computer planning and a computer-assisted design/computer-assisted manufacturing (CAD/CAM) technique for milling stereolithographic models has been used to aid in the process of repairing highly comminuted segments. This is particularly useful for patients with gunshot wounds to the face in which dozens of fractured segments are often found and it is impossible to piece all of them together. By providing a virtual construct, a locking reconstruction plate can be prebent and the segments simply made to fit the plate in situ (Figure 18-29).

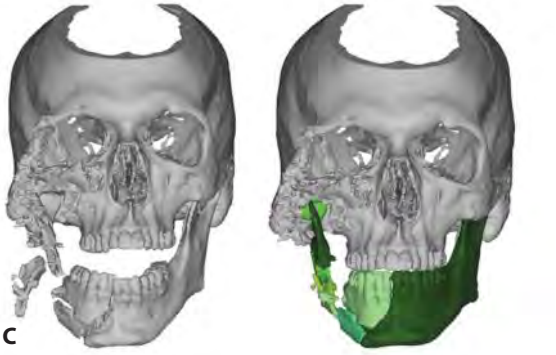
Edentulous Atrophic Mandibular Fractures

Fractures of the atrophic edentulous mandible have plagued surgeons for years. The attached muscular forces often cause significant displacement, patients are unable to wear dentures, and associated pain and masticatory dysfunction quickly render these patients oral cripples. Furthermore, fracture healing potential is impaired because there is little endosteal or periosteal blood supply and there is a severely diminished surface area that allows no load-sharing capacity.

Various treatment modalities have been used in the management of edentulous atrophic mandibular fractures.[152–160] For many years, closed reduction using Gunning type splints







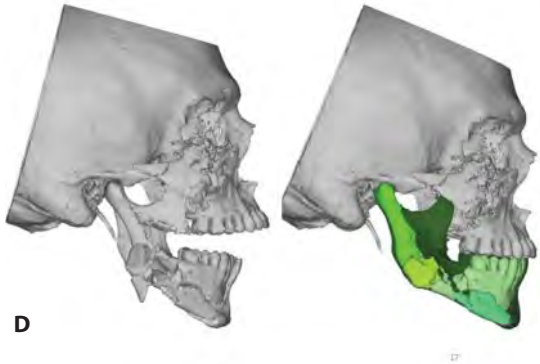
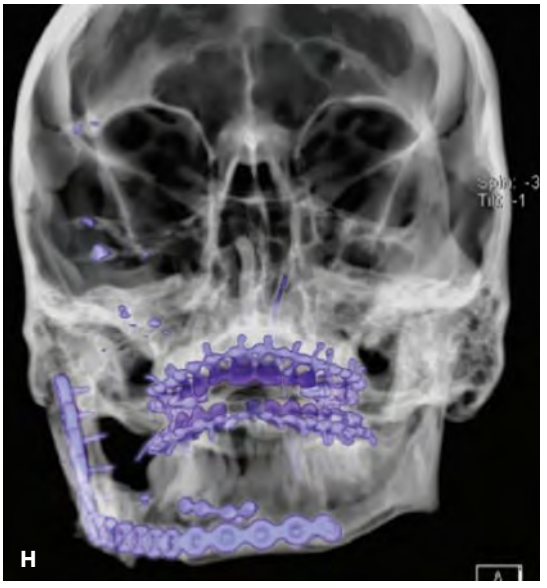








FIGURE 18-29. A 24-year-old man with self-inflicted gunshot wound to the face. **A,** Preoperative appearance. **B,** Preoperative CT angiogram demonstrates a highly comminuted mandibular fractures. **C,** Virtual reconstruction, anterior view. **D,** Virtual reconstruction, lateral view. **E,** CT scans of virtually reconstructed mandible to be milled into stereolithographic model. **F,** Stereolithographic model of "reconstructed" mandible with prebent 2.3-mm locking reconstruction plate. **G,** Intraoperative view of fracture reduction, stabilization, and fixation with prebent locking reconstruction plate. *(continued)*



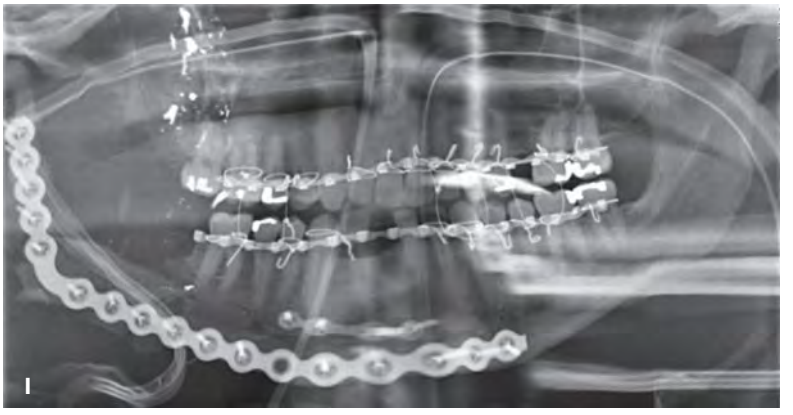


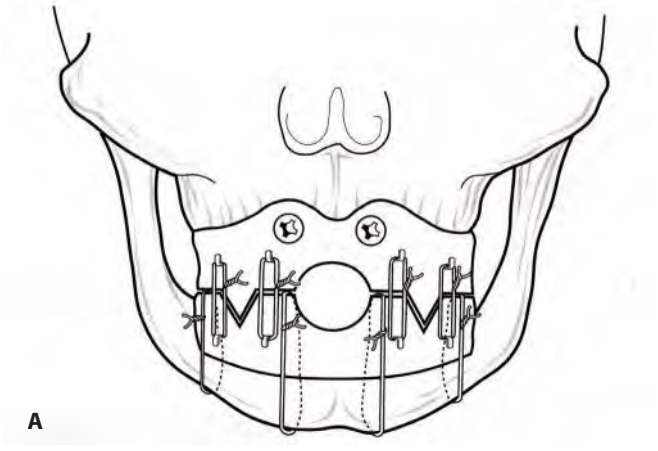
FIGURE 18-29. *(continued)* **H,** Postoperative CT reconstruction demonstrates favorable restoration of mandibular contour. **I,** Postoperative panoramic radiograph.

with circummandibular wires plus or minus IMF was the standard of care (Figure 18-30). Unfortunately, this technique suffered from a high degree of unpredictability, malunion, and nonunion and is no longer recommended.

Obwegeser and Sailer[153] popularized a technique in the 1970s of using split rib grafts fixed with circummandibular wires to stabilize the fractures. Marciani and Hill[155] advocated skeletal pin fixation as a method of minimizing periosteal stripping. Neither of these techniques, however, yielded consistent results.

Luhr and coworkers[158] later described favorable results in a large series of patients with atrophic edentulous mandible fractures using compression plating for fracture stabilization. These experiences led to the development of specialized instrumentation and locking reconstruction plates for the purpose of providing load-bearing fixation to edentulous atrophic mandibles. Ellis and Price,[161] Tiwana and colleagues,[162] and Van Sickels and Cunningham[163] have each recently reported excellent results using locking reconstruction plates placed at various locations on the atrophic mandible and have provided a sound rationale for management.

Contemporary treatment of atrophic edentulous mandibular fractures is performed via a submental incision with skeletonization of the entire mandible. Care is taken to minimize dissection on the lingual surface. The fractured segments are then reduced and temporarily stabilized with



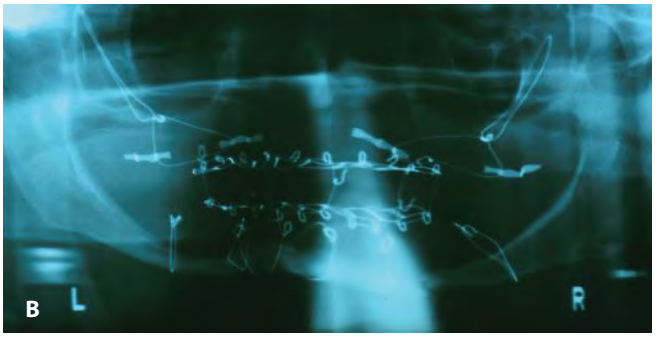


FIGURE 18-30. Traditional technique of using Gunning style splints with circummandibular wires and IMF to treat edentulous atrophic mandibular fractures. **A,** Diagram of technique. **B,** Postoperative panoramic radiograph of patient managed with closed treatment. Note the poor reduction of the mandibular segments. This technique is no longer advocated.

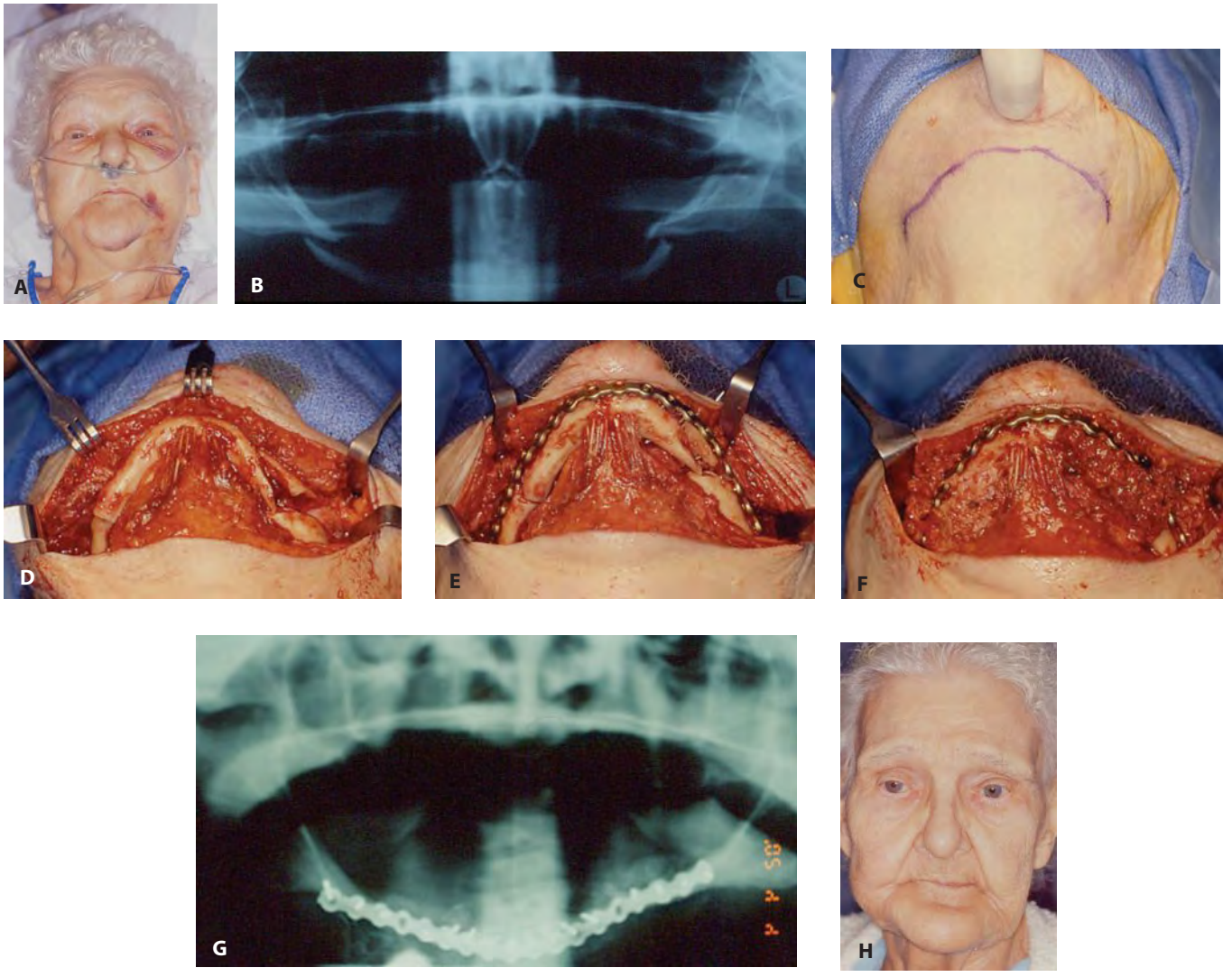


FIGURE 18-31. An 86-year-old woman with severely atrophic edentulous mandibular fractures, also called a "bucket handle" fracture. **A,** Preoperative appearance. **B,** Preoperative panoramic radiograph. **C,** "reverse smile" submental incision. **D,** Subperiosteal exposure. Note the displacement and comminution. **E,** Favorable reduction, stabilization, and fixation with a single 2.0-mm locking reconstruction plate and screws. **F,** Immediate anterior iliac crest bone graft. **G,** Postoperative panoramic radiograph. **H,** Postoperative appearance.

miniplates. Following anatomic reduction and temporary stabilization, load-bearing rigid internal fixation is applied utilizing a locking reconstruction plate. At least three or four screws should be placed on either end of the proximal and distal segments (Figure 18-31). Although some authors utilized two plates for bilateral bucket-handle fractures, a single load-bearing plate that extends from angle to angle may be more appropriate.

Controversy still exists with regard to the efficacy or necessity of simultaneous bone grafting.[161–162] The theoretical advantage of primary bone grafting the severely atrophic mandible (<10 mm) is that a corticocancellous bone graft would augment the osteogenic potential of the atrophic bone and enhance healing. Rh-BMP-2 may also be used for this purpose on an off-label technique with or without an allogeneic bone graft.[164] Bone grafts can also be used to provide additional bone stock in preparation for dental implant–supported prosthetic rehabilitation. Clinical experience in the nonfracture patient suggests that if bone grafting for this purpose is contemplated, then the implants should be placed between 4 and 6 months after grafting.[164] Failure to provide functional support to the bone graft within this timeframe will result in rapid bone resorption and complete loss of the graft.[165,166]

A 2007 Cochrane database review on the management of fractured edentulous atrophic mandibles demonstrated inadequate evidence for the effectiveness of a single approach.[167] Treatment decisions should, therefore, be based on the training and experience of the clinician. Many surgeons utilize a 2.3- or 2.4-mm locking reconstruction plate for Luhr class I

and class II edentulous atrophic mandibles and the 2.0-mm locking reconstruction plate for Luhr class III (severely atrophic) mandibles. In the case of the severely atrophic patient, consideration of primary bone grafting is made depending on the patient's desire or ability to afford dental implants. Rh-BMP shows promise in this population of patients as well, but thus far, its use remains experimental.

Pediatric Mandibular Fractures

Less than 5% of all facial fractures occur in children.[168] Of these, approximately 32% involve the mandible and less than 1% occur in children under age 5.[167,168 169] The discrepancy between children and adults is partially due to the greater elasticity of the pediatric mandible and the presence of developing tooth buds. This fact generally accounts for the fracture patterns seen in children, who often present with minimally displaced fractures in the form of a "greenstick."

The healing potential of pediatric mandibular fractures is advantaged owing to a rich endosteal blood supply. Therefore, nondisplaced fractures of the mandible in very young children (<5 yr old) can often be treated with observation and a nonchew diet. Fractures in older children who are in the mixed dentition or permanent dentition stage are typically managed in a manner similar to that for adults. Children younger than 5 years of age, however, represent a separate subset of pediatric mandibular fractures and are the subject of this section.

It is unusual for a very young child to present with an operative mandibular fracture.[169] Most fractures of the primary dentition are nondisplaced and can be managed nonsurgically. Displaced fractures of the dentate mandible in children younger than age 5 do occur, however, and when present should be surgically treated.

The surgical management of pediatric mandibular fractures has evolved significantly over the last 2 decades.[171–173] Various techniques have been advocated to repair displaced fractures of the dentate mandible of very young children,[172] including IMF using interdental wires or arch bars, lingual splints,[173] or ORIF utilizing both titanium[174] as well as biodegradable plates and screws.[176,177, 176] The ideal method depends upon the patient's age, fracture pattern, degree of displacement, concomitant injuries, and surgeon training and/or experience.[177] A number of experienced surgeons have described favorable results using modern craniofacial techniques and various combinations of open and closed treatment.[171,174–176,178,175–180]

Contrary to popular opinion, it is not particularly difficult to apply interdental wire fixation or even Erich arch bars to primary teeth. Care must be taken to avoid tooth avulsion in the late primary or mixed dentition phase. The advantage of utilizing interdental wire fixation is that it helps to establish a stable platform on which to apply internal fixation or IMF if indicated. The problem with the latter is that many small children cannot be expected to thrive in IMF; thus, lingual splints do have efficacy in the early primary dentition (age 1-3) (Figure 18-32).

It is acceptable to perform ORIF for even the youngest of patients with significantly displaced mandibular fractures. Because titanium has the potential to cause growth retardation and may become encased in bone in a very short period of time, biodegradable fixation is used.[176] Care must be taken to apply the fixation to the inferiormost aspect of the mandible to avoid the developing tooth buds (Figure 18-33).

Children with fractures of the mandibular condyle should virtually always be managed with closed treatment and active physiotherapy to normalize range of motion and prevent complications of hypomobility, growth disturbance, and ankylosis. The pediatric mandibular condyle has tremendous regenerative capacity and will often form a morphologically normal appearing condyle within a few years after injury in patients in the primary and mixed dentition.

COMPLICATIONS

Complications in the treatment of mandibular fractures occur regularly and, even in the most experienced of hands, should be expected.[181] The most common complications include infection, malunion, nonunion, tooth injury, and the need for hardware removal. Adherence to proper surgical techniques and the principles of reduction, stabilization, and fixation with adequately rigid plates and screws generally minimizes risk, even in high-risk populations.

Infection

Infection is the most common complication in patients undergoing treatment of mandibular fractures, occurring between 1% and 32% of the time.[141–151] Numerous risk factors have been associated with postoperative infection, including substance abuse or patient noncompliance with postoperative regimens,[143] and a significant delay in treatment.[152] Definitive treatment within 3 to 5 days after trauma has been shown to be optimal in terms of minimizing the rate of infection.[153] Other risk factors include high-velocity injuries and severe comminution, gross contamination, and high-velocity ballistic injury.

Most infections related to mandibular fractures are polymicrobial, with both aerobes and anaerobes routinely cultured. The most common organisms are *Staphylococcus*, alpha-hemolytic *Streptococcus*, and *Bacteroides*, as well as gram-negative organisms. Penicillin G (with or without Flagyl depending on gram stain) or clindamycin is the drug of choice.

Successful management of the infection requires adequate drainage, removal of the source, and appropriate antibiotic therapy. If the cause of the infection is related to loose hardware and there is favorable bony union, removal of the loose hardware is all that is necessary. Conversely, if inadequate

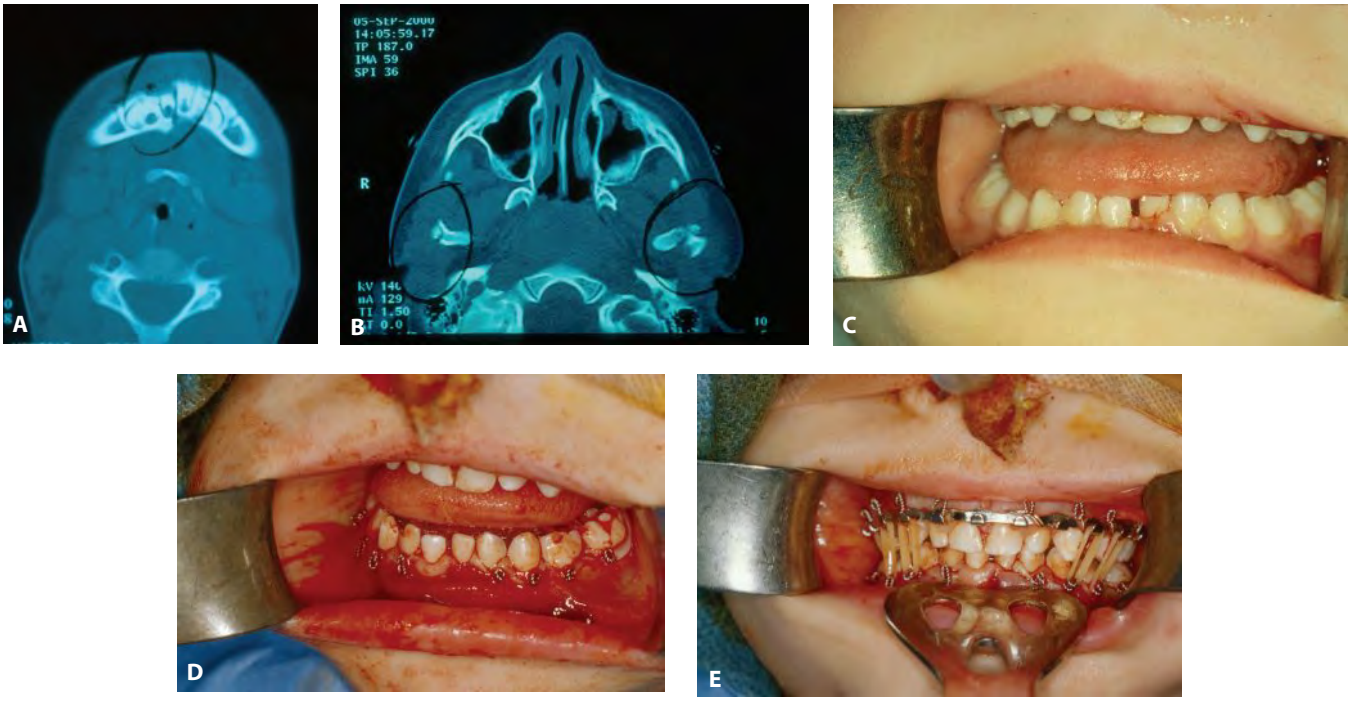


FIGURE 18-32. A 2-year-old male with displaced mandibular fractures involving the symphysis and condyles bilaterally treated with a lingual splint and interdental wire fixation. **A,** Preoperative CT scan demonstrating displaced symphysis fracture. **B,** Preoperative CT scan demonstrating bilateral mandibular subcondylar fractures. **C,** Preoperative occlusion. **D,** Application of interdental wires and lingual splint. **E,** Intraoperative intermaxillary fixation with elastic bands which are removed at the completion of the procedure.

stability of the fractured segments is apparent, the previously placed fixation should be removed and replaced with more rigid fixation, usually in the form of a locking reconstruction plate. Although careful patient selection is necessary, immediate bone grafting of infected mandibular fractures used in conjunction with rigid internal fixation and appropriate intraoperative débridement can be an effective treatment modality, which allows a single surgical procedure and successful bony union.[154]

Malunion

Malunion occurs typically because the injury complex was particularly complicated, the patient was noncompliant with postoperative instructions, and/or the surgeon violated basic principles of reduction, stabilization, and fixation. The excep-tion to this rule is with regard to closed treatment of mandibular condyle fractures, where malunion is an inherent risk to the procedure itself and malocclusion results in a significant number of patients, even those who are compliant and in whom the procedure was carried out expertly. When malunion occurs, mandibular osteotomies are generally indicated to correct the occlusion and restore facial symmetry/projection. A description of mandibular osteotomies for post-traumatic malocclusion is beyond the scope of this chapter.

Nonunion

Nonunion of mandibular fractures is an uncommon sequelae of treatment, but it may occur even in the most experienced hands. High-velocity injuries resulting in severe comminution, inadequate or improperly placed fixation, and poor patient compliance are common etiologies. Treatment typically involves reoperation with débridement of soft tissue and nonviable bone, followed by stabilization and application of rigid internal fixation with a locking reconstruction plate and immediate bone grafting if necessary.[182–183]

CONCLUSIONS

Technical advances in maxillofacial fracture repair have turned what used to be one of the great challenges in trauma surgery (i.e., treatment of a mandibular fracture) to a matter of routine. The vast majority of patients with mandibular fractures can go on to bony union and return to premorbid form and function very predictably by applying the principles of reduction, stabilization, and fixation with modern titanium plates and screws. Both open and closed techniques have a role to play in mandibular fracture treatment and will result in predictable outcomes as long as the surgeon respects the physiologic and biomechanical properties of the patient's mandible and the fixation material.

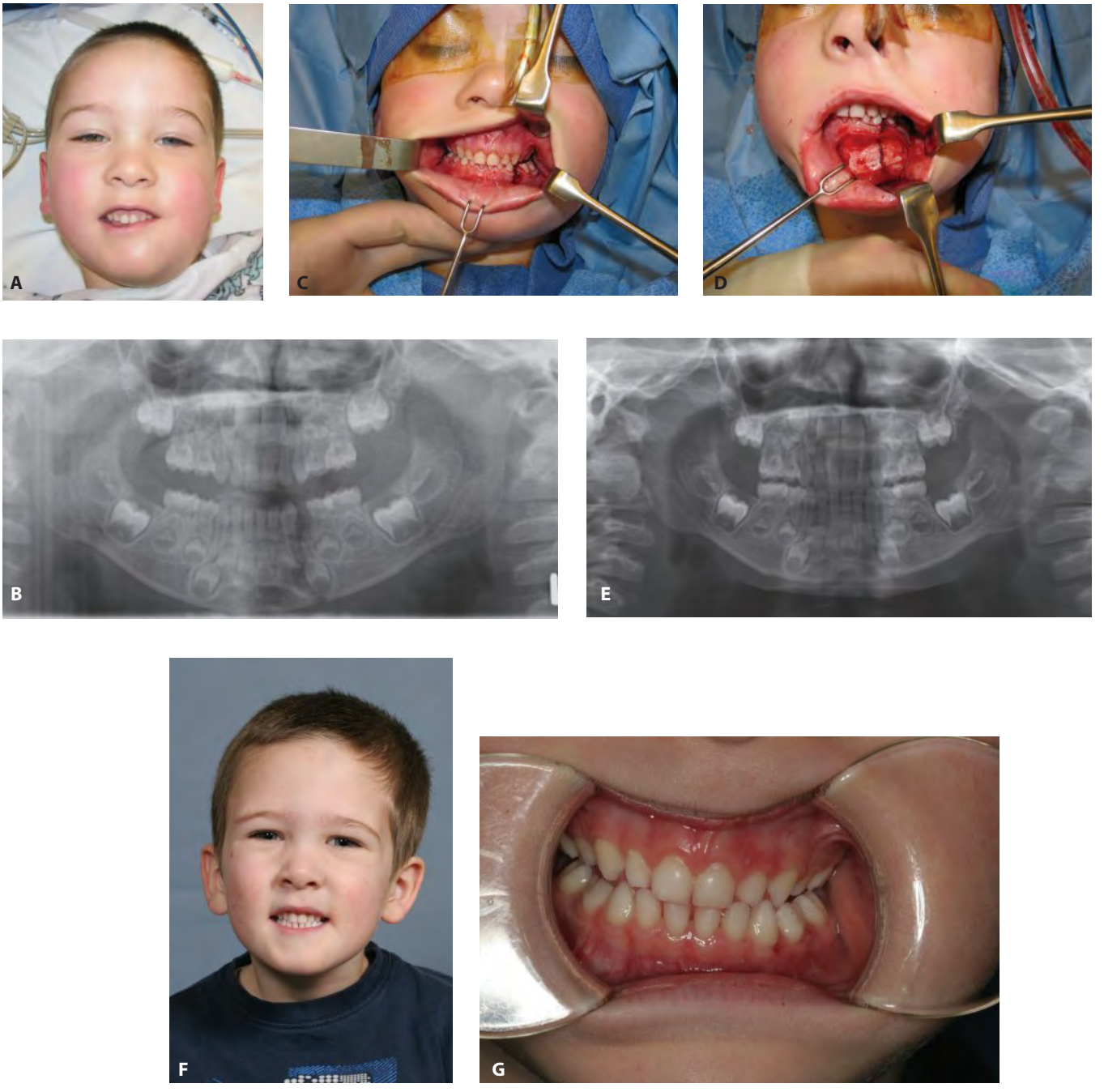


FIGURE 18-33. A 3-year -old with displaced mandibular fracture treated with ORIF utilizing biodegradable plate and screws. **A,** Preoperative appearance. **B,** Preoperative panoramic radiograph. **C,** Preoperative occlusion. **D,** ORIF using biodegradable plate and screws. **E,** Postoperative panoramic radiograph. **F,** Postoperative appearance. **G,** Postoperative occlusion.

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CHAPTER 19

Fractures of the Mandibular Condyle

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INTRODUCTION

Fractures of the mandibular condyle are unique among maxillofacial injuries in that they affect mandibular function in a manner unlike other traumatic injuries (Table 19-1). Condylar fractures differ significantly from other fractures of the mandible owing to the functional derangement that may affect occlusion, mastication, airway, and speech resulting from condylar positional changes. Although infection with subsequent nonunion and step occlusal deficits resulting from malunion may be typical adverse sequelae of fractures of the mandibular symphysis, body, angle, and ramus, condylar fractures are unique among mandible fractures in that infection and nonunion are rare after condylar fractures. However, functional derangements unique to condylar fractures are very frequent sequelae of these injuries and subsequent events. This is due to the unique functional anatomy of the mandibular condyles, the specific challenges of surgical treatment of these fractures, and the various patient-specific factors affecting the outcomes of condylar fractures. An understanding of these variables will guide the clinician toward a knowledge-based and evidence-driven approach to patient management of condylar fractures. Condylar fracture outcome also depends upon the application of specific surgical repair techniques. A detailed understanding of techniques for both closed and open treatment is needed to obtain favorable treatment outcomes.

TABLE 19-1. **Injuries of the Articular Apparatus**

Effusion
Hemorrhagic or serous
Soft tissue injury
Disk
Capsule
Ligaments
Dislocation of the condyle from the fossa
Without fracture
With fracture other than condyle
With associated condylar fracture
Fracture
Nondisplaced
Deviated
Displaced
Dislocated
Comminuted
Involving adjacent bony structures
Combinations of the above

FUNCTIONAL ANATOMY

The condyles form the distal portion of the temporomandibular articulation, which consists of the condylar heads, interpositional disks, glenoid fossae, and articular eminences. Functionally, these articulations are bilateral, diarthroidial (freely movable joints held together by a complete joint capsule), and ginglymoidial joints in that they permit a broad range of movement in the mouth opening-closing axis including the glenoid fossa and articular eminence. This ginglymoidial articulation is first a hinge axis movement of the joint forming the primary articulation throughout the first 15 to 20 mm of rotational condylar movement as measured at the incisor. With further movement beyond this range, subsequent translation of the condyles down the articular eminence occurs, permitting full range of motion to 40 to 50 mm of mouth opening. Translation of the condyles depends upon the posterior (retrodiskal) elasticity of the interposed

articular disks. The elasticity of the joint capsule and the forward sloping of the lateral and medial suspensatory capsular ligaments permit anterior translation of the condyles and inhibit posterior movement. Posterior and superior movement of the condyles is limited from an evolutionary standpoint in that posterior and superior movement of the condyle would cause injury to the middle cranial fossae. Therefore, fracture of the condylar head and tympanic plate protect the cranium from injury. Also, displacement of the condyle in a posterior direction is a very rare clinical event.[1] With the biped status and binocular vision of humans, the condylar necks form a valuable breakpoint that protects the neurocranium.

Translation of the mandible is another important evolutionary adaptation associated with maintenance of bite clenching force throughout the range of motion of the mandible by improving the mandibular lever action of the masseter, temporalis, and medial pterygoid muscles as the mouth is opened. As a result, stability of the condyle against the articular eminence during condylar translation with the interposed articular disk elegantly allows for incising food when the mouth is open wide and the mandible is translated laterally to the side of the food bolus.

During mastication, the articulation of the condyles in the glenoid fossae results in a translatory movement permitting disocclusion of the ipsilateral dentition and functional working side occlusion to permit canine contact on the opposite/working side as well as working side and balancing side contact of the molars and premolars during lateral excursion of the jaw. All functional dental contacts result from a combination of condylar guidance of movement along with neuromuscular influences. This complex, harmonious masticatory system is capable of broad adaptation over time, but the sudden alterations produced by condylar fractures can create certain expected typical clinical findings of functional alterations.

FUNCTIONAL ANATOMIC ALTERATIONS WITH CONDYLAR FRACTURES

A patient with a condylar fracture will variably demonstrate one or more of the following clinical alterations in mandibular function:

Ipsilateral (Side of Injury) Occlusal Prematurity

Owing to loss of vertical height of the mandible on the injured side, the teeth will contact first, or exclusively, on the side of injury. Often, this is a first contact followed by a rocking motion of the mandible into a position of maximum intercuspation. Thus, although the final occlusion may appear normal, careful observation of the path of closure will reveal this functional derangement.

Inability to Achieve Maximum Intercuspation

Typically, this includes a single posterior contact on the ipsilateral side and a wedge-shaped open bite, indicating that the change in the occlusion has occurred acutely (Figure 19-1). In bilateral fractures of the mandible, including bilateral condylar fractures, a large anterior open bite (gag bite) may be present (Figure 19-2). In bilateral condylar fractures with an associated mandibular symphysis fracture, facial widening may result with posterior buccal cross-bites and anterior open bite (Figure 19-3).



FIGURE 19-1. Occlusion in a left subcondylar fracture: note the single contact in the left posterior region and complete disocclusion of other teeth

Ipsilateral Laterognathia

For unilateral fractures, there may be no obvious open bite malocclusion, whereas there may be a laterognathia toward the side of the condylar injury. The molar relationship on the side of injury may tend toward class II malocclusion due to retrodisplacement of the mandible on that side.

Ipsilateral Deviation on Opening

The opening path of the mandible often becomes an arc-shaped deviation toward the side of the condylar injury. The



FIGURE 19-2. Occlusion in a bilateral condylar fracture: a wedge shape open bite with bilateral posterior contacts.



FIGURE 19-3. Occlusion in a bilateral condylar fracture with symphyseal fracture. The patient exhibits bilateral posterior cross-bites and open bite with wide face.

terminus, or late point, of the opening cycle can produce a more abrupt and sharper deviation of the mandible toward the side of the injury.

Balancing Side Occlusal Interferences during Contralateral Mandibular Translation

Patients with unilateral condylar fractures may manifest occlusal interferences on the balancing side during attempted contralateral translation of the mandible (during movement to the side opposite the injury). When performing contralateral jaw movement, the functionally shortened ramus on the injured side will cause balancing side interferences, often not allowing unimpeded working side contacts. In addition, limitations in the amount of contralateral lateral excursion may be present.

Limitation of Mouth Opening

Trismus in the condylar fracture patient may be due to functional interferences of the fracture segments, coronoid impingement, concomitant joint hemarthrosis or to pain from splinting. Displacement and rotation of the condylar segment can produce trismus (Figure 19-4).

Anatomic Types of Condylar Fractures

Various classifications of condylar fractures have been proposed based upon location, displacement, or resultant functional alterations. There are numerous condylar fracture classifications including those of Brophy,[2] Thoma,[3] Rowe and Killey,[4] and Dingman and Natvig.[5] These are based upon anatomic findings, usually present on radiographic examination. A classification system based upon functional aspects has not been developed, although Zide and Kent[6] attempted to outline those fracture types that would benefit from open versus closed reduction techniques. Simple anatomic classification schemes offer little information to guide treatment recommendations because all fractures can be treated via



FIGURE 19-4. Trismus and open bite due to a subcondylar fracture.

open or closed techniques based upon various functional clinical indicators. Perhaps the most straightforward classification system from a clinical perspective is discussed in the following sections.

Subcondylar Fracture

A fracture extending from the coronoid notch cranially to the posterior border of the ramus caudally (Figure 19-5).

Condylar Neck Fracture

A fracture cranial to the coronoid notch, extending across the condylar neck, located in an extracapsular position (Figure 19-6).



FIGURE 19-5. Subcondylar fracture as seen on coronal computed tomography (CT).

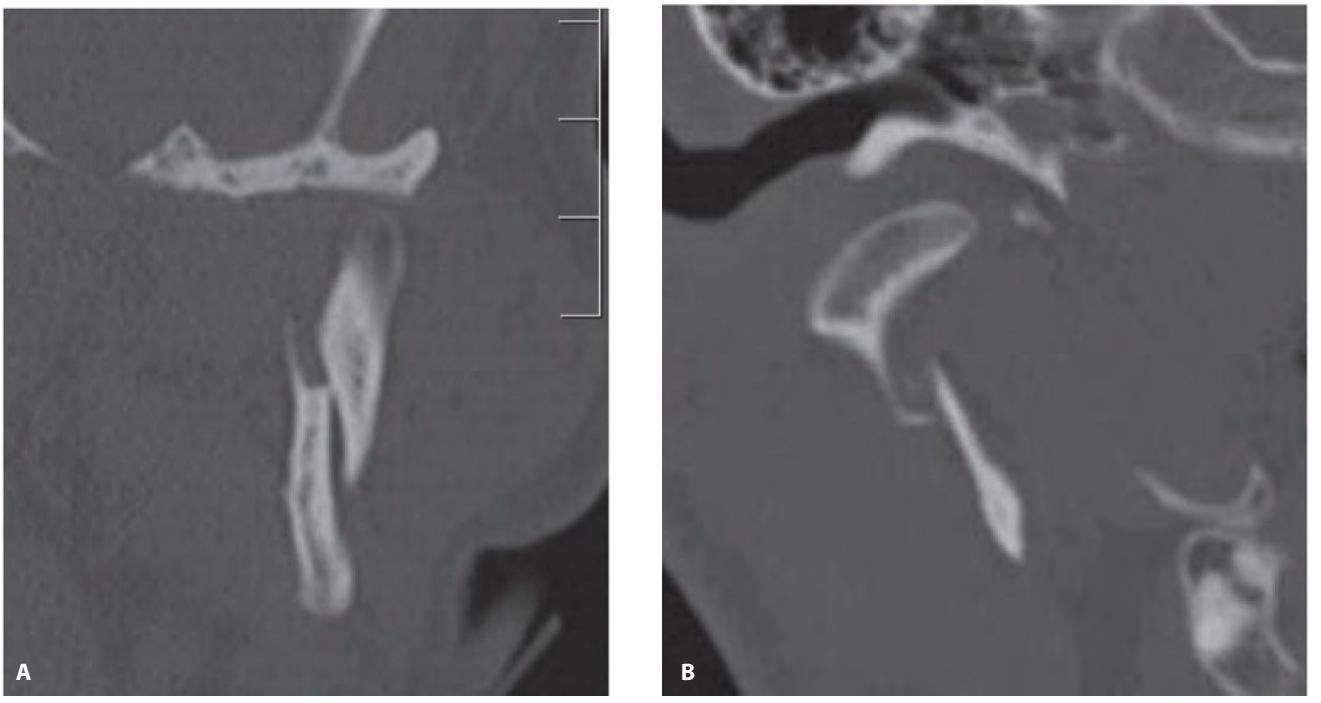


FIGURE 19-6. **A,** Condylar neck fracture as seen in closed position on coronal CT. **B,** Condylar neck fracture as seen on axial CT.

Condylar Head Fracture

An intracapsular fracture of the condyle, including the articulating surface (Figure 19-7).

TREATMENT OF CONDYLAR FRACTURES

The goals of treatment of condylar fractures are to restore function to the preinjury state and to do so with undisturbed healing of the injured site. An understanding of the preinjury state is important in assessing the results of treatment of subcondylar fractures. Such an evaluation performed in population research studies indicates that pain, deviation, other jaw excursion problems, and joint noise are very frequently present. Although Pullinger and coworkers[7] reported on the orofacial and temporomandibular assessment of dental students without condylar fractures and found that 29% had joint noise and 14% had pain, the reader of outcome studies of fractures of the mandibular condyle must consider how a treated population might differ from one that has no injuries.

What are the optimal methods of treating condylar fractures that will accomplish the functional goals and also address the various factors affecting outcome that have been

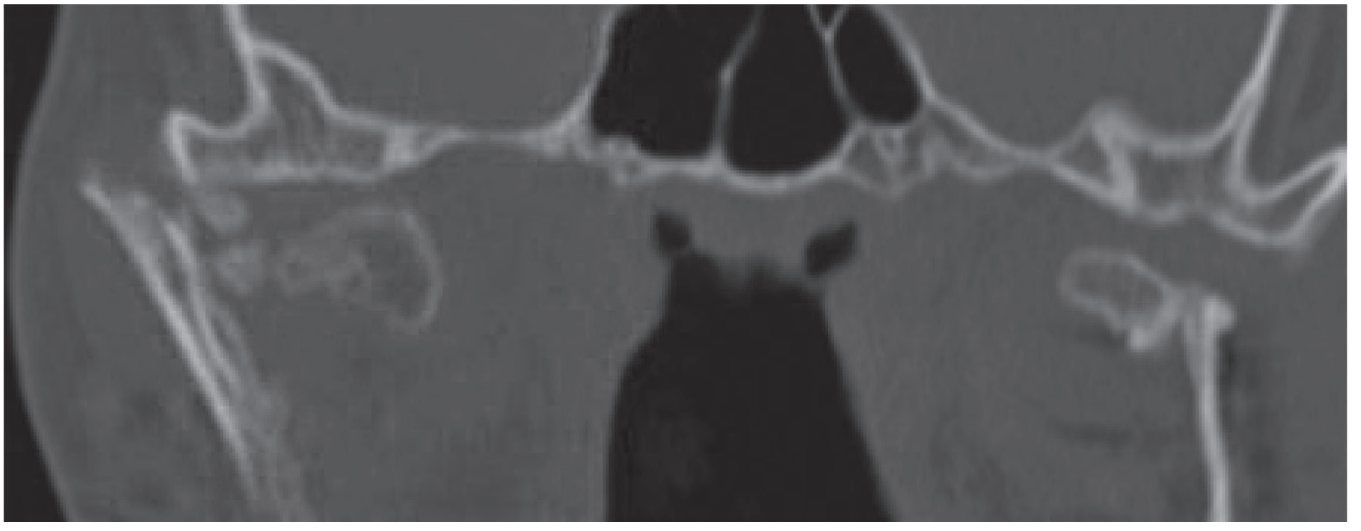


FIGURE 19-7. Bilateral condylar head fractures as viewed on coronal CT.

identified for these injuries? A review of the pertinent studies of condylar fracture treatment will offer an interpretation of the evidence. Each of the clinical studies of condylar fractures suffer from the same biases of addressing too many variables in an inconsistent manner and the problems of developing a comparative study with so many critical success factors that may influence outcome. The surgeon benefits by developing a detailed understanding of the complicated mixture of factors affecting the outcome of condylar fracture treatment.

Closed Treatment of Condylar Fractures

The goals of closed treatment, a more appropriate designation than closed reduction because in most cases no "reduction" of the fracture is performed, include rehabilitation of mandibular function along with restoration of the preinjury occlusion. Closed treatment consists of reestablishing the preinjury occlusion with arch bars or other appropriate appliances. Whereas some clinicians use a brief 1- to 3-week period of maxillomandibular fixation, as described by Walker,[8] many contemporary surgeons no longer use continuous maxillomandibular fixation. In the alternative, immediate guiding elastics are used to guide a functional path of movement and correct occlusion. Typically, elastics on the side of injury are oriented in a class II direction and anterior transverse elastics guide the mandible away from the side of injury (e.g., left on mandible to right on maxilla for a left-sided condylar fracture). Early functional rehabilitation with range of motion exercises and promoting excursion of the mandible away from the fracture site are additional hallmarks of closed treatment. Active physiotherapy can also include passive movement with or without task-specific devices and other manual manipulation of the jaw.

As mentioned, closed treatment of a condylar fracture is not the same as closed reduction. Although improvements in radiographic appearance and displacement do occur, mandibular function is generally restored owing to functional neuromuscular adaptation and also to the condyle functioning further down the articular eminence. Muscle and joint proprioception will guide mandibular movement against a physiologic range of osseous and dental relationships. Thus, small changes in bone contact and tooth occlusion can be accommodated by this response.

Does closed treatment of a condylar fracture carry with it a risk of complication and treatment failure that is decreased with open reduction? If closed reduction results in an unacceptable incidence of complications with real clinical significance, then overcoming the obstacles to open reduction is an important goal of treatment. To understand the sequelae of condylar fractures, a clinician should include evidence from a nonconcurrent control group, namely, patients who have not sustained condylar fractures. Understanding the baseline of temporomandibular joint function in noninjured adults offers help in assessing how the macrotrauma of a fracture is adapted to over the course of time after closed treatment. In other words, the studies of functional derangements in closed reduction patients should be compared with studies in normal subjects.

One of the first, largest, and most historically important evaluations of closed treatment was performed by MacLennan in 1952.[9] His series of 180 cases included 67 patients who were available for direct examination. He identified a clinically apparent deformity or malfunction in only 6% of the cases in his series. These were not subjective complaints of the patient, but only those signs on physical examination observed by the clinician. Indeed, no patient complained about their clinical outcome on recall visits. This is not surprising, considering that the occlusal disturbances resulting from closed treatment are usually subtle. Small deviations of the chin, laterognathia, or modestly decreased range of motion are rarely detected by the patient.

Park and colleagues[10] demonstrated that adverse outcome of closed treatment was dependent upon the location of the fracture and that intracapsular condylar head fractures have the highest incidence of malfunction. Neizen and associates[11] recently evaluated closed treatment of condylar fractures in 114 patients assessed before treatment and 1 year posttreatment. The results of closed treatment included 9% of patients still complaining of pain, and 24% describing their occlusion as "fair" or "poor." However, the average mouth opening was excellent at 52 mm.

Studies performed since McKlennan's study have focused on deviation on mouth opening as an important outcome of closed treatment of condylar fractures.[2] The importance of deviation on opening has never been studied from a patient-centered approach. Because the mandible is not in function during opening and appears only to be limited functionally by the amount of opening, the value of studying deviation remains unclear. In addition, the path of mandibular movement is highly variable. McKlennan was the first investigator to systematically demonstrate the efficacy of closed treatment. The most important aspect of McKlennan's seminal study is that both surgeons and patients can expect a low incidence of obvious complications after closed treatment of condylar fractures.

Subsequently, further objective evaluation of closed treatment has included studies of facial symmetry using radiographic clinical analysis. Ellis and Throckmorton[12] performed this evaluation after closed and open reduction of condylar fractures. Towne's and panoramic radiographs were used to measure changes in posterior facial height immediately and in subsequent follow-up evaluations. In the 2 years of the study, the mean shortening of ramus height in the closed reduction group was only 2 to 5 mm. No loss of ramus height was noted in the open reduction group. This study does not indicate a functional shortening of the ramus in that the articulation of the condyle simply moved further down the articular eminence and in general permitted maintenance of function and occlusion through a new articular position of the condylar head. This is effectively a neoarthrosis formation that permits adequate jaw function.

Conservative therapy is evaluated by the patients themselves in Blevins and Gores's study[13] of 90 condyle fracture patients who answered a questionnaire and 45 patients who appeared for examination an average of 5.8 years postinjury. The patients available for long-term follow-up offered patient-centered views of the results of their conservative therapy. These patients reported a self-perception of normal mouth opening, but when measured clinically, the maximum interincisal opening was only 34 mm. Whereas a report of restriction of jaw movement by a patient is variable, many clinicians use 35 mm as a guide to maximum interincisal opening that will not be a concern to patients and as a guide to successful functional recovery. Thus, in Blevins and Gores's paper, only 1 in 8 patients managed with closed treatment of their condylar fracture reported difficulty chewing or any restriction in jaw movement.

To understand the nature of patient's findings and complaints after treatment for condylar fracture, an understanding of the findings of temporomandibular function offers a useful comparison.[14] The Helkimo index and the Mandibular Functional Impairment Index are used to assess relevant clinical signs and symptoms in contemporary outcome studies of treatment of subcondylar fractures.[15] Using this method, Klatt and coworkers[15] demonstrated very good functional outcomes of condylar fractures. From the treating surgeon's perspective, standard assessments of masticatory function such as occlusion, range of motion, and lateral excursions are typically used. In a careful study of closed treatment condylar fracture patients and controls, Murakami and colleagues[16] identified only subtle, and seemingly insignificant, differences at 6 months postinjury, and these differences included a greater bite force on the side of injury and diminished lateral excursion away from the side of injury. These findings had continued to diminish in the 6 months after injury and indicated that closed treatment is a viable contemporary option for condylar fracture rehabilitation.

There are also many attempts to define those cases of condylar fracture at risk for problems after closed treatment. This information could guide surgeons into determining who would benefit from open reduction. In 1947, the Chalmers J. Lyons Club used patient self-assessment as an essential feature of their review of 120 patients.[17] Although none of these patients reported pain, about 5% had functional complaints after condylar fracture management with closed treatment. Silvennoinen and associates[18] noted malocclusion in 13% of patients; other complications of treatment were also noted. They contended that "problematic condylar fractures can often be identified preoperatively." The most important variable that determined a need for open treatment was a loss of ramus height on the fracture side. Detecting a shortening of ramus height and quantifying its functional effect depends upon clinical measurements of occlusion and radiographic evaluation comparing posterior ramus height from condyle to gonion (Figure 19-8).

An alternative method for measuring ramus height is determining a patient's ability to achieve maximum occlusal intercuspation. A patient with a condylar fracture who cannot obtain a full occlusion will most likely have a shortened ramus on the affected side. In Silvennoinen and associates' study, 54% of patients could obtain a normal occlusion pretreatment, but 9% had fracture displacement severe enough to prevent achieving a good occlusion. These were the cases that were at risk for poor outcome after closed treatment. Thus, it would appear that from a ramus height standpoint, the majority of patients without a ramus height decrease may be managed effectively with closed treatment.

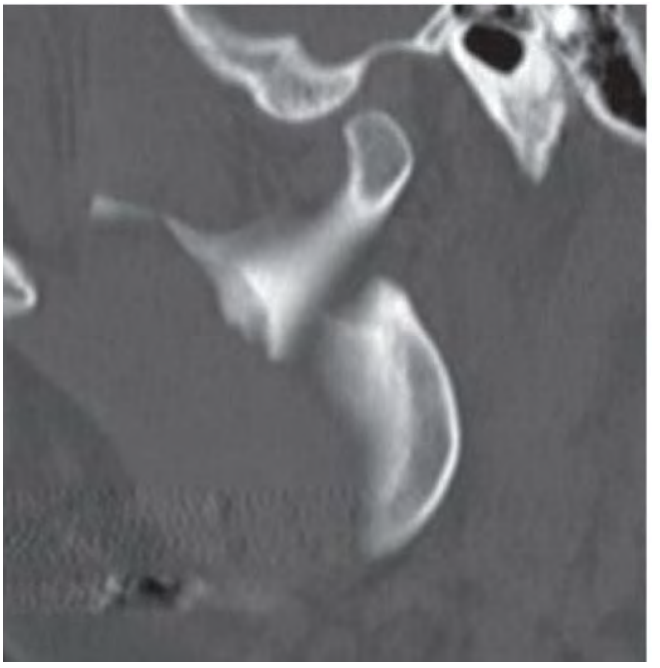


FIGURE 19-8. Condylar neck fracture with shortening of the ramus and severe rotation of the proximal segment as seen on sagittal CT.

Open Reduction and Internal Fixation of Condylar Fractures

Open reduction of condylar fractures has the advantage of immediate and complete restoration of the anatomical relationships of the joint, and rigid fixation allows for immediate and full function of the mandible (Table 19-2). The main obstacle to the application of internal fixation remains the technical complexity, unless specialized training and equipment are available. The emergence of endoscopic open reduction of condylar fractures is an important advance in this regard over the past decade. Brandt and Haug[19] reviewed the efficacy of open reduction versus closed reduction of condylar fractures and stated that "under similar indications and conditions, ORIF [open reduction and internal fixation] is the preferred approach" for the treatment of condylar fractures.

The variety of surgical approaches to the condyle region remain limited by poor visibility through remote sites and the restrictions to direct access caused by both aesthetic

TABLE 19-2. **Indications for Open Reduction of Fractures of the Mandibular Condyle**

1. Absolute indications
   A. Limitation of function secondary to the following:
      1. Fracture into middle cranial fossa
      2. Foreign body within the joint capsule
      3. Lateral extracapsular dislocation of the condylar head
      4. Other fracture-dislocations in which a mechanical stop is present on opening, which is confirmed radiographically
   B. Inability to bring the teeth into occlusion for closed reduction
2. Relative indications
   A. Bilateral condylar fractures with comminuted midface fractures in which rigid internal fixation of the midface is not possible
   B. Situations in which intermaxillary fixation is not feasible as a result of the following:
      1. Medical restrictions
         a. Poorly controlled seizure disorder
         b. Psychiatric disorders
         c. Severe mental retardation
         d. Concomitant injuries such as head injury or chest injury (unless tracheostomy is planned)
      2. Displaced fractures in which dentures or splints are not feasible because of severe mandibular atrophy
   C. Bilateral fractures in which it is impossible to determine what the proper occlusion is as a result of loss of posterior teeth or the presence of a preinjury skeletal malocclusion
   D. In fracture dislocation in adults to restore the position and function of the meniscus (controversial)

concerns and the location of the facial nerve and parotid gland (Figure 19-9). Thus, all approaches variously address these anatomic concerns while attempting to offer adequate access for direct reduction and fixation. Traditional approaches include the Risdon approach that suffers from poor direct access to the fracture. The retromandibular approach is perhaps the most common method used today and is also employed for endoscopic access techniques. An incision behind the vertical ramus is used to lift the tail of the parotid and incise the aponeurosis of the vertical ramus at the posterior border of the mandible. Subperiosteal dissection to the coronoid notch allows the placement of a notch retractor and direct and/or endoscopic access to the condyle. Recently, new approaches have taken a very detailed look at cranial nerve VII anatomy and access to the condyle. The high cervical transmasseteric approach offers access between the cervical and the buccal branches of the facial nerve and permits a direct view of the condylar region, albeit through the parotid capsule.[20] Klatt and coworkers[21] reported a modest morbidity of the transparotid approach, with temporary cranial nerve VII palsy in 4 of 48 patients and parotid cutaneous fistulae in 2 of 48 patients. For intracapsular fractures, a standard preauricular approach gains access to an area as far as the condylar neck. When combined with a retromandibular approach, full access to the ramus-condyle unit is possible.

Even with wide surgical access, retrieval of the medially dislocated condylar segment can be difficult. The problematic nature of retrieving the proximal segment in some cases has led surgeons to utilize extracorporeal reduction and fixation of the segments (i.e., on the back table) and immediate replacement. Park and colleagues[22] demonstrated reasonable outcomes in a series of 35 extracorporeal ORIF cases. With this method, the condylar head, along with an osteotomized portion of the posterior ramus, is removed and fixed ex vivo. These segments are subsequently replaced and fixated to the distal portion of the mandibular ramus.

Regarding intracapsular and condylar head fractures, a greater utility of the preauricular approach is employed. Whereas there is traditional resistance to ORIF of condylar head fractures, He and associates[23] reported on a large series with good outcomes.

Malposition of the proximal condylar segment after open reduction is a problem that may occur even with the most skilled surgeons owing to limited visualization of the operative site. Various methods of assessing the adequacy of fracture reduction during surgery are emerging using navigation techniques and intraoperative computed tomography (CT) guidance. Klatt and coworkers[24] experienced this improvement in 12% of cases when evaluated by intraoperative cone beam CT scan. In addition to intraoperative imaging, other methods to ensure correct fracture reduction include endoscopy-assisted examination, the use of intraoperative navigation, and more extended exposure through multiple access incisions or the use of an extended rhytidectomy incision.

There has been an increased interest in endoscopic assistance for condylar fractures owing to the significant improvement in visualization that is possible. The endoscope permits direct access to a narrow operative field with limited access, and with a three-dimensional assessment, endoscopic confirmation of fracture reduction and fixation is often very effective (Figures 19-10 and 19-11).

Factors Affecting Outcome of Condylar Fracture Treatment

Minimal morbidity with open reduction is essential to offering it as a common option, and therefore, outcomes must be assessed in order to guide appropriate treatment decisions. With regard to endoscopy-assisted open reduction techniques, outcome studies have demonstrated low morbidity with the use of this technique. For example, Troulis and Kaban[25] demonstrated effective open reduction with an endoscopy-assisted, minimally invasive surgical technique. However, a prospective study of open reduction by Trost and colleagues[26] revealed that only 73% of patients had anatomic reduction, and 80% had a normal occlusion 6 months after open reduction and plate fixation. The AO group compared transoral endoscopic open reduction to the retromandibular approach for subcondylar fracture access and found lower morbidity, albeit with an increased surgical time in the endoscopy-assisted group.[27]

All surgical approaches to the condyle, including preauricular, postauricular, retromandibular, transoral, and Risdon

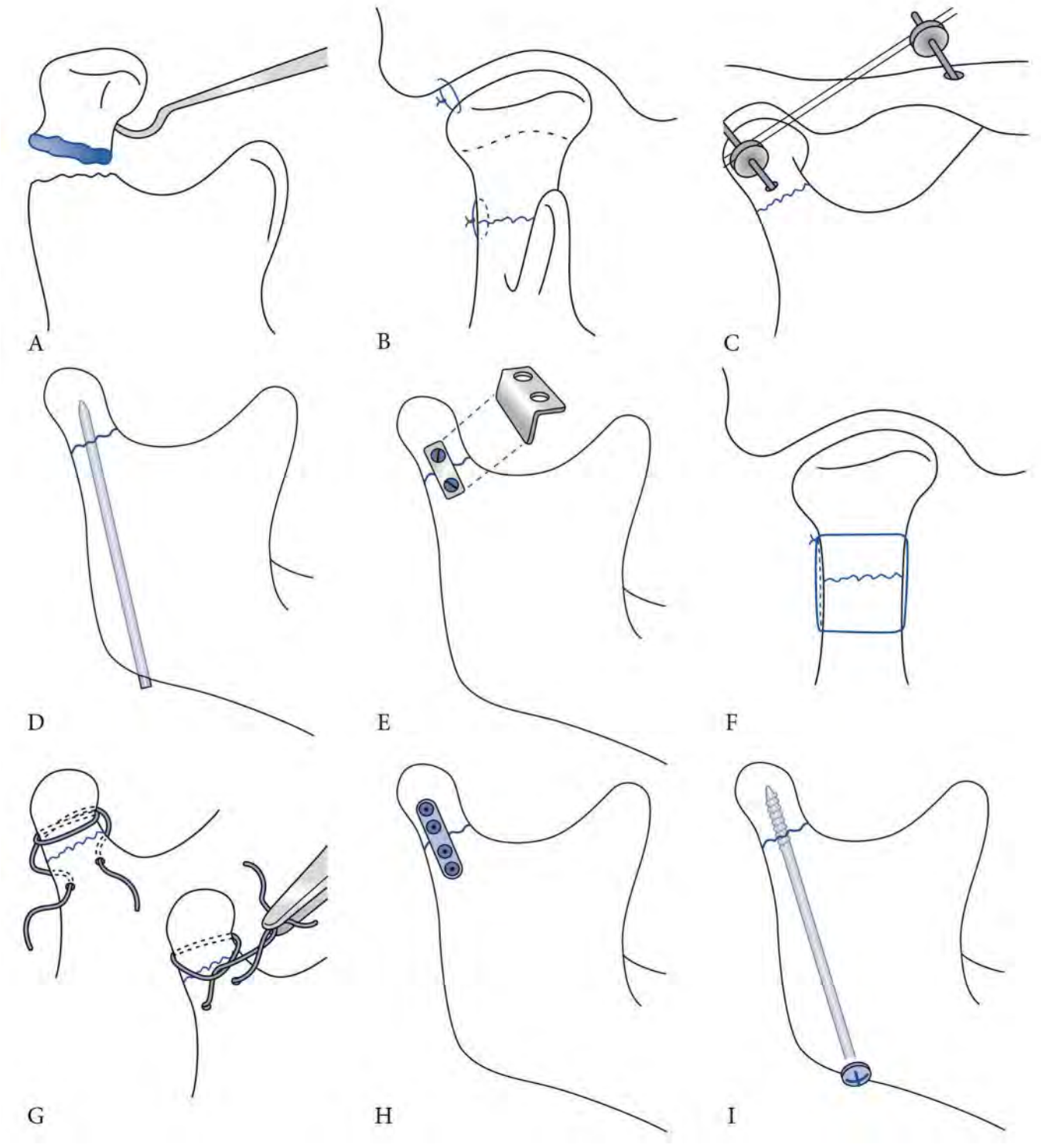


FIGURE 19-9. Previously reported techniques for direct stabilization of condyle fractures. **A,** Silverman (1925). **B** and **C,** Thoma (1945). **D,** Stephenson (1952). **E,** Robinson (1962). **G,** Messer (1972). **H,** Kobert (1978). **I,** Petzel (1982). *(Continued)*

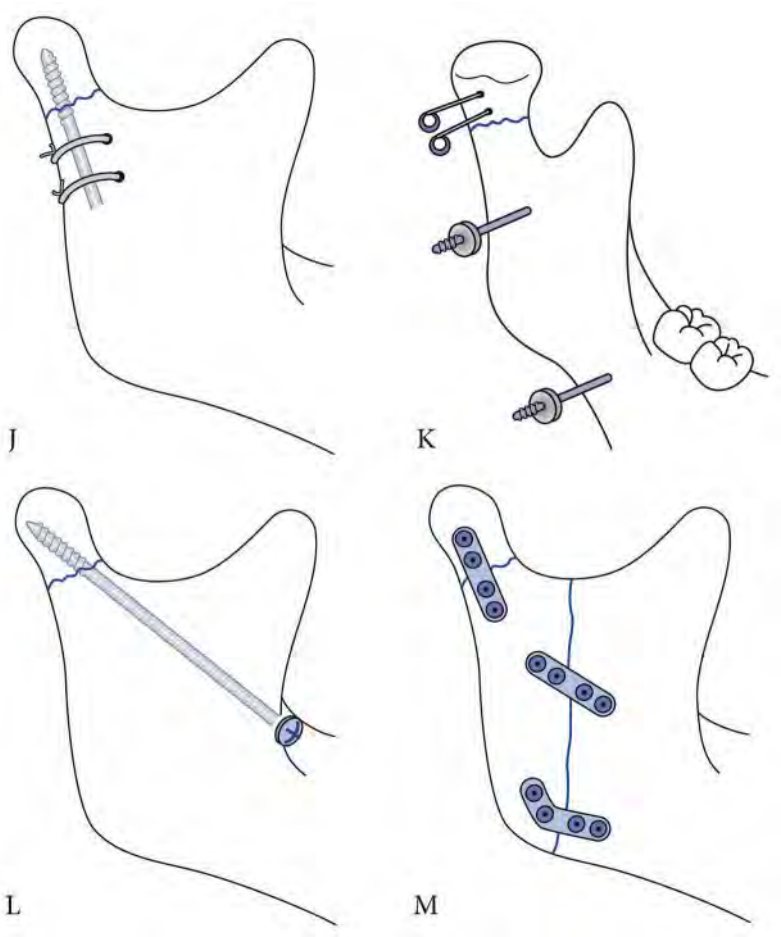


FIGURE 19-9. *(Continued)* **J,** Brown (1984). **K,** Fernandez (1987). **L,** Kitayama (1989). **M,** Ellis (1989). (**A–M,** Adapted from Larsen PE. Traumatic injuries of the condyle. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 1. Philadelphia: JB Lippincott; 1992; p. 460.)



FIGURE 19-10. Condyle fracture with shortened ramus amenable to endoscopic reduction.

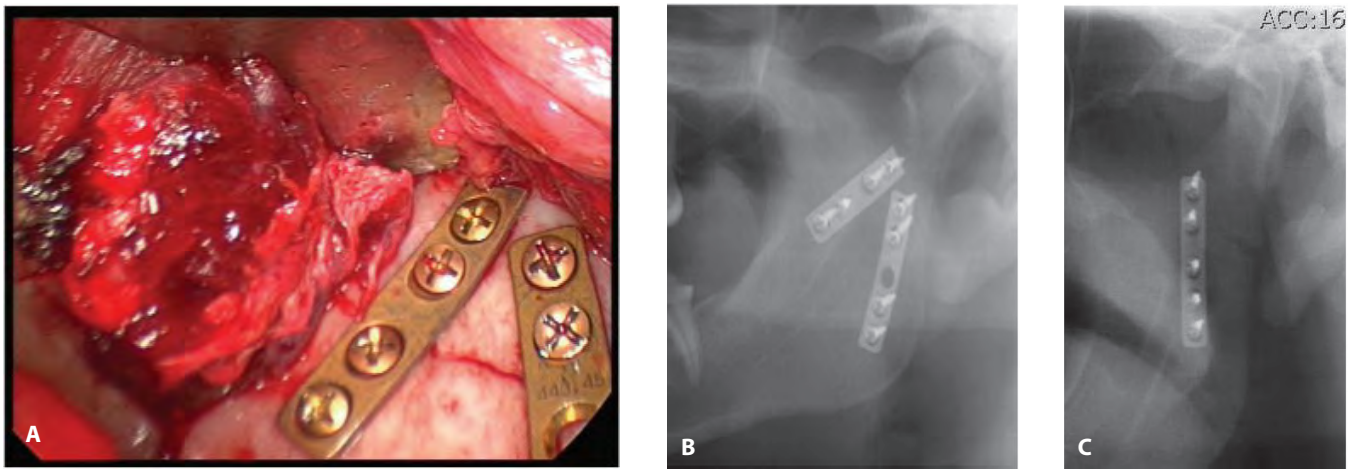


FIGURE 19-11. **A,** Intraoperative endoscopic clinical view of treatment. **B,** Radiographic assessment of endoscopic reduction. **C,** Case demonstrating postoperative view and challenges to positioning plate via endoscope.

incisions, place both cranial nerves V and VII at risk for iatrogenic injury.[28] For retromandibular and Risdon approaches, the marginal branch of VII is at greatest risk, whereas the preauricular approach places temporal and zygomatic branches of VII at highest risk. Ellis and associates[29] reported that 17.2% of open reduction patients had temporary facial nerve weakness with the use of a retromandibular approach. However, Schon and coworkers reported no VII nerve derangements 18 months after open reduction and plate fixation via an endoscopic technique.

With advances in technology and improvement in surgical skill and experience, open reduction of condylar fractures is a reasonable option for any fracture that is causing a functional derangement. However, all surgery carries an inherent cost as well as risks including anesthetic risks and the cost of hospitalization and surgery. Decision to proceed with open reduction of condylar fractures should remain dependent upon factors that will affect the outcomes of treatment.

Independent of the method of condylar fracture treatment, many known variables will affect the outcome. These factors were reviewed by Assael[30] and triaged regarding their contribution to outcome. The relationship of these variables results in a complex interaction that shows that the ability of small cohort studies to make meaningful statements about treatment remains problematic. The relative weight, or importance, that each of these variables carries in an individual case will vary. These variables include

1. **Patient age:** It is generally understood that condylar fractures can inhibit the vertical growth of the face according to Moss's theory of functional matrices whereby associated parts will not grow if condylar growth is inhibited by injury. Therefore, a resultant cant of the maxilla, asymmetry of the mandible, and occlusal disturbances can occur in patients sustaining condylar fractures during growth. Lindahl and Hollender[31,32] showed that younger children up to age 11 had better results owing to the potential for considerably more adaptation and remodeling of the bone than teenagers or adults. This makes sense when considering that the greatest vertical growth of the condyle occurs during adolescence. Landes and associates[33] demonstrated this restriction in growth with a 20% complication rate for open treatment of condylar fractures in children of average age 14.

   Condylar fractures in the elderly and edentulous patient are known to result in poor adaptation and less compensation and remodeling than in younger adults.
2. **Patient gender**: Adolescent females appear to demonstrate more functional derangements than male cohorts. Because temporomandibular disorders occur with a frequency far greater in female patients, internal derangement, idiopathic condylar resorption, and myofascial pain dysfunction may be related to issues separate from, or in addition to, the acute trauma of a condylar fracture.
3. **Systemic diseases:** The effects of specific systemic diseases have not been well studied relative to condylar fractures. Anecdotally, however, diabetes mellitus, osteoarthritis, smoking, alcoholism, bisphosphonate use, and steroid use may each play an adverse role in outcomes of condylar fracture treatment.
4. **Patient compliance:** Walker[34] has emphasized the need to have a good patient-doctor rapport in order to gain compliance in an arduous path to rehabilitation in many patients. Poor compliance was shown by Mitchell[35] who reported that just 73 of 142 patients with condylar fractures returned for both the 6- and the 12-week posttreatment visits. With such a poor recall rate, surgeons often consider whether compliance will be an issue when initially deciding upon whether closed or open treatment is indicated in an individual patient.
5. **Risk of infection:** There remains a substantial risk of infection after open reduction of condylar fractures.[36] This may be associated with avascular necrosis of the condylar head owing to periosteal reflection and possible muscular detachment. Fractures that extend into the external auditory canal, delayed treatment, and multiple-injury patients

may be at higher risk of infection among other medical comorbidities.

6. **Risk of operative site injury:** Whereas immunocompromise may predispose to poor wound healing such as with diabetes mellitus, obese patients offer special challenges for open reduction condylar fractures. Cranial nerve traction injuries and pressure necrosis are more likely when access is difficult owing to excessive overlying soft tissues.
7. **Risk of scarring:** Haug and Assael[37] noted that the perception of a scar was the most frequent complaint of patients who underwent open reduction of condylar fractures. Developing the skill for transoral endoscopy-assisted fracture treatment may mitigate this complaint. For older patients, an extended face-lift incision, or even simultaneous bilateral rhytidectomy procedures, if indicated, has been performed to minimize the appearance of scar and offer improved aesthetic outcomes.
8. **Risk for chronic pain:** The investigation of postoperative pain is very important in assessing the most appropriate treatment for a patient. Surprisingly, open reduction appears to offer a relatively lower impact on chronic pain. Umstadt and coworkers[38] used the Helkimo Anamnestic Index to demonstrate the outcomes of open treatment compared with those of closed treatment Whereas the incidence of chronic pain was low in both groups, it was lower in the open treatment group.
9. **Comminution:** Intracapsular comminution with loss of blood supply to the segments is particularly associated with pain, limitation in opening, infection, and malunion. When treated with open reduction, fixation failure is more likely to occur in the comminuted fracture patient.[39]
10. **Hemarthrosis:** Hemarthrosis is better detected today with the advent of magnetic resonance imaging (MRI) and CT evaluation of patients sustaining head and neck injuries. Arthroscopic intervention in the acutely injured joint patient is now widely reported. Piper and Chuong[40] first reported this with and without use of open reduction of the associated condylar fracture.
11. **Disc injury:** In condylar fractures with displacement or dislocation, the disk typically follows the condylar head due to the attachments of the medial and lateral collateral ligaments as well as the anteromedial influence of the lateral pterygoid muscle. Further understanding of meniscal injury associated with mandibular trauma is a separate topic in this chapter but is well understood to produce tearing, displacement, contusion, adhesion, arthritis, and necrosis. Choi and colleagues[41] and Neff and associates[42] have found that about 20% of open reduction patients demonstrate disk displacement on postoperative MRI. Whether the anatomic reduction of the condylar fracture helps or exacerbates this phenomenon is not known.
12. **Osteoarthrosis:** Adaptive changes of the condyle fossae complex occur after both open and closed treatment. For open treatment, Iizuka and coworkers[43] demonstrated that osteoarthrosis occurred in 13 patients 18 months after open treatment. Whereas these problems are typically noted on imaging (e.g., osteophyte formation, flattening of the condylar head, and joint space narrowing) and on clinical examination (crepitus), only a minority of patients, in this case, 1 of 13, have any complaints of related symptoms.
13. **Presence of other facial fractures:** He and colleagues[44] have shown that mandibular corpus fractures, particularly those of the symphysis, if allowed to be fixated in a position that results in mandibular widening, are associated with a higher rate of temporomandibular joint ankylosis due to the lateral impingement of the condylar head. Since the publication of Zide and Kent,[45] it has been understood that the posterior facial height is dependent on establishing proper ramus height via operative management of condylar fractures in patients with panfacial fractures.[46]
14. **Glenoid fossa fracture:** A condyle displaced into the middle cranial fossa is an indication for reduction of the fracture as well as fossa reconstruction (and dural repair, if indicated) with an appropriate bone graft and alloplast.[45]
15. **Occlusal considerations:** High mandibular plane angle patients and those with shallow intercuspal relationships due to bruxism are more prone to posttreatment malocclusion and open bite after closed treatment. High mandibular plane angles allow for fulcrum changes after condylar fracture that promote the development of an open bite occlusion. Conversely, patients with a deep bite and a low mandibular plane angle are less prone to open bites even with an anatomically shortened ramus due to a subcondylar fracture. Those without posterior bite support owing to loss of molars are more likely to sustain loss of vertical dimension.
16. **Bruxism and masseteric hypertrophy:** Patients may fracture a bone plate after open reduction and displace the bony segments if they suffer from bruxism and clenching habits. Masseteric hypertrophy is often present in these cases. Botox injections of up to 20 units per affected muscle can decrease these parafunctional loads during the course of bony healing.
17. **Functionally shortened ramus:** Whereas an anatomically shortened ramus can be measured as a decrease in the distance from condylion (Co) to gonion (Go), a functionally shortened ramus is one in which the condyle-ramus unit does not support the vertical dimension requirements for adequate occlusion and mastication. The slope of the articular eminence and the neuromuscular response to positional change of the condyles permits retention of acceptable function even for patients with an anatomically shortened ramus in many cases. However, many patients do exhibit functional derangements in mastication or occlusion (e.g., a functionally shortened ramus). This is an important indication for open reduction. Findings of an ipsilateral persistent occlusal prematurity, inability to achieve maximum intercuspation, and

inability to achieve contralateral occlusal working side contacts are some findings indicating the presence of a functionally shortened ramus.

Ellis[47] looked at the functionally shortened ramus in a different way in his evaluation of 332 fractures intraoperatively in which he assessed "drop back." Intraoperatively, if the mandible would collapse posteriorly with gentle pressure on the symphysis, then the mandible was felt to be unstable and ORIF is indicated. Inn this study, 105 stable mandibles had no "drop back" from maximum intercuspation, and all of those were treated successfully with closed treatment.

In another method for assessing ramus height, Schneider and colleagues[48] looks at ramus length from the perspective of angulation of the proximal segment. In their randomized clinical trial, they demonstrated morbidity with closed treatment of condylar fractures with angulations of 10 to 45 degrees. It is generally accepted that 30 to 35 degrees of proximal segment angulation should mandate consideration for ORIF.

18. **Surgeon experience:** Experienced surgeons need to obtain procedure-specific training to properly treat subcondylar fractures surgically. Those who are well trained and experienced are more able to achieve anatomic reduction and fixation of fractures with minimal morbidity.[49,50] Thus, the surgeon must understand her or his own skills and the capabilities of the setting and team before considering open reduction as the best means of management. Nearly all condyle fractures have the ability to be treated to the benefit of the patient via closed treatment methods.
19. **Economic constraints:** In the present economic climate, medicine is only supporting treatment based upon proven benefit at reasonable cost. Without a cogent means to support one method over another, the less expensive method will be chosen. Although condylar fractures are not a significant component of trauma care, they will be looked at in the context of other maxillofacial trauma treatment.[51]

Evidence-based Approach to the Management of Condylar Fractures

The level of evidence to offer treatment recommendations that are evidence-based remains inadequate. Sharif and associates,[52] perhaps optimistically, performed a Cochrane analysis of treatment of condylar fractures searching for randomized clinical trials meeting CONSORT criteria. No studies met their qualifications and they concluded that there remained a strong need for valid clinical evidence supporting the various modes of treatment for these injuries. Despite this rigorous assessment, there have been attempts to prospectively evaluate condylar fracture treatment randomized for open versus closed reduction. Singh and coworkers[53] performed a randomized clinical trial attempting to meet CONSORT criteria to compare open versus closed treatment. The limitations provided by a necessarily heterogeneous population of patients remain an obstacle to such studies.

In 2010 Singh and coworkers[53] randomized 40 patients and found that open reduction offered similar occlusion to closed reduction, but without further explanation, the closed treatment group had an MIO of only 33 mm, compared with 39 mm in the open group; and the closed treatment group was 4 points higher on a visual analogue scale (VAS) for pain. Conversely, Danda and colleagues[54] performed a nearly identical investigation on 35 patients randomized to closed versus open reduction. They found no difference in occlusion, function, pain, or any other clinical parameter.

Many problems specific to condylar fracture remain to be explored. For example, an issue that remains in open reduction is the long-term effect of the anatomic relationship of the fracture on the temporomandibular joint apparatus. Saponaro and associates[55] found that postoperative MRI demonstrated 25% of patients with anterior disk displacement without reduction after surgical treatment of a condylar fracture. Long-term follow-up of open reduction patients has been shown to demonstrate significant condylar remodeling, possibly due to vascular disturbance, although function and absence of pain were generally good.[56]

A careful assessment of the literature and the experiences of surgeons indicate that both open reduction and closed treatment of condyle fractures of the mandible deserve a role in the management of these patients.[57] Also, the facility created with open reduction has increased its indications greatly in this decade.[58] Despite a meta-analysis by Laskin and coworkers[59] and the previously cited Cochrane paper, there are no coherent recommendations regarding the optimal treatment of condylar fractures of the mandible. Acceptable outcomes that permit full, pain-free function, with good aesthetics have been extensively demonstrated with both closed and open techniques.

The adverse sequelae of condylar fractures occur in both the open and the closed reduction patient. These include pain, malocclusion, traumatic arthritis, and ankylosis among others. The relative risk of these accepted complications cannot be adequately compared based upon mode of treatment. Early full function and anatomic reduction remain the most important advantages of open treatment. There are complications due to anesthesia and surgery, such as bleeding or infection, that are generally limited to the surgical patient.

Whereas selection of treatment aspires to be an evidence-based process, the surgeon must develop a treatment plan individualized to the patient, the injury, and the environment for care.

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CHAPTER 20

Management of Maxillary Fractures

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As is true of other facial trauma, maxillary fractures are associated more frequently with motor vehicle and motorcycle accidents than with any other cause.[1–5] These fractures most often occur in conjunction with other facial fractures and are most often associated with injuries such as lacerations, other facial fractures, orthopedic injury, and neurologic injury.[1,2,5,6] Most maxillary fractures occur in young men aged 16 to 40 years; they are most common among patients between 21 and 25 years of age, and the risk of sustaining facial bone fractures increases as the age of the patient increases.[6]

HISTORY

Although maxillary fractures are commonly classified according to the Le Fort system, these fractures were described and treated thousands of years before René Le Fort was born. The first clinical examination of a maxillary fracture was recorded in 2500 BC in the Smith Papyrus.[7] Many other early records describe treatments for maxillary fractures or the iatrogenic fracture of the maxilla for therapeutic purposes. In 1822, Charles Fredrick William Reiche provided the first detailed treatise of maxillary fractures, entitled *De Maxillae Superiors Fractura*.[7] In 1823, Carl Ferdinand van Graefe described the use of a head frame for treating a maxillary fracture.[7] His device was as technically complex as those currently in use. In 1859, Bernhard R. K. Von Langenbeck described a technique for the osteoplastic resection of the maxilla.[8] In 1867, David Cheever discussed complete mobilization of the maxilla with the use of chisels for the removal of a nasopharyngeal tumor.[9]

It was not until 1901 that René Le Fort published his landmark works, a three-part experiment using 32 cadavers that were either intact or decapitated.[10–12] The heads of the cadavers were subjected to various types of trauma; the soft tissue was then removed and the bones were examined. Le Fort noted that, generally, if the face was fractured, the skull was not. He then stated that fractures occurred through three weak lines in the facial bony structure: those that protect the cranial cavity, those that circumscribe the midface, and those that cut across the face. From these three lines, the Le Fort classification system was developed (Figure 20-1).

LE FORT CLASSIFICATION SYSTEM

In his description of maxillary fractures, Le Fort considered several factors: the vector of force overcoming the inertia of the face; the thickness of the bone and buttresses counteracting the mass, velocity, and point of application; and the maxilla, which he noticed was unaffected by muscle pull, unlike the long bones. These considerations resulted in a classification of three levels of fracture.

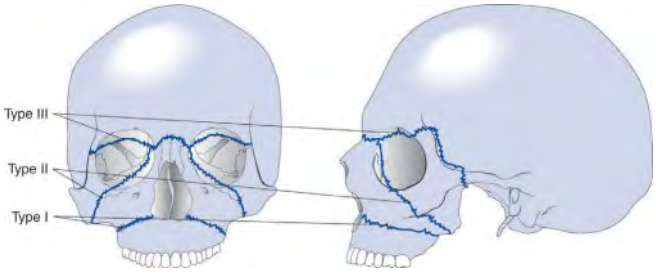


FIGURE 20-1. Anteroposterior and lateral views of the skull show the Le Fort classification system of maxillary fractures.

Le Fort I Level

Maxillary fractures at the Le Fort I level traverse the lateral antral wall, the lateral nasal wall, and the lower third of the septum, and they separate at the pterygoid plates. Thus, the entire mobilized segment consists of the maxillary alveolar bone, the palatine bone, the lower third of the nasal septum, and the lower third of the pterygoid plates. The superior two thirds of these bones remain associated with the face.

Le Fort II Level

Maxillary fractures at the Le Fort II level involve most of the nasal bones, the maxillary bones, the palatine bones, the lower two thirds of the nasal septum, the dentoalveolus, and the pterygoid plates. Unlike the horizontal separation noted in the Le Fort I fracture, the Le Fort II fracture is pyramidal in shape. The fracture extends from below the nasofrontal suture through the nasal bones along the maxilla to the zygomaticomaxillary suture and includes the medial inferior third of the orbit. The fracture then continues along the zygomaticomaxillary suture to and through the pterygoid plates. The septum is also separated superiorly. The segments may be intact below this line of fracture, but they are most often comminuted.

Le Fort III Level

Fractures at the Le Fort III level involve the nasal bones, the zygomas, the maxillae, the palatine bones, and the pterygoid plates. These fractures essentially separate the face along the base of the skull. The fracture line extends from the nasofrontal suture along the medial wall of the orbit through the superior orbital fissure. It then extends along the inferior orbital fissure and the lateral orbital wall to the zygomaticofrontal suture. The zygomaticotemporal suture is also separated. The fracture then extends along the sphenoid bone, separating the pterygoid plates. The septum becomes separated at the cribriform plate of the ethmoid. Le Fort III fractures are most often comminuted. With highly comminuted fractures, patients may sustain fractures at more than one level. Combinations of Le Fort I, II, and III fractures are possible on either side of the face.

Garretson's 1898 treatise[13] stated that the primary method of treating fractures of the maxillae was to construct a bandage or dressing that elevated the mandible into occlusion and to secure the mandible there. A number of materials were used to add stability to these bandages, including plaster of Paris, wood, gutta-percha, and vulcanized rubber. In addition to splinting the jaws, Garretson advocated the use of interdental splints, stating, "As a means of dressing in any complicated jaw fracture, the interdental splint is as invaluable and reliable as it is simple of construction and easy of application."[13]

Blair[14] gave a very good description of the anatomy of maxillary fractures and of the examination to be used for diagnosing such fractures. He noted that mandibular bandages were insufficient to stabilize maxillary fractures, and he advocated a maxillary splint, quoting an authority of the day, Dr. John L. Marshall:

> Impressions of the upper and lower teeth were taken with the modeling compound by first molding it upon the upper teeth and while it was yet soft forcing the lower jaw upward until a correct occlusion of the teeth was obtained. This impression was trimmed to the desired shape; a one-eighth-inch steel wire was imbedded in the sides on a line with the ends of the teeth, then bent backward upon itself opposite the cuspid teeth. . . . From this was constructed a hard-rubber splint, with the wires attached. . . . The splint is held in position by means of double elastic straps attached to the wire on each side and buckled to a close-fitting leather or net cap, which is reinforced with leather and laced firmly on the head. . . . The object of [the splint] was to furnish a sure guide to the normal position of the superior maxillae. Without this the correctness of the adjustment of the bones could not have been verified. Its importance therefore cannot be overestimated.[14]

Similar treatment modalities were presented by Brophy in 1918[15]; he presented illustrations of the splints and preoperative and postoperative images of a patient.

ANATOMY

The two maxillae are paired structures connected by a midline suture; the bones together compose a five-sided pyramid. The anterior surface slopes downward from its superior contact with the frontal and nasal bones at an angle of approximately 15 degrees. The most prominent point at the anterior surface is the anterior nasal spine. A number of protuberances exist on the maxilla, formed by the alveolar base and the origins of the small facial muscles. The lateral surface of the maxillae forms the infratemporal fossae and the buccal vestibule and attaches to the zygoma. Most of the superior surface forms the majority of the orbital floor.

The medial surface of each maxilla forms the midline suture and the lateral nasal walls. This includes the nasal concha and sinus ostia. The ostium of the nasolacrimal duct is beneath the inferior concha. The ostia of the maxillary sinus and middle ethmoids, as well as the opening of the nasofrontal duct, lie beneath the middle concha.

The inferior border composes the palatal vault and the alveolus, which contain the teeth. The posterior border abuts the sphenoid bone and the pterygomaxillary suture.[16] Within the maxilla is the maxillary sinus. This $34 \times 33 \times 25$-mm air cavity is responsible for the weakness of the maxilla. The sinus is present at birth but does not pneumatize to its mature extent until the patient reaches 14 to 15 years of age. Minor changes in the sinus continue throughout life.[17] The strong buttresses of the maxilla are the lateral piriform buttress, the zygomatic buttress, the greater palatine buttress, and the floor of the nose.

The palatine bone is L-shaped and abuts the posterior maxilla as a paired structure. These bones assist the maxilla in forming the posterior sinus, the posterior lateral nasal wall,

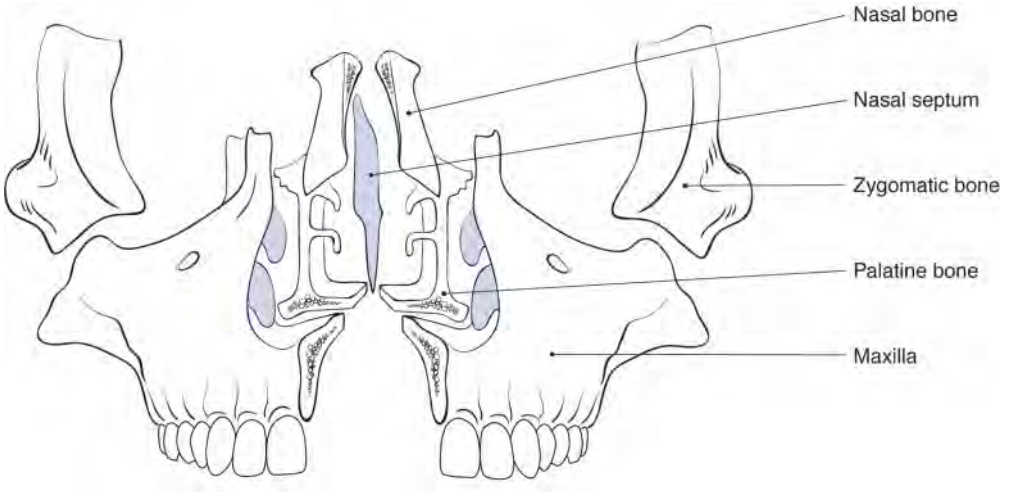


FIGURE 20-2. Disarticulated midfacial skeleton demonstrates the anatomy of the maxilla, the zygoma, the nasal bones, and the nasal septum.

and the pterygomaxillary suture. When joined to the maxilla, the four bones compose one unit (Figure 20-2).[16]

The nasal bones are paired structures that abut the frontal bone superiorly, the maxilla laterally, the septum posteriorly and medially, and each other anteriorly and medially. The bones are thicker superiorly; therefore, fractures at the Le Fort II level may occur inferior to the nasofrontal suture. The nasal septum is a thin trapezoidal bone lying perpendicular to and joining the maxillae and the palatine bones. The superior border is thick and articulates with the ethmoid bone.[16]

The ethmoid bone is cuboidal and extremely pneumatized; thus, it can be easily fractured and comminuted. The cribriform plate of the ethmoid composes the roof of the nasal cavity and communicates with the anterior cranial fossae through multiple foramina for the olfactory nerves. Lateral to the crista galli is a slit through which dura mater is exposed. Posterior and superior movements of the midface can easily comminute this bone, thus disrupting the dura mater and resulting in a cerebrospinal fluid leak.[16]

The zygoma abuts the frontal bone at the frontozygomatic suture and the temporal bone at the zygomaticotemporal suture. The maxilla and zygoma form two thirds of the orbital rim and, along with the palatine bone, one third of the walls and floor of the orbit.

The infraorbital nerve traverses the orbital floor and exits through the infraorbital foramen. The maxillary bone, along with the zygoma, forms the inferior orbital fissure. Through this fissure run the maxillary nerve, the infraorbital vessels, and the ascending branches of the pterygopalatine ganglion. The frontal process of the maxilla contains the lacrimal apparatus, which is housed between the medial canthal ligaments.

The blood supply to the maxillae and the palatine bones is through the periosteum, the incisive artery, and the greater and lesser palatine arteries. The internal maxillary artery, a source of potentially devastating hemorrhage, lies posterior to the maxillae and the palatine bones and anterior to the pterygoid plates of the sphenoid.[18] The blood supply to the nasal septum and the lateral nasal walls is provided by the anterior and posterior ethmoidal arteries, the sphenopalatine artery, and the greater palatine and superior labial arteries.[16]

DIAGNOSIS

Clinical Examination

Advanced trauma life support protocols should be followed for all trauma patients. Detailed examination of maxillofacial fractures is completed in the secondary survey, after the primary survey and successful resuscitation have been completed. As has been done historically, the clinical examination should begin with the initial observation of the patient, followed by palpation of the fractures.[14,19] As Blair wrote in 1914, ". . . In all cases of injury of the face the dental arches and the palate should be inspected, and the facial bones outlined digitally."[14] Lacerations, abrasions, and ecchymotic areas should be recorded. Periorbital ecchymosis and facial edema should be noted and are very typical of these fractures. The patient should be evaluated for epistaxis with any evidence of cerebrospinal fluid leakage. Intraorally, the examiner may see fractured teeth, vestibular ecchymosis and edema, palatal ecchymosis, mucosal lacerations and bleeding, steps or diastema in the maxillary teeth, and malocclusion.

The skeletal framework of the face should be carefully palpated. With respect to the maxilla, the alveolus should be palpated and any fractures or mobility noted. While palpating the forehead, the nasal bridge, and the zygomaticofrontal sutures, the examiner should also observe the maxilla for movement as a unit. A nasal speculum should be used to detect compound fractures of the septum or septal hematoma. Both hands should be used to palpate the orbital rims and, in particular, the zygomaticomaxillary suture. Abnormal occlusion with an anterior open bite and posterior prematurities should be noted and correlated with pretraumatic occlusion if possible (using family members, photographs, dental records as sources). The most obvious sign of maxillary fracture is a mobile maxilla, which can be observed by grasping

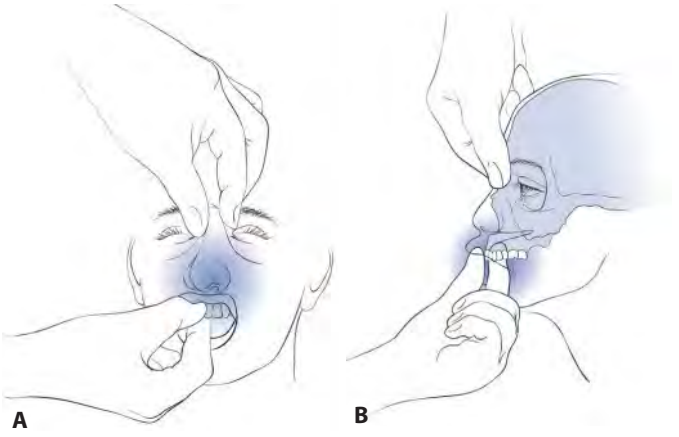


FIGURE 20-3. **A** and **B,** When the maxilla is examined for fracture, the head is stabilized and the dentoalveolar process is manipulated so that gross movements of fractured segments can be detected. Checking for Le Fort II or III fractures requires that one hand holds the bridge of the nose while the other manipulates the maxilla. Movement at the nasofrontal suture suggests a Le Fort II or III fracture.

the anterior maxilla with thumb and forefinger and moving the bone while stabilizing the patient's nasal bridge with the opposite hand (Figure 20-3).

Imaging

Fractures are detected clinically and confirmed radiographically. In the past, the Waters' view and lateral facial radiographs were used for visualizing maxillary fractures and may still be used today in remote areas without access to a computed tomography (CT) scanner (Figure 20-4). The standard imaging modality for diagnosing facial fractures is axial and coronal CT (Figure 20-5). If clinical evidence strongly indicates a maxillary fracture (midface mobility and malocclusion with intact mandible), then CT imaging is a confirmatory test. Important indications for CT scanning are suspected orbital floor fractures (best diagnosed in the coronal view) and surgical planning. CT scans can also demonstrate the soft tissue differences of hematoma or edema of the subcutaneous tissue, muscle, and fat. For severe midface trauma or maxillary displacement, the three-dimensional CT scan is a valuable tool (Figure 20-6).

TREATMENT

As is true of all injuries, initial attention should be directed toward establishing an airway and controlling hemorrhage. The most frequent cause of hemorrhage associated with any Le Fort fracture is a fractured septum. This bleeding may be addressed by placing nasal packs composed of one of a number of materials, including gauze, Merocel (Medtronic Xomed), Rhinorocket (Shippert Medical Technologies Corp.), and Epistat (Medtronic Xomed). Bleeding from sites of laceration or abrasion may be controlled by tamponade. In the setting of maxillary trauma, nasal bleeding that is not



FIGURE 20-4. Waters' radiograph for evaluation of maxillary fractures.

immediately controllable by adequate nasal packing indicates the need for consultation with an interventional radiologist. Exsanguinating hemorrhage is rarely associated with facial fractures; however, its occasional occurrence has long been noted to be due to laceration or rupture of the internal maxillary artery.[15,20] Should uncontrollable bleeding be encountered, the patient should undergo angiographic evaluation with embolization of the injured artery if indicated.[21–27]

Maxillary fractures isolated to the dentoalveolar process and involving bone should be manually reduced and rigidly fixated with arch bars and ligature wires. If the segment is too large to be stabilized with arch bars alone, acrylic can be added to the facial surface of the arch bar or an occlusal splint can be constructed and secured in place. Complications include bone resorption, ankylosis of teeth, external root resorption, and tooth loss.[28,29]



FIGURE 20-5. Computed tomography scan of midface trauma, including maxillary fracture.



FIGURE 20-6. Three-dimensional computed tomography scans provide an overall view of the injuries and offer valuable information.

In more extensive injuries, the sequence of treatment of maxillary fractures depends largely on the associated injuries. Nasotracheal intubation is preferred when it is not contraindicated by the need for complicated repair of nasal and nasoethmoidal injuries. In such cases, a submental intubation technique or tracheotomy is indicated (Figure 20-7).[30–34]

The initial step in the reduction and fixation of maxillary fractures is the placement of arch bars. If teeth are deemed unsalvageable, they should be removed at this time. The sequence of treatment depends on the surgeon's philosophy and the presence of other facial fractures. Whether the surgeon prefers to work from the "bottom up" or from the "outside in," anterior projection of the maxilla is most easily obtained when the mandible is intact. For this reason, strong consideration should be given to repairing any mandible fracture before the maxilla is stabilized. Intermaxillary fixation (to an intact mandible) is the most reliable technique for establishing anterior projection of the maxilla (Figure 20-8).



FIGURE 20-7. The patient's airway is protected by using a submental intubation technique.

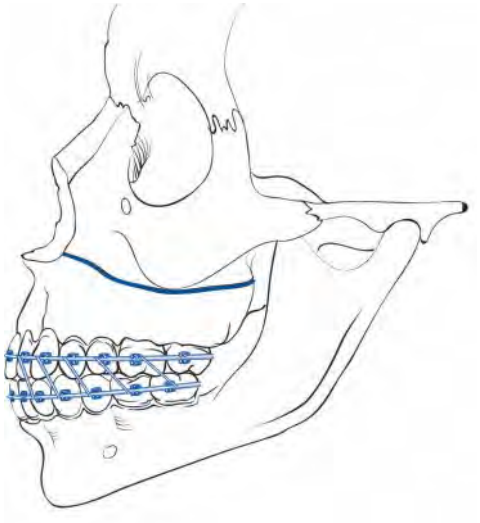


FIGURE 20-8. Arch bars and intermaxillary fixation.

Although many wiring techniques have been described in the past, rigid internal fixation is the standard of care.[19,35–38] The maxilla should be stabilized to the next highest stable facial structure, which varies with Le Fort fracture level. At the Le Fort I level, fixation is placed along the vertical buttresses of the maxilla at the piriform and zygomatic buttresses. At higher Le Fort levels, fixation to the nasal bones, the orbital rims, or the zygomaticofrontal sutures may be necessary. Although Le Fort levels are frequently referred to in discussions of patient treatment, high-quality CT scans and widespread use of rigid fixation have led to the treatment of multiple facial fractures as separate units. For example, a Le Fort I/II fracture would be treated as a Le Fort I fracture, a left orbital fracture, or a left zygomaticomaxillary complex fracture. In these cases, it is advisable to restore midface projection by repairing orbital or zygomatic fractures before performing fixation of the maxilla.

Contemporary bone plates and screws are made of titanium. For maxillary reconstruction, these plates must be of sufficient rigidity to overcome the effects of gravity; the forces of mastication are resisted primarily by bone contact. For providing rigidity, screws with an outer diameter of 1.5 mm are adequate. In areas such as the orbital rim or nasal bone, 1.3- or 1.0-mm systems may be used. In cases in which bone contact is decreased because of comminution, 1.7- or 2.0-mm systems may be used.

If resistance is encountered during mobilization of the maxilla, Rowe disimpaction forceps may be used to help reduce the fracture (Figure 20-9). The paired forceps are placed with the fat end in the nose and the bowed end on the palate. The surgeon stands over the patient's head and disimpacts the maxilla with an inferior-anterior movement. Further assistance may be provided by Hayton-Williams forceps used in conjunction with the Rowe disimpaction forceps.

If the maxillary fracture is incomplete (e.g., greenstick fracture), the surgeon may have difficulty in mobilizing the maxilla. The fractured hemimaxilla may be impacted or

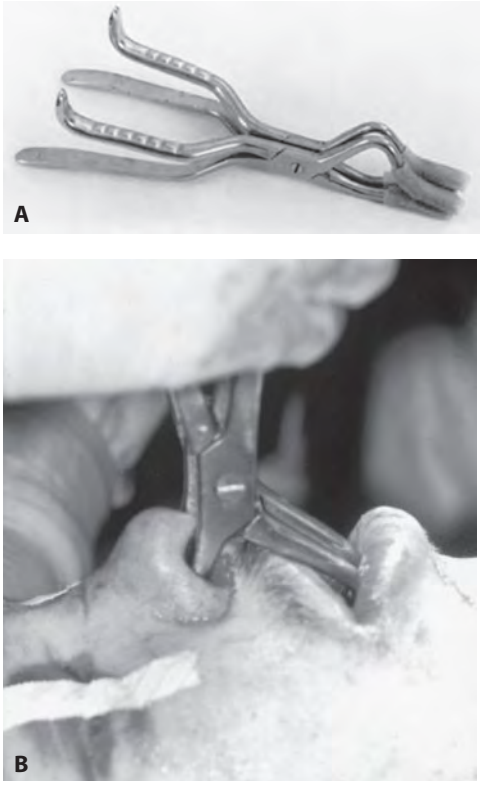


FIGURE 20-9. **A,** Rowe disimpaction forceps. **B,** Application of the forceps.

telescoped, thus causing severe malocclusion with minimal mobility. In a case such as this, severe difficulty with disimpaction of Le Fort level fractures can be easily overcome by completing the fracture with an osteotomy. This concept was described in 1914 by Blair, who wrote, ". . . if the impaction cannot be broken up . . . resort may be had to a small, sharp chisel."[14] After down-fracture, the maxilla can easily be moved into appropriate occlusion and stabilized without further difficulty (Figure 20-10).

Immediate bone grafting has been advocated for the severely comminuted maxillary antrum.[39] This treatment prevents prolapse of the facial soft tissue into the maxillary sinus. Titanium mesh also works for this procedure: it is malleable, can be quickly fixated, resists pressure of the soft tissues of the face, becomes osseointegrated, and allows regrowth of the native tissue (i.e., ciliated respiratory epithelium, goblet cells, squamous epithelium) (Figure 20-11).[40]

Surgical Splints

In the most difficult cases of gross comminution, periodontal disease, or inadequate partial dentition (fewer than three occluding teeth per sextant), occlusal wafers or palatal splints may be helpful. These splints are fabricated after impressions have been taken and model surgery has been completed. The occlusal wafer should be fabricated to cover the occlusal surfaces and the heights of contour, but it should not encroach

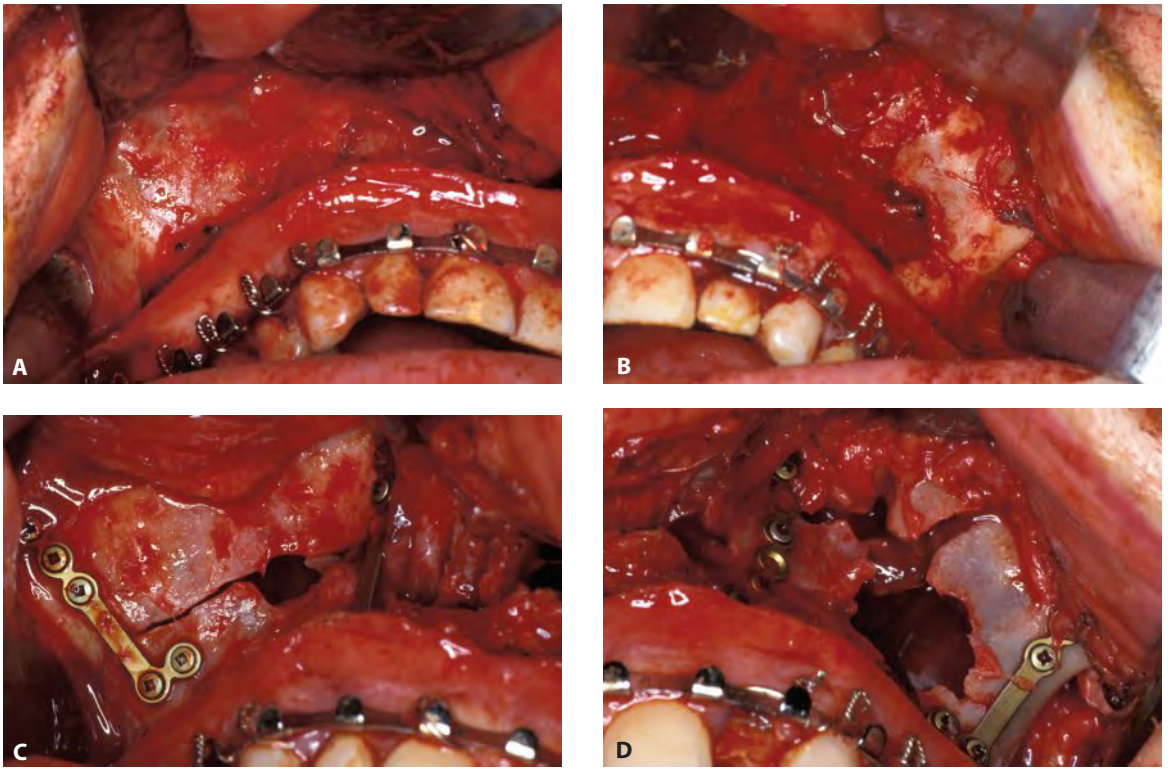


FIGURE 20-10. **A** and **B,** Clinical images show an unfractured right maxillary antrum and a comminuted telescoped left maxillary fracture that was very difficult to reduce. **C** and **D,** After an osteotomy was performed at the Le Fort I level on the right side, the maxilla could be easily mobilized, and the fracture was reduced and fixated without further difficulty.

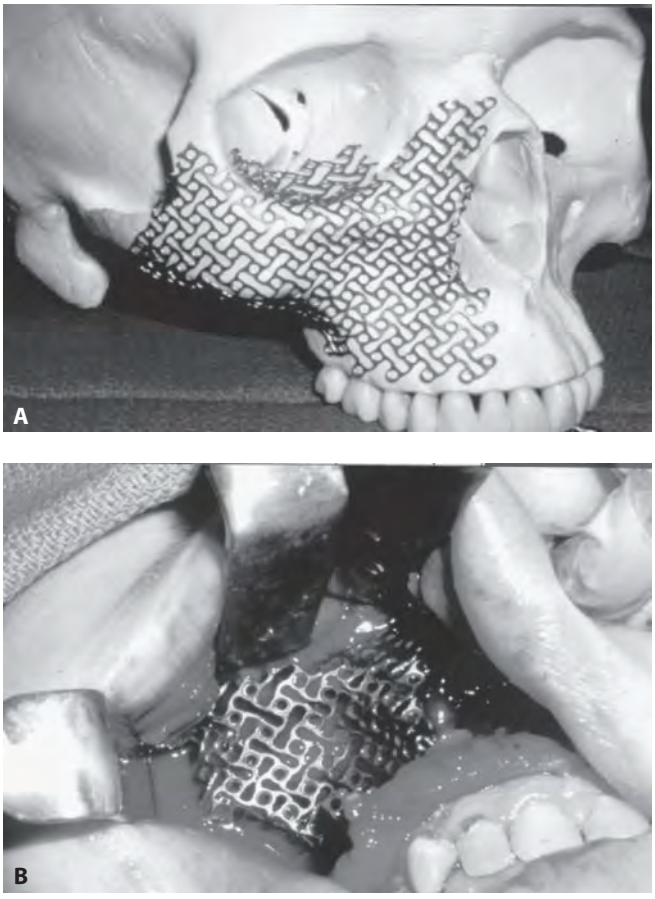


FIGURE 20-11. **A,** Titanium mesh is preformed before it is sterilized and used in maxillary reconstruction. **B,** Intraoperative view of the use of titanium mesh. (**A,** Reproduced with permission from Haug RH, Jenkins WS, Brandt MT. Advances in plate and screw technology: thought on design and clinical applications. Semin Plast Surg 2002;16:219–227.)

on the soft tissues. Holes should be placed between occlusal surfaces in the splint so that it may be ligated separately to the arch bar, perhaps with an orthognathic surgical splint.

The Gunning splint has been used to establish intermaxillary fixation for edentulous patients; this splint is essentially a denture baseplate fabricated to the existing edentulous or partially edentulous ridge; to this baseplace are attached arch bars or suspension brackets.[41] This splint, or even the patient's own dentures, if available, can be secured to the jaws with bone screws or with circummandibular or maxillary wires before maxillomandibular fixation is accomplished.

Special Considerations

High-Force or Avulsive Injuries

High-caliber, high-velocity gunshot wounds, blast injuries, and high-speed motor vehicle accidents with unrestrained victims cause most of the avulsion injuries associated with maxillary fractures. The priority in treating these injuries is to preserve as much of the remaining tissue as possible. Narrow-spectrum antibiotics directed at oral and nasal contaminants, as well as tetanus prophylaxis, are a priority in these injuries. As is true of all injuries, these wounds should be thoroughly evaluated for bleeding, foreign bodies, and extent of damage. Extensive irrigation with pulsed fluids should be used to remove debris.

Life-threatening hemorrhage should be addressed early for homeostasis and for airway management.[42] Hemorrhage that cannot be controlled by local measures such as packing (anterior and posterior) and electrocautery is an indication for angiography and embolization of the injured artery or arteries. Because of the collateral blood supply of the face, most tissues remain viable with only a small isthmus of blood supply. Fractures should be repaired with rigid fixation. Voids in bone should be addressed with a secondary reconstruction. Multiple lacerations with comminuted fractures will be associated with edema and substantial venous congestion. This tissue may provide satisfactory blood supply to existing segments but not to large bone grafts. Next, the soft tissue lacerations should be addressed. Advancement flaps should be used only to cover exposed bone or to correct oronasal or oroantral fistulas. If too little soft tissue exists, flaps should not be advanced; such repairs should be addressed during a secondary reconstruction. Consideration should be given to the use of vascularized free flaps in this situation.[21]

For cases of avulsion, whether free flaps are used or not, implant reconstruction should be considered. Implants with obturators can be used, as is often seen in partial maxillectomy after tumor resection. Implant restorations can also be placed in bone from composite flap reconstructions.[43]

Injuries to Geriatric Patients

The geriatric maxilla is less vascular and has more pneumatized antra, less alveolar bone, and less dense trabeculation. Should reduction and fixation be required, existing dentures may be modified by relining and affixing arch bars or intermaxillary fixation buttons. A Gunning splint may also be fabricated. Such a splint may be fixed to the zygoma, the anterior nasal spine, the piriform rim, or the palate, with either wires or cortical bone screws.

For geriatric patients, additional medical illnesses and disabilities may increase the risk of morbidity due to general anesthesia. The surgeon should exercise judgment when morbid medical conditions coexist with minimally displaced fractures in edentulous patients. A new prosthesis may be more effective than reduction and fixation of the fracture.

Pediatric Maxillary Fractures

Pediatric maxillary fractures are infrequent, and children are nearly five times less likely than adolescents to have facial fractures.[44,45] Because the pediatric sinuses are not highly pneumatized, these fractures tend to be less comminuted in children than in adults. One large review of more than 3000 cases of craniomaxillofacial trauma in children reported 615 facial bone fractures in 389 pediatric patients.[44] Of these fractures, midface fractures accounted for 64.5%, and

fractures assoicated with the maxilla accounted for approximately 20% (defined as one of the following: Lefort I, Lefort II, Lefort III, maxilla right, maxilla left, maxillary process right, maxillary process left). In this review, only 4% of patients were children with facial fractures. In this study, older children (9.7 ± 4 yr) were more likely than younger children to have facial fractures, and boys with fractures outnumbered girls by a ratio of 1.8:1.

A more recent study reviewed 291 pediatric patients with craniomaxillofacial fractures.[46] In this report, most fractures were to the skull vault, and 6% were either maxillary sinus or Le Fort fractures.

Skeletal fixation is indicated for mobile, displaced maxillary fractures in pediatric patients. No long-term studies have definatively reported what alterations in maxillary growth will occur after pediatric maxillary fractures. Avoiding the developing permanent tooth buds is an obvious concern when fixation devices are placed. The use of occlusal splints and skeletal fixation should be considered. Metal fixation systems carry theoretical complications of translocation, extrusion, and growth restriction, and consideration should be given to the removal of hardware in the growing patient.[47]

Resorbable plating systems have been advocated for use in pediatric patients so that potential complications of translocation, extrusion, and growth restriction can be avoided.[47,48] Extensive experience with the use of resorbable plating systems for pediatric patients has been described, and although their specific use for treating maxillary fractures is relatively uncommon (primarily because of the uncommon nature of these injuries), experience with the material, especially in craniofacial surgery, provides strong justification for the use of resorbable materials for this indication.[49–51]

Complications

Complications associated with maxillary fractures and their repair are listed in Table 20-1. A number of these complications may not be readily apparent until weeks or months after injury, but the potential for their occurrence should be borne in mind during evaluation and treatment of the patient.

TABLE 20-1. **Complications Associated with Maxillary Fractures**

Infraorbital nerve paresthesia
Enophthalmos
Infection
Exposed hardware
Deviated septum
Nasal obstruction
Altered vision
Nonunion
Malunion or malocclusion
Epiphora
Foreign body reactions
Scarring
Sinusitis

Adapted from Haug RH, Bradrick JP, Morgan JP. Complications in the treatment of midface fractures. In Kaban LB, Pogrel MA, Perrott DH, editors. Complications in Oral and Maxillofacial Surgery. Philadelphia: WB Saunders; 1997; p. 153.

Perioperative and postoperative airway obstructions are unusual in cases of maxillary fracture alone. However, these conditions may occur in association with extubation while the patient is obtunded, with a septal hematoma or nasal packing, and with excessively edematous soft tissues that do not allow breathing through the nasal airways. Patients with intermaxillary fixation and complete dentition may have difficulty breathing during this time. Reintubation, opening nasopharyngeal airways, or merely removing the intermaxillary fixation may be effective. Uncorrected nasal septal fractures can lead to postoperative airway obstruction that remains after all soft tissue swelling has resolved. Acute sinusitis can result from prolonged nasotracheal intubation.[52] Acute or chronic sinusitis may also occur in the ethmoid, sphenoid, frontal, and maxillary sinuses because fractures may obliterate or obstruct the sinus ducts or ostia.

Postoperative hemorrhage occurs if arterioles and veins are not ligated when lacerations are repaired, if inadequate bone reduction allows continued oozing of blood, if an aneurysm is present, or if an artery is partially transected. Lacerations should be reexplored so that hemorrhage can be controlled. Hematomas should be drained. Oozing of blood from bone requires repeated reduction or the use of bone wax. Hemorrhage from a major artery requires emergency tamponade; if the source of bleeding cannot be identified, then arteriography and embolization are indicated. Aneurysms and pseudoaneurysms are complications of maxillofacial trauma but rarely occur as the result of isolated maxillary fractures. They can also result in postoperative bleeding and are indications for angiography and embolization.[53]

Because of the proximity of the maxilla to the orbits, complications associated with vision can occur. Blindness is rarely associated with midface fractures and is most frequently seen in fracture patterns involving the orbit, often with a more severe mechanism of injury.[54] Immediate postoperative blindness can be a complication of the reduction of high Le Fort fractures (Le Fort III or fractures involving the orbits) and occurs because of increased intraorbital hemorrhage or pressure, a retinal artery spasm, retrobulbar hemorrhage, or the impingement of bone fragments on the optic nerve.[55] An undiagnosed or inadequately treated orbital floor fracture (alone or in combination with a zygomatic component) can lead to enophthalmos and diplopia.

The most obvious postoperative complications are misplaced bone segments or fixation devices. These complications are readily identified by clinical examination (e.g., malocclusion) or postoperative radiographic examinations. A second surgical procedure will correct such complications. Other complications related to rigid internal fixation include palpability, infection, extrusion or exposure, translocation, stress shielding, cortical osteopenia, and nonunion.[56,57] Nonunion of the fractured segments can occur as the result of inadequate blood supply, inaccurate position, movement of

segments, infection, or nutritional deficiencies.[58] Infections may be caused by contaminated soft tissue lacerations or foreign bodies, hematomas, or odontogenic infections from previously diseased or fractured teeth. Injury to teeth can be due to the original trauma or to placement of fixation screws. Infection around bone plates and screws can occur years after their placement.

Malunion of maxillary fractures can obstruct the nasolacrimal ducts. This obstruction causes epiphora and may lead to episodes of dacryocystitis. Bone segments from fractured or improperly reduced maxillary fractures can also impinge on the infraorbital nerve, causing numbness of the distribution of the second division of the trigeminal nerve.

Although the reduction and fixation of maxillary fractures may at times seem straightforward, the proximity of complicated anatomic structures and the consequences of inaccurate repair make it incumbent on the surgeon to follow sound surgical principles in the management of these fractures.

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21

CHAPTER

Management of Zygomatic Complex Fractures

Jonathan S. Bailey, DMD, MD, FACS

The zygoma articulates with the frontal, sphenoid, temporal, and maxillary bones and contributes significantly to the strength and stability of the midface. The forward projection of the zygoma causes it to be injured frequently.[1] The zygoma may be separated from its four articulations. This is called a *zygomatic complex fracture.* The terms *trimalar* or *tripod fracture* are, therefore, inaccurate. These terms reflect an inability to easily identify the orbital (zygomaticosphenoid) portion of the injury before the advent of computed tomography (CT). The zygomatic arch may be fractured independently or as part of a zygomatic complex fracture.

The cause of zygomatic injuries varies with patient demographics and the location of the reporting institution. Matsunaga and and coworkers[2] at Los Angeles County/University of Southern California Medical Center found that a majority of the 1200 zygomatic fractures studied were the result of motor vehicle accidents (MVAs). In contrast, Ellis and colleagues[3] found that 80% of zygomatic fractures in Glasgow, Scotland, resulted from assaults, falls, or sports injuries. Only approximately 13% of fractures in this series involved MVAs.

In this chapter, the anatomic features, diagnosis, management, and complications of zygomatic complex fractures are discussed.

SURGICAL ANATOMY

The zygoma has four projections, which create a quadrangular shape: the frontal, temporal, maxillary, and infraorbital rim. The zygoma articulates with four bones: the frontal, temporal, maxilla, and sphenoid. A zygomatic complex fracture includes disruption of the four articulating sutures: zygomaticofrontal, zygomaticotemporal, zygomaticomaxillary, and zygomaticosphenoid sutures (Figure 21-1A and B).

All zygomatic complex fractures involve the orbital floor, and therefore, an understanding of orbital anatomic features is essential for those treating these injuries. The orbit is a quadrilateral pyramid that is based anteriorly. The orbital floor slopes inferiorly and is the shortest of the orbital walls, averaging 47 mm.[4] It is composed of the orbital plate of the maxilla, the orbital surface of the zygomatic bone, and the orbital process of the palatine bone.

The medial and lateral walls converge posteriorly at the orbital apex. The medial wall consists of the frontal process of the maxilla, the lacrimal bone, the orbital plate of the ethmoid, and a small portion of the sphenoid body. The lateral orbital wall is the thickest and is formed by the zygoma and the greater wing of the sphenoid.

The orbital roof is composed of the frontal bone and lesser wing of the sphenoid (see Figure 21-1C).

The zygomatic arch includes the temporal process of the zygoma and the zygomatic process of the temporal bone. The glenoid fossa and articular eminence are located at the posterior aspect of the zygomatic process of the temporal bone.

The sensory nerve associated with the zygoma is the second division of the trigeminal nerve. The zygomatic, facial, and temporal branches exit the foramina in the body of the zygoma and supply sensation to the cheek and anterior temporal region. The infraorbital nerve passes through the orbital floor and exits at the infraorbital foramen (see Figure 21-1C). It provides sensation to the anterior cheek, lateral nose, upper lip, and maxillary anterior teeth. Muscles of facial expression originating from the zygoma include the zygomaticus major and labii superioris. They are innervated by cranial nerve VII. The masseter muscle inserts along the temporal surface of

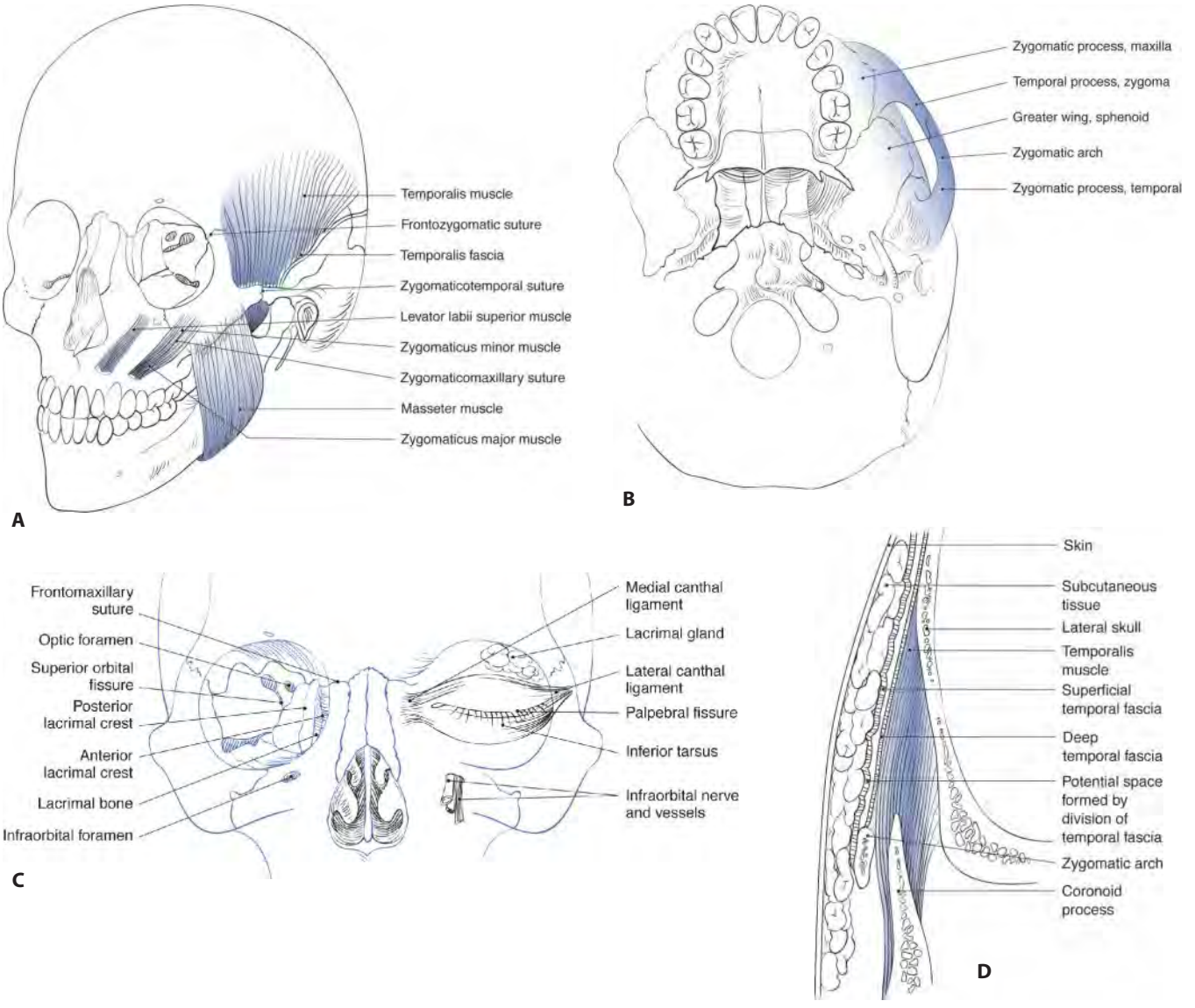


FIGURE 21-1. **A,** Relation of muscles and cranial bones to the zygomatic complex as seen in frontal or lateral view. **B,** Relation of the skull to the zygomatic complex as seen from a submental view. **C,** Relation of soft tissues, muscle, and nerves to the orbit as seen from a frontal view. **D,** Frontal view of fascia and muscle attachment to the skull, zygomatic arch, and coronoid process. (**A–D,** Adapted from Perrott DH, Kaban LB. Management of zygomatic complex fractures. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 1. Philadelphia: JB Lippincott; 1992; pp. 490–491.)

the zygoma and arch and is innervated by a branch of the mandibular nerve (see Figure 21-1A).

The temporalis fascia attaches to the frontal process of the zygoma and zygomatic arch (see Figure 21-1D). The fascia produces resistance to inferior displacement of a fractured fragment by the downward pull of the masseter muscle.

The position of the globe in relation to the horizontal axis is maintained by Lockwood's suspensory ligament. This attaches medially to the posterior aspect of the lacrimal bone and laterally to the orbital (Whitnall's) tubercle (which is 1 cm below the zygomaticofrontal suture on the medial aspect of the frontal process of the zygoma). The shape and location of the medial and lateral canthi of the eyelid are maintained by the canthal tendons. The lateral canthal tendon is attached to Whitnall's tubercle. The medial canthal tendon is attached to the anterior and posterior lacrimal crests. Zygomatic complex fractures are often accompanied by an antimongoloid (downward) cant of the lateral canthal region caused by displacement of the zygoma (see Figure 21-1C).

DIAGNOSIS

Zygomatic fractures are not life-threatening and are usually treated after more serious injuries are stabilized and swelling has resolved 4 to 5 days after injuries.

Initial evaluation of the patient with a zygomatic fracture includes documentation of the bony injury and the status of surrounding soft tissue (eyelids, lacrimal apparatus, canthal

tendons, and globe) and cranial nerves II to VI. Visual acuity and the status of the globe and retina should be established; an ophthalmologist should be consulted for suspected or questionable ophthalmic injury.

History

The nature, force, and direction of the injuring blow should be determined from the patient and any witnesses. A direct lateral blow, as in an assault, often results in an isolated zygomatic arch or an inferomedially displaced zygomatic complex fracture. A frontal blow usually produces a posteriorly and inferiorly displaced fracture.

The patient with a zygomatic complex fracture complains of pain, periorbital edema, and ecchymosis. There may be paresthesia or anesthesia over the cheek, lateral nose, upper lip, and maxillary anterior teeth resulting from injury to the zygomaticotemporal or infraorbital nerves. This occurs in 18% to 83% of all patients with zygomatic trauma.[3,5–8] When the arch is medially displaced, the patient may complain of trismus. Epistaxis and diplopia may be present.[3]

Physical Examination

Ecchymosis and edema are the most common early clinical signs and are present in 61% of all zygomatic injuries.[2] Depression of the malar eminence and infraorbital rim produce flattening of the cheek. Subconjunctival hemorrhage is often noted. Downward displacement of the zygoma produces an antimongoloid slant to the lateral canthus, enophthalmos, and accentuation of the supratarsal fold of the upper eyelid (Figure 21-2). Lacerations in the facial region should lead the surgeon to suspect underlying fracture.

Palpation of the zygomaticofrontal suture, the entire 360 degrees of the orbital rim, and the zygomatic arch should be carried out in an orderly fashion. Tenderness, a step-off, or separation at the sutures are indicative of a fracture. Intraorally, disruption at the zygomaticomaxillary buttress area is palpable, and ecchymosis in the region of the canine fossa may be visible. The range of mandibular motion is evaluated to rule out impingement of the zygomatic arch on the coronoid process.



FIGURE 21-2. **A,** A 22-year-old male who sustained a blow to the right cheek. Frontal photograph illustrates the typical signs of zygomatic complex fracture: periorbital ecchymosis, edema, antimongoloid slant, and subconjunctival hemorrhage. **B,** A 38-year-old male who sustained a blow to the left cheek 2 weeks before presentation. Frontal photograph demonstrates resolving periorbital ecchymosis and malar depression.

In isolated zygomatic arch fractures, a depression is observed and palpated anterior to the tragus (Figure 21-3). Pain and decreased mandibular motion are commonly present with these injuries, whereas orbital signs are usually absent.

Evaluation of the eye includes documentation of visual acuity, pupillary response to light, fundoscopic examination, ocular movement, and globe position. Limitation of motion of the extraocular muscles, diplopia, and enophthalmos may be noted if significant fractures of the orbital floor or medial or lateral walls are present. Lack of pupillary response and ptosis are present if cranial nerve III has been injured. Injuries to the optic nerve, hyphema, injury to the globe, retroorbital hemorrhage, retinal detachment, and disruption of the lacrimal ducts may also be present.

Neurologic examination includes careful evaluation of all cranial nerves, with special attention directed toward cranial nerves II, III, IV, V, and VI.

A retrospective study by Barry and associates[9] evaluated 138 patients with orbitozygomatic complex fractures and identified 23 patients with associated ocular injuries. The most common injuries included diplopia, enophthalmos, retinal tear, hyphema, angle recession, and traumatic mydriasis. Likewise, Jamal and coworkers[10] evaluated 98 patients with zygomaticomaxillary complex fractures to identify minor and major ocular injuries. The authors categorized the ocular injuries as "minor" if the injury would likely not result in permanent visual sequelae and as "major" those with a potential for permanent visual compromise. In this population, 66.6% sustained minor ocular injuries such as subconjunctival hemorrhage and microhyphema. Ten percent suffered major ocular injuries such as ruptured globe, retinal hemorrhage or detachment, and hyphema. Both studies strongly recommend prompt preoperative ophthalmologic consultation for all patients with zygomaticomaxillary complex fractures.

Radiographic Evaluation

The diagnosis of zygomatic fractures is usually established by history and physical examination. CT scan of the facial bones, in axial and coronal planes, is standard for all patients with suspected zygomatic fractures.[11–13] Radiographs are helpful for confirmation of the extent of the injury and for medicolegal documentation.

CT is the gold standard for radiographic evaluation of zygomatic fractures. Axial and coronal images are obtained to define fracture patterns, degree of displacement, and comminution and to evaluate the orbital soft tissues. Specifically, CT scans allow for visualization of the buttresses of the midfacial skeleton: nasomaxillary, zygomaticomaxillary,

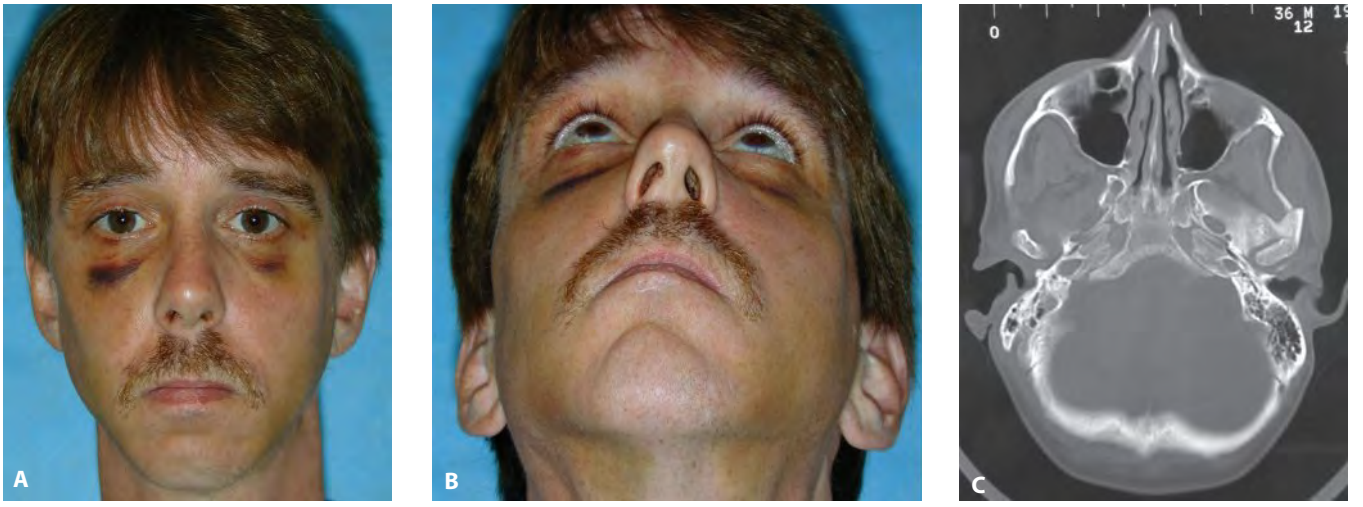


FIGURE 21-3. A 36-year-old male who sustained a blow to the left cheek. **A,** Frontal photograph illustrates the typical findings of a zygomatic arch fracture: preauricular depression. **B,** Worm's eye view. **C,** Axial computed tomography (CT) scan demonstrates an isolated depressed left zygomatic arch fracture.

infraorbital, zygomaticofrontal, zygomaticosphenoid, and zygomaticotemporal buttresses. Coronal views are particularly helpful in the evaluation of orbital floor fractures (Figure 21-4A).[12] Soft tissue windows, in the coronal plane, are useful to evaluate the extraocular muscles and to evaluate for herniation of orbital tissues into the maxillary sinus.

Plain Radiographs

CT scans have replaced plain films for the diagnosis and management of zygomatic complex fractures. However, a fundamental working knowledge of this technique is required. In many emergency rooms and hospitals, trauma patients will still have plain film radiographic evaluation. The ability to read and interpret these films to diagnose and treat these patients is mandatory.

Waters' View

The single best radiograph for evaluation of zygomatic complex fractures is Waters' view. It is a posteroanterior projection with the head positioned at a 27-degree angle to the vertical and the chin resting on the cassette. This projects the petrous pyramids off the maxillary sinuses, permitting visualization of the sinuses, lateral orbits, and infraorbital rims (see Figure 21-4B) When this is combined with an erect Waters view, a stereographic view of the fracture can be obtained. In patients who are unable to assume a facedown position, a reverse Waters projection provides similar information.

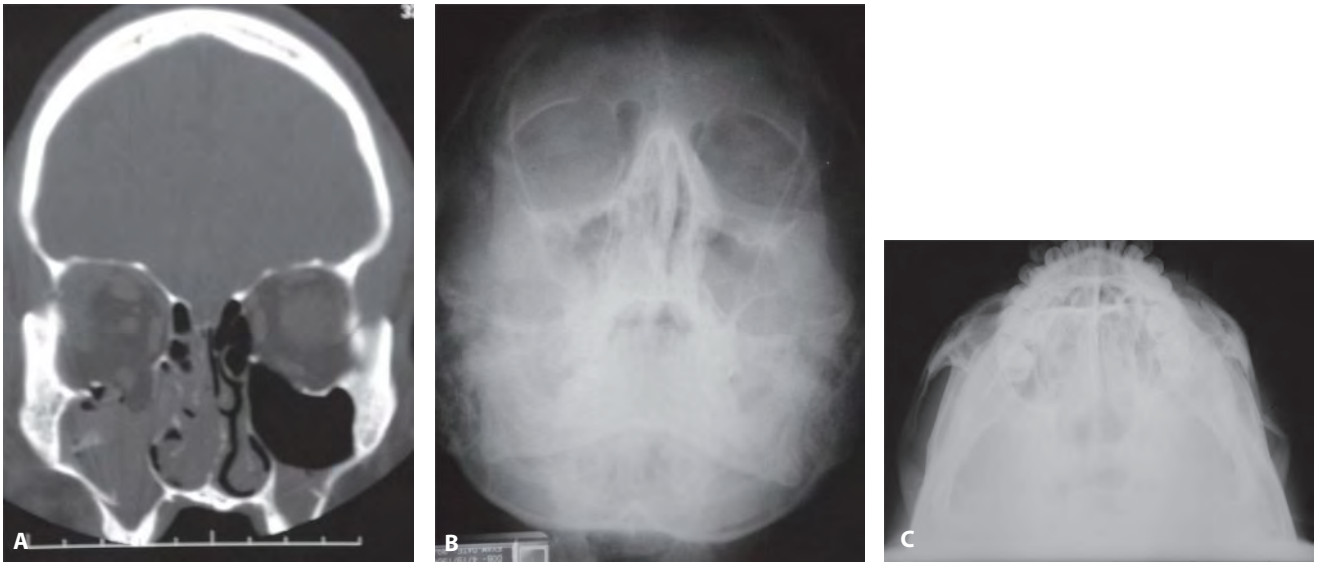


FIGURE 21-4. **A,** Coronal CT scan demonstrates a right zygomaticomaxillary buttress and orbital floor fracture with herniation of orbital contents into the maxillary sinus. **B,** Waters' view demonstrates a right zygomatic complex fracture. **C,** Submentovertex view demonstrates a displaced left zygomatic arch fracture.

Caldwell's View

Caldwell's view is a posteroanterior projection with the face at a 15-degree angle to the cassette. This study is helpful in the evaluation of rotation (around a horizontal axis).

Submentovertex View

The submentovertex (jug-handle) view is directed from the submandibular region to the vertex of the skull. It is helpful in the evaluation of the zygomatic arch and malar projection (see Figure 21-4C).

Classification of Fractures

Historically, the classification of zygomatic fractures was used to predict which fractures would remain stable after reduction. Clinically, this would allow the surgeon to identify those fractures that would require open reduction and some method of fixation.

In 1961, Knight and North[14] classified zygomatic fractures by the direction of displacement on a Waters view radiograph. With the advent of CT scans and the increased use of rigid internal fixation, more modern classification schemes aim to identify those fractures that require aggressive surgical approaches.

In 1990, Manson and colleagues[11] proposed a method of classification based on the pattern of segmentation and displacement. Fractures that demonstrated little or no displacement were classified as *low-energy injuries.* Incomplete fractures of one or more articulations may be present. *Middle-energy fractures* demonstrated complete fracture of all articulations with mild to moderate displacement. Comminution may be present (Figure 21-5). *High-energy injuries* were characterized by comminution in the lateral orbit and lateral displacement with segmentation of the zygomatic arch (Figure 21-6).

Gruss and associates[15] proposed a system that stressed the importance of recognizing and treating zygomatic arch fractures in association with the zygomatic body. Like Manson and colleagues,[11] Gruss and associates stressed the importance of identifying and treating segmentation, comminution, and lateral bowing of the zygomatic arch.

Zingg and coworkers,[7] in a review of 1025 zygomatic fractures, classified these injuries into three categories. *Type A fractures* were incomplete low-energy fractures with fracture of only one zygomatic pillar: the zygomatic arch, lateral orbital wall, or infraorbital rim. *Type B fractures* were designated complete "monofragment" fractures with fracture and displacement along all four articulations. *Type C "multifragment" fractures* included fragmentation of the zygomatic body.

Although all three classification schemes vary to some degree, each method notes that as the amount of displacement and comminution increases, the role of open reduction and internal fixation increases.

SECTION 3

PRINCIPLES OF SURGICAL TREATMENT

Treatment of zygomatic fractures must be based on a complete preoperative evaluation. This includes a CT scan with axial and coronal images to fully appreciate the nature of the injury. Classification techniques, if they are accepted, are helpful to standardize terminology, plan treatment, and predict prognosis. However, the surgeon must individualize treatment based on a combination of history, physical examination, radiographic findings, and sound clinical judgment.

Management of zygomatic complex and zygomatic arch fractures depends on the degree of displacement and the resultant aesthetic and functional deficits. Treatment may, therefore, range from simple observation of resolving

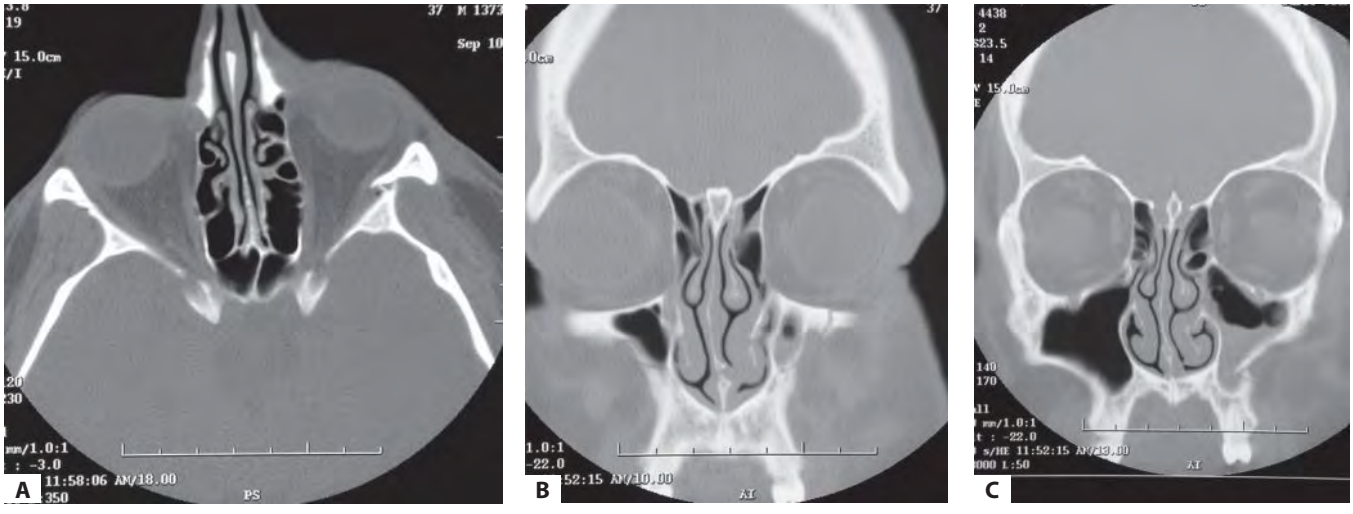


FIGURE 21-5. Middle-energy fracture. **A,** Axial CT scan demonstrates displacement of the lateral orbital wall. **B,** Coronal CT scan demonstrates fracture and minimal displacement of the infraorbital rim. **C,** Coronal CT scan demonstrates mild displacement of the zygomaticomaxillary buttress.

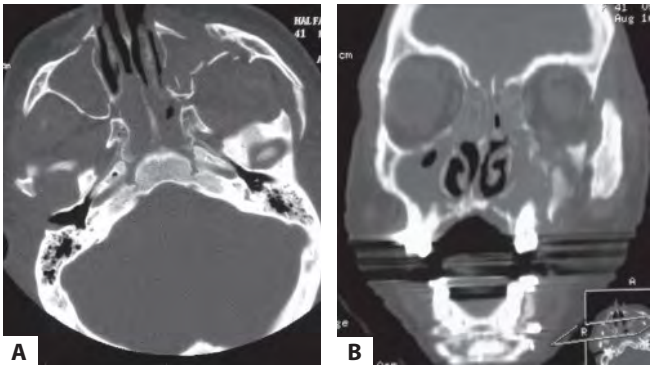


FIGURE 21-6. High-energy fracture. **A,** Axial CT scan demonstrates lateral bowing and segmentation of the zygomatic arch. **B,** Coronal CT reconstructions demonstrate comminution of the zygomaticomaxillary buttress, infraorbital rim, and orbital floor. This patient was unable to be positioned for true coronal scans because of a cervical spine injury. Note disruption of the right globe on coronal images resulting from orbital globe rupture.

swelling, extraocular muscle dysfunction, and paresthesia to open reduction and internal fixation of multiple fractures.

Zygomatic Arch Fractures

Nondisplaced and minimally displaced zygomatic arch fractures may require no surgical correction. Because these injuries usually do not result in significant functional deficits, it may be appropriate simply to observe the patient.

Duverney was the first surgeon to describe an operative technique for treatment of a fractured zygomatic arch.[16] He used intraoral finger pressure to elevate the depressed arch. Alternatively in this technique, the patient is instructed to bite on a block of wood, which results in temporalis muscle and tendon tension. This force, along with finger pressure in an outward direction, reduces the fracture.[16]

Goldthwaite in 1924[17] was the first to describe an intraoral approach to the zygomatic arch through a stab wound in the buccal sulcus. A sharp elevator is passed superiorly through the vestibule and behind the maxillary tuberosity and forward pressure is applied to reduce the arch.

Quinn[18] modified this technique by making an incision in the mucosa at the level of the maxillary alveolus and extending it inferiorly along the anterior border of the ramus. The dissection continues along the lateral aspect of the coronoid process, ending at the level of the maxillary alveolus and extending it inferiorly along the anterior border of the ramus. The dissection continues along the lateral aspect of the coronoid process, ending at the level of the zygomatic arch at the site of the fracture. An elevator is placed between the coronoid process and the zygomatic arch, and the fracture is reduced.[18]

The standard technique for treatment of zygomatic arch fractures, first described by Gillies and colleagues in 1927,[19] can also be used to reduce zygomatic complex fractures. A temporal incision (2 cm in length) is made behind the hairline. The dissection continues through the subcutaneous and superficial temporal fascia down to the glistening white deep temporal fascia (Figure 21-7A). The temporal fascia is incised horizontally to expose the temporalis muscle. A sturdy elevator, such as a urethral sound or Rowe zygomatic elevator, is inserted deep to the fascia, underneath the temporal surface of the zygoma. The elevator must pass between the deep temporal fascia and the temporalis muscle or it will be lateral to the arch. The bone should be elevated in an outward and forward direction, with care taken not to put force on the temporal bone (see Figure 21-7B). The arch should be

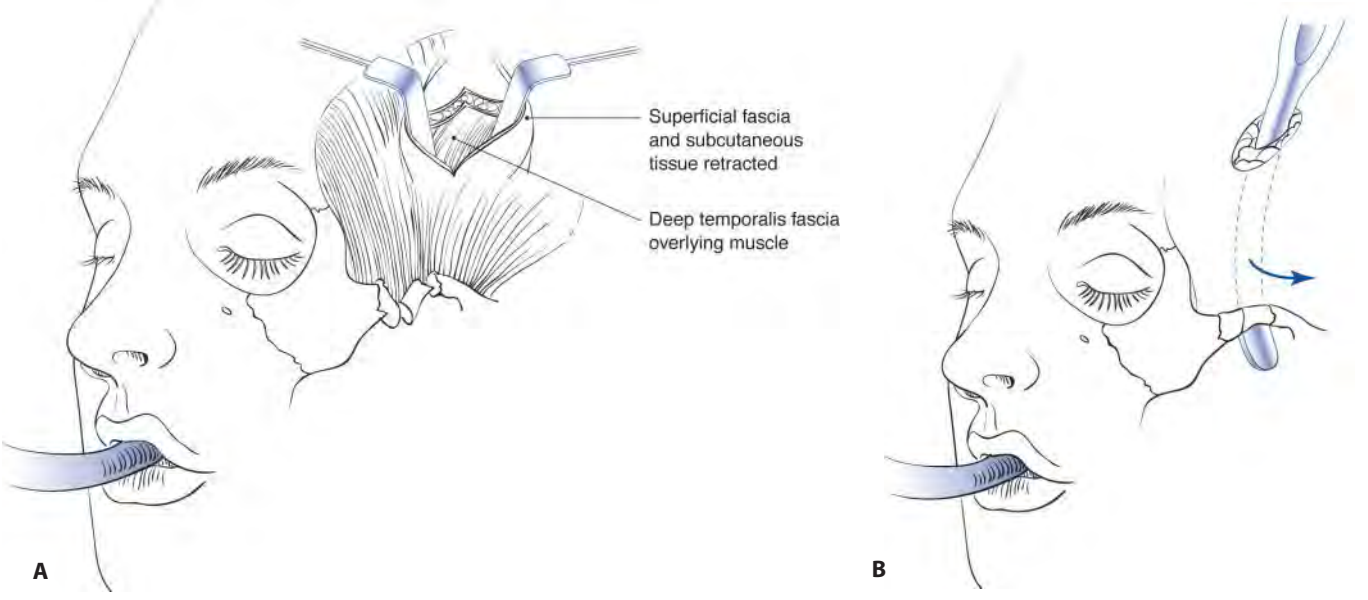


FIGURE 21-7. Gillies' approach to reduce a zygomatic arch fracture. **A,** Temporal incision through the subcutaneous and superficial fascia down to the deep temporal fascia. **B,** Reduction of fracture with an elevator. (**A** and **B,** Adapted from Perrott DH, Kaban LB. Management of zygomatic complex fractures. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 1. Philadelphia: *JB Lippincott;* 1992; p. 498.)

palpated at all times as a guide to proper reduction. The wound is closed in layers.

In a series of 2067 zygomatic fractures, Ellis and colleagues[3] found 10 of 136 isolated zygomatic arch fractures required some form of fixation. Numerous methods of stabilization for zygomatic arch fractures have been proposed. These include temporarily packing the temporal fossa with $^1/_2$-inch gauze, a nasogastric tube, or a urinary catheter.[20–22] More conveniently, a transcutaneous circumzygomatic arch wire can be passed and tightened over a foam-backed aluminum eye shield to suspend the arch.

Open reduction with internal fixation is seldom necessary for treatment of isolated zygomatic arch fractures. However, internal fixation with miniplates may be required as part of the management of high-energy comminuted zygomatic complex or panfacial fractures.

Zygomatic Complex Fractures

Low-Energy Zygomatic Complex Fractures

Low-energy, nondisplaced or minimally displaced zygomatic complex fractures may require no operative correction. The patient should be observed longitudinally for signs of displacement, extraocular muscle dysfunction, and enophthalmos after swelling resolves. Stable, minimally displaced zygomatic complex fractures without significant clinical findings may require no treatment. The patient should be made to appreciate the risk of residual asymmetry of the cheek, orbit, and eyelid if the fracture is not reduced. Documentation, including photographs, is recommended.[23]

Middle-Energy Zygomatic Complex Fractures

Middle-energy, displaced zygomatic complex fractures require reduction and internal fixation. Since the 1990s, there has been an increase in the use of open reduction and internal fixation. In 1984, Zachariades and associates managed 45% of all zygoma fractures with the Gillies technique. At the same institution, in 1995, only 2.5% of these fractures were treated by this same method.[24]

In 1996, Ellis and Kittidumkerng[25] proposed an algorithm of treatment for isolated middle-energy zygomatic complex fractures that did not require orbital reconstruction (Figure 21-8). The initial step in this algorithm is reduction of the fracture. Ellis and others recommend the use of a Carroll-Girard screw, which is inserted into the malar eminence (Figure 21-9). The Carroll-Girard screw provides excellent three-dimensional control to reduce the fracture.

If the reduction is unstable, or if there is question regarding the accuracy of the reduction, the author recommends proceeding to open reduction and internal fixation. The zygomaticomaxillary buttress is exposed first and stabilized with a plate if necessary.

The zygomaticofrontal buttress is exposed next and also stabilized with a plate if required. This method requires proper patient selection, experience, and meticulous technique to ensure accurate reduction and stabilization.

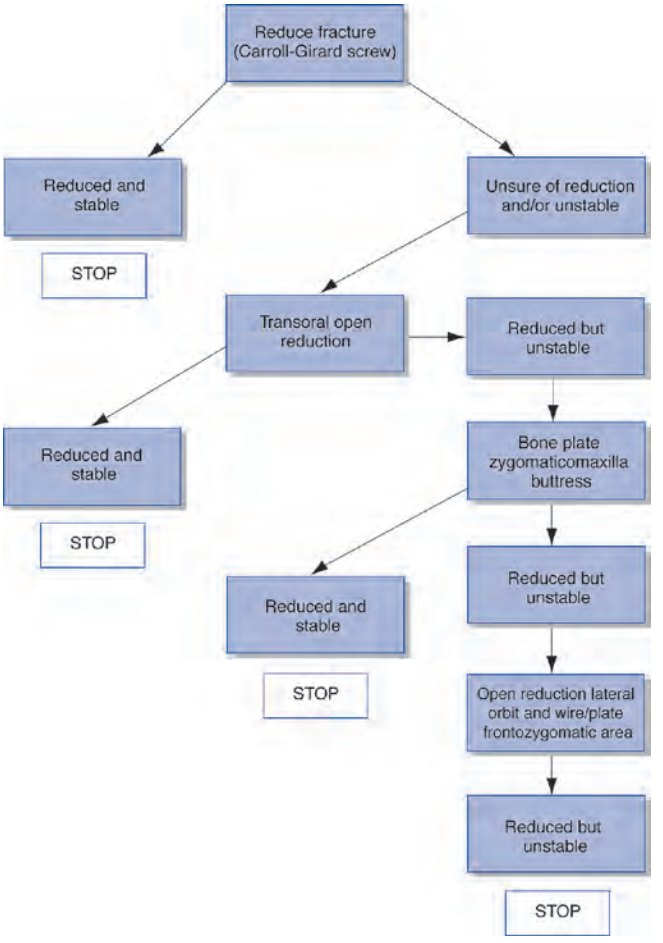


FIGURE 21-8. Zygomatic complex fracture without need for internal orbit reconstruction. (Adapted from Ellis E, Kittidumkerng W. Analysis of treatment of isolated zygomaticomaxillary complex fractures. *J Oral Maxillofac Surg* 1996;54:386.)

Other authors recommend routine exposure of two or more of the three anterior buttresses for middle-energy injuries: the zyomaticomaxillary buttress, zygomaticofrontal buttress, and the infraorbital rim (Figures 21-10 to 21-12). In this manner, multiple buttresses are visualized and the three-dimensional accuracy of the reduction can be confirmed.[26–30]



FIGURE 21-9. Carroll-Girard screw placed transcutaneously for reduction of a zygomatic complex fracture.

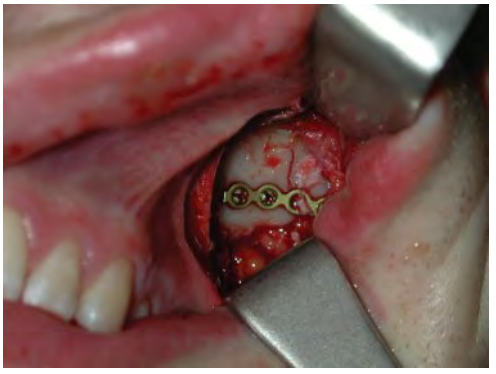


FIGURE 21-10. Intraoral exposure and fixation of a zygomaticomaxillary buttress fracture.



FIGURE 21-11. Exposure and fixation of a zygomaticofrontal buttress fracture via a supratarsal fold incision.



FIGURE 21-12. Exposure and fixation of an infraorbital rim fracture via a subciliary incision.

High-Energy Zygomatic Complex Fractures

A more aggressive surgical approach should be planned to treat high-energy fractures (Figure 21-13).[15,25,26,31] There is often significant comminution of the anterior buttresses, making anatomic reduction difficult. With segmentation of the zygomatic arch, it is impossible to control this posterior buttress. In addition, these fractures often require orbital reconstruction.

To restore proper projection, facial width, and orbital volume, exposure of the zygomatic arch and orbital floor is often required in addition to exposure of the anterior buttresses. A coronal flap may be used to gain access to the zygomatic arch. A transcutaneous or transconjunctival incision is used to explore and reconstruct the internal orbit. With wide intraorbital exposure, the broad sphenozygomatic suture may also be visualized to aid in anatomic reduction and may also provide another point of fixation.[15,25,26,31,32]

Internal Fixation of Zygomaticomaxillary Complex Fractures

Historically, many methods have been used for stabilization of zygomatic complex fractures. These have included antral packing, percutaneous wire fixation, and wire osteosynthesis. It is now accepted that miniplate or microplate fixation provides the best results and minimal complications.[24,33–35]

Controversy exists regarding the best location for internal fixation and the number and type of plates required. Multiple studies have tried to characterize the forces placed on the zygomatic complex and the amount of fixation required to achieve "stability."[25,26,32,36–43] These forces include the masseter and temporalis muscles and fascia and soft tissue contracture, which cause rotational movements in multiple axes around the zygomatic buttresses. Internal fixation must provide enough strength to resist these forces.

For low- and middle-energy fractures, stable fixation can be achieved at one or more of the anterior buttresses. The location of fixation and number of sites of fixation depends on the fracture pattern, location, vector of displacement, and degree of instability. Occasionally one-point fixation may be adequate.[7,25,28,33,40] More commonly, two- or three-point stabilization is required.[7,25,26,28,29,33,44]

For high-energy injuries, a fourth point of fixation is required. The zygomatic arch is typically comminuted and laterally displaced. Open reduction and internal fixation is required to restore proper facial width and projection.[7,15,25,26,33,44]

Sequence of Internal Fixation

As in the treatment of panfacial fractures, a systematic approach is helpful to ensure accurate restoration of facial height, width, and projection.[31,41]

For more complex middle-energy injuries that may require exposure of all three anterior buttresses, the zygomaticofrontal fracture may be first stabilized temporarily with an interosseous wire.[28,31] This is followed by fixation of the zygomaticomaxillary fracture and the infraorbital rim if indicated. The temporary wire at the zygomaticofrontal fracture

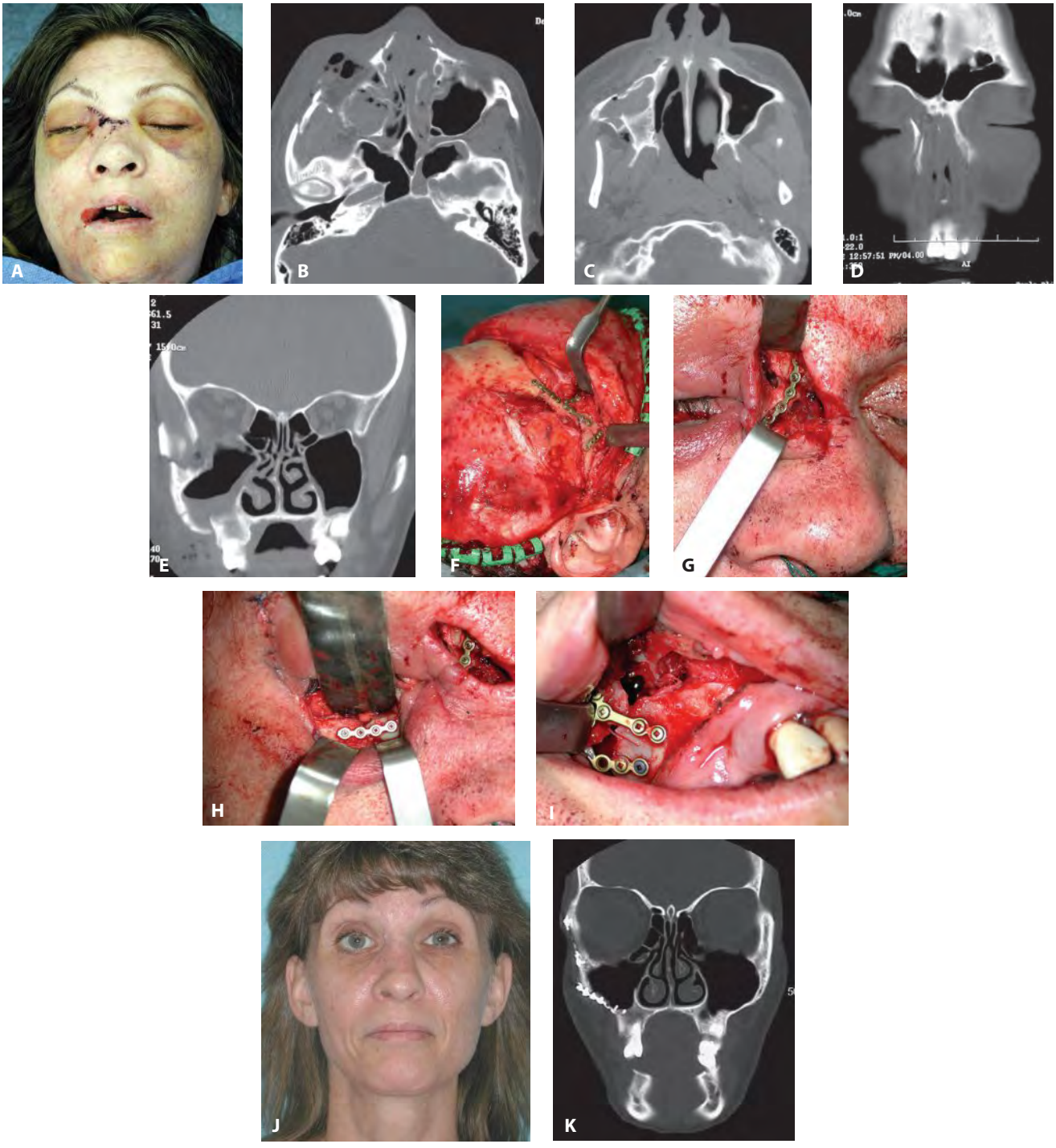


FIGURE 21-13. A 36-year-old female sustained a high-energy right zygomatic complex fracture from a motor vehicle accident. **A,** Note periorbital ecchymosis and flattening of the right malar eminence. The widened intercanthal distance results from a concomitant heminasoethmoid fracture with lateral dislocation of the medial canthal ligament. **B,** Axial CT scan demonstrates zygomatic arch fracture. **C,** Axial CT scan demonstrates posterior displacement of the malar prominence and comminution of the zygomaticomaxillary buttress. **D,** Coronal CT scan of heminasoethmoid fracture. **E,** Coronal CT scan of comminution of the zygomaticofrontal and zygomaticomaxillary buttresses. Note the orbital floor fracture and significant enlargement of the orbital volume. **F,** Coronal flap to restore sagittal projection of the zygomatic arch. Because of comminution of the zygomaticofrontal, zygomaticomaxillary, and infraorbital rim, and heminasoethmoid fracture, this was required to ensure anatomic reduction. Note exposure of the temporal fat pad to protect the facial nerve and exposure of zygomatic arch. **G,** Anatomic reduction of the heminasoethmoid fracture via a preexisting laceration. Note the insertion of the medial canthal ligament. **H,** Anatomic reduction of the orbital rim via a subciliary incision. **I,** Intraoral exposure of the zygomaticomaxillary buttress. **J,** Postoperative frontal photograph demonstrates restoration of facial width, intercanthal distance, and malar projection. Note increased scleral show secondary to subciliary incision. **K,** Postoperative coronal CT scan demonstrates restoration of the orbital floor and the zygomaticofrontal and zygomaticomaxillary buttresses.

is then replaced with a plate. The orbital floor is reconstructed after the zygoma has been restored to its correct three-dimensional position.[28,41]

In high-energy fractures, the zygomatic arch should be reconstructed first.[15,26,31,33,41]

Open Reduction and Internal Fixation of the Zygomaticomaxillary Buttress

After a throat pack is placed and local anesthesia infiltrated, an incision is made in the maxillary vestibule 3 to 5 mm above the mucogingival junction. The incision extends from the canine area to the first or second molar region. The use of electrocautery may reduce bleeding. The periosteal incision is made, and a mucoperiosteal flap is elevated to expose the infraorbital nerve, piriform rim, and zygomaticomaxillary buttress (see Figure 21-10). Additional superior dissection is used to visualize the infraorbital rim.[45,46]

The zygomaticomaxillary buttress provides an ideal location for internal fixation for middle- and high-energy fractures.[41,42] Anatomic reduction of this fracture assists in restoring malar projection, but this is difficult if the buttress is comminuted. The overlying soft tissue is thick, and plate palpability is not a concern. Therefore, this fracture may be stabilized with 1.5 or 2.0 plates.[25,28,44,47]

Open Reduction and Internal Fixation of the Zygomaticofrontal Buttress

Access and exposure for open reduction of the zygomaticofrontal buttress can be achieved through a supratarsal fold or lateral eyebrow incision (Figure 21-14A and B). If present, a preexisting laceration may be used for exposure of this region.

In 1996, Kung and Kaban[48] described the use of a supratarsal fold incision for approach to the lateral orbit (see Figures 21-11 and 21-14B). The incision is placed in a skinfold parallel to the superior palpebral sulcus above the tarsal plate. It is placed approximately 10 to 14 mm above the margin of the upper eyelid. A 2.0-cm incision is usually adequate but may be extended laterally into the crow's foot for increased exposure. Blunt dissection parallel to the orbicularis oculi muscle fibers separates them and exposes the lateral orbital rim. The dissection is continued, superficial to the orbital septum and over the lateral orbital rim. A vertical periosteal incision is made, and subperiosteal dissection will expose the fracture. The incision provides access to the frontozygomatic suture and results in a less notic1.5 or 2.0 plates able scar.

A lateral brow incision is performed by first palpating the frontozygomatic suture. A 2.0-cm incision is made within the confines of the lateral eyebrow parallel to the superior lateral orbital rim (see Figure 21-14A). Dissection is continued through the orbicularis oris and the periosteum to the fracture site.

The zygomaticofrontal buttress contains excellent bone for fixation and can accommodate a 2.0 plate.[44] The reduction and fixation of this fracture will reestablish the vertical height of the zygomatic complex. However, because of its narrow interface, this buttress may not be as helpful in evaluating reduction of a rotated fracture.[15,42] The thickness of the soft tissue overlying this region is variable. In some instances, it may be quite thin and a large plate may be palpable. If stable fixation can be achieved at other sites, a smaller plate may be used.[44]

Open Reduction and Internal Fixation of the Infraorbital Rim and Orbit

Exposure for open reduction of the infraorbital rim and orbital floor can be achieved through a transcutaneous or transconjunctival incision. Protection of the globe with a scleral shield or tarsorrhaphy is recommended.

Multiple transcutaneous techniques have been described for exposure of the orbital rim and floor. These variations include a subciliary skin-only flap, a stepped skin-muscle flap, and a subtarsal approach. The subtarsal incision and dissection is perhaps the easiest and most effective.[47] The subtarsal incision is placed 5 to 7 mm below the lid margin in a preexisting crease. The incision is through skin and orbicularis oculi and the dissection is then carried inferiorly to the orbital rim. The periosteum is then incised inferior to the orbital rim and a subperiosteal dissection of the rim and floor is completed. Exposure of the medial wall is limited with this technique. The various transcutaneous approaches have been compared with each other and with the transconjunctival incision.[49–51] Regardless of technique, transcutaneous approaches may be associated with a higher incidence of ectropion, increased scleral show, and cutaneous scarring.[52–55]

To avoid the problems associated with cutaneous incisions, many authors recommend the transconjunctival approach.[52–58] Tessier described this approach in 1973[59] (Figure 21-15; see also Figures 21-14C). The lower lid is retracted, and an incision is made below the lower border of the tarsus. Dissection is extended inferiorly, and a preseptal dissection (superficial to the orbital septum) is used to expose the infraorbital rim. Variations of this technique include a retroseptal dissection. This approach maintains the integrity of the lower lid but requires retraction of the orbital fat during fracture reduction and fixation (Figure 21-16).[46]

A lateral canthotomy can be used to increase exposure. If a canthotomy is used, it should be completed first to improve exposure for the transconjunctival incision and dissection. Meticulous repair of the lateral canthotomy is required to prevent asymmetry.[46,53,55,56]

Manson and coworkers[60] described a method to expose the entire lateral orbit, infraorbital rim, and orbital floor through a single incision. This may be performed with a transcutaneous or transconjunctival approach and requires extended subperiosteal dissection with mobilization of the lateral canthal tendon.

Internal fixation of the infraorbital rim poses a surgical dilemma. Unlike the zygomaticofrontal buttress, the infraorbital rim has poor-quality bone for internal fixation.[44] In addition, the lower eyelid skin is quite thin, and the plate may be palpable. Despite these concerns, fixation of this site may

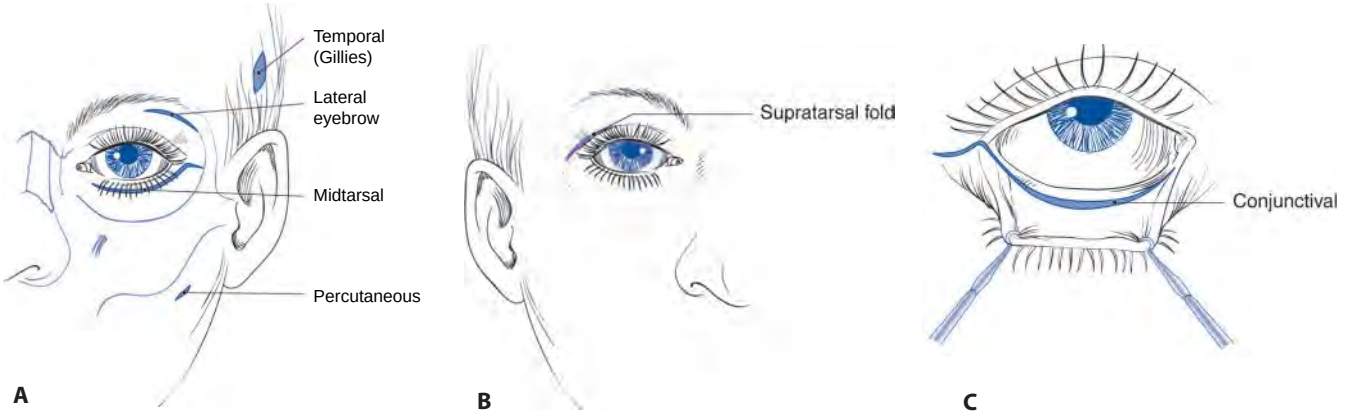


FIGURE 21-14. Frontal view illustrates periorbital incision sites. **A,** Four different incisions for repair of zygoma fractures. **B,** Upper eyelid incision within the lateral supratarsal fold. **C,** Transconjuctival incision below the lower border of the tarsus. (**A–C,** Adapted from Perrott DH, Kaban LB. Management of zygomatic complex fractures. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 1. Philadelphia: JB Lippincott; 1992; p. 500.)

be required to define the orbital volume and facial width.[61–63] The infraorbital rim is typically displaced posteriorly and inferiorly.[31] The fracture should be mobilized anteriorly and superiorly and stabilized with a 1.0 or 1.5 microplate.[28,29,44,45] A potential pitfall in reduction of this fracture is an unappreciated heminasoethmoid fracture (see Figure 21-13D). If the infraorbital rim is secured to this undiagnosed displaced segment, postoperative facial widening may occur.[25,42,64]

Management of the Orbital Floor

Patients with middle-energy zygomatic complex injuries and no clinical or radiographic evidence of orbital floor disruption may not require orbital exploration.[25,65] Clinical indications for orbital exploration include enophthalmos, limitation of extraocular muscle function with a positive forced-duction test, and persistent diplopia. Radiographic indications for exploration of isolated orbital floor fractures include CT evidence of a displaced fracture and herniation of orbital tissue (see Figure 21-4A).[33,62,64,66–69] High-energy fractures require a more aggressive approach, and the orbital rim and floor should be explored and reconstructed.[25,26,28,33,44,65,66]

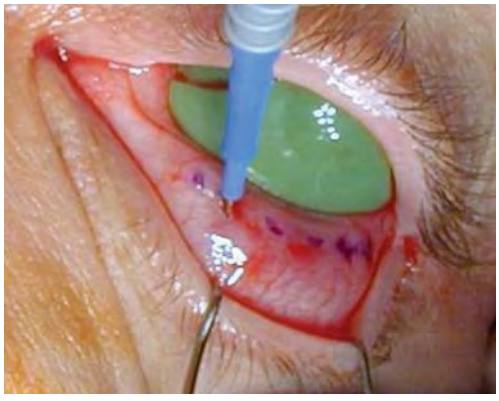


FIGURE 21-15. Transconjunctival incision for approach to the infraorbital rim.

Fujino and Makino[70] classified orbital floor injuries as *linear* and *pure blow-out fractures* (Figure 21-17). A linear fracture occurs when the infraorbital rim is struck, displacing the orbital contents and floor posteriorly. The orbital septum is torn, herniating soft tissue into the maxillary sinus. When the force is removed, the orbital floor returns to its original position and the soft tissues are entrapped in the fracture site. Comminution of the orbital floor is produced by a force 10 times greater than that required for a linear fracture. Fragments are forced inferiorly into the sinus, producing bony discontinuity.

After obtaining exposure, simple linear fractures may require only removal of entrapped tissue. Larger defects require reduction of the soft tissue and bone fragments from the sinus and reconstruction of the floor with a bone graft or implant. Exploration of the orbital floor is completed before

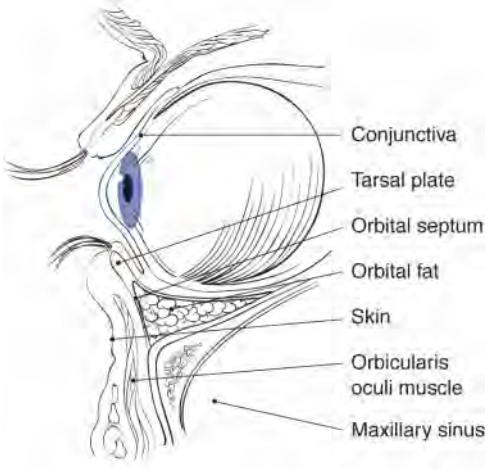


FIGURE 21-16. Sagittal view of the eye illustrates the relation of the conjunctiva, orbital septum and fat, and orbicularis muscle to the eye and infraorbital rim. (Adapted from Perrott DH, Kaban LB. Management of zygomatic complex fractures. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 1. Philadelphia: *JB Lippincott*; 1992; p. 501.)

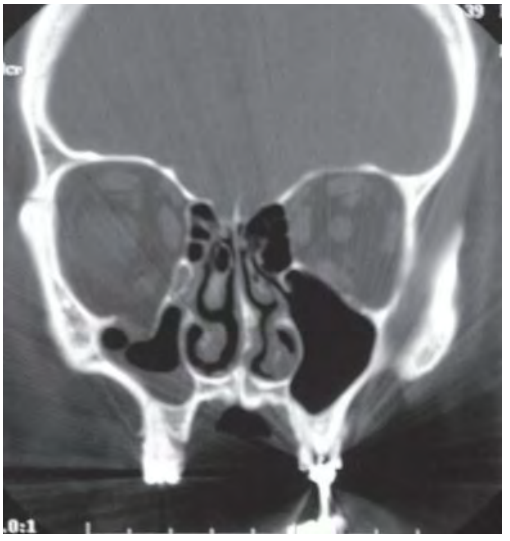


FIGURE 21-17. Isolated blow-out fracture with herniation of the orbital contents into the maxillary sinus.

reduction of the fracture. Reconstruction of the floor is completed after reduction and stabilization of the orbital rim if it is a component of a zygomatic complex.[28,44,66]

The orbital floor may be reconstructed with an autograft, an allograft, or a prosthetic implant. Autograft sources include calvaria, iliac crest, or nasal septal cartilage.[71,72] Allograft sources include lyophilized dura and cartilage.[73] Alloplastic material such as titanium mesh[29,71,74,75] offers a strong, malleable material that can accurately be adapted to span the orbital defect. Porous polyethylene implants and resorbable polydioxanone sheets have also been used for orbital reconstruction.[33,76,77]

Regardless of the technique, anatomic restoration of the orbital volume is required to prevent postoperative enophthalmos.[61–63,71] In complex fractures, a significant portion of orbital floor may be comminuted or missing. The defect must be completely defined, and the graft or implant must be placed on an intact posterior "ledge," which may be 35 to 38 mm posterior to the rim.[78,79]

Forced-duction tests should be performed before and after the orbital floor exploration as well as after reconstruction.[44,66]

Pitfalls in Surgical Approach to the Infraorbital Rim and Orbit

All approaches to the infraorbital rim may result in complications such as ectropion, entropion, and increased scleral show. Advocates of the transconjunctival approach cite increased rates of ectropion and scleral show with transcutaneous incisions (see Figure 21-13J).[52–55,58] In 1993, Appling and colleagues[54] found a 12% rate of transient ectropion and 28% rate of permanent scleral show with a subciliary approach. In comparison, the transconjunctival approach had no transient ectropion and a 3% rate of permanent scleral show.

Multiple factors have been cited as the cause of increased scleral show and ectropion. During the dissection to the orbital rim, care should be taken to ensure that the placement of the periosteal incision is on the anterior surface of the maxilla. An incision placed on the superior rim or posterior to the orbital rim may violate the orbital septum. Subsequent scarring and contracture of the septum may result in increased scleral show or ectropion.[60]

Improper wound closure may also contribute to lower lid complications. Following wide subperiosteal exposure, which is often required for complex fracture repair, the facial soft tissues may descend caudally, resulting in loss of anterior projection, accentuation of the nasolabial fold, increased scleral show, and ectropion. Phillips and associates[79] recommend resuspension of the periosteum, muscle, and subcutaneous tissue. Multiple holes are drilled in the inferolateral orbital rim. The edge of the periosteum, muscle, and subcutaneous tissue is sutured to the orbital rim. This may minimize traction on the infraorbital tissue and subsequent ectropion or increased scleral show.[80]

Lastly, postoperative support for the lower eyelid with a frost stitch has been proposed as a technique to prevent ectropion. This may encourage redraping of the lower eyelid tissues.[25,60]

Open Reduction and Internal Fixation of the Zygomatic Arch

In high-energy zygomatic complex fractures or secondary correction of zygomatic deformities, access is limited with conventional incisions. To obtain adequate exposure, a coronal incision combined with a lower eyelid approach is recommended (see Figure 21-13F).

The initial incision is through the skin, subcutaneous tissue, and galea of the scalp. Elevation of the coronal flap proceeds in the subgaleal loose areolar connective tissue superficial to the pericranium. The temporal and preauricular plane of dissection is along the temporal fascia, which can be identified by its characteristic glistening white appearance. A horizontal periosteal incision is made 2 to 3 cm above the supraorbital rim, and a subperiosteal plane of dissection is developed to the superior and lateral orbit. An incision is made in the superficial layer of the temporal fascia from the posterior zygomatic arch to the previously exposed supraorbital region. The temporal fat pad should be identified (see Figure 21-13F). The dissection is extended inferiorly at this depth to the zygomatic arch and anteriorly to the lateral orbital rim. Subperiosteal dissection of the internal orbit allows for exposure of the sphenozygomatic suture. The facial nerve is protected within the flap.[15,46]

Internal fixation of the zygomatic arch is required for high-energy fractures that demonstrate comminution and lateral displacement.[15,25,26,31,44] Restoration of this sagittal buttress assists in restoring facial projection and facial width. When exposed, the zygomatic arch is often reduced and stabilized first in the sequence of repair of high-energy injuries. Caution must be used in restoring a "straight" arch and not a "curved" arch, which will decrease facial projection. This fracture typically requires a large plate to resist deformational forces.[44,47]

Role of Bone Grafting in Zygomatic Complex Fractures

Early bone grafting is indicated for severe injuries in which there is loss of bone or extensive comminution. Comminution of the orbital floor and zygomatic buttresses is common in high-energy injuries. These zygomatic complex fractures are often associated with other severe midface fractures that require treatment. Grafts may help to achieve anatomic reduction and stability as well as to prevent soft tissue contraction.[81,82]

Postoperative Care

Zygomatic complex fractures violate the maxillary sinus. For this reason, periorperative antibiotics and decongestants are recommended particularly if a transoral approach is used or an implant placed. Scientific evidence is scant to support any particular regimen or course. Ampicillin, amoxicillin, clindamycin, or cephalosporin may be used.[66] A decongestant such as pseudoephedrine may also be indicated.

Incisions are observed carefully for signs of infection, and the eye is examined to document visual acuity and to rule out complications such as corneal abrasion. Postoperative imaging should be obtained to document reduction of the fracture and orbital reconstruction.[42,66]

COMPLICATIONS OF ZYGOMATIC COMPLEX FRACTURES

Although complications of zygomatic complex and zygomatic arch fractures are uncommon, the surgeon must recognize their signs and symptoms to provide appropriate care. Complications may occur in the early postoperative period or may become manifest only later in recovery.

Infraorbital Paresthesia

The incidence of sensory alterations of the infraorbital nerve after zygomatic trauma ranges from 18% to 83%.[3,5–8] Renzi and coworkers,[8] in a prospective study of 97 patients with 103 facial fractures, noted 70.9% of nondisplaced midfacial fractures had post-traumatic infraorbital nerve impairment with a mean recovery time of 4 weeks. The authors suggest that the likely mechanism is moderate indirect injury by diffusion of traumatic forces through the overlying soft tissue. In displaced midfacial fractures, 18/20 (90%) had altered sensation within the infraorbital nerve distribution with a mean recovery time of 13 weeks. Incomplete recovery was frequently associated with intraoperative evidence of direct nerve injury. The authors support early open reduction and internal fixation to improve recovery of post-traumatic nerve dysfunction.

Malunion and Asymmetry

Inadequate reduction or stabilization of zygomatic fractures may result in malunion or asymmetry.[7,15,25,31] Poor malar projection is the result of uncorrected inferior and posterior rotation of the zygoma. Increased facial width, in addition to decreased malar projection, results from inadequate reduction of the zygomatic arch as part of a high-energy orbitozygomatic injury.[31] As technology has continued to evolve, routine use of intraoperative CT guidance may improve reduction of complex fractures to minimize postoperative malunion and asymmetry.[83–85]

Malunion that is recognized up to 6 weeks after injury may be corrected using routine zygomatic reduction techniques. Correction of mild late deformities includes autogenous onlay grafts or placement of alloplastic implants such as porous polyethylene.[26] Severe late post-traumatic deformities may require zygomatic osteotomy and repositioning. Cranial bone grafting may also be required. Scarring and contraction of the periorbital soft tissue may also occur.[86] Lid retraction, entropion, ectropion, and canthal repositioning may need to be addressed in addition to osseous reconstruction.

Enophthalmos

Enophthalmos is one of the most troubling complications after orbitozygomatic fractures. An increase in orbital volume is the most common etiology.[61–63]

Grant and colleagues[62] described this clinical problem eloquently by comparing the shape of the orbit with that of a cone. The volume of a cone is $^1/_3(\pi r^2) \times h$. The orbital rim position determines the radius of the cone and the anteroposterior orbital length is the height of the cone. In this equation, the radius is squared and small increases in the radius result in dramatic increases in volume. Clinically, poor alignment of the orbital rim may significantly increase the orbital volume and result in enophthalmos.[62]

Orbital floor blow-out fracture also may result in enophthalmos by increasing the orbital volume (Figure 21-18). With improved CT technology, calculation of orbital volume and its implication regarding orbital floor fractures is possible.[79,87–90] Raskin and associates[88] demonstrated that a 13% increase in orbital volume, at 4 weeks, results in significant enophthalmos (>2 mm). The critical size of the orbital defect and herniation of orbital tissues have also been studied. In 2002, Ploder and coworkers[90] reported that a mean fracture area of 4.08 cm or a mean displaced tissue volume of 1.89 mL was associated with greater than 2 mm of enophthalmos. In general, approximately 1 cm^3 of displaced tissue equals 1 mm of enophthalmos.[91]

Late repair of enophthalmos is technically challenging. Wide access with osteotomy of the zygoma, repositioning, and grafting is usually required. Redraping of the periorbital soft tissue including a canthopexy may be required.[61–63]

Diplopia

Diplopia is a common sequela of midfacial fractures. The incidence varies between 17% and 83% and depends on the time of presentation after the injury and the pattern and severity of the injury.[3,73,92–96] In a review of 2067 zygomatic complex fractures, Ellis and colleagues[3] noted a 5.4% to



FIGURE 21-18. **A,** A 27-year-old female presented with late enophthalmos and diplopia after an undiagnosed orbital floor fracture. Note vertical dystopia and prominent supratarsal fold. **B,** Coronal CT scan demonstrates displacement of the orbital floor. **C,** One-year postoperative frontal photograph after transconjunctival reconstruction of the orbital floor with titanium mesh. Note the symmetry of the vertical globe position and the supratarsal fold. **D,** Postoperative coronal CT scan demonstrates titanium mesh reconstruction of the orbital floor.

74.5% incidence of diplopia. Nondisplaced zygomatic complex fractures and isolated zygomatic arch fractures had the lowest incidence of diplopia, whereas pure blow-out fractures had the highest incidence.

The principal causes of diplopia include edema and hematoma, entrapment of the extraocular muscles and orbital tissue, and injury to cranial nerve III, IV, or VI. Histologic studies by Iliff and coworkers[97] have shown post-traumatic fibrosis of the extraocular muscles in response to injury. They hypothesize that this may impair contractility and decrease excursions of the muscles.

Axial and coronal CT scans and ophthalmologic consultation are recommended to assist in evaluation.[33,92] Diplopia related to edema, hematoma, or neurogenic causes may resolve without intervention. Diplopia resulting from entrapment requires exploration and reduction of herniated orbital tissue (Figure 21-19).[33,64,66,67–69,92]

Persistent bothersome diplopia that does not resolve may require treatment by an ophthalmologist. The condition may respond to exercise or surgery.[33,66]

Traumatic Hyphema

Trauma to the eye may result in bleeding into the anterior chamber—the area between the clear cornea and the colored iris (Figure 21-20). Ophthalmology consultation is recommended. Goals of treatment include prevention of rebleeding, which may occur in 5% to 30% of patients, and maintenance of normal ocular tension.[98,99]

Management of hyphema consists of supportive therapy including elevation of the head of the bed and patching of the injured eye. Medical management includes topical cycloplegics, corticosteroids, and beta blockers. Systemic antifibrinolytics, carbonic anhydrase inhibitors, and osmotic agents may also be required. Rarely, surgical intervention by the ophthalmologist is required. Repair of fractures may be delayed.

Traumatic Optic Neuropathy

Traumatic optic neuropathy may manifest as conditions ranging from mild visual deficit to complete visual loss. An

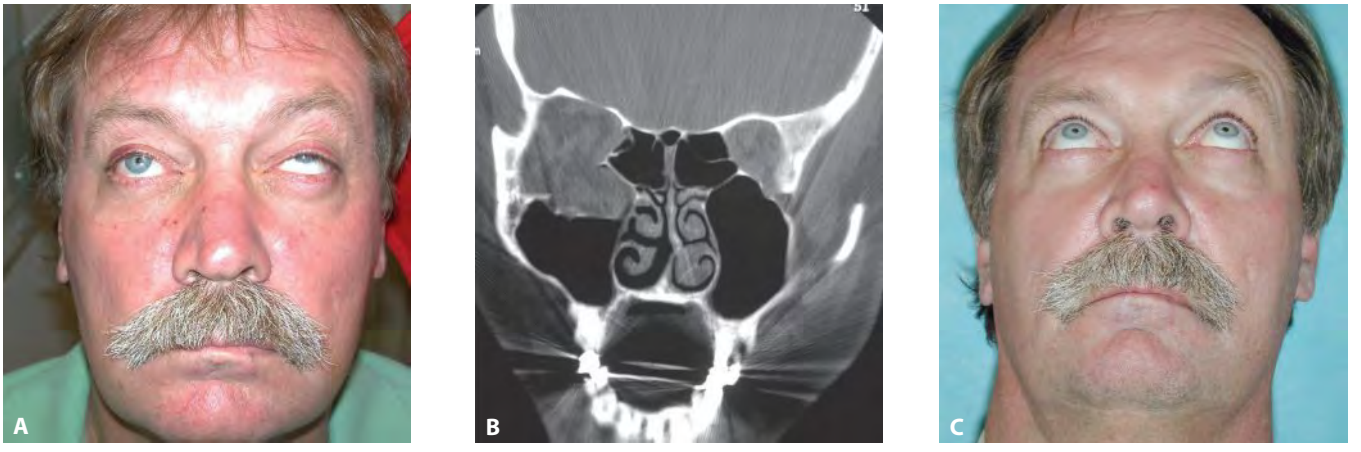


FIGURE 21-19. **A,** A 45-year-old male suffered a fall and presented with right orbital floor blow-out fracture and significant restriction of the inferior rectus and diplopia. **B,** Coronal CT scan demonstrates a large orbital floor blow-out fracture with herniation of the orbital contents into the maxillary sinus. **C,** Postoperative view after transconjunctival reconstruction of the orbital floor with titanium mesh and return of normal extraocular muscle function. Note projection of the globes without evidence of enophthalmos.

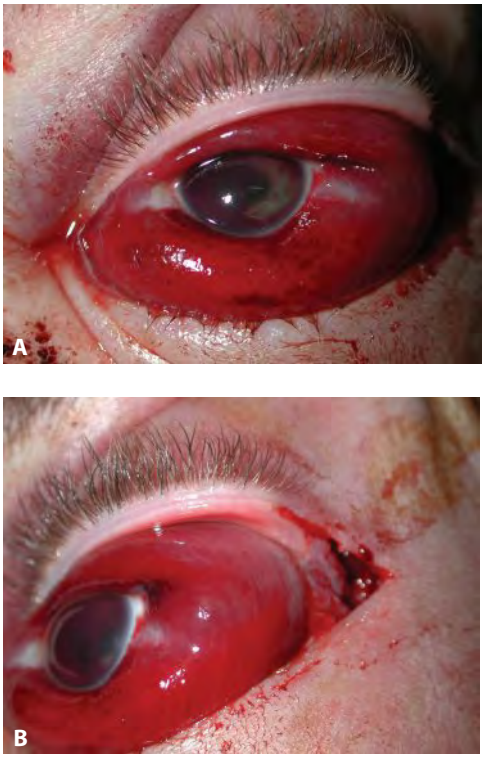


FIGURE 21-20. Retrobulbar hemorrhage. **A,** This patient presented with periorbital pain, fixed and dilated pupil, proptosis, and acute progressive loss of vision. Note hyphema. **B,** Immediate lateral canthotomy and cantholysis were performed.

ophthalmologic consultation is mandatory. Treatment varies depending on the cause but may include systemic steroids or surgery with orbital or optic nerve decompression. Treatment of facial fractures may be delayed.[33,100]

Superior Orbital Fissure Syndrome

Superior orbital fissure syndrome is an uncommon complication after facial trauma. Presentation may include ptosis, ophthalmoplegia, forehead anesthesia, and a fixed dilated pupil. Proptosis may be present. Treatment may include reduction of fractures, steroids, orbital apex exploration, and aspiration of retrobulbar hematoma if present.[101]

Retrobulbar Hemorrhage

Retrobulbar hemorrhage is a rare but severe complication that may be the result of either the initial injury or the operative correction. Disruption of the retinal circulation may lead to irreversible ischemia and permanent blindness. In a review of 1405 orbitozygomatic fractures, Ord[102] reported a 0.03% incidence of postoperative retrobulbar hemorrhage with visual loss. An emergent ophthalmologic consultation is necessary; however, decompression with lateral canthotomy and cantholysis should not be delayed (see Figure 21-20).

Trismus

Patients with zygomatic fractures commonly present acutely with a complaint of trismus. However, there are few cases of long-term reduced mandibular range of motion after zygomatic complex fractures reported in the literature. The most likely cause is impingement of the zygomatic body on the coronoid process of the mandible. Trismus may also occur secondary to fibrous or fibro-osseous ankylosis of the coronoid to the zygomatic arch. A CT scan should be obtained to confirm the diagnosis. Coronoidectomy is the most common treatment. If the zygoma is improperly reduced, zygomatic osteotomy and repositioning may be necessary to restore unrestricted motion of the mandible.[66,103]

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22

CHAPTER

Orbital and Ocular Trauma

Deepak G. Krishnan, DDS, and Mark W. Ochs, DMD, MD

ORBITAL FRACTURES

Anatomy

The orbit is the bony vault that houses the eyeball, or globe. It is a quadrangular-based pyramid that has its peak at the orbital apex. The average adult orbit has a volume of 30 cc; the globe averages 7 cc (Figure 22-1). Even a modest change in the position of one of the bony walls can have a significant impact on the orbital volume and, thus, globe position. The orbit serves to house and protect the globe. By age 5 years, orbital growth is 85% complete, and it is finalized between 7 years of age and puberty.[1,2]

The orbital rim is composed of dense cortical bone that generally protects the orbital contents and globe from direct blunt trauma. Seven bones form the orbit: maxillary, zygomatic, frontal, ethmoidal, lacrimal, palatine, and sphenoid. Besides forming a protective socket for the globe, these bones also provide origins for the extraocular muscles and foramina and fissures for cranial nerves and blood vessels.[3]

The orbital walls vary considerably in their thickness. Whereas the superior lateral and inferior rims tend to be rather thick, the bones just posterior to these and the medial rim are usually fairly thin (<1 mm). Fractures of the anterior and middle thirds of the bony orbit are fairly common. The orbital floor and medial wall are most frequently fractured owing to their thinness and lack of support. This is fortunate since inward or medial displacement of midfacial or zygomatic bones can reduce the orbital volume and be accompanied by orbital hemorrhage. The subsequent increased intraorbital pressure is most often relieved by traumatic expansion of the walls with herniation of orbital tissue into the maxillary sinus and/or ethmoid air cells adjacent to these walls. In essence, the paranasal sinuses and ethmoid air cells serve as air bags or shock absorbers to the globe and orbital contents. This protective mechanism explains why globe perforation is relatively uncommon after midfacial trauma. Orbital fractures that involve the frontal sinuses more commonly result in serious eye injuries.[4,5] These fractures, following blunt trauma, and the associated blindness are probably not seen as often owing to the severity of forces and concomitant neurologic, cervical spine, and multisystem trauma. In short, they generally are not survivable events.

The orbital roof consists mainly of the frontal bone, with the anterior cranial fossa superior to it. The lesser wing of the sphenoid has a minor contribution posteriorly. The superior orbital rim is generally rather thick and then rapidly becomes quite thin (<1 mm) posterior from the edge. In elderly patients, the orbital roof may be resorbed in select areas, allowing the dura to become confluent with the periorbita. This should be kept in mind during orbital dissection and elevation in this region for both trauma and tumor work. Generally, the anterior portion of the orbital roof is occupied by the supraorbital extension of the frontal sinus. The frontal sinus begins to form around the age of 6 years and is unilateral in 5% of adults and lacking in another 5%. Anterolaterally, there is a smooth broad fossa that houses the lacrimal gland. At the most medial extent is the trochlea, approximately 4 mm behind the rim. There, the cartilaginous pulley has a dual insertion for the superior oblique muscle tendon. At the junction of the medial one third and lateral two thirds of the superior rim is the supraorbital notch. In one fourth of adults, a supraorbital foramen is found, secondary to the ossification of the ligament crossing the inferior extent.[6] When reflecting bicoronal flaps, a small triangular wedge ostectomy should be performed in these individuals to relieve the encased supraorbital nerve and vessels and to allow for a relaxed reflection of tissues at the rim.

The orbital floor is bordered laterally by the inferior orbital fissure. However, there is no distinct border medially (Figure 22-2). The orbital floor is formed primarily by the orbital process of the maxilla—anterolaterally by a portion of the zygomatic bone and posteriorly by a small portion of the palatine bone. The maxillary sinuses are present at birth and

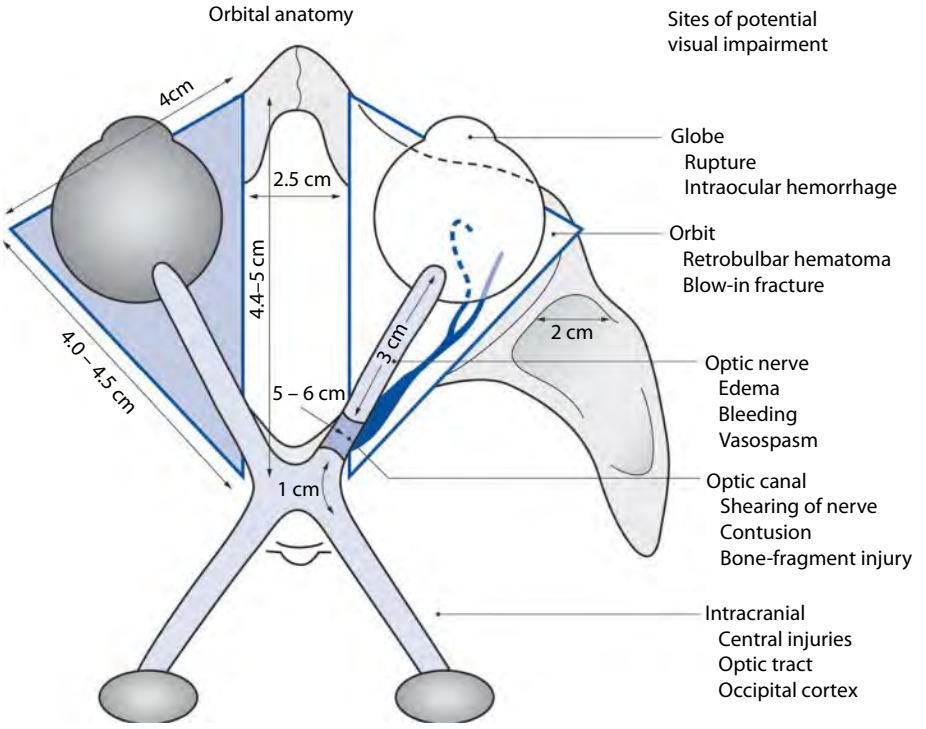


FIGURE 22-1. Orbital configuration with potential sites for traumatic injuries leading to visual impairment. (Adapted from Ochs MW, Johns FR. Orbital trauma. In Fonseca RJ, Marciani RD, Hendler BH, editors. Oral and Maxillofacial Surgery: Trauma. Vol. 3. Philadelphia: WB Saunders; 2000; p. 207.)

reach the orbital floor and infraorbital canal by age 2 years.[7] The inferior orbital fissure gives rise to the infraorbital groove from its midportion, which is about 2.5 to 3 cm from the infraorbital rim. The infraorbital fissure converts to a canal halfway forward, carrying the infraorbital nerve and vessels and opening approximately 5 mm below the rim of the maxilla as the infraorbital foramen (Table 22-1).[8] The infraorbital nerve provides sensory innervation to the upper lip, lateral nose, and anterior maxillary teeth and mucosa.



FIGURE 22-2. Right bony orbit. The inferior orbital fissure can be seen converting to a canal angling medially at the Y-shaped divide. The lacrimal fossa is characteristically thin. The lamina papyracea occupies the majority of the medial wall, with the frontoethmoidal suture at the superior extent.

The orbital floor can be as thin as 0.5 mm, with its weakest portion just medial to the infraorbital groove and canal. This explains the phenomenon that most blunt traumas resulting in orbital floor blow-outs are manifested primarily with injury and sagging of the medial orbital floor and orbital contents into the underlying maxillary sinus with extension laterally to the infraorbital canal.

The lateral wall of the orbit is formed mainly by the greater wing of the sphenoid and portions of the zygoma. Although this tends to be the strongest wall, it is fairly commonly fractured along the frontozygomatic junction, extending slightly posteriorly and then running vertically along the thinnest portion of the suture line, where the greater wing of the sphenoid and zygoma meet. This wall separates the orbit from the temporalis muscle. Owing to the heavy nature of this muscle and the direction of blunt forces, generally there is some mild degree of inward displacement. The lateral orbital walls, if they were to be extended posteriorly, would form a 90-degree angle to each other. Each lateral orbital wall forms a 45-degree angle at the orbital apex, with its medial wall counterpart. This is important to bear in mind when attempting to realign or reconstruct fractured walls. The superior orbital fissure separates the greater and lesser wings of the sphenoid and serves as the delineation between the orbital roof and the lateral wall. At the orbital apex, the lesser wing of the sphenoid forms the lateral portion of the ring of the optic canal. One centimeter below the frontozygomatic suture, and just internal (3–4 mm) to the lateral orbital rim, is Whitnall's tubercle (lateral orbital tubercle).

TABLE 22-1. **Orbital Fissures/Canals and Their Contents**

Location	Contents
Superior orbital fissure—lesser and greater wings of sphenoid	Motor nerves: III (superior and inferior divisions), IV (trochlear),V (abducens) Sensory nerves:V1 (frontal, lacrimal, nasociliary), sympathetic fibers Vessels: superior ophthalmic vein, anastomosis of recurrent lacrimal and middle meningeal arteries
Inferior orbital fissure—greater wing of sphenoid; palatine, zygomatic, and maxillary bones	Sensory nerves:V2 (infraorbital and zygomatic), parasympathetic branches of pterygopalatine ganglion Vessel: inferior ophthalmic vein and branches to pterygoid plexus
Optic canal—lesser wing of sphenoid	Optic nerve, meninges, ophthalmic artery, sympathetic fibers
Anterior ethmoid canal—frontal and ethmoid bones	Vessel: anterior ethmoid artery
Posterior ethmoid canal—frontal and ethmoid bones	Nerve: anterior ethmoid becomes dorsal nasal
Vessel: posterior ethmoid artery	Nerve: posterior ethmoid
Nasolacrimal fossa—lacrimal and maxillary bones	Nasolacrimal sac and duct

This gentle outcropping of bone functions as the insertion point for the lateral retinacular structures. The lateral retinaculum is composed of the lateral horn of the levator aponeurosis; the lateral canthal tendon of the eyelids; and the inferior suspensory (Lockwood's) ligament and multiple fine check ligaments of the lateral rectus muscle. These soft tissue attachments are found anatomically in this order proceeding inferiorly and posteriorly from the rim. These multiple structures become confluent to form the common lateral retinaculum, which is the actual insertion to the tubercle.[6] Clinically, the point to remember is that reattachment of the lateral canthal tendon should be to the lateral orbital tubercle.

The medial wall of the orbit is by far the most complex and potentially problematic to manage in severe trauma. The medial orbital wall is composed anterior-to-posterior by a portion of the maxillary, lacrimal, ethmoid, and sphenoid bones. The majority of the medial wall is formed by the extremely thin (0.2–0.4 mm) lamina papyracea of the ethmoid bone. Housed along the frontoethmoidal junction are the anterior and posterior ethmoidal foramina. The anterior ethmoidal foramen is 20 to 25 mm behind the medial orbital rim; 12 mm beyond this is the posterior ethmoidal foramen. The foramina can be found approximately two thirds of the way up the medial orbital wall, within the frontoethmoidal suture line; they serve as important surgical landmarks identifying the level of the corresponding cribriform plate. Orbital surgeons use these arteries as the landmarks for the superior extent of orbital wall decompression. The anterior ethmoidal foramen transmits the anterior ethmoidal artery and anterior ethmoidal branches from the nasociliary nerve from the orbit coursing into the nasal cavity. This is why otolaryngologists sometimes use a medio-orbital approach to ligate or cauterize the anterior ethmoidal artery to control recalcitrant nasal bleeding. Although the anterior ethmoidal vessel can be cauterized with few ill effects, the contents of the posterior ethmoidal foramen (posterior ethmoidal artery and, variably, a sphenoethmoidal nerve from the nasociliary nerve) are generally allowed to remain intact since they serve as a useful delineation to the posterior extent of safe medial wall dissection.

Once beyond the orbital rims, subperiosteal dissection generally proceeds fairly easily, except for points of nerves or vessels perforating through foramina, orbital fissures, or muscle origins such as that of the inferior oblique. When encountering resistance, surgeons should attempt to identify the exact anatomic reason for the resistance, such as structures that may need to be preserved or periorbital tissues that have become entrapped in fracture lines. Knowledge of the limits of safe subperiosteal dissection is mandatory. Also important is knowing the distance from the intact orbital rim, where vital structures can be identified. Generally, a subperiosteal dissection from the inferior lateral rims can be safely extended for 25 mm. An exploration distance of 30 mm from the superior orbital rim or anterior lacrimal crest (found on the frontal process of the maxilla) can be safe.[5] A high medial wall dissection places the orbital apex and optic canal at risk. One caveat to these "safe surgical exploration distances" is that they are averages of known landmarks to intact adult orbital rims. When traumatic forces displace a portion of a rim, it is generally in a posterior or medial direction, which effectively reduces these distances. Knowledge of the bony orbital anatomy, with its foramina, fissures, and attachment areas, helps the surgeon to avoid injuries to vital structures contained within them.[1] Average distances for locating these critical structures as they relate to identifiable bony landmarks are contained in Table 22-2. Surgeons should avoid disrupting the medial canthal tendon, lacrimal apparatus, pulley of the superior oblique muscle, supraorbital

TABLE 22-2. **Distance of Vital Orbital Structures from Bony Landmarks**

Structure	Reference Landmark	Mean Distance (Mm)
Midpoint of inferior orbital fissure	Infraorbital foramen	24
Anterior ethmoidal foramen	Anterior lacrimal crest	24
Superior orbital fissure	Zygomaticofrontal suture	35
Superior orbital fissure	Supraorbital notch	40
Optic canal (medial aspect)	Anterior lacrimal crest	42
Optic canal (superior aspect)	Supraorbital notch	45

SECTION 3

TABLE 22-3. **Eyelid Layers: Cutaneous Anterior) to Conjunctival (Posterior)**

Skin
Subcutaneous areolar tissue
Striated muscle (orbicularis oculi)
Submuscular areolar tissue (contains main sensory nerves to lids)
Fibrous layer with tarsal plates
Nonstriated smooth muscle
Mucous membrane or conjunctiva

nerves and vessel, attachments to Whitnall's tubercle, and the origin of the inferior oblique muscle.

The anterior boundary of the orbit is defined by the orbital septum. The upper and lower eyelids are anatomically similar in their composition, with corresponding layers anteriorly to posteriorly. When one is looking downward, the lid retractors enable the lower eyelid to roll with the globe, thus avoiding a visual field cut. The lids have a very thin keratinized epithelium that is loosely attached to the underlying orbicularis oculi muscle (Table 22-3). The orbicularis oculi muscle is innervated by cranial nerve VII and acts as a sphincter and closing force for the eyelids. In the relaxed state, the orbicularis oculi is opposed in the upper eyelid by the levator palpebrae superioris, which is innervated by cranial nerve III. The resting tone and level of the upper eyelid are partly determined by the amount of sympathetic input to Müller's muscle. The orbicularis oculi has two distinct layers: the outer superficial fibers (orbital portion) and the deeper fibers (palpebral portion). The palpebral section medially has intricate insertions and envelops the lacrimal sac by dividing into intertwined deep and superficial heads. The superficial portion inserts onto the anterior lacrimal crest. The inner deep head inserts into the fascia of the lacrimal sac and posterior lacrimal crest. The medial canthal tendon is formed by the condensation of the orbicularis muscle fibers. It is the superficial head of the canthal tendon that has a tenacious insertion into the anterior lacrimal crest. This is beneficial during orbital approaches because the anterior insertion offers considerable resistance to dissection, which helps one avoid inadvertent injury to the lacrimal sac. At the lateral edge of the orbicularis oculi, the superficial fibers form an indistinct raphe, and it is the deeper fibers that comprise the lateral canthal tendon, inserting onto Whitnall's tubercle.[9] The upper and lower lids should form a 30 to 40-degree angle at the lateral canthus, which is situated 1 cm below the frontozygomatic suture. Typically, the lateral canthus is situated 2 to 4 mm above the medial canthus.

Just posterior to the orbicularis oculi is the orbital septum. The orbital septum is continuous with the orbital periosteum and the periosteum of the facial bones overlying the rims. One to 2 mm below the inferior rim, where these layers converge on the facial aspect, is a periosteal thickening called the *arcus marginalis*.[5] This is a useful landmark when performing an infraciliary or preseptal transconjunctival approach to the inferior rim. If one stays in front of the orbital septum and incises below the arcus marginalis, orbital contents and fat do not herniate into the field. The distal edges of the orbital septum insert into the superior edge of the tarsal plates. The orbital septum and these insertions prevent the preaponeurotic orbital fat from herniating out into the eyelids. Superiorly there is a central and medial fat pad, and inferiorly there are three distinct fat pads (medial, central, and lateral). With aging, the orbital septum can become lax and, particularly in the lower lids, result in "baggy lids." Severe sagging of the lower lids is referred to as *festooning*.

The primary elevator of the upper eyelids is the levator palpebrae superioris muscle. Inferiorly, it forms an aponeurosis below Whitnall's ligament that attaches broadly over the anterior tarsal plate. Approximately 15 to 20 mm above the tarsal plate, the aponeurosis consists of a thickened fascial band, which is termed *Whitnall's ligament*. This is a suspensory ligament of the lid. Müller's muscle arises beneath the levator muscle and inserts into the superior border of the tarsal plate. Müller's is a smooth muscle that receives sympathetic input for its tone and helps regulate the resting position of the upper eyelids while the eyes are open. Increased stimulation or sympathetic input causes a "wide-eyed" look and a more alert appearance.[10] The capsulopalpebral fascia and the inferior tarsal muscle in the lower eyelids are also termed the *lower lid retractors*. The lid retractors are formed from the fibrous attachments of the inferior rectus and inferior oblique muscles and fuse with Lockwood's inferior suspensory ligament.

The tarsal plate is formed by dense fibrous connective tissue and is primarily responsible for the convex form of each of the lids. The tarsal border parallels the free margin of the eyelid. The horizontal length of each tarsus is approximately 30 mm. The height is greatest in the midportion of the lid. The height of the upper tarsus is 10 mm, whereas in the lower lid it is 4 mm. Embedded within the tarsal plates are a fine network of meibomian (sebaceous) glands. When obstructed and chronically inflamed, these glands can form a cystlike mass called a *chalazion*.

The lacrimal system is responsible for the lubrication and wetting of the globe. Accessory lacrimal glands perform normal wetting of the eye, and the lacrimal gland produces reflex tearing. The lacrimal gland, which is situated in the anterior aspect of the superior lateral orbit, is divided into two lobes by the levator aponeurosis. The larger orbital lobe lies above the levator aponeurosis, and its tear ducts traverse the palpebral lobe, which has 6 to 12 tear ductules that empty into the superior lateral fornix. When drilling in this region, such as during a repair of a frontozygomatic fracture, one must take care not to injure the palpebral lobe or to inadvertently remove it, thinking that it is herniated fat; this error often results in a problematic dry eye. Lacrimal secretions, or tears, traverse medially and inferiorly across the globe, wetting the cornea, and accumulate at the medial inferior aspect of the eye. The fluid is then either drawn or pumped into the lacrimal puncta of the upper and lower eyelids. These puncta are only 0.2 to 0.3 mm in diameter. The upper punctum is usually just slightly medial in relation to the lower punctum. When the lids close, the puncta come into contact. The upper

and lower canaliculi travel within the lids, first vertically (2 mm), then horizontally for 8 to 10 mm, paralleling the lid margin. They join to form a common canaliculus just before entering the lateral aspect of the lacrimal sac, which is one third of the way down from the upper portion of the sac. Typically, the lacrimal sac is 1 cm in length and 5 mm in diameter. The palpebral portion of the orbicularis oculi has dense intertwined insertions that envelope the lacrimal sac. Inferiorly, the sac drains into the nasolacrimal duct, which has a 12-mm intrabony canal coursing inferiorly and posteriorly that opens into the inferior meatus of the nasal cavity below the inferior concha. This opening is 30 to 35 mm from the edge of the external nares. Reflux of tears and nasal mucus back up into the nasolacrimal duct is prevented by a mucosal fold called *Hasner's valve.* With persistent epiphora after trauma or surgical intervention, it is important to establish the precise point of mechanical obstruction that exists within the lacrimal drainage system. Irrigation of the inferior canaliculus may relieve temporary obstruction owing to dry or thickened secretions. A dye disappearance test, Jones I or II, nasolacrimal irrigation, or dacryocystography can help one determine the precise point of obstruction and guide surgical planning. After trauma or operative intervention, epiphora may be due to hypersecretion from a corneal abrasion, lash ptosis, foreign bodies, or entropion, all of which serve as persistent stimuli leading to reflex lacrimal gland secretion.

Fracture Configurations

Isolated orbital wall fractures account for 4% to 16% of all facial fractures. If fractures that extend outside the orbit are included, such as those of the zygomatic complex (ZMC) and naso-orbitoethmoid (NOE), this accounts for 30% to 55% of all facial fractures.[11,12]

ZMC fractures are the most commonly occurring facial fracture, second only to nasal fractures. By definition, ZMC fractures are the most common fracture with orbital involvement.[13] The ZMC, or tripod, often hinges about the frontozygomatic suture with a medial, inferior, and posterior vector of rotational displacement. This is due to the direction and force of blunt trauma and the variable thicknesses of the components of the ZMC. The frontozygomatic area offers the thickest pillar. When fractured, there is usually a slight vertical displacement with a reasonable anteroposterior alignment. The much thinner anterior maxillary and lateral orbital floor offers little resistance to fracture and displacement.

Fractures of the NOE are most often due to severe blunt midface trauma. These fractures create cosmetic deformities with a flattening of the nasal dorsum and a widening of the intercanthal distance; they can also be accompanied by a violation of the underlying dura with a cerebrospinal fluid (CSF) leak. Any persistent or copious clear nasal drainage should be tested to determine a beta$_2$-transferrin level to rule out a CSF leak. It is uncommon for the canthal tendons to become disinserted from the bones. This is particularly true of the lateral canthal tendon. Traumatic telecanthus with NOE fractures is a result of a flattening of the nasal bridge and a lateral splaying of the orbital rims and anterior lacrimal crest. Reduction and fixation of these bony segments and, less frequently, direct transnasal wiring are necessary for adequate restoration of medial intercanthal distance and alignment. In adult whites, this is typically 29 to 32 mm; it is slightly more in black and Asian individuals. Lacrimal drainage problems can also arise from severe NOE fractures owing to canalicular or lacrimal sac disruption or scarring.

Internal orbital fractures occur in numerous patterns. These fractures are typically described by their location and the size of the defect. Three basic patterns of internal orbital fractures have been described: linear, blow-out, and complex.[14] Linear internal orbital fractures maintain periosteal attachments and typically do not result in a defect with orbital content herniation; however, they can result in a significant enlargement of the orbital volume with a resulting late enophthalmos. Blow-out fractures are the most common. By definition, these are limited to one wall and typically are 2 cm or less in diameter. The most commonly involved wall with a blow-out fracture is the anterior medial orbital floor, followed by the medial wall, and less frequently, the orbital roof, which can present as a *blow-in* fracture. Exploration, repair, or reconstruction of an orbital roof fracture may be indicated if a dural tear is suspected or to prevent a "pulsatile globe." This rhythmic inward and outward movement of the eye is due to the cerebrovascular pulsation and the influence of respiration on the overlying cerebral hemispheres. This phenomenon is typically not present acutely but occurs after resolution of edema, with the recovered patient complaining of persistent blurred or double vision. Complex internal orbital fractures consist of extensive fractures affecting two or more orbital walls; they often extend to the posterior orbit and may involve the optic canal. These complex fractures are usually associated with more severe trauma and surrounding fractures such as Le Fort II, Le Fort III, and frontal sinus fractures.

Clinical Examination

Even in the most severely injured patient, the mechanism of injury and surrounding history should be ascertained before performing a clinical examination of the orbit and globe. A systematic approach assessing both the globes and the orbits further defines functional and cosmetic defects. The initial ophthalmologic evaluation should include periorbital examination, visual acuity, ocular motility, pupillary responses, visual fields, and a fundoscopic examination. Often, clinical exam is made difficult by edema, blood, debris, and other distractions. (Figure 22-3)

Visual acuity should be independently tested on each eye using a Snellen chart at a standard 6 m (20 feet) distance or with reading of standard-type print at 40 cm (16 inches). The patient should wear his or her corrective lenses during this examination. If older than 40 years of age, the patient should be wearing his or her reading glasses. The eyelids and periorbital region should be inspected for edema, chemosis, ecchy-

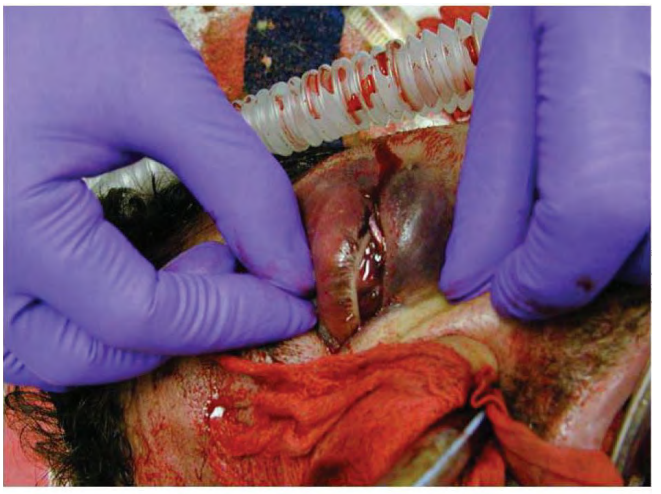
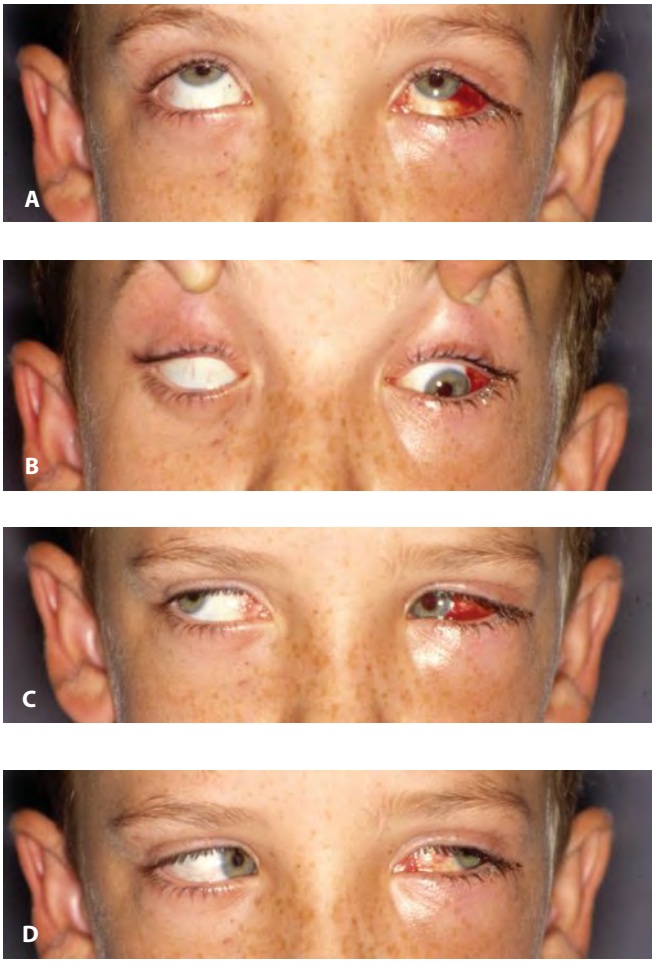


FIGURE 22-3. Clinical exam is difficult when the patient is intubated, edematous and covered in blood and debris.

mosis, lacerations, ptosis, asymmetrical lid drape, canalicular injury, and canthal tendon disruption. With significant acute periorbital ecchymosis, there should be an increased suspicion of a direct blunt globe injury or an internal orbital wall fracture. A lid retractor (Desmarres) is useful for separating swollen tight lids so that the globe and pupil can be adequately examined. Also, this retractor may serve to lift the edge of the lid to examine its inner aspect. With an upper eyelid laceration, any fat that is herniating below the level of the brow through the wound should cause concern that an underlying injury has occurred to the levator muscle. Likewise, if the palpebral conjunctiva has been violated, it is prudent to consult an ophthalmologist to rule out a globe perforation. With a medial vertical laceration of the lids, particularly the lower, gentle lateral retraction may reveal a cut canaliculus or medial canthal tendon disinsertion. Canalicular disruption warrants an urgent ophthalmology consult and usually requires surgical reanastomosis and silicone tube placement into the nasolacrimal system and surrounding supportive repair to prevent outflow obstruction and epiphora.

Extraocular movements are evaluated to rule out mechanical entrapment or paresis. Diplopia, and the field of gaze in which it occurs, should be noted (Figure 22-4 A-F). Of greatest concern is diplopia in the primary (straight-ahead) and downward gazes. These are the two fields that are used most



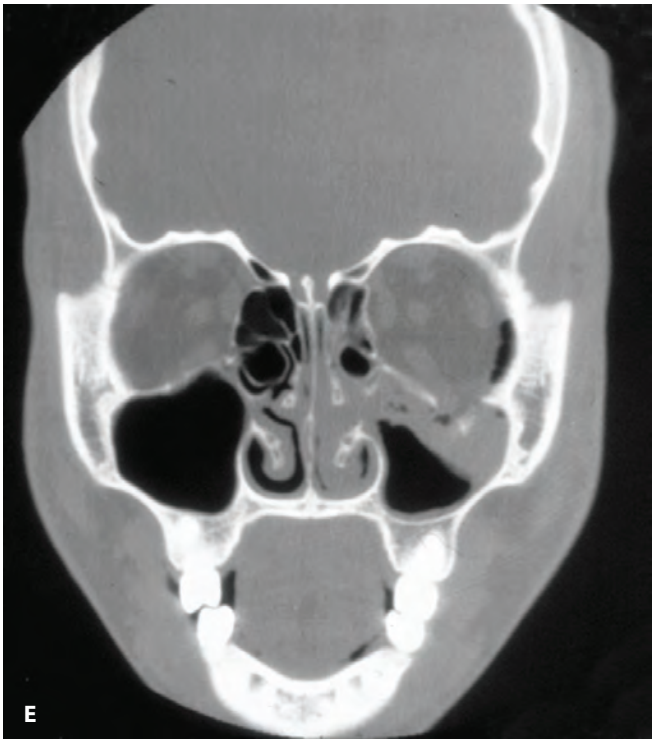


FIGURE 22-4. This 9-year-old child presented with complaint of "double vision and cheek numbness" after being struck in the left orbital region with a hardball. **A,** Note the lateral subconjunctival hemorrhage and that there was no difficulty in the upgaze. **B,** In downgaze, he had severe firm fixed restriction of the left eye that was positive to a forced duction test. **C,** The right lateral gaze had trace restriction. **D,** The left lateral gaze was unremarkable. **E,** Direct coronal computed tomography (CT) scan of the bony window revealed a trapdoor fracture of the left orbital floor with herniation and a probable impingement of the inferior oblique muscle and fascial framework. *(Continued)*

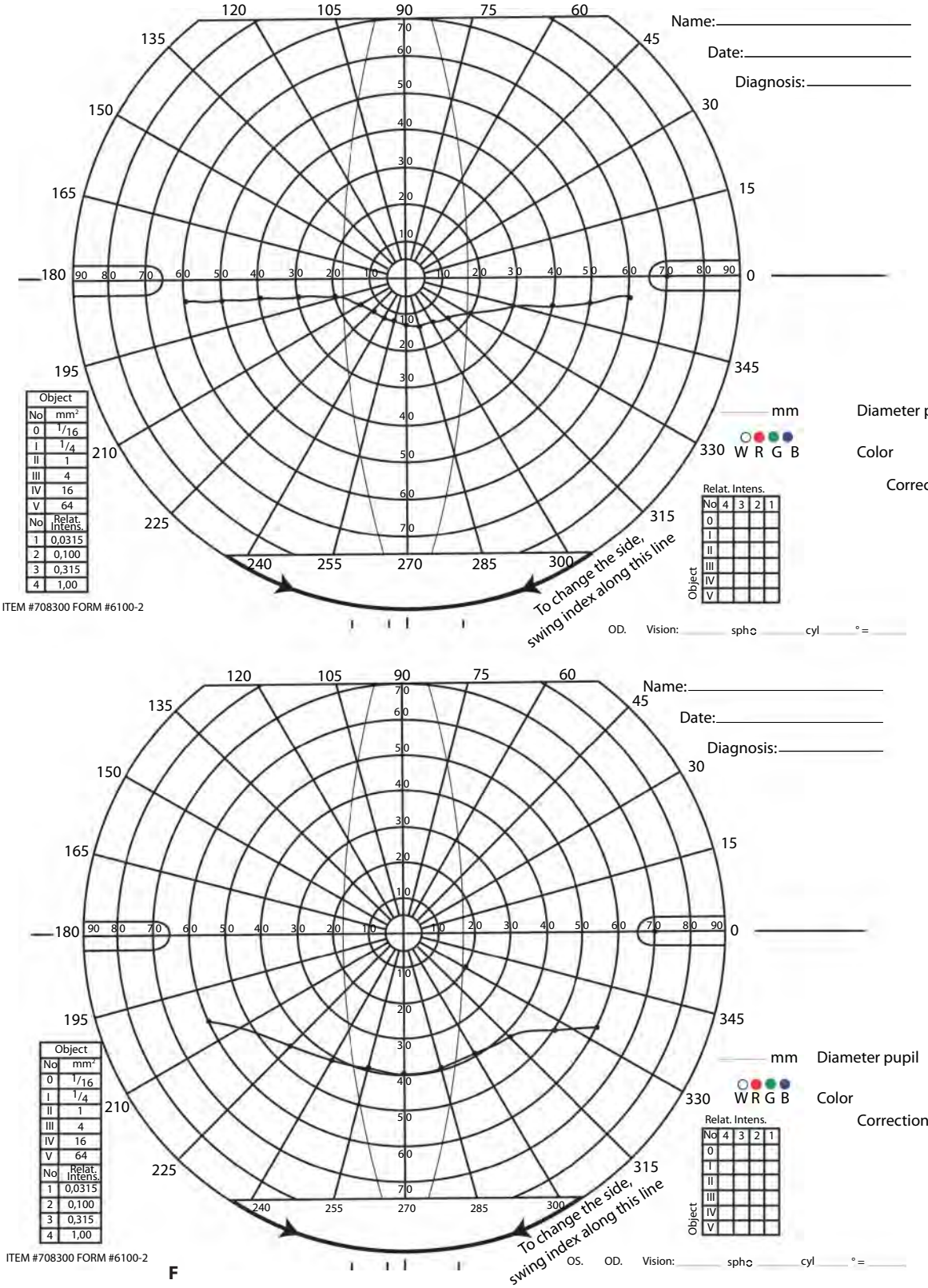
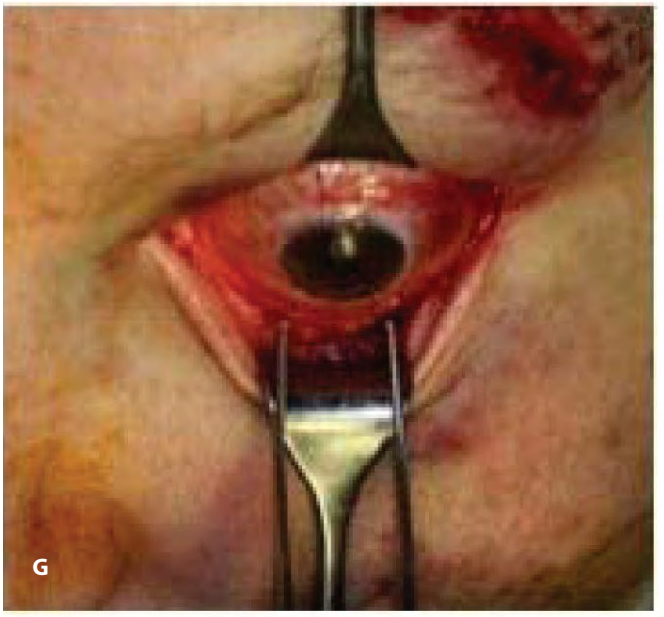
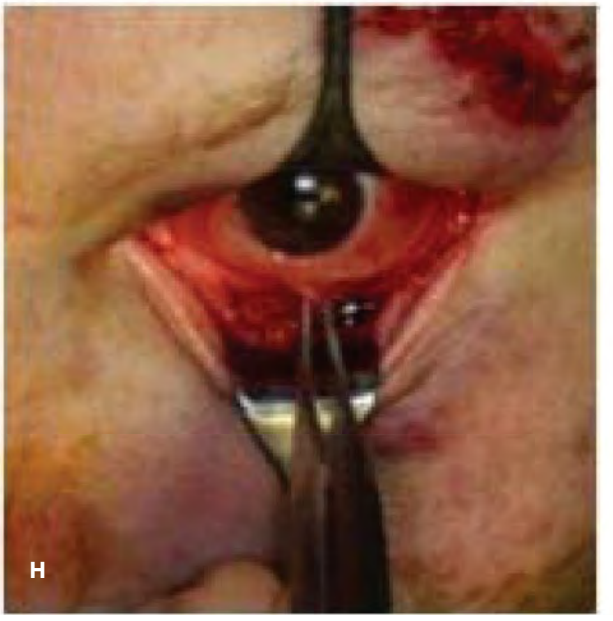


FIGURE 22-4. *(Continued)* **F,** Diploic visual fields (Goldman visual field test). With binocular testing, patients are asked to look at the grid and track a pointed light that is shown from behind the chart. When patients experience double vision, they respond to the examiner who charts the abnormality. In this case, the *upper grid* was recorded at the initial presentation. Diplopia was experienced in all areas below the line (10–12 degrees). This child's severely limited downgaze, correlated with the CT findings, prompted surgical exploration and orbital floor repair within 12 hours. The *lower grid* was recorded at 10 days postoperatively and showed marked improvement in the downgaze, with diplopia occurring at 40 degrees inferiorly *(Continued)*.





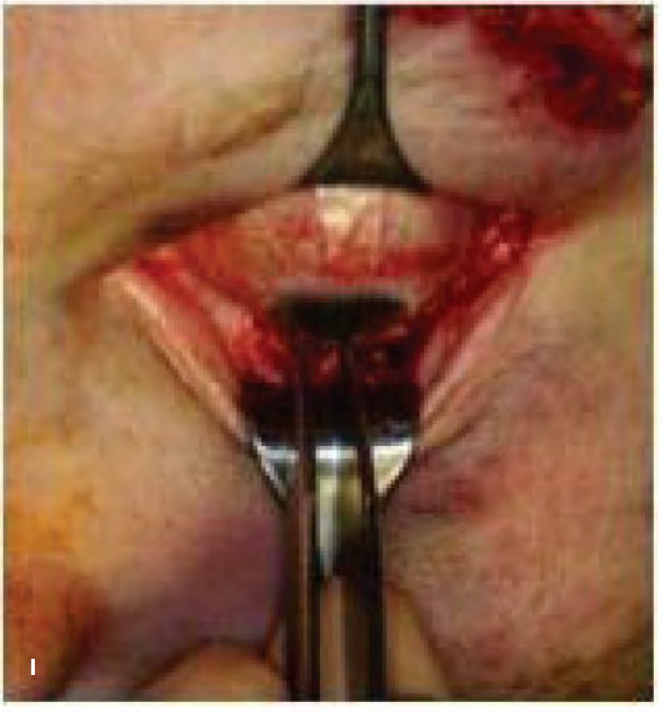


FIGURE 22-4. *(Continued)* **G,** An Adson forceps is used at the inferior fornix with the beaks open, pressing inward against the depth of the fornix and toward the globe side, until the globe rolls downward slightly. **H,** The beaks are then pressed together, grasping the insertion of the inferior rectus. **I,** Upward, downward, and lateral motions can be evaluated.

often. Mild or equivocal restriction (<5 degrees) in extreme fields of gaze is common in the setting of severe orbital trauma with hemorrhage or edema. Computed tomography (CT) scan findings should be correlated with any clinically noted entrapment. If mechanical entrapment is suspected, the eye should be topically anesthetized and a forced duction performed with a fine-toothed forceps. Typically, an Adson forceps is used at the inferior fornix with the beaks open, pressing inward against the depth of the fornix and toward the globe side, until the globe rolls downward slightly. The beaks are then pressed together, grasping the insertion of the inferior rectus. IUpward, downward, and lateral motions can be evaluated (Figure 22-4 G-I). The point of doing a forced duction test is to determine whether the diplopia is due to a restriction of a muscle or paresis of a muscle.

Pupillary light reactivity, size, shape, and symmetry should all be assessed and noted. If unequal pupils (anisocoria) or an irregularly pointing pupil is found, the patient should be queried regarding previous ocular trauma or eye surgery (cataracts). An irregular pupil often points toward the site of a globe penetration or injury. This is often teardrop-shaped, with the narrow portion pointing toward the perforated side of



FIGURE 22-5. Laceration of the right lower medial eyelid that extends through the margin. At first, the examiner thought there was simply a strand of clotted blood on the medial globe. Recognition of the irregular-pointing pupil led to the suspicion of a globe perforation, which was confirmed with a dilated ophthalmologic examination.

the globe, which is usually concealed beneath the lid (Figure 22-5). An ophthalmologist should be consulted immediately and precautionary measures instituted, including protective Fox shield over the eye, head-of-bed elevation, bed rest, analgesics, and antiemetics to avoid sudden increases in intraocular pressure owing to Valsalva forces.

Both globes should be evaluated for any acute enophthalmos, exophthalmos, or vertical dystopia. This is often ascertained from above or by standing directly in front of the patient. Visual fields are tested for each eye, one at a time, by confrontation. The examiner and patient faces should be positioned directly toward each other, 0.6 m apart. The patient is asked to stare directly into the examiner's eyes, while the examiner's hand is held in their own extreme field of gaze, midway between the patient and the examiner. The patient is then asked to detect numbers of fingers showing, motion, or the digit displayed. In essence, the examiner's peripheral field of gaze is serving as a control for the patient.

Quadrant defects are indicative of postchiasm injury. A fundoscopic examination should be performed in a dimly lit room to help maximize pupillary dilatation and ease of the examination. Lens dislocation, vitreous hemorrhage, retinal detachment, and foreign bodies may be noted or may be the cause for not being able to view the fundus. If history and initial clinical findings warrant a dilated fundoscopic examination, neurologic status should be reevaluated and confirmed, and clearance from the primary treating physician or neurosurgeon first obtained. A dilated fundoscopic examination with indirect ophthalmoscopy is generally performed by an ophthalmologist to rule out more occult injuries or examine a greater portion of the globe toward the equator. The ophthalmologist may elect to perform tonometry or a slit-lamp examination. A loss of red reflex is one of the earliest fundoscopic findings of vitreous hemorrhage and must make the clinician aware of possible ocular trauma. Tonometry indirectly measures intraocular pressure by placing the instrument on the surface of the eye. Normal (10–20 mm Hg) or symmetrical bilateral readings are reassuring. However, this does not rule out a penetrating injury. With elevated pressures but an otherwise unremarkable examination, a history of glaucoma should be elicited. An acute abnormally high intraocular pressure with exophthalmos, limited globe movement, and resistance to retropulsion are indicative of a retrobulbar hematoma, which may require acute evacuation via a lateral canthotomy. A "soft eye" with a relatively low pressure or deep anterior chamber is suggestive of a posterior scleral rupture.

A slit-lamp examination is generally performed with the patient in an upright position; if the patient is confined to a bed, a modified examination can be performed with a penlight. A handheld portable slit lamp can be used in the trauma setting. The purpose of this examination is to evaluate the surface contour of the globe and cornea to rule out conjunctival chemosis (swelling), hemorrhage, emphysema, and foreign bodies. The anterior chamber should be evaluated for depth, clarity, and hyphema (blood in the anterior chamber). Hyphema, if found, should be evaluated by an ophthalmologist so that surgical evacuation or medical management may be instituted in an effort to avoid occlusion of the trabecular meshwork, which may lead to glaucoma or a fixed iris.

The iris's shape and reactivity should also be noted. If a corneal abrasion or laceration is suspected, this may be more thoroughly evaluated with fluorescein dye and a Wood's lamp (cobalt blue light). The fluorescein dye pools in the laceration or abrasion and fluoresces with a bright lime-green hue under the lamplight (Figure 22-6).

Finally, the bony orbital rim should be palpated for steps, crepitus, and mobility. The patient should be queried about altered or lack of sensation, and neurosensory testing should be performed to evaluate the supraorbital, supratrochlear, and infraorbital nerves.

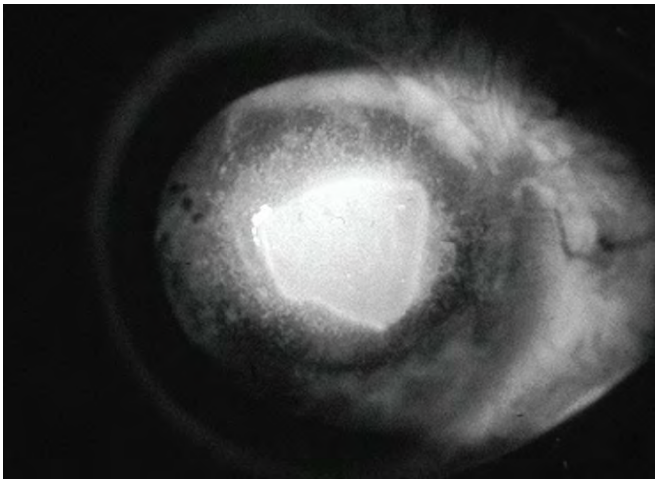


FIGURE 22-6. A broad corneal abrasion of the right eye illustrated with the pooled fluorescein dye under a cobalt blue (Wood's) lamp.

Imaging

Once a complete ophthalmologic and oral examination has been performed, selected studies such as CT or magnetic resonance imaging (MRI) can be ordered with defined parameters to provide meaningful results. Imaging is essential for proper diagnosis and treatment of orbital trauma. Noncontrasted CT is the primary imaging modality currently used for evaluating injuries from blunt or penetrating trauma as well as for localizing most orbital foreign bodies.[15] Other imaging modalities, such as plain radiography, reconstructed three-dimensional CT, MRI, ophthalmic ultrasonography, color Doppler imaging, and angiography, may provide necessary additional information in select instances. CT scans have become the standard of care in evaluating acute orbital injuries. Standard radiography is a readily available and inexpensive method for primary evaluations of orbital fractures. Plain radiography, however, is inadequate when used in evaluating internal orbital fractures, and it is difficult to localize foreign bodies with plain films alone.

If plain films reveal an internal orbital fracture that possibly warrants surgical intervention, CT scans should be obtained. The fracture can then be fully evaluated for surgical treatment planning. CT allows excellent visualization of orbital soft tissues and permits one to simultaneously assess the cranial vault and brain during a "trauma scan." A trauma CT scan series generally involves 10 10-mm axial cuts of the cranium and 5 5-mm cuts through the facial region. If finer detail or three-dimensional reconstructed images are desirable, then 1 1-mm fine cuts can be ordered. Internal orbital fractures are best evaluated when the imaging plane is perpendicular to the fracture line. Thus, images are usually obtained in both the axial and the coronal planes to fully evaluate the fracture lines, patterns, and volume changes. This is particularly useful for comparison to the contralateral or uninjured side. The standard imaging approach for facial trauma is to obtain direct (non-reformatted) 3- to 5 5-mm sections in the axial and coronal planes. Intravenous contrast offers no advantages to the evaluation of acute bony facial injuries. Direct coronal views with 3 3-mm sections are preferred for evaluating orbital roof or floor fractures; however, they may be unobtainable owing to cervical spine precautions or the patient's inability to extend the neck and adequately 6 position himher- or herself himself for the coronal CT In these patients, reformatted coronal images can be obtained based on the axial image data set. However, with this technique, there can be a loss of spatial resolution on the reformatted images. The axial images with fine detail (1 1-mm slices) must be obtained to allow for meaningful reformatted image quality. If an optic canal fracture is suspected, then 1- to 1.5 5-mm axial cuts should be obtained.[16,17] This allows a better determination and correlation of any afferent visual defect owing to possible bony impingement. The standard imaging approach for facial trauma is to obtain direct (non-reformatted) 3 to 5 mm sections in the axial and coronal planes. Intravenous contrast offers no advantages to the evaluation of acute bony facial injuries. A dedicated orbital CT provides 1 to 1.5mm axial cuts. The axial images with fine detail (1 mm slices) must be obtained to allow for meaningful reformatted image quality. If an optic canal fracture is suspected, then 1 to 1.5 mm axial cuts should be obtained.[16,17] This allows a better determination and correlation of any afferent visual defect owing to possible bony impingement. Newer helical CT scanning techniques now allows for reformatting that is very precise. Reformatted views in the sagittal plane allows for better visualization of the antero-posterior extent of the orbital floor defect, that may sometimes be missed or not as well appreciated on coronal or axial views. (Figure 22-7)

Although MRI is generally accepted as a superior soft tissue imaging modality, CT scans adequately assess lens dislocation, vitreous hemorrhage, ruptured globe, retrobulbar hemorrhage, or avulsion of the optic nerve. CT is the imaging of choice in localizing metallic and most nonmetallic foreign bodies in relation to the globe, muscular cone (area inside the extraocular muscles), and the optic nerve.[15,18] The location and extent of any subperiosteal hematoma formation, with possible mass effects, can also be adequately assessed with CT imaging. Computer-generated three-dimensional CT imaging can provide superior views and spatial orientation of fragments for complex orbital and facial fractures. In the majority of acute facial fractures, three-dimensional CT scanning is unnecessary. However, with complex facial trauma with severe displacement, or for secondary reconstruction, three-dimensional CT scanning is invaluable for surgical treatment planning.[19] Generally, 1- to 1.5-mm fine axial cuts are obtained; the patient must remain motionless for the entire scan, which may include more than 100 slices.

Obtaining CT scans in a trauma victim may have some technical difficulties. Sedation may be warranted in pediatric or uncooperative trauma patients. However, with facial bleeding, possible concomitant mandible fractures, or obtundation from alcohol or street-drug use, a secure airway must be maintained throughout the radiology procedure. This may require endotracheal intubation. CT scans may fail to reveal radiolucent foreign bodies such as wood or vegetative matter.[20] In these instances, ultrasonography and MRI are most useful in detecting the radiolucent foreign body and localizing it. These studies should be obtained when the CT scans are equivocal or when physical examination suggests the presence of foreign bodies.

MRI can be useful in the setting of orbital trauma to assess soft tissue injury or entrapment of extraocular muscles in the area of the orbital suspensory framework. Standard radiographs or CT scans should be obtained before MRI is performed on patients with suspected intraocular or intraorbital ferromagnetic bodies because of the potential for displacement of the metallic fragments, resulting in further significant ocular or brain injury.[21,22] With CT imaging, wood can appear isodense with fat or mimic intraorbital air. If the history or clinical examination indicates that fragments of wood may have penetrated the orbit or globe, an MRI should be ordered. An MRI should also be performed when an

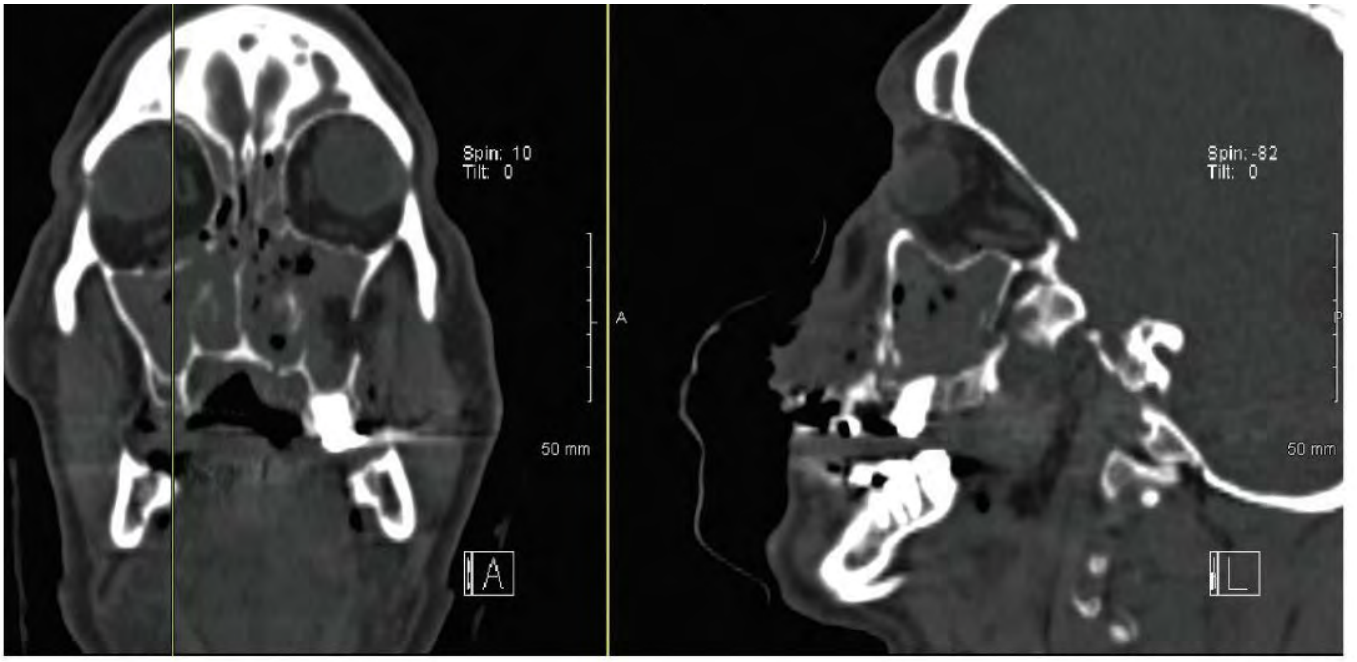


FIGURE 22-7. Reformatted views in the sagittal plane allows for better visualization of the antero-posterior extent of the orbital floor defect, that may sometimes be missed or not as well appreciated on coronal or axial views.

apparent orbital emphysema (focal air collection) fails to resorb rapidly (within several days); this may suggest a space-occupying foreign body.[23]

Ophthalmic ultrasonography is seldom used but is a readily available, safe, inexpensive, and noninvasive imaging modality.[24] Foreign bodies located in the orbit can be identified with ultrasonography but are much more difficult to detect when located in the orbital apex owing to signal reflection. Wood and other radiolucent materials can be detected with ultrasonography.[25] Color Doppler imaging is an ultrasound technique that provides simultaneous two-dimensional images and visualization of blood flow.[26] It can be useful in evaluating a post-traumatic high-flow carotid cavernous fistula. However, angiography remains the study of choice for definitively establishing this diagnosis. With the advent of cone beam CT scanners in offices and outpatient areas, this is becoming a useful entity. The images of a cone beam CT of the orbit however miss much detail especially of the soft tissue but could be useful adjunct in intra-operative navigation assisted reconstruction of the bony structures of the orbit.[83]

OCULAR INJURIES AND DISTURBANCES

Patients who sustain midfacial trauma, particularly in motor vehicle accidents, often have concomitant neurologic and multisystem injuries. A neurologically impaired or uncooperative patient presents additional challenges in performing an adequate orbital and ophthalmologic examination. It is paramount that the primary tenets of advanced trauma life support be adhered to in securing the airway and protecting the cervical spine. When orbital fractures caused by severe blunt force trauma are detected, additional associated injuries should be sought, such as orbital canal or apex involvement, retrobulbar hematoma, or globe perforation. When there are multiple midface fractures, such as those of the ZMC, NOE, and frontal sinus, and Le Fort II or Le Fort III fractures, then more severe intraorbital injury, bleeding, and globe perforation are likely. Basilar skull fractures, as evidenced by clinical signs such as CSF otorrhea or rhinorrhea, Battle's sign, or CT evidence such as fracture lines or intracranial air, are generally caused by high-velocity impact and are often associated with severe neurologic injury.

Superior orbital fissure syndrome is characterized by impairment of cranial nerves III, IV, V, and VI secondary to compression by a fractured bony segment or hematoma formation in the region. Orbital apex syndrome has all the hallmarks of superior orbital fissure syndrome, with the addition of optic nerve (cranial nerve II) injury. Between 0.6% and 4% of patients suffering orbital fractures have a globe injury or optic nerve impairment, resulting in a significant or total loss of vision in one eye.[27,28] This fact highlights the need for a thorough initial ophthalmologic and visual acuity assessment, with follow-up serial examinations as indicated.

Visual Impairment

Visual impairment or total vision loss can occur at various levels along the optic pathway. Direct injury or forces transmitted to the globe by displaced fracture segments can result in retrobulbar hematoma, globe rupture, hyphema, lens displacement, vitreous hemorrhage, retinal detachment, and optic nerve injury. Patients with orbital fractures and any



FIGURE 22-8. Axial CT scan of a right retrobulbar hematoma. This diffuse infiltrative pattern is characteristic, whereas the discreet clot mass is less common.

degree of visual impairment who complain of severe ocular pain should be evaluated for retrobulbar hematoma. It is often the "less impressive" orbital fracture that leads to retrobulbar hematoma formation (Figure 22-8). This is due to bleeding within a relatively closed compartment and the lack of a potential drainage pathway through paranasal sinuses, such as the ethmoids or maxillary sinus. In essence, there is a compartment syndrome resulting from elevation of intraorbital pressure, which leads to central retinal artery compression, or ischemia of the optic nerve. The increased intraorbital pressures can secondarily raise the intraocular pressure, which, in turn, compromises the ocular blood supply.[29–31] In most instances requiring emergent treatment, there is a degree of exophthalmos and excessive tension of the lids. Although CT scanning to confirm the diagnosis is desirable, there should not be unnecessary delay in the surgical management. The immediate or urgent surgical management for retrobulbar hematoma evacuation consists of a lateral canthotomy, with or without inferior cantholysis, and disinsertion of the septum along the lower eyelid in a medial direction. A small Penrose drain is left in place for 24 to 48 hours to ensure adequate drainage and to prevent reaccumulation. Additional maneuvers to lower the intraocular pressure include administration of intravenous mannitol or acetazolamide or application of various glaucoma medications. Typically, blow-in fractures or inward rotation of the ZMC does not result in increased intraorbital or intraocular pressures with visual impairment. This is most likely due to pressure relief and volume expansion provided by additional orbital wall fractures such as the medial wall into the ethmoid or the floor sagging into the maxillary sinus or a large ZMC component to the fracture complex (Figure 22-9).

A penetrating globe injury can result from what appears to be an innocuous small laceration or from horrific blunt-force trauma. When an eyelid laceration is accompanied by an asymmetrical pupil, without a prior history of surgery, a globe perforation likely exists (Figure 22-10). Blunt trauma

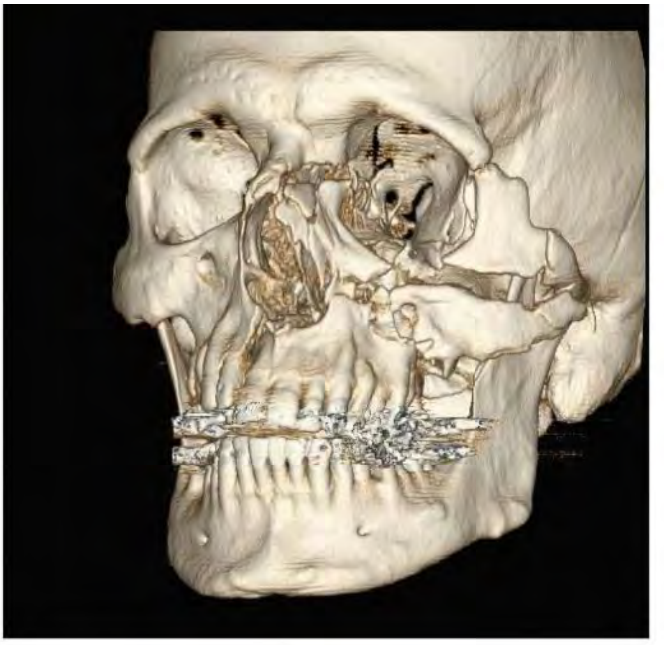


FIGURE 22-9. Accompanying medial orbital fractures as well as ZMC fractures can cause significant expansion of the orbital volume.

can lead to globe perforation owing to a scleral rupture from the sudden instantaneous increased intraocular pressure. The most common site for scleral rupture is at the site of previous cataract surgery, at the limbus, or just posterior to the insertion of the rectus muscles onto the globe, which is 5 to 7 mm from the edge of the limbus. The area under the muscle insertion is anatomically the weakest and thinnest portion of the sclera. With suspected globe perforation, pupillary dilatation and inspection by an ophthalmologist is mandatory. The inspection may be difficult—the injury may not be visible on fundoscopic examination because it is anterior to the equator of the globe and externally may be hidden underneath the rectus muscle insertion. Detection and surgical access for repair may require dissection of the bulbar conjunctiva with retraction of the extraocular muscles and external globe

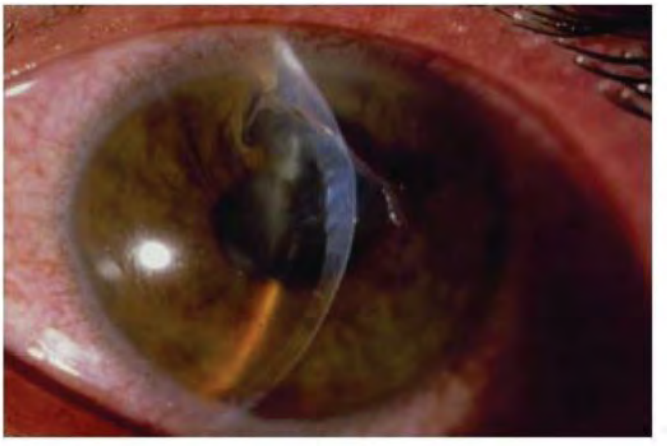


FIGURE 22-10. A full-thickness corneal laceration and an irregular pupil of the right eye is seen during a slit-lamp examination

inspection. The penetrating injuries should be treated emergently, or within 12 hours, to decrease the risk of infection or ocular content herniation. The ultimate visual outcome directly correlates with the presenting visual acuity. Few eyes that cannot detect hand motions or have no light perception (NLP) regain useful vision. Globe injuries should be addressed before any facial lacerations are repaired. The exception is significant active blood loss from a severed vessel.

Sympathetic Ophthalmia

Sympathetic ophthalmia is a rare, bilateral granulomatous uveitis that occurs after either surgical or accidental trauma to one eye. It occurs in less than 0.2% of penetrating globe injuries. The ocular inflammation in the normal eye becomes apparent usually within 3 months after injury, often much earlier. Clinical presentation is an insidious or acute anterior uveitis with gradual decreasing visual acuity in the contralateral uninjured globe Sympathetic ophthalmia is thought to represent an autoimmune inflammatory response, mediated by T-cells,against choroidal melanocytes;which are structurally shielded from the peripheral circulation until injury "exposes them". Diagnosis is based on clinical findings and a history of previous ocular trauma or surgery. Treatment of sympathetic ophthalmia consists of systemic anti-inflammatory agents or immunosuppression. The role of enucleation after the diagnosis of sympathetic ophthalmia remains controversial. Visual prognosis is reasonably good with prompt wound repair and appropriate immunomodulatory therapy.[90]

Hyphema is blood in the anterior chamber of the eye. It can be as severe as complete obliteration of the anterior chamber, termed "eight-ball hyphema," or more commonly a thin 1 to 2 mm layering at the inferior margin in the upright position (Figure 22-11). Some hyphemas are termed *microhyphemas*, with red blood cells floating in the anterior chamber and not layering out. The level and severity of the hyphema should be noted and recorded. The bleeding is from the rupture of an iris or ciliary body vessel and usually is the result of blunt trauma. Patients often complain of eye pain and, occasionally, visual loss if the amount of bleeding is severe. Medical management of hyphema is aimed at preventing rebleeding and venous congestion and promoting clearance of the existing blood. This may include hospitalization, bed rest, head-of-bed elevation, and longer-acting cycloplegics (topical agents such as scopolamine or atropine). Cycloplegics maintain a dilated pupil and thus immobilization of the iris, which discourages further rebleeding. Topical steroids may be administered to decrease further rebleeding and reduce intraocular inflammation. Oral aminocaproic acid is an antifibrinolytic recommended to reduce the incidence of rebleeding into the anterior chamber. In moderate to severe cases, there should be daily monitoring of intraocular pressures and control of any high pressure increases with intravenous carbonic anhydrase inhibitors (acetazolamide, which limits aqueous humor production) or hyperosmotics (mannitol). With severe hyphema, intraocular surgery to irrigate, aspirate, and evacuate the clot may be necessary to prevent optic atrophy owing to elevated pressures or to avoid permanent corneal blood staining.[32] The anterior chamber washout is the most commonly performed procedure for this purpose.



FIGURE 22-11. This partial hyphema of the right eye resulted from a punch to the face; a CT scan showed a minimally displaced orbital floor fracture. The slit-lamp examination shows early layering. This patient received nonoperative management.

Vitreous hemorrhage can result from blunt trauma with the rupture of ciliary, retinal, or choroidal vessels. If, during fundoscopic examination, the retina cannot be visualized despite a normal-appearing anterior chamber and lens, vitreous hemorrhage is most likely present. An early loss of red reflex is also an indication of vitreous hemorrhage. As with hyphema, initial management typically involves hospitalization, bed rest with head-of-bed elevation, and serial clinical examinations. Vitreous hemorrhage is slow to resolve, and it may take months for this to clear, with symptomatic visual improvement.[33] A vitrectomy may be required after 6 months if satisfactory resorption has not occurred.

Lens dislocation may be detected by fundoscopic or slit-lamp examination. The lens, in its normal anatomic position, physically separates the anterior and posterior chambers, but it can be dislocated either partially or totally into either one. Symptoms include monocular diplopia and blurred vision; thus, it is important to check each eye's visual acuity independently. Posterior dislocation may be well tolerated; however, complete anterior dislocation can result in glaucoma and usually requires emergency extraction of the lens.

Rhegmatogenous retinal detachment and peripheral tears result from blunt force trauma. Characteristic symptoms include flashing lights and a field loss best described as a "curtain" or "window shade" coming over the eye. On fundoscopic examination, the retina may not be clearly visualized, or undulations may be present. Retinal detachments require surgery.[33] An emergency consultation with an ophthalmologist and initial maneuvers should be instituted. Maneuvers involve bed rest in a head-up position and assurance that there is no Valsalva-type exertion; these prevent

further extension of the detachment. Operative management may include any or all of the following: a scleral buckle, cryotherapy a vitrectomy, or endolaser. In-office pneumatic retinopexy works well with superior detachments: an inert expandable gas is injected into the vitreous and indirect laser treatment is applied. Retinal ischemia also presents itself similar to a retinal detachment. Should a fundoscopic exam rule out detachment, a high index of suspicion should be maintained for venous or arterial occlusion from micro-emboli causing retinal ischemia and the patient should be worked up to find causes of the same. Orbital ischemic syndrome may be a delayed manifestation of carotid dissection and precede cerebral hypotension in trauma patients[84].

Optic nerve injury or compromise can result from orbital fractures in the posterior region or optic canal. Optic nerve injury or vascular compromise is characterized by decreased visual acuity, diminished color vision, and a relative afferent pupillary defect. It is possible to retain very good vision and yet still have an optic nerve injury manifested by color deficits, afferent papillary defect, and visual field loss. Detection of early subtle changes require that a cooperative patient undergoes visual acuity testing, consisting of testing with a Snellen chart, finger counting, detection of motion, or light perception. Patients may present with NLP, which mandates an emergency consultation with an ophthalmologist and a fine axial CT imaging of the orbital apex. If NLP persists longer than 48 hours, then rarely does any meaningful vision return to the affected eye. Patients with NLP or severely decreased visual acuity may be suffering from traumatic optic neuropathy and should be given high-dose systemic methylprednisolone therapy for at least 48 to 72 hours (initial loading dose of 30 mg/kg IV methylprednisolone sodium succinate, followed by 15 mg/kg IV 2 hr later and q6h thereafter).[34–36] If the patient is uncooperative, heavily sedated, or unconscious, pupillary reaction can be monitored and followed as a sensitive test of optic nerve (cranial nerve II) function. This is best achieved in a dimly lit room; a penlight is moved alternating from one eye to the other every 2 to 3 seconds, and the pupillary response is observed. With the light shining into the normal eye, both pupils should exhibit a brisk constriction. If the light is then directed from the uninjured to the injured eye, the pupil on the injured eye will dilate. This is indicative of an optic nerve injury (relative afferent pupillary defect). A unilateral, fixed, dilated pupil is usually due to an efferent pathway injury (cranial nerve III) or some form of intracranial injury or bleed, which is usually accompanied by other neurologic lateralizing signs. Early loss of monocular dyschromatopsia (impaired color vision) especially red desaturation that can be demostrated using a "red card test" and may be a very early sign of optic neuritis after an ocular injury.

Diplopia

When a patient complains of seeing a double image of the same object, the examiner should first test each eye independently by covering the opposite eye to determine whether the diplopia is monocular or binocular. Monocular diplopia is usually due to lens dislocation or opacification or another disturbance in the clear media along the visual axis. Acute binocular diplopia, secondary to trauma, derives from one of three basic mechanisms: edema or hematoma, restricted motility, or neurogenic injury. The most common cause of binocular diplopia after trauma is orbital edema and hematoma. This is usually found in peripheral fields of gaze, and if other findings are absent, diplopia in the primary and downward gazes usually resolves along with the edema in 7 to 10 days. Slight diplopia in extreme peripheral fields of gaze may persist for months but is rarely problematic because individuals seldom require these extreme views for everyday function. Also the patient may complain that the phenomenon is transitory and that suddenly looking "upward and outward" (superiorly and laterally, such as when looking in a rearview mirror) may cause instantaneous but brief diplopia. Binocular vision without diplopia is most important in the primary (straight-ahead) and downward fields of gaze. The majority of our daily activities, such as conversing, reading, and walking, use these visual fields. If diplopia persists, an ophthalmologic consultation should be sought. Systemic corticosteroids hasten the resolution of orbital edema and the resulting diplopia, which is fairly common after blunt trauma to the orbit.

Persistent post-traumatic diplopia is best evaluated by an ophthalmologist. It is important to establish an accurate diagnosis and precise etiology. The basic evaluation should include assessing symmetry of the corneal light reflexes and testing of ductions (following a finger in all eight fields of gaze) including a selective forced duction. The forced duction helps distinguish between restricted motion from entrapment, scarring, or fibrotic contractures versus a neurogenic motility disorder (cranial nerves III, IV, or VI). Ophthalmologists use diplopic visual fields (see Figure 22-4F) to quantify and categorize the diplopia; serial examinations allow accurate tracking of spontaneous recovery or postsurgical progress. In the acute setting, restrictive disorders are managed with early bony orbital surgery and reconstruction, whereas neurogenic disorders are managed with the injection of botulinum toxin into select extraocular muscles whose forces are unopposed by the injured or restricted muscles. Following bony orbital reconstruction or selective botulinum toxin injections, there should be a 6- to 12-month waiting period for the diplopia to stabilize. Then, any residual and stable diplopia can be addressed with strabismus (extraocular muscle) surgery. Strabismus surgery has two basic maneuvers: a repositioning of muscle insertions onto the sclera or a weakening of the opposing muscles. After a period of healing, selective botulinum toxin injections or more minor revision strabismus surgery may be required to fine-tune the result. The important point to stress is that a healed abnormal bony wall position or orbital volume changes, resulting in enophthalmos or vertical dystopia, typically do not cause stable significant diplopia. In fact, vertical dystopia of up to

1 cm can be accommodated by the brain and should not result in diplopia in the primary fields of gaze. Therefore, any bony wall revision or reconstruction should be performed to correct a cosmetic or other functional defect without promise of correction or improvement in any coexisting diplopia. These reconstruction procedures should be performed and allowed to heal, and the diplopia allowed to stabilize for 6 months prior to the strabismus surgery, which would address the diplopia.

In the trauma setting, diplopia may be due to restricted ocular motility from a prolapse of the periorbital contents into the medially fractured ethmoid air cells or underlying maxillary sinus. Such diplopia may also be due to entrapment or direct impingement on the fine suspensory ligamentous system of the orbit or, less frequently, of the extraocular muscles. Restricted motility or entrapment is commonly found with orbital floor and medial wall fractures, less frequently with roof fractures, and rarely with lateral wall fractures. Significant medial wall fractures are manifested primarily by enophthalmos owing to volume expansion.

When testing range of motion, if there is repeatedly a firm fixed limited stop of unilateral eye motion, the eye should be anesthetized topically and a forced duction test performed. Occasionally, the entrapment or incarceration of the supporting structures or muscles is mild, and during the forced duction, initial resistance may be encountered and then relieved. In such an instance, the positive forced duction test was both diagnostic and therapeutic. However, if the forced duction test is positive and mimics the voluntary active point of restricted motion, this should be correlated with CT scan findings (see Figure 22-4).[37] A repeatable fixed point of limitation is usually due to direct entrapment of the extraocular muscles or the capsulopalpebral fascia (fascia of Tenon). This is more common in linear floor fractures than in comminuted multiple wall fractures. Patients with muscle or tenon capsule incarceration confirmed by CT are candidates for urgent exploration and repair (within 12 hr). Prolonged muscle entrapment with ischemia can lead to fibrosis (Volkmann's contracture) with permanent diplopia, despite surgical release of the entrapped tissues. When exploring these fractures, the entrapped fascia or muscle can be difficult to release. This classically occurs in the pediatric patient with an anteroposterior linear fracture of the orbital floor with no accompanying rim fracture. When an area of resistance is encountered initially and correlates to this same anatomic location on CT, consideration should be given to inserting an instrument within the anterior fracture line and gently twisting or prying to open up the fracture, or taking a fine osteotome or instrument to fracture away a small adjacent strip of orbital floor so that a thin blunt malleable retractor on either side of the entrapped area can gently lift and reduce the entrapped soft tissues back into the orbit. Direct grasping of the tissues and tugging to reduce them back into the orbit may result in further contusion and injury.

Diplopia can be due to a central ophthalmoplegia owing to impairment of cranial nerves III, IV, or VI. The fourth nerve is the most commonly injured at the point where it passes over the petrous ridge of the temporal bone. This results in vertical diplopia and a compensatory head tilt to the opposite shoulder. These nerves have fairly long intracranial tracts and can be injured by direct skull fractures or be compressed by intracranial bleeds or diffuse cerebral edema after blunt head trauma. Cranial nerve palsies often spontaneously recover within 6 to 9 months. Recovery is quite variable and is dependent on severity and the type of injury.

Eyelid Lacerations

Eyelid lacerations, particularly those extending to the lid margin and gray line, should be thoroughly evaluated for lacrimal drainage system injury, canthal tendon disruption, or injury to the tarsal plate and levator aponeurosis. After antibiotics and tetanus prophylaxis have been administered as necessary, the wound should be cleansed and débrided, taking care to protect the globe, possibly with a contact lens. The eyelid laceration should be repaired in a layered fashion, starting with the tarsal plate repair (with 6-0 polyglycolic acid), lid margin (two to three interrupted sutures with 6-0 silk, which is nonirritating to the cornea), orbicularis muscle re-apposition (multiple 6-0 plain gut sutures), and finally skin (with 6-0 nylon or 6-0 fast-absorbing gut). Topical ophthalmic ointment should be prescribed because these agents come in contact with the globe frequently, and sutures should be removed in 5 or 6 days. Patients should be followed up and monitored for potential complications such as scar contracture or lid notching. Several weeks after repair, if significant lid contracture or focal thickening is noted, selective judicious steroid injections (triamcinolone acetonide, 40 mg/mL) can be administered with accompanying daily massage by the patient.

Avulsion or loss of eyelid soft tissue is rare. When this occurs, it is usually from an abrasive crushing macerated-type laceration sustained in such accidents as a rollover in an all-terrain vehicle or ejection from a motor vehicle. In evaluating these injuries, the examiner should moisten the rolled edges of the laceration and attempt to gently realign them. One should not abnormally align the tissues, borrowing them from the periphery and shortening them in the vertical dimension. This can result in lid retraction or lagophthalmos, with risks of corneal exposure and ulceration. It is best either to leave a small amount of denuded underlying tissues, which will reepithelialize secondarily, and possibly perform a temporary tarsorrhaphy, or, for larger defects, to harvest a thin defatted skin graft for primary reconstruction (Figure 22-12).

Lacrimal Injuries

Injuries to the lacrimal drainage system most often result from direct eyelid lacerations at the medial edge of the lid, which traverses the lid margin and disrupt the inferior canaliculus. Canalicular lacerations also occur indirectly when strong forces are applied to the lateral aspect of the lids. This tension directed laterally causes the eyelid to split at the



FIGURE 22-12. **A,** This young male sustained a macerated forehead and eyelid and nasal lacerations after being ejected from a motor vehicle in an accident. **B,** After moistening, redraping, and suturing the soft tissues, it was apparent that there was a significant defect (8 × 10 mm) of skin on the right upper lid. **C,** A full-thickness skin graft was harvested from the right posterior auricular area, which was closed primarily with the aid of releasing Z-plasty incisions. **D,** The undersurface (dermal side) of the graft was thinned and defatted. **E,** The graft was first perforated and inset over the skin defect. A temporary tarsorrhaphy was maintained for 1 week to minimize motion and shearing forces. **F,** Facial appearance 3 months after repair. **G,** Passive lid patency was achieved. There was no further revision surgery.

weakest point, which is just medial to the punctum (Figure 22-13). Damage to the lacrimal drainage system can also be seen with severe medial rim and orbital wall fractures. A disruption in the lacrimal system can be detected by passing a lacrimal probe through the punctum and visualizing the blunt-tipped probe within the laceration or wound. It is especially important to detect this with the inferior canaliculus because this system is dominant in the vast majority of patients.

Repair involves reanastomosis of the canaliculus and either mono- or bicanalicular intubation. With bicanalicular intubation, repair is performed by passing a silicone intubation tube through the puncta into the laceration and then locating the distal cut end of the drainage system for passing the tube into the nose, which is retrieved with a hook beneath the inferior turbinate. Typically, both the superior and the inferior canaliculi are intubated (usually one is uninjured); both silicone tubes are passed into the nose and are tied to each other. This allows for retention of the looped tube for 6 to 12 weeks. Intraoperatively, the silicone tubes are stretched toward the external nares, tied together, and typically oversewn or tied with a fine silk suture to allow for long-term retention. If no tension is applied to the cut ends of the silicone tubing while tying, then, postoperatively, the loop formed at the canaliculi puncta will migrate laterally toward the cornea, causing irritation or an annoying visual field disturbance.

Telecanthus

Traumatic telecanthus typically results from severe midfacial trauma (NOE) with displacement and splaying of the bones that serve as attachments for the medial canthal tendons. It is less frequently due to laceration and actual physical disruption and disinsertion of the canthal tendons from the underlying bone. Therefore, traumatic telecanthus from these injuries

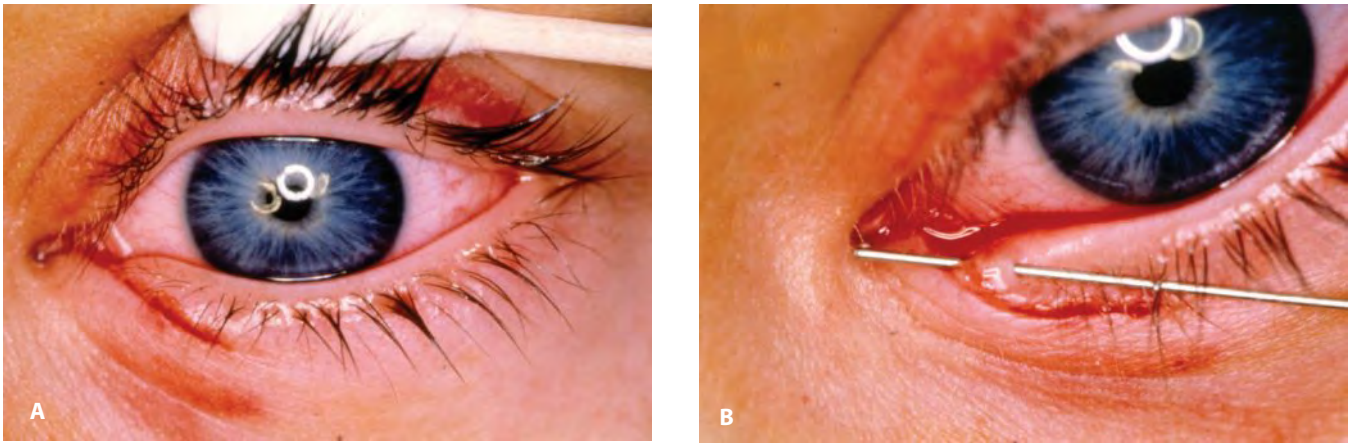


FIGURE 22-13. **A,** Innocuous-appearing small left lower medial lid laceration sustained from an incidental grab along the cheek during a touch football game. **B,** Slight lateral traction on the lower lid and probing of the inferior punctum revealed a full-thickness lid laceration medial to the punctum with severance of the inferior canaliculus. An oculoplastic surgeon repaired and managed this injury within 8 hours.

is best treated early (within 7–10 days) after injury to prevent scarring and secondary maladaptive changes that compromise the reestablishment of the more normal narrow intercanthal distance. Preoperatively, one should determine whether the increased intercanthal distance is due to either a unilateral or a bilateral injury. Treatment typically includes an approach via a coronal incision, a Lynch (lateral nasal) approach, or a combination, with reduction and fixation of the displaced bones or direct transnasal wiring. External splinting rarely yields satisfactory results.

An effective technique to reattach direct canthal tendon reattachment with transnasal wire fixation is best performed by passing a double-end loop of 30-gauge stainless steel wire transnasally from the contralateral medial orbital wall and then suturing the medial canthus to the wire loop. The wire is then drawn to the opposite side by gradually twisting the two ends around a short section of titanium microplate situated in the opposite medial orbital wall (Figure 22-14).

NONOPERATIVE MANAGEMENT OF ORBITAL FRACTURES

Indications for nonoperative or, as it has previously been termed, *conservative* management of orbital fractures, have been controversial for many years. Some historic perspective and review is warranted because it provides insight into the evolution and current thinking regarding nonoperative orbital fracture treatment. In 1957, Smith and Regan[38] coined the term *blow-out fracture* and advocated early surgical intervention for orbital floor fracture repair. Following this, Converse and Smith[39] endorsed surgical exploration and repair of all orbital fractures within the first 3 weeks of injury. Even with surgical exploration and repair, they found that enophthalmos or functional difficulties would develop, and they attributed this to the blunt trauma forces and tissue damage rather than the surgical intervention. Crikelair and colleagues in 1972[40] promoted the concept that orbital floor fractures were overdiagnosed on plain films and, thus, were overoperated. They introduced the concept of repairing only select orbital floor fractures, which were confirmed by tomography and only if diplopia or enophthalmos persisted after an observational period of 2 weeks. This marked an important change in thinking toward a more selective approach for surgical intervention of orbital floor fractures. This change was, in part, prompted by reports and articles documenting unacceptable complications such as a total loss of vision after surgical exploration of asymptomatic floor fractures.[41] In 1974 Putterman and colleagues[42] reported on a series of 57 patients whom they had observed and on whom they had performed no surgical intervention whatsoever. Only a few of these individuals had any persistent diplopia, and there were no visual acuity disturbances 4 months after the trauma. This landmark article created a drastic shift in thinking—nonsurgical treatment of all orbital fractures was advocated. Putterman and colleagues proposed that patients with persistent diplopia should be managed by contralateral eye muscle surgery, or contralateral fat resection, to mask the enophthalmos or altered visual access of the injured side. Although this retrospective study and series of patients received much criticism from both the ophthalmology and the facial trauma specialties, it did reveal that many orbital floor fractures healed uneventfully without surgical intervention and with the performance of eye movement exercises.

Following Putterman and colleagues' report were a series of articles by various practitioners who attempted to refine and delineate the indications for surgical exploration and repair of orbital floor fractures. Dulley and Fells[43] reported that only 50% of all patients with orbital floor fractures required surgical intervention. All patients underwent a 2-week observational period; an individual would then undergo surgical intervention if one of the following criteria was present: enophthalmos greater than 3 mm, large herniation of tissue into the antrum, entrapment with limited





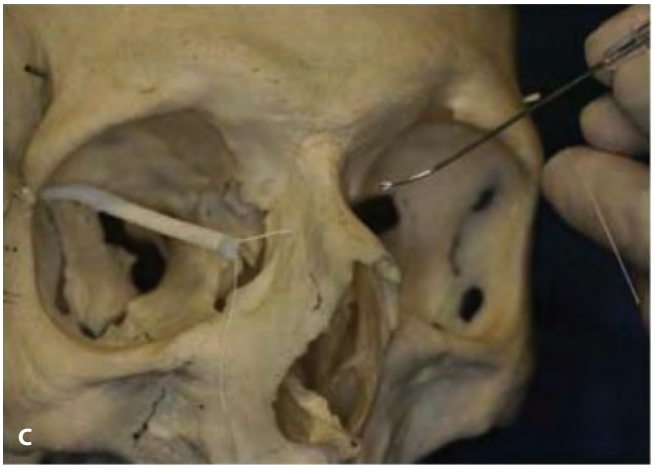






FIGURE 22-14. **A,** Reattachment and repositioning of the left medial canthus is fine-tuned by twisting a 30-gauge wire over a section of microplate situated along the right medial orbital wall just behind and above the posterior lacrimal crest. **B,** Reattachment and repositioning of the left medial canthus is finetuned by twist-ing a 30-gauge wire over a section of microplate situated along the right medial orbital wall just behind and above the posterior lacrimal crest. **C,** The looped 30 gauge wire is placed transnasally, drilling previously through the septum and uninjured side. **D,** The half-round needle that has sutured the medial canthal tissues is then passed through the wire loop and tied **E,** The free wire limbs are then passed on either side of a microplate and then twisted while placing tension so as to draw the medial canthus inward and "fine tune" its position.

upward gaze, or significant diplopia. Nevertheless, these criteria were somewhat subjective and were limited by the current imaging techniques. Crumley and colleagues[44] used indications for surgery similar to those of the Putterman group, but based on these criteria, almost 90% of all their patients with orbital floor fractures underwent surgical repair. Converse and Smith[45] developed and further refined these same indications for orbital floor surgery and reinforced the need and importance of serial clinical examinations in patients who had shown no initial indications for surgery. This group promoted the concept that serial examinations revealing the development of enophthalmos should be the

criterion for surgical intervention and not simply that a large or comminuted floor fracture existed. They proposed that the development of significant postinjury enophthalmos is variable and could be due to either resolving hemorrhage and edema or orbital fat atrophy. In 1982, a survey by the American Society of Ophthalmic, Plastic, and Reconstructive Surgery revealed that two thirds of oculoplastic surgeons were operating within 2 weeks of injury with few serious complications or sequelae.[46] Although this was reassuring that current surgical approaches and techniques were safe, there was no inquiry into what the criteria or determinates were for undertaking surgical repair.

What was helpful was that several ensuing studies began to delineate which patients exhibiting functional deficits might benefit from surgical exploration as opposed to observation. Koorneef,[47] in an anatomic study, showed that fine connective tissue septa surrounded the extraocular muscles. He advocated eye movement exercises in patients with mild or moderate restrictive motility as long as there was demonstrated serial improvement in motility. He purported that edema, hemorrhage, and connective tissue entrapment were responsible for the majority of limited motility in patients with orbital floor injuries.

In 1984, Smith and colleagues[48] introduced the concept that Volkmann's contracture might occur as a result of elevated intraorbital compartment pressures. Although this phenomenon was well-known, documented, and proven in the orthopedic literature to occur with extremities, it was unproven to occur in the orbit. Volkmann's contracture is a paresis from muscle shortening and fibrosis that results in limited mobility. Applying this concept to the orbit, Smith and colleagues recommended surgical intervention in the elderly, in individuals who are hypotensive, and for small or linear orbital floor fractures with coexisting diplopia. They felt that these situations left patients at an increased risk for orbital compartment syndrome, thus developing permanent limited mobility owing to Volkmann-like contractures. Concurrent with these theories and recommendations was the report by Hawes and Dortzbach[49] that emphasized the need for surgical repair within 2 weeks after injury in patients with persistent diplopia within a 30-degree range of the primary visual (straight-ahead) gaze. They based this on their findings that there were poor results when late repairs were performed in this patient group.

Clearly, the advent and ready availability of CT for use in diagnosing "trapdoor" fractures with mechanical impingement of the orbital structures helped to refine diagnostic capabilities and to aid treatment planning. Several groups of authors emphasized the need for correlating a positive forced duction test with CT evidence of incarceration or impingement.[50,51] Without specific evidence of a trapdoor phenomenon or direct impingement, orbital floor fractures with limited motility were observed for 2 weeks. Persistent symptoms or findings then prompted surgical intervention. Trapdoor fractures or fine linear breaks without rim fractures are much more common in pediatric patients. When severe limitation of movement is encountered (typically upward or downward gaze, or both) and is correlated with CT findings, this is a true emergency that should be treated surgically to relieve the entrapment as soon as possible.

Since his initial controversial 1974 article, Putterman and colleagues[52] have revised their indications for surgical intervention. Their indications are now comparable to those of other surgeons. They advocate 7 days of systemic corticosteroids to speed the resolution of diplopia within the first 3 weeks. This may aid in resolving edema and helping determine who might benefit from surgery. Although persistent functional limitations are usually clear indications for surgery, controversy remains in treating those patients who demonstrate a steady but slow resolution of their diplopia that persists beyond 3 weeks.

When the surgeon is confronted with any orbital fracture, it is helpful to categorize the clinical deficits and goals of surgical treatment as being either functional or cosmetic. Simply operating on a radiographic finding because it exists is not satisfactory. The surgeon, with the assistance of his or her ophthalmology colleagues, should determine what, if any, functional deficits and cosmetic deformities exist. A specific anatomic reason for these should be sought. Then, if the magnitude of the functional deficit or cosmetic deformity warrants surgery, the type of surgical approach, repair, and materials should specifically address the structural causes. In a patient with the clinical findings of only "soft" indications for surgery, a 2-week observational period seems prudent. Several studies have addressed cosmetic deformities as they relate to orbital floor fractures, offering indications for surgery versus observation. Hawes and Dortzbach[49] used tomography and felt that orbital floor fractures involving greater than 50% of the surface area should be reconstructed within the first 2 weeks to avoid the predictable development of enophthalmos. They also stated that patients with smaller orbital floor fractures but with greater than 2 mm of enophthalmos present at 2 weeks postinjury should undergo orbital floor reconstruction. This recommendation is based on the fact that later repair is technically more difficult with less optimal outcomes owing to scar contracture and muscle shortening. Parsons and Mathog[53] were able to demonstrate, using a laboratory model, that orbital floor fracture and displacement of equal magnitude with the medial wall fracture and displacement had a much greater effect on globe position. This study supports the practice of most surgeons, which is nonsurgical and observational management of isolated displaced medial wall fractures.

When orbital fractures are associated with other facial fractures such as Le Fort or ZMC fractures, several authors have advocated orbital floor exploration and repair with any evidence of prolapse of the orbital contents into the sinus.[54,55] In 1991, Putterman and colleagues[52] advocated following patients closely for the development of enophthalmos, using objective measurement with a Hertel exophthalmometer or serial measurements for vertical dystopia by aligning the top of a clear ruler to both undisturbed medial canthi and

noting where the ruler bisects each eye. Despite numerous reports, clinical series, and author suggestions, controversy still remains regarding the management of those patients who develop only mild enophthalmos or hypo-opthalmos (1–2 mm) without any functional deficits during the acute observational period.

OPERATIVE MANAGEMENT OF ORBITAL FRACTURES

Indications

It is imperative that the surgeon has a complete understanding of the mechanism of injury and potential complications to make a full diagnosis and an appropriate treatment plan in each type of orbital fracture. Patients with a suspected or known orbital fracture should undergo thorough clinical examination, including fundoscopic examination; visual acuity; pupillary reactivity; detection of diplopia, extraocular movement with any limitations noted, enophthalmos, and vertical dystopia; forced duction testing; and recording of paresthesias. Radiographic studies should determine the full extent of the orbital fracture and any surrounding and associated facial fractures. CT scans, especially in the direct coronal plane, are the gold standard for use in orbital surgery treatment planning. Contraindications for surgery are hyphema, retinal tears, globe perforation, the patient sees only with the eye on the injured side, and life-threatening instability.

Indications for surgery can be divided into functional and cosmetic categories. A logical systematic approach is prudent in selecting patients who are suitable for acute or early surgical repair versus those who deserve an observational period with intervention when signs or symptoms warrant it (Figure 22-15). With regard to function, diplopia and decreased visual acuity are the two main areas of concern. The majority of surgeons and articles in published literature support early surgical intervention in a patient with an orbital floor fracture that has mechanical restriction of gaze and a positive forced duction test with a CT scan that has a trapdoor appearance or suggestions of inferior rectus muscle incarceration.[56,57] This phenomenon occurs more in children with linear fractures owing to the elasticity of their bones.[58] Pediatric or adult patients with these findings warrant early intervention to free up the tissues and hopefully prevent any permanent restriction owing to ischemic necrosis or scar contracture. In patients with less impressive restrictive motility (10–15 degrees), a positive forced duction test, and no CT evidence of muscle entrapment, an observational period of several weeks is reasonable. These patients may only have entrapment of some of the fine connective tissue septa supporting the globe, and with routine daily function and/or eye exercises, this restriction typically steadily improves. Clinical follow-up with a series of examinations (two or three) within the first 14 days, steroid therapy, and eye movement exercises should optimize the outcome. In any patient with an orbital fracture that has persistent mechanical restriction or diplopia within 30 degrees of their primary gaze, especially the downgaze (used during reading), surgical exploration is warranted. Before undertaking surgery, however, any neurogenic or central component should be ruled out. Although infrequently employed, electromyography can be used to distinguish neurogenic diplopia from mechanical restriction in problematic or brain-injured patients. Neurogenic or neuromuscular injuries are more suitably treated by strabismus surgery. With regard to decreased visual acuity, an ophthalmologist should assess the patient serially for resolution or improvement. In more severe cases—patients who can only see shadows or figures or who have NLP—the fine-cut axial CT scans of the orbital apex and canal should be reviewed with the radiologist to determine whether there is bony mechanical impingement, hematoma, and/or edema compressing the optic nerve or vascular supply. With the increasing popularity of endoscopic approaches to the cranial base (typically for tumor removal), most major medical centers have neurosurgeons and/or otolaryngology head and neck specialists who are competent in performing transnasal endoscopic optic canal decompression. If at all possible, this should be performed within 12 to 24 hours of the confirmed diagnosis of external optic nerve compression within the canal proper.

Cosmetic deformities such as enophthalmos or hypo-ophthalmos result from a bony orbital volume increase, extrusion of intraconal fat into extraconal spaces, or prolapse of orbital contents into the maxillary sinus or ethmoid air cells. Contrary to longstanding dogma, post-traumatic fat atrophy does not play a significant role in the development of these deformities.[59] Most surgeons currently undertake surgical intervention in orbital floor reconstruction if there is 2 to 3 mm or greater of enophthalmos or hypo-ophthalmos in the presence of orbital edema or hematoma. The rationale is that early repair offers the most favorable outcome and that the cosmetic deformity only worsens as the edema and hematoma resolve. Orbital floor defects of greater than half of the surface area with concomitant CT evidence of the disruption or prolapse into the underlining antrum generally should be repaired. Again, the rationale for this is that as the edema resolves, eventually there is some degree of enophthalmos or vertical dystopia that creates a cosmetically unacceptable or, less frequently, functional problem requiring surgery. With minimal floor disruption (<50%) and no entrapment or significant herniation, observation for 2 weeks is prudent. If the patient develops any functional problems or enophthalmos greater than 2 mm, surgery can be undertaken to treat the functional or cosmetic defect. Unnecessary delays approaching 6 weeks and beyond make the surgical repair more difficult and the ultimate outcome less desirable owing to scarring and muscle shortening.

Surgical Approaches

Once it has been determined that a patient requires surgical intervention, a well-thought-out plan and sequential approach should be developed. Of paramount importance is the deter-

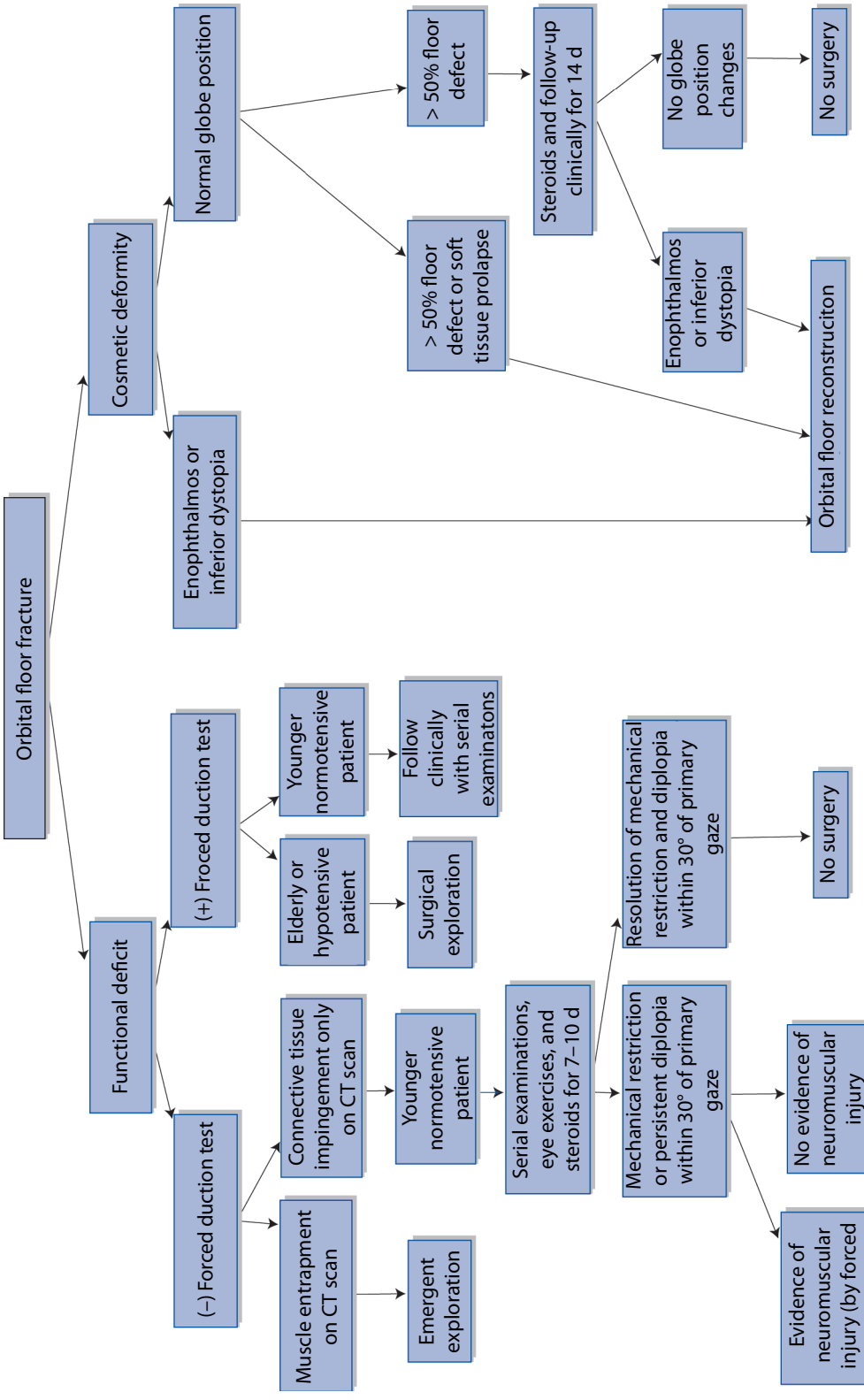


FIGURE 22-15. Orbital floor fracture evaluation and treatment decision diagram. CT = computed tomography; (–) = negative; (+) = positive.

mination of which of the anatomic areas need to be accessed with direct visualization and which intact bony edges or landmarks need to be found or fixated to accomplish the repair. This helps the surgeon determine which soft tissue incision should be employed. In general, most surgeons prefer to first grossly reduce and usually fixate all periorbital and facial fractures before accomplishing internal orbital repairs. The most commonly used surgical approaches and methods of reconstruction are presented here so that the surgeon can make an individualized and informed decision.

Inferior and Lateral Orbital Approaches

There are three basic incisions used for accessing the orbital floor: infraorbital, subciliary, and transconjunctival (Figure 22-16). Although there are three basic approaches, there are numerous technical variations based on surgical training and individual preference. Clearly, the subciliary and transconjunctival incisions are the most popular owing to their superior aesthetics and generous access, and the fact that surgeons are familiar with their use. The infraorbital or rim incision results in compromised aesthetics and offers no advantages over the two former approaches.

The subciliary incision was popularized by Converse in 1944.[60] Typically, a gently curved linear skin incision is made several millimeters below the lid edge or eyelash margin, preferably in a skin crease. The skin flap is then undermined in an inferior direction for several millimeters before traversing deeper inward directly through the orbicularis oculi muscle fibers and stopping when the orbital septum is encountered. The rationale for the division of the skin and muscle at different levels (stepping the incision lines) is that it helps to prevent direct or full-thickness scarring and tethering of the eyelid. Once the orbital septum has been encountered, the preseptal approach is then carried out inferiorly to the orbital rim, and the periosteum is incised just below the arcus marginalis. The periosteum of the orbital rim is then reflected upward and inward, and dissection is carried out over the orbital rim. One must bear in mind that the orbital floor drops off several millimeters toward the inferior direction before heading straight posteriorly. The orbital floor dissection can then be extended posteriorly for a safe distance of 30 mm. With an intact adult rim, the optic canal is only 40 mm from the anterior lacrimal crest, and with any rim displacement inward, this margin of safety is further decreased. A modification of the subciliary approach is the "skin-only" incision. This technique is comparable to the technique just described, except that after dividing the skin, the inferior dissection is carried out superficially to the orbicularis oculi muscle fibers until the inferior orbital rim is reached, and then the muscle is divided at the same level as the periosteal incision. This approach is used less often owing to the amount of stretching on the unsupported large skin flap and the resultant high rate of ectropion (permanent in 8%) and potential skin necrosis, particularly in the elderly patient who has a history of heavy smoking.[61] These complications prompted the development of an alternative technique called the "skin-muscle flap." With this procedure a similar incision is accomplished 1 to 2 mm below the lid margin but is carried through both the skin and the muscle at the same level down to the tarsal plate. Again, the plane of dissection is carried out anterior or superficial to the orbital septum (preseptal) until the orbital rim is encountered. This approach results in excellent

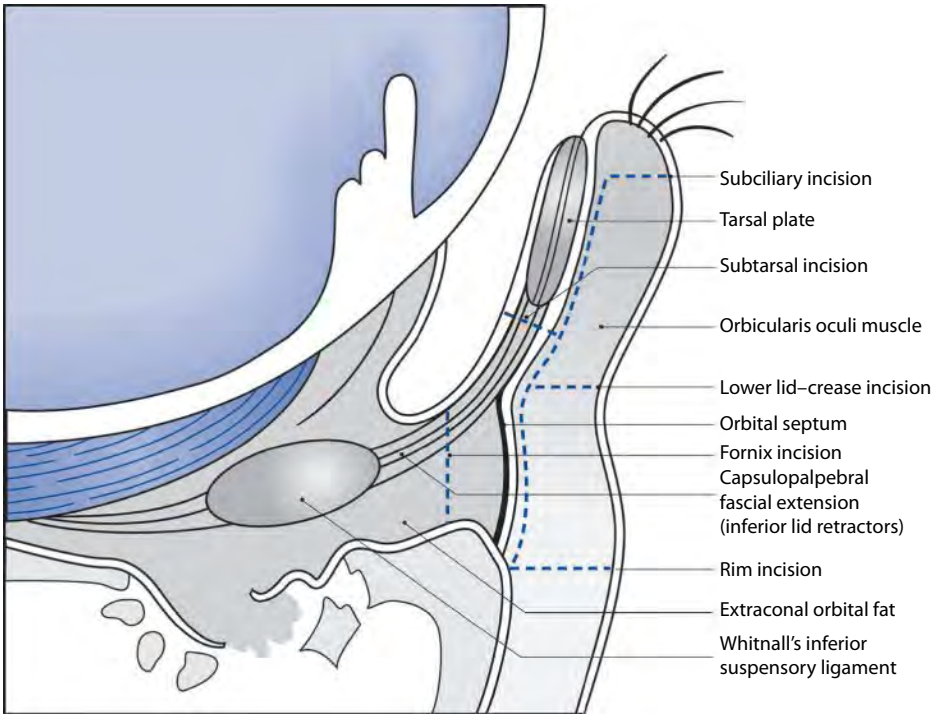


FIGURE 22-16. Cross-sectional view of the inferior lid and various floor approach incisions. (Adapted from Ochs MW, Johns FR. Orbital trauma. In Fonseca RJ, Marciani RD, Hendler BH, editors. Oral and Maxillofacial Surgery: Trauma. Vol. 3. Philadelphia: WB Saunders; 2000; p. 208.)

aesthetics, a simplified dissection, and a decreased incidence of hematoma formation or skin necrosis. This skin-muscle flap still carries a 6% rate of early ectropion[62]; however, it is generally temporary and resolves within several weeks with gentle massage. This was confirmed by several investigators who correlated preoperative periorbital edema and increased age positively with the development of this temporary ectropion with the subciliary approach.[63] A revision of this approach or technique is to use a relaxed skin tension line incision.

The transconjunctival approach for orbital floor fractures was first popularized by Tessier[64] and Converse and associates[65] in 1973 for orbital floor fractures. The two basic variations of this approach to the orbital rim are retroseptal or preseptal approaches. Although the retroseptal approach is a more direct approach to the rim, it exposes the orbital fat, which herniates into the surgical field and may interfere with the surgery and result in more fat atrophy, especially with cautery, and hence enophthalmos. For this reason, the preseptal approach is generally favored.[66] The preseptal approach (see Figure 22-16) as described by Tessier[64] involves an incision through the palpebral conjunctiva just 2 to 3 mm below the inferior edge of the tarsus that is extended through the inferior lid retractors and orbital septum. Next, a preseptal vertical dissection is carried out down several millimeters below the orbital rim, and the periosteum is incised. The dissection of the facial aspect of the rim and the floor is then carried out. This obviates orbital fat herniation in a fairly bloodless field. The necessity for a periosteal closure is controversial owing to the possibility of entropion or ectropion with inadvertent suturing of the periosteum to the orbital septum or other layers.[64,67] Some surgeons advocate a Frost suture for a period of 24 to 48 hours to allow for proper lower lid redraping during early healing. Most surgeons find this unnecessary. If there is any difficulty in identifying opposing edges of the cut periosteum, no suturing should be performed rather than an inappropriate tethering of more superficial or superior eyelid layers and structures to the underlying rim. Many instances of "early ectropion" or a "shortened lid" are the result of improper suturing. The transconjunctival preseptal approach enjoys a low incidence of unfavorable scarring with ectropion or entropion (1.2%).[55] However, one drawback to this approach remains a somewhat-limited view during the preseptal dissection and limited exposure once the orbital floor has been accessed. For this reason, the lateral canthotomy and complete severance of the lower limb of the lateral canthal tendon (inferior cantholysis) was introduced by McCord and Moses in 1979.[68] This procedure allows for a generous tension-free exposure to the orbital floor, lateral orbital wall, and medial area. The surgical exposure obtained with the transconjunctival approach with the inferior cantholysis is superior to that of a subciliary incision. Also, the much smaller cutaneous incision is placed in a more favorable area of the crow's-feet.

The majority of surgeons currently use the transconjunctival incision with or without canthotomy or the subciliary incision (preseptal approach) for orbital rim and floor access.[69] Both of these basic incisions provide good exposure with excellent aesthetics and an extremely low rate of complications. Each surgeon's own training, familiarity, and personal preference should guide which rim approach is used.

Superior and Medial Orbital Approaches

Access to the superior orbital rim and zygomaticofrontal (ZF) suture can be accomplished via a lateral eyebrow incision, upper blepharoplasty incision, coronal incision, or lateral canthotomy incision that is an extension of a subciliary or transconjunctival incision with a superior cantholysis. The eyebrow incision, if performed properly, results in excellent aesthetics and is quickly and easily performed; therefore, it is one of the more common approaches used for the lateral orbital rim or ZF suture area. The other incisions described are used more often when extensive facial fractures are present that require extensive skeletal exposure of the superior rim, cranial vault, or zygomatic arch.

The lateral brow incision is placed on the extreme outer aspect of the eyebrow, usually just superior to the ZF suture. The ZF suture line is usually approximately 1 cm above the lateral canthus. Generally, the skin of the lateral brow is tented over the superior lateral orbital rim, and a 1.5-cm curvilinear incision is made in a beveled fashion paralleling the hair follicles. Double-pronged skin hooks are then placed on the skin margins, and traction is maintained with digital palpation of the internal edge of the orbital rim. The skin incision opening is then gently retracted inferolaterally more directly over the ZF suture, and a needle-tipped Bovie cautery is used to divide the orbicularis oculi muscle fibers overlying the rim and ZF suture. Additional undermining and dissection is carried out in an inferolateral direction to provide full and adequate access to the fracture and enough adjacent bone to allow for rigid fixation. The advantages of not extending the skin incision beyond the brow obviously involve aesthetics (placing it in the well-camouflaged and hidden area of the hair follicles) but also include that the skin is stepped and muscle incisions are made in distinct layers, which provide for more favorable healing. This incision also allows access for placing a blunt curved instrument deep to the zygomatic arch for the reduction of the ZMC or arch fractures. Closure should be accomplished in three distinct layers of periosteum, subcutaneous tissue, and skin. The periosteal, muscle, and deep subcutaneous closures are particularly important in that they provide the bulk of soft tissue over any plates and screws in the region.

The upper blepharoplasty incision can also be used for access to the ZF suture. The incision is placed in one of the upper eyelid skin creases, preferably the deepest crease (which can be marked preoperatively, with the patient awake). The skin incision is then carried down through subcutaneous tissue, retracted somewhat laterally, and extended through the orbicularis oculi and periosteum by sharp dissection. Generally, a 1-cm length of the lateral blepharoplasty inci-

sion is all that is required for complete access to the lateral orbital rim. This is due to the suppleness and mobility of the thin eyelid skin. Care should be taken to not over-retract the tissue, and the skin incision should be extended slightly laterally if excessive retraction forces are apparent. Separate suturing of the periosteum and skin is all that is required.

The coronal incision allows for excellent access to the entire supraorbital rim, roof, frontal sinus, superior aspects of the nasal bone, lateral orbital rim and wall, medial orbital rim and wall, and zygomatic arch.[70] This approach is generally necessary for extensive facial fractures involving the zygoma, frontal sinus, and NOE complex and for Le Fort III fractures. Numerous variations of the incision design exist, but generally, a curvilinear incision is placed at least 2 cm posterior to the hairline (in the midline) and then extended posteriorly, paralleling the hairline, and finally inferiorly into the preauricular region. It is generally helpful to carry the vertical component of the coronal incision overlying the temporalis muscle just posterior to the junction of the superior helix and the scalp. It is then sharply angled forward, hugging the anterior helix and preauricular skin crease down to the pretragal area. By doing so, the superficial temporal vessels are generally not encountered or violated and retracted forward with the flap, allowing for a much drier field. It is not necessary to shave the scalp, but a 1-cm area of hair can be trimmed at the incision to allow for ease of closure, postoperative hygiene, and suture removal. Local anesthesia with vasoconstrictors is helpful for hemostasis and often obviates the need for compression (Raney) clips. The incision is carried out through the skin, subcutaneous connective tissue, and galea aponeurotica into the loose areolar tissue in the midline. The subgaleal plane of dissection is contiguous with a plane deep to the parietotemporal fascia in the area of the temporalis muscle. The incision is then extended laterally in the supraperiosteal plane; it is helpful to insert a Metzenbaum or curved Mayo scissors in this plane before extending the incision laterally. This prevents inadvertent incising or nicking of the temporalis in an otherwise dry field. The dissection is carried out laterally to the superior temporal line bilaterally. Dissection is then carried anteriorly to the frontal bone, and a horizontal incision is made through the periosteum approximately 2 cm above the superior orbital rim. The incision is carried laterally to the superior temporal line and joined with the preauricular area inferiorly through the superficial layer of the deep temporal fascia to protect the temporal and frontal branches of the facial nerve.[71] The facial nerve courses in a plane superficial to the deep temporal fascia approximately 1 to 3 cm from the tragus along the zygomatic arch.[72] This approach provides complete access to the medial, lateral, and superior orbital rims. When a more extensive view of the medial orbital wall is required, subperiosteal dissection and release of the superior trochlea can be performed—the flap is retracted more inferiorly over the nasal dorsum, with a direct view of the medial wall. No attempts should be made to reattach the trochlea because, when the soft tissues are redraped, the trochlea readheres on its own. Suturing may actually pierce or violate the trochlear tendon and result in ocular motility disturbances. Closure of the coronal flap should include suspending the deep temporal fascia over the temporalis muscle, deep closure of the galea aponeurotica, subcutaneous buried suturing, and closure of the skin. It is important to remember that when a hemicoronal incision is employed, the medial extent of the incision should be carried beyond the midsagittal plane and extended completely to the hairline. This allows for adequate reflection and retraction over the entire zygoma and orbital rim structures.

When a transconjunctival incision is used with a lateral canthotomy, an extension of the dissection superiorly can be used for access to the ZF suture by severing the superior limb of the canthal tendon.[67] This approach provides good access to the lateral and infraorbital skeleton; however, it is less frequently used because it requires a more complex closure and reanchoring of the lateral canthal tendon complex. Any misalignment results in canthal dystopia, usually in an inferior direction, and a rounded-out "almond-shaped" eye appearance. If the superior canthal tendon and its origin to the internal rim are allowed to remain intact, it provides a highly reliable landmark to which the inferior canthal limb can be sewn, resulting in excellent sharp-angled (30–40 degree) aesthetics.

The entire lateral wall and rim is easily accessed through a standard blepharoplasty incision that extends only to the lateral orbital rim. This approach is commonly used for lateral orbital decompressions in cases of severe thyroid orbitopathy, and it affords excellent exposure also to portions of the orbital roof and to the apex of the orbit laterally.

Medial Orbital Approaches

Access to the medial orbital rim and superior aspect of the medial orbital wall can be accomplished through a coronal incision, as previously described. However, a separate lateral nasal incision can be used for isolated medial wall exploration or to access the inferior aspect of the medial orbital floor. This can be a transconjunctival or subciliary approach to the inferior rim and floor. The entire medial wall can be visualized by extending the transconjunctival incision through the caruncle. The medial orbital wall and rim, by definition, are involved in fractures of the NOE complex, Le Fort II and III fractures, extensive frontal sinus fractures, and occasionally, large blow-out fractures. The lateral nasal incision is most often used for access to the medial orbital rim to reconstruct a detached medial canthal tendon with direct transnasal wiring. This type of injury often occurs with NOE fractures and Le Fort III fractures. As stated earlier, medial orbital wall fractures generally do not result in any entrapment or ocular mobility problems. Generally, the upper third of the medial orbital wall is uninvolved or nondisplaced, simply because it is the very thick extension of the cranial base. The lower two thirds of the medial orbital wall overlie the ethmoid air cells and can be displaced inward, resulting in volume expansion. Unless there is extensive involvement, generally the resulting increase in orbital volume does not

result in the development of enophthalmos. If the inferior two thirds of the medial wall or orbital floor are involved and require surgical repair, the previously described approaches to the orbital floor should suffice. However, fractures that extend farther superiorly (above the frontoethmoidal suture/anterior ethmoidal foramen) may require a lateral nasal approach or coronal incision. The lateral nasal approach involves a vertical gentle curvilinear 1-cm incision approximately 5 to 10 mm medial to the insertion of the medial canthus. Care should be taken not to place this incision too close to the medial canthus because this can result in a scar contracture with "webbing" and an abnormal epicanthal fold postoperatively. The incision should be placed over the lateral nasal structures properly, and after the skin incision is made, the dissection should be carried straight medially through skin, subcutaneous tissue, and a rudimentary portion of the orbicularis oculi muscle and periosteum. There is no need to step these layers. The periosteum can then be reflected posteriorly and superiorly to the medial orbital rim and wall. The medial canthal tendon and lacrimal sac lie posterior and just inferior to the incision. The anterior ethmoidal vessels lie posteriorly and superiorly approximately 24 mm from the anterior lacrimal crest. These vessels can be gently divided with bipolar cautery, providing excellent hemostasis and improved access for identifying an intact bony ledge. However, one should bear in mind that any bony violation or entry superior to this line carries the potential risk for entry into the anterior cranial fossa. When an orbital implant is required along the medial wall, anterior fixation of the implant is recommended.

The transcaruncular approach is not a very popular choice for most surgeons to approach the medial orbit, but it has its indications. The plica semilunaris is the crescent-shaped fold of conjunctiva and is considered a vestigial remnant of the nicitating membrane. The caruncle is the fleshy keratinized structure attached to the medial side of the plica semilunaris and the common canaliculis is just medial to this. Traction sutures placed on the medial portion of both the upper and lower lids and holding the plica semilunaris with forceps taking care not to damage the puncta and canaliculi, an incision is made through the caruncle with Westcott scissors and extended inferiorly and superiorly by about 10–12 mm. Blunt dissection is then carried medially by opening the Stevens scissors, and the exposed periosteum is incised in a superior inferior fashion. Retractors are placed to provide an unobstructed view of the medial wall and the floor of the orbit. (Figure 22-17 A,B,C) The incision is closed using 4.0 fast resorbing suture. The transcaruncular approach to the medial orbital wall allows excellent access and is considered by some to be cosmetically superior to the Lynch approach.[87, 88]

Endoscopic approach

The endoscopic approach to the orbit has gained popularity over the years.[89] The approach utilizes a 30 degree 3 mm transantral endoscope to assist with visualization of the orbital floor through a Caldwell-Luc antrostomy window. The sinus mucosa is then dissected around the fractured orbital floor using sinus intruments, and the fracture delineated. A 0.4mm Medpor (Porex Surgical, Inc, College Park, Georgia) is then folded and inserted into the orbital defect taking support of the intact ledges when unfolded. This approach provides the greatest advantage of avoiding a skin or conjunctival incisions (Figure 22-18).

Acute Repair

Internal orbital fractures have varied patterns and degrees of severity. It is helpful to attempt to classify them either as linear, blow-out, or complex fractures. Linear fractures are those in which the bone fragments and walls remain intact. However, owing to angulation or overlap, they may result in either a bony orbital volume increase or decrease. Overlap fractures generally result in a bony defect of one orbital wall (typically the medial orbital floor) and are the most common orbital fracture. Blow-in fractures can occur in any orbital wall but most commonly occur in the roof and are associated with frontal sinus fractures. Blow-in and blow-out fractures of the orbital roof occur with equal frequency. Complex fractures are those that involve two or more walls, are greater than 2 cm in diameter, or are comminuted with displaced and unretrievable segments. Often, these complex fractures are associated with fractures that extend beyond the orbital frame such as Le Fort II or III and frontal sinus fractures. These are termed *combined fractures.* The goals of acute or primary reconstruction of primary orbital fractures are to alleviate any functional deficit and to restore the facial aesthetics.

Linear fractures are generally caused by blunt forces directly to the globe or partially to the rim and most often result in an aesthetic deformity such as enophthalmos or hypo-ophthalmos. Functional deformities with entrapment are less common with linear orbital fractures. However, isolated linear fractures can have an instantaneous trapdoor effect owing to momentary expansion and entrapment of the edge of soft tissues including the inferior rectus. Once tightly pinched between these bony segments, this manifests itself as severe ocular motility restriction that is reproducible on serial examinations at the same point of limitation. There is also a positive result to the forced duction test. This kind of entrapment of the muscle can also lead to the vagally mediated oculo-cardiac reflex.[85] This type of fracture necessitates immediate surgical intervention to prevent the ischemic necrosis of the extraocular muscles. The majority of linear fractures in the orbit do not result in aesthetic deformities such as enophthalmos or hypo-ophthalmos unless there is an associated facial fracture such as a fractured ZMC with a medial and downward rotation. It is the volume changes that account for the abnormal globe position. The goal of reconstruction is to restore the anatomic position of the bony rim and associated facial bones and to reapproximate, to the





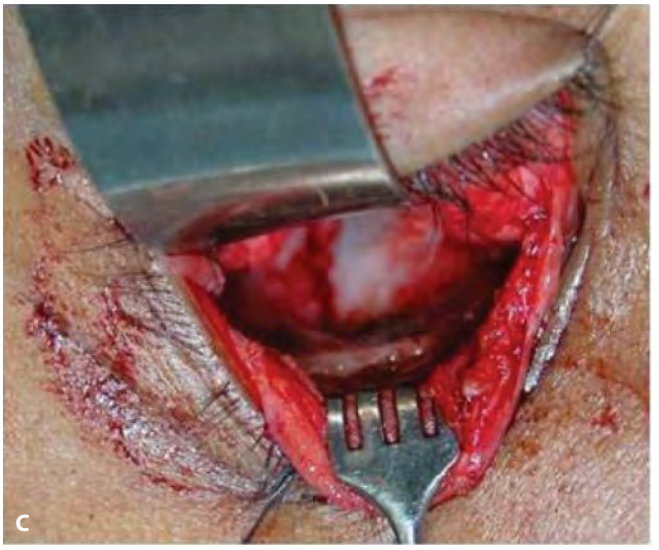


FIGURE 22-17. Transcaruncular approach to medial orbital wall and floor **A,** Incision through the caruncle with Westcott scissors **B,** The incision is extended inferiorly and superiorly 10 –12 mm **C,** Blunt dissection medially by opening the Stevens, periosteum is incised in a superior inferior fashion, and retractors provide unobstructed view of the medial wall and the floor of the orbit.

best of one's ability, the normal bony orbital volume with a reconstructive material. Numerous materials have been described in the literature for these purposes, such as porous polyethylene, bioresorbable polydioxanone, nylon, gelatin film, titanium mesh, and autogenous bone grafts (split-thickness calvarium and, less frequently, iliac crest).[73–79] Each material has advantages and disadvantages related to the strength, application, reactivity, infection rate, biointegration, and complication rate associated with its use.

For linear and blow-out fractures, thin (0.85-mm) porous polyethylene sheeting could be used. This alloplastic material is extremely biocompatible and nonresorptive. It has more than adequate tensile strength and does not cause any capsule formation such as that seen with polymeric silicone sheeting. It has considerable flexibility (which can be improved with placement in an autoclaved saline) and little memory properties. The pore size allows tissue ingrowth, which reduces the risk of migration.[80] Despite that, it is often recomennded to anchor the porous polyethylene sheeting to the anterior lateral orbital floor with a single titanium screw (Figure 22-19). The greatest advantages of this material are its ease of contouring, in situ carving, burring, and ability to be layered posteriorly behind the orbital equator to achieve proper orbital volume and contour.[52]

Titanium mesh, with fixation to surrounding intact orbital rims, is quite useful when there are severe or comminuted injuries and a cantilevering is required because intact internal medial or posterior bony margins have not been identified or accessed. However, the possibility of unacceptable postoperative scarring to the mesh may occur, resulting in limited ocular motility. Therefore, when titanium mesh is employed, it is recommended to overlay it with either a split-thickness calvarial graft or a sheet of porous polyethylene sheeting. These materials are secured to the underlying mesh with either 30-gauge stainless steel wire or suturing (Figure 22-20).

Blow-out fractures typically involve one orbital wall (usually the anterior or medial portion of the orbital floor) and are less than 2 cm in diameter. Enophthalmos associated with orbital blow-out fractures is due to an enlargement of the orbital bony volume that allows the orbital fat to be distributed within a larger compartment.[39] Fat atrophy contributes

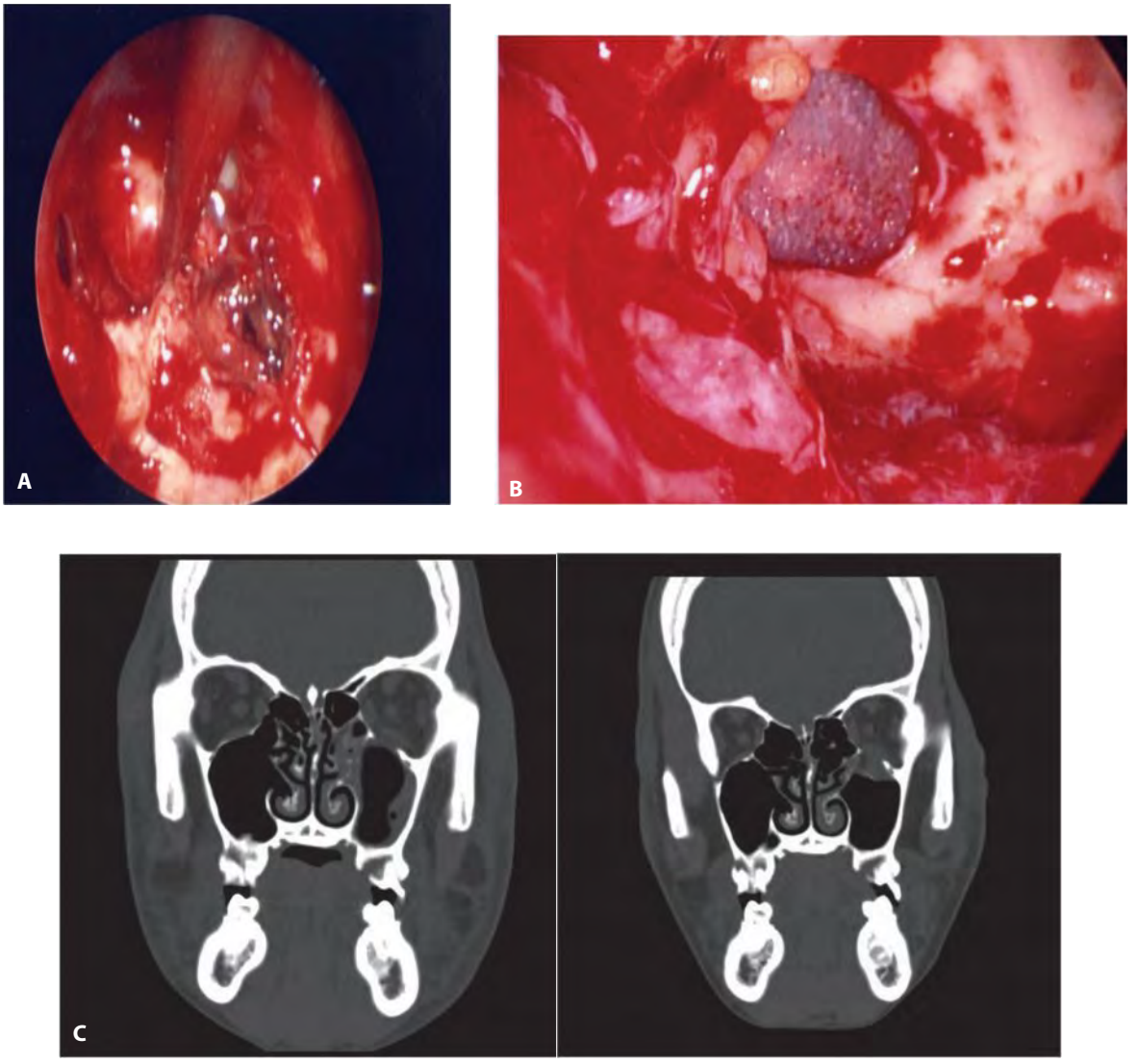


FIGURE 22-18. **A,** Endoscopic approach to orbit. Endoscopic view through maxillary sinus looking up at the fractured orbital floor and herniated orbital contents. **B,** Good reduction of orbital contents, removal of all bone, and appropriate placement of 0.4mm porous polyethylene orbital floor implant spanning the entire defect. **C,** Pre- and post-operative views of orbital floor fracture repaired endoscopically. Photos courtesy Dr. Rui Fernandes, Jacksonville, Florida.

little, if anything, to the development of early or late enophthalmos.[81] The reverse mechanism, often referred to as blow-in fracture, may result in a decreased orbital volume. Exophthalmos and ocular motility disturbances are uncommon unless there are surrounding severe associated fractures such as ZMC or frontal sinus fractures.

In 1960, Converse and Smith[39] introduced the concept of "pure" (isolated floor) and "impure" (floor and rim) blow-out fractures. Pure fractures are thought to be caused by a sudden instantaneous increase in intraorbital pressures from direct blunt-force trauma to the globe itself. Impure fractures are purported to be caused by direct trauma and compression of the bony rim and collapse of the surrounding facial bones, and result in the disruption of the internal orbital walls. What is most disconcerting is the finding of associated globe trauma such as hyphema, iridoplegia (ciliary body paralysis), and retinal hemorrhage in 90% of patients with pure blow-out fractures. This supports the notion that pure blow-out fractures are created by substantial instantaneous direct globe trauma. This fact should heighten one's awareness of the potential for serious globe injury when dealing with isolated or pure blow-out fractures.

The goal of primary reconstruction of blow-out fractures is to restore the configuration of the orbital walls, return prolapsed orbital contents to the orbit proper, and eliminate any impingement or entrapment of orbital soft tissues. In contrast to the orbital floor blow-out fractures, isolated blow-out fractures to the roof or medial walls usually do not contribute significantly to the development of cosmetic deformities or result in entrapment or limited ocular motility. As a result, medial and roof defects are managed by observation, serial examinations, and intervention when symptoms warrant.

The most difficult area of the orbital floor blow-out fracture to repair is the posterior medial extent, which is beyond

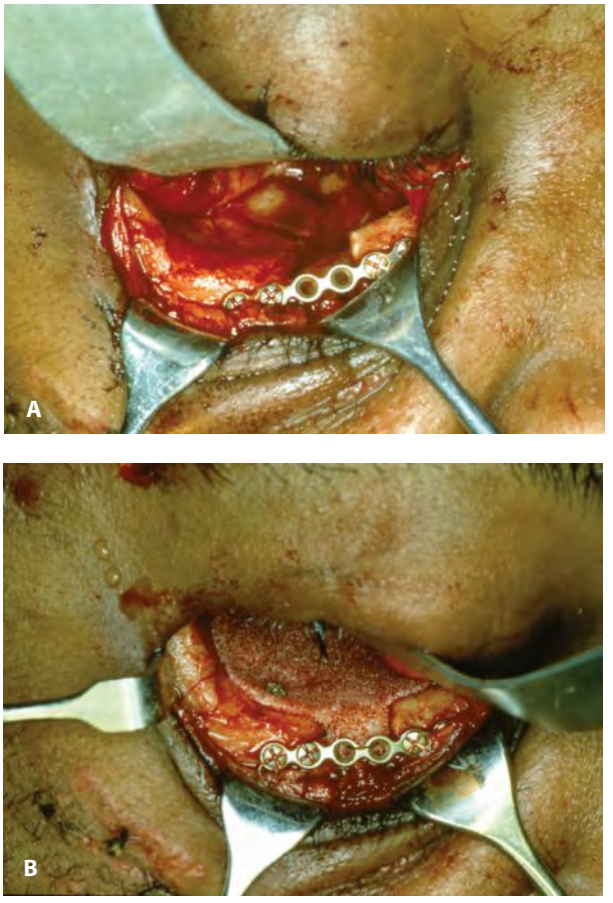


FIGURE 22-19. **A,** Right inferior orbital rim and floor fracture reduced and fixated with a 1.7-mm microplate. A portion of the mid-rim was suctioned away from the antrum and was missing. **B,** The floor defect was reconstructed with 0.85-mm-thick porous polyethylene sheeting secured with a single 4-mm-long 1.7-mm screw at the anterior lateral intact floor. A tab extension of the sheeting was fashioned at the rim defect, curved, and secured with a 5-0 nylon mattress suture.

the globe axis. Often, an intact bony ledge cannot be identified or the graft material is not extended posteriorly enough to support the orbital contents in this region. This area is often responsible for a failed enophthalmos repair in orbital blow-out fractures. The reconstruction of this posterior medial floor to its normal contour is the key to restoring normal globe position both anteroposteriorly and vertically.

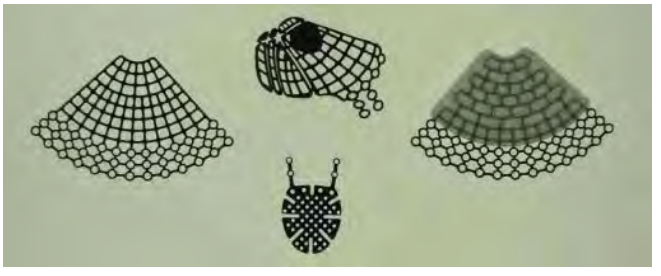


FIGURE 22-20. Different types and thicknesses of titanium orbital floor implants.

This scenario is problematic in delayed reconstructions because attempts to create a normal anteroposterior position of the globe may result in inappropriate overpositioning of the globe in a superior direction. The authors prefer to use gelatin film as a temporary barrier for small or linear defects simply to prevent entrapment during normal active ocular motion. This film is resorbed rather rapidly and does not provide much structural support; therefore, it is not used for larger defects in which herniation of contents into the underlying sinus is a possibility. Generally, the orbital blow-out fracture is explored in all of the intact bony walls identified. Once the malleable ribbon or globe retractors have supported the globe and orbital contents superiorly, the reconstructive material can be slid underneath them and overlap the intact bony margins slightly at the majority of areas to provide adequate support. The newly developed orbital retractor (Fernandes orbital retractor; Biomet, Jacksonville FL.) offers the advantage of adjustable working ends and a graduated surface that allow the operator to gauge the depth of retraction. (Figure 22-21). Using a transparent Jaeger retractor – the lucite lid plate has the particular advantage of constantly being able to inspect the pupil intra-operatively while retracting the contents of the orbit while working on the orbital floor. Additionally, it is non-conductive and inadvertent



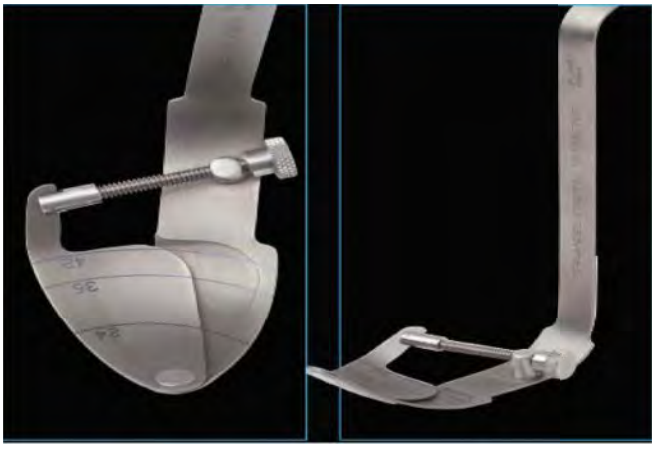


FIGURE 22-21. Fernandes orbital retractor; Biomet, Jacksonville FL.

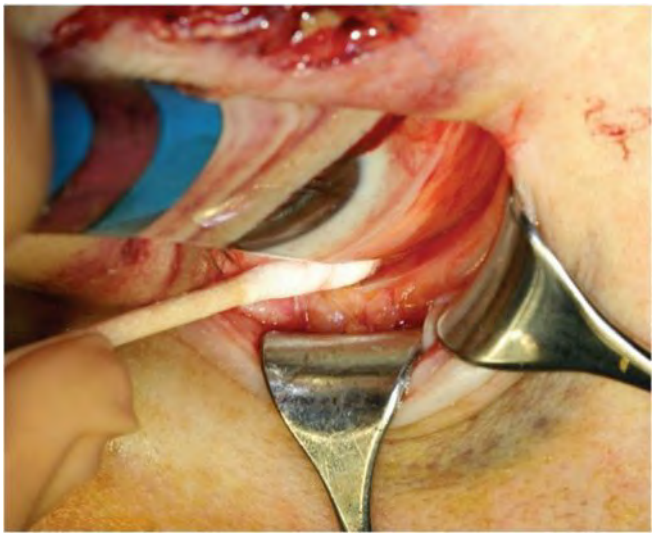


FIGURE 22-22. Using the Jaeger lid retractor made of Lucite.

touching of monopolar or bipolar cautery against the retractor surface will not cause harm to the cornea or globe. (Figure 22-22).

The authors prefer to use porous polyethylene for moderate to large blow-out fractures. The porous polyethylene sheeting can be secured with a single positional screw (usually 1.7-mm external thread diameter), or an extended tab of this material can be sutured to the orbital rim orbital plate (see Figure 22-19). Care should be taken to not extend the grafts up to the orbital rim or over the edge because these will be palpable and would improperly reconstruct the normal anatomic contour to the floor, which should dip down behind the rim for several millimeters before proceeding posteriorly. Also, the extension of semirigid grafts onto the orbital rim has an undesirable ramping effect, which tends to position the globe in an abnormal posterior direction, resulting in enophthalmos. After the floor graft is placed and secured, trimming or smoothing should be accomplished and a forced duction test performed before any wound closure to ensure that no impingement of the soft tissues has occurred.

Complex orbital fractures are generally associated with additional surrounding midfacial and frontal sinus fractures. Primary reconstruction of these defects is challenging owing to the extent of these injuries, the lack of any normal identifiable anatomy, and poor surrounding bony support for rigid fixation and anchoring of reconstructive materials. However, in this group of individuals, primary repair with normal anatomic realignment is critical for acceptable aesthetic and functional outcomes. Delaying the primary repair beyond 7 to 10 days usually results in some secondary soft tissue changes, the inability to completely retrieve small bony segments, and a less-than-desirable outcome. The initial step in the reconstruction of complex facial fractures is adequate exposure of all midfacial structures with adequate alignment and reduction before rigid fixation of any components with plates and screws (Figures 22-23 and 24). This helps one avoid misalignment, overreduction, or improper angulation of these segments. Achieving adequate exposure requires more extensive subperiosteal dissection than is done for most other orbital fractures. It may be desirable to also completely dissect and expose all internal orbital fractures before fixation of the surrounding periorbital or midfacial fractures. Generally, the orbital rim is plated with 1.7 mm or finer plating systems. Care should be taken at the inferior orbital rim and especially the lateral orbital rim to keep the plates several millimeters from the edge of the rim; otherwise, they will be annoyingly palpable once the soft tissue edema has subsided. Once the orbital rims and midfacial bones have been fixated, the moderate to large orbital floor defects are generally repaired with porous polyethylene and anchored to the



FIGURE 22-23. **A,** An elderly woman sustained a severely displaced left zygomatic complex (ZMC) fracture with > 75% orbital floor disruption. She was on warfarin sodium and had moderately decreased left visual acuity with increased ocular pressures. **B,** Axial CT scan revealed a ZMC fracture with a severe posterior, medial, and moderate inferior displacement. **C,** The patient was taken urgently (within 12 hr) for surgical treatment to reduce the fracture and re-expand the orbital volume. Serial examination and ocular pressure checks were performed every 2 hours pre- and postoperatively. Owing to cardiac risk factors, the anticoagulation was not reversed, nor was the patient treated with fresh frozen plasma. The zygomaticofrontal (ZF) suture area was first approached through a lateral brow incision. After the intraoral vestibular and then transconjunctival approaches were accomplished, the ZF fracture was plated. *(Continued)*

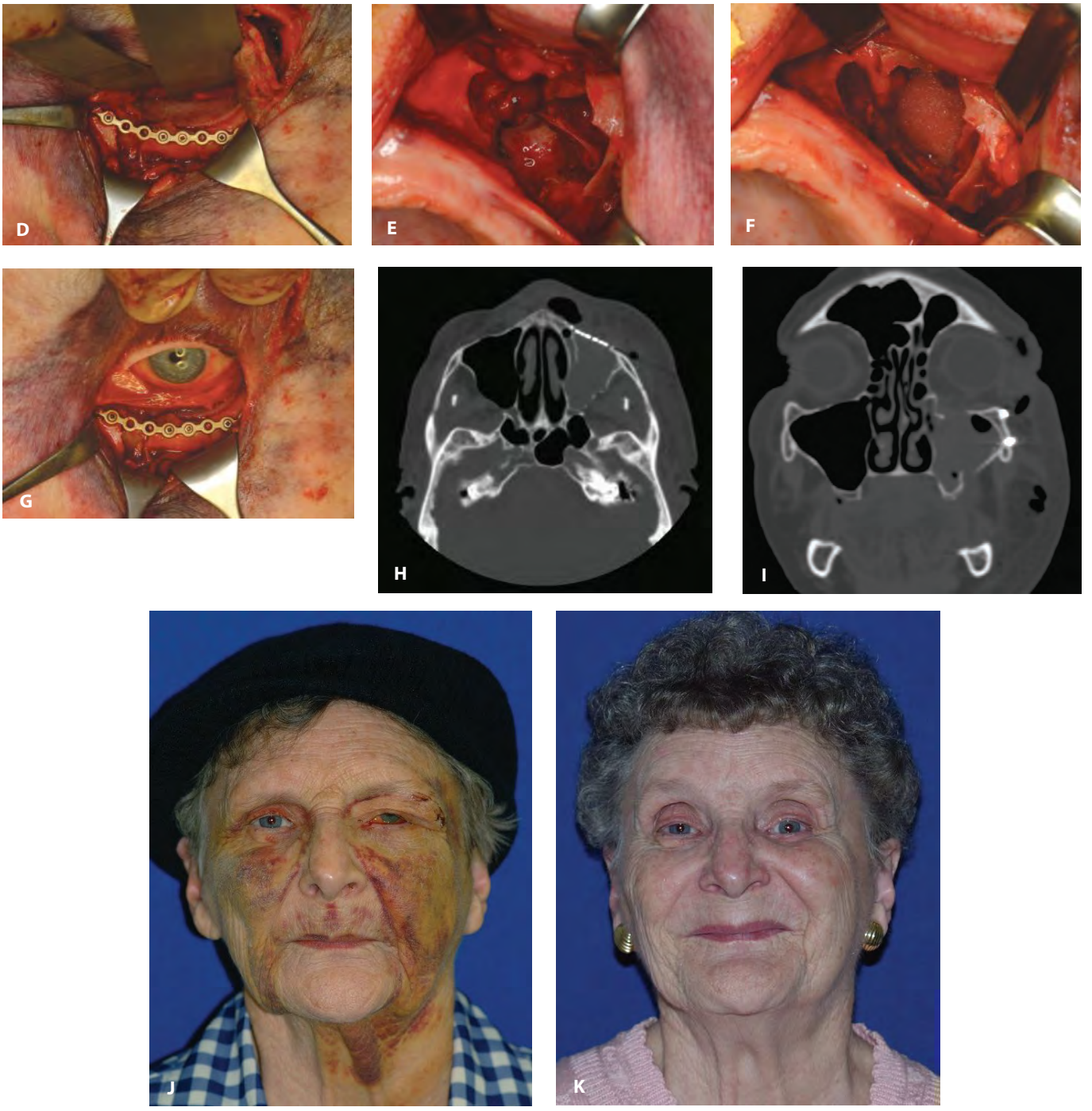


FIGURE 22-23. *(Continued)* **D,** The infraorbital rim was fixated with a 1.2-mm titanium plate, and the floor was reconstructed with 0.85-mm porous polyethylene sheeting. **E,** The left maxillary sinus anterior wall defect visualized through the vestibular incision along the edentulous ridge. Note the herniated orbital soft tissues. **F,** After retrieval of the orbital soft tissues from above and insertion of the porous polyethylene floor graft, the repair was inspected from below ensuring that there was no tissue prolapse or entrapment. The fracture was then spanned from the buttress to the intact medial maxilla with a 1.7-mm plate. The anterior maxillary wall defect was not grafted. **G,** The eye position was assessed with the contralateral side, and a forced duction test revealed a free and full range of motion. **H,** The patient had a routine 24-hour follow-up CT scan of the head, as per the request of the neurosurgeon. The images of the patient's face demonstrated excellent realignment. Postoperatively, she had greatly improved vision and no neurologic impairment. She was discharged home on postoperative day 2 on warfarin sodium. **I,** The reformatted coronal images show good orbital floor support of the globe. **J,** Facial appearance at 1 week postoperatively. **K,** Six weeks postoperatively, this patient had no complaints and her baseline visual acuity had returned.

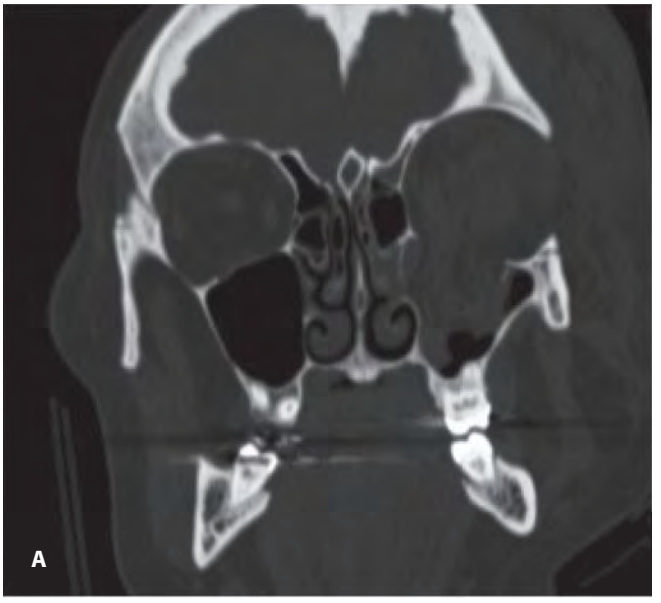
anterior inferior floor with a single screw. Sometimes, layering of this material with an additional sheet posteriorly is required to achieve correct anteroposterior globe positioning. More extensive defects may require titanium mesh or orbital floor plates with screw fixation to the rims and autogenous bone grafts. Several bone grafts can be secured to the metallic mesh framework to independently reconstruct the floor, medial wall, and less frequently, the lateral orbital walls. The advantage of having bone overlie the metallic mesh is that remodeling can occur—secondary revision surgery is enhanced when dissecting along a healed bony surface versus bare mesh. In severe or large defects with comminution, overcorrection of the enophthalmos component (but not a hyperophthalmic deformity) by several millimeters is often necessary to take into account the orbital edema that exists. In addition, with bone grafts, some mild resorption can take place with subtle settling. However, the resolution of the edema accounts for the majority of postoperative globe position changes.

ZMC fractures are second only to nasal fractures in incidence. These fractures are described in greater detail in Chapter 21, "Management of Zygomatic Complex Fractures" and Chapter 23, "Management of Frontal Sinus and Naso-orbitoethmoid Complex Fractures." Some discussion is warranted here, because ZMC fractures relate to orbital involvement and appropriate intraoperative sequencing. Nonfragmented or single-piece ZMC fractures are generally displaced in an inferior, medial, and posterior direction, with a pivot-point rotation about the ZF suture. As a result, the orbital floor suffers the most disruption. On initial inspection, the coronal CT scans may not reveal the degree of orbital floor disruption, but if one envisions the outward reduction of the zygomatic buttress and the resulting medial floor void, the magnitude of the injury can be appreciated. Only after reduction and stabilization of the entire external orbital framework and surrounding facial bones should the internal orbital defects be repaired (see Figure 22-23C-G). The internal orbital injuries associated with fragmented ZMC fractures usually involve multiple orbital walls and larger defects. Therefore, more extensive exposure is generally necessary and more rigid materials are usually required for reconstruction.

NOE injuries result mainly from extreme blunt force trauma and have a high degree of associated intracranial and neurologic injuries. In addition, injuries to the nasal airway and lacrimal system can occur.[82] Injuries to the lacrimal system can be managed by the placement of small silicone tubes. Even though canalicular disruption is more common with laceration-type injuries, these tubes can still be inserted with blunt trauma when a fair amount of edema is present and the surrounding anatomy is obscured. This prophylactic intubation of the superior and inferior canaliculi and the lacrimal system helps to avoid iatrogenic injury during the extensive dissection required to treat this type of injury. The tubes can be allowed to remain in place several weeks postoperatively during the resolution of edema. Repair of NOE injuries is recommended within the first 7 to 10 days after injury, before the soft tissues have had the chance to readapt with significant scarring contracture and generally a flattened and splayed appearance to the orbits and midface. NOE injuries generally do not cause entrapment simply because of the orbital walls involved and the degree of comminution. However, entrapment of the medial rectus can occur during reconstruction, fixation, and suturing. Therefore, a forced duction test should be performed at the completion of these phases. The primary defects associated with NOE injuries are medial canthal disruption with telecanthus and increased bony volume resulting in enophthalmos. If there are no other indications for coronal dissection, such as frontal bone or zygomatic arch fractures, the medial orbital component of the NOE fracture is best approached directly through a lateral nasal (Lynch) incision. Often, accessing the inferior medial wall or positioning the inferior edge of the medial wall graft requires an additional inferior rim and orbital floor approach, such as the subciliary or transconjunctival approach. Traumatic telecanthus should be treated by direct fixation techniques, using 1.0- to 1.7-mm plating systems. External splinting may provide some reasonable nasal bone molding, but it generally does little to improve traumatic telecanthus. Generally, the medial canthal ligament heals in a position that is too superficial and inferior. Postoperatively, the entire area fills with dense scar tissue, and it is difficult to secondarily dissect and reposition the canthus in its normal position. With NOE fractures, the medial canthal tendons usually maintain their attachment to the bony segments. Therefore, proper reduction and fixation of the bony skeleton to the surrounding stable bone (maxilla, orbital, and frontal) often corrects the telecanthus deformity. This should be accomplished and the medial canthal position reassessed. If the canthal position is still unacceptable, a fine stainless steel wire (30-gauge) can be secured directly to the canthal tendon or, preferably, sutured to the wire that has been passed transnasally.

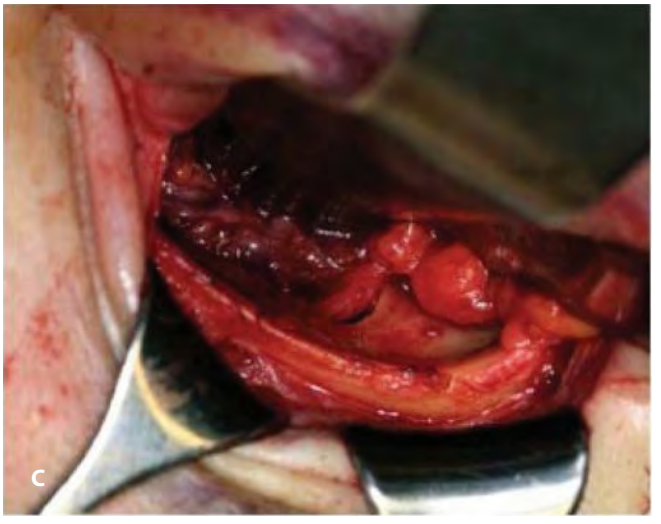
The double-armed wire is inserted from the contralateral orbit to the side that will be anchored, with the entry point on the medial wall being just posterior and superior to lacrimal fossa. This can be accomplished by prethreading the double-armed wire into a gently curved 16-gauge needle, passing it transnasally through small bur holes, retrieving the double-armed wire on the side to be fixated, and withdrawing the needle canula. The canthus is then sutured to the wire loop with a half-round needle (4.0 Mersilene S-2 needle), and the wire is drawn to the contralateral side and the limbs twisted gradually around a short section of plate to fine-tune the canthal position (see Figure 22-14). This is a much easier way to accomplish precise canthal positioning than are direct suturing techniques.

Secondary reconstruction of the peri-orbita can be a challenge in itself. The use of stereo-lithographic modelling, computer assisted treatment planning, the use of 3-D imagery and technology and intra-operative navigational surgery can be utilized to improve outcomes.[86]











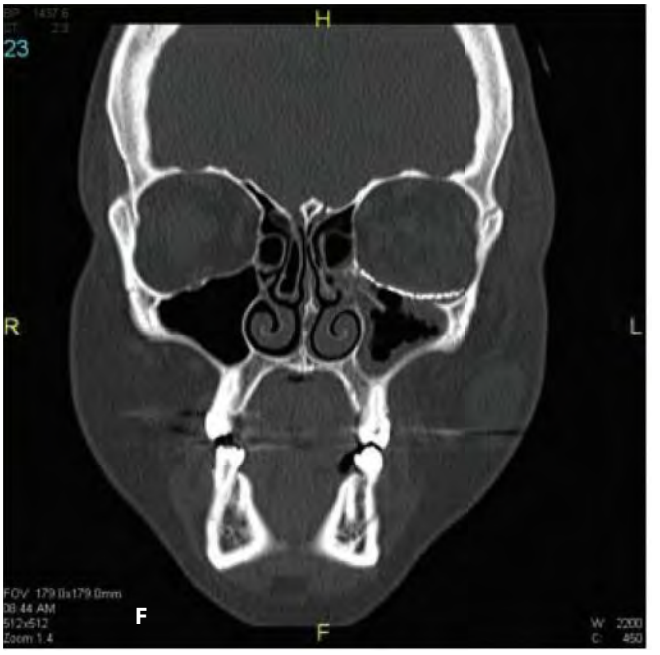


FIGURE 22-24. **A,** Coronal CT showing large orbital floor defect **B,** pre-operative bird's eye view of severe enophthalmos resulting from the large orbital floor defect **C,** Herniated contents released and fracture reduced **D,** Orbital floor reconstructed using titanium orbital floor mesh fixated at inferior orbital rim **E,** post-operative bird's eye view of repaired orbital floor defect correcting the severe enophthalmos. Note, the left eye (repaired side) is slightly forward owing to the orbital edema and the eye typically is looking downward slightly with the patient under general anesthesia. This is due to swelling and stiffness of the contused inferior rectus. This typically resolves gradually in several months time. **F,** Coronal CT of repaired orbital floor.

SUMMARY

Orbital fractures are often associated with ocular injuries and midfacial fractures. A thorough ophthalmologic evaluation is mandatory to detect ocular injuries and to preserve vision. Surgical intervention should be based on either a functional deficit or a cosmetic deformity. The surgical sequencing and timing of the repair should be well thought out. When visual compromise exists, an ophthalmologist should be involved in the treatment planning.

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CHAPTER 23

Management of Frontal Sinus and Naso-orbitoethmoid Complex Fractures

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The frontal bone and naso-orbitoethmoid (NOE) complex have central aesthetic and functional roles. Thankfully, fractures in this region are infrequent, occurring among only 2% to 15% of patients with facial fractures,[1–4] because when these fractures do occur, they can cause devastating complications because of their proximity to the brain, eyes, and nose. Complications include blindness or other forms of visual disturbance, orbital cellulitis or abscess, meningitis, brain abscess, and facial deformation. Although reports of the surgical management of the diseased frontal sinus have existed for more than 100 years,[5] no consensus has yet been reached on ideal patient care after traumatic injury.[5–7]

Most frontal sinus and NOE injuries (44–85%) are sustained in motor vehicle or motorcycle collisions,[1,3,7–21] with young males most commonly affected (66–91%).[1,7–17,22–24] NOE fractures can occur in isolation, but they generally occur in association with other midface fractures.[20,25] As many as 60% of patients with NOE fractures also have associated nonfacial injuries.[21]

For NOE fractures, the distribution between fractures of the supraorbital rim and fractures of the frontal sinus is almost equal. The published frequency of fractures of the anterior wall, the posterior wall, and the floor of the frontal sinus varies rather widely: 43% to 61% of reported patients had anterior table fractures only; 19% to 51% had anterior and posterior table fractures; 2.5% to 25% had injuries to the nasofrontal duct; and 0.6% to 6% had posterior fractures only.[2,3,8]

ANATOMY AND PHYSIOLOGY

Embryology of the Sinus

The frontal bone is an intramembranous bone that develops from two paired structures that begin to ossify at the 8th or 9th week in utero.[9,26] The ossification begins in the frontal processes of the squamous regions, progresses to the orbital and squamous regions, and reaches the frontal and temporal regions by the 12th week. The metopic suture in the midline closes during the second year of life.[27] The forehead is displaced anteriorly by sutural growth, inner table resorption, and outer table deposition.[28]

The frontal sinus is a small outpouching at birth and undergoes almost all of its development thereafter. The sinus may develop from one or several different sites: as a rudiment of the ethmoid air cells, as a mucosal pocket in or near the frontal recess, as an evagination of the frontal recess, or from the superior middle meatus.[17] Initial pneumatization begins during the fourth month in utero. Secondary pneumatization begins between the ages of 6 months to 2 years and develops laterally and vertically. The sinus is radiographically identifiable by the time the child reaches the age of 6 years.[29] Most pneumatization is completed by the time the child is 12 to 16 years old, but continues until the age of 40.[5,17,26,30] The configuration of the sinus and the position of the septa are extremely variable between individuals.

Physiology of the Sinus

The entire surface area of the frontal sinus is covered with respiratory epithelium ranging in thickness from 0.07 to 2.0 mm.[31] The mucosa consists of pseudostratified ciliated epithelium, mucus-producing goblet cells, a thin basement membrane, and a thin lamina propria that contains seromucous glands.[31] When the mucosa is healthy, a blanket of mucin overlies the epithelium. The cilia flow at 250 cycles/min and push the mucin blanket in a spiral fashion and in a medial to lateral direction. The flow is slowest at the roof and fastest at the nasofrontal duct[32] where the mucin empties at a rate of 5.0 g/cm^2. The physiologic characteristics of the sinus and the status of the nasofrontal duct dictate the treatment of the frontal sinus in trauma.[33] Although described as the "nasofrontal duct," a true duct is seen in only 15% of people, with most having only a recess draining into the middle meatus. Some authors prefer to use the term *nasofrontal outflow tract (NFOT)* to describe the drainage system. Recognition of the variability in duct size and shape is important in management of impaired drainage after injury.[34]

Osteology

The frontal bone is shaped as a concave disk with a horizontal table forming the orbital rim. From the nasion, the bone extends approximately 12.5 cm superiorly, 8.0 cm laterally, and 5.5 cm posteriorly. Two frontal tuberosities are noted lateral to the midline and superior to the supraorbital run. The thickest area of the bone is the supraorbital rim, running from the frontozygomatic process to the nasal bones. The ethmoid plate is bound on three surfaces along the floor of the frontal bone in the midline. As the floor of the frontal bone extends laterally, it becomes concave and forms the orbital roof. The supraorbital and frontal foramina are located at the most superior portion of the orbital rim. The supratrochlear foramen is located medial to the supraorbital foramen or notch and lateral to the nasal bones. A spine or concavity exists on the frontal bone along the medial anterior orbital roof and forms the attachement for the trochlea of the superior oblique muscle[35,36] (Figure 23-1).

Paired triangular sinuses are found within the frontal bone. These sinuses are asymmetrical and are separated by a frontal septum. The average height of the sinuses is 32 mm, and their average width is 26 mm.[29,36] The surface area within is approximately 720 mm.[2,32] The frontal bone is thinnest in the region of the glabella at the anterior wall and floor of the sinus. The duct of the frontal sinus empties into the ethmoid air cells of the middle meatus of the nose.

The internal concave surface of the frontal bone forms the anterior cranial fossa that houses the brain. The floor of the

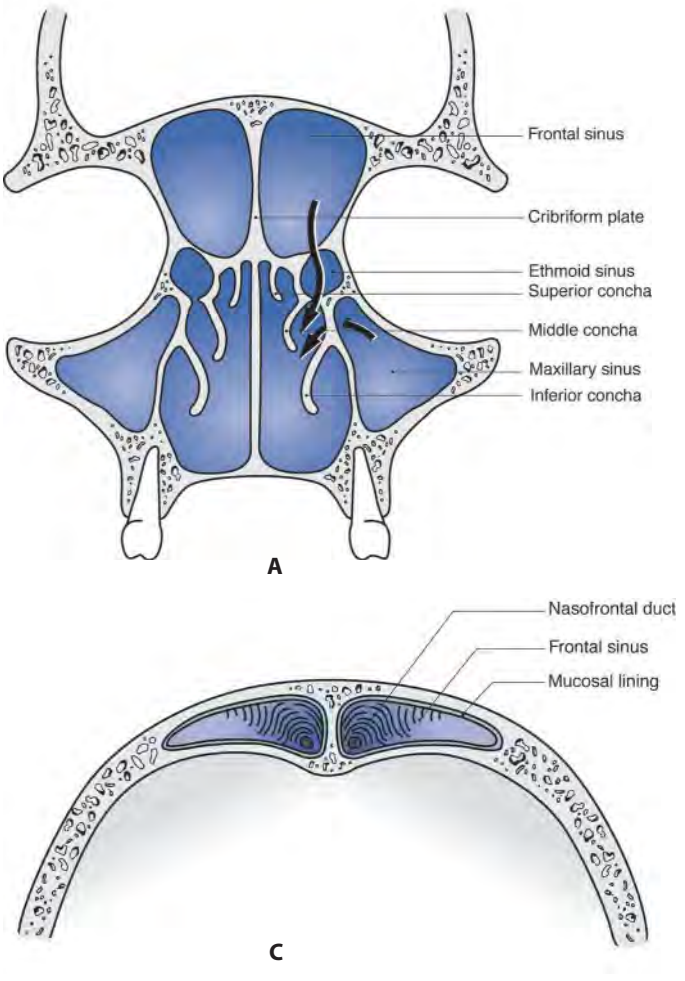


FIGURE 23-1. **A,** Frontal bone and frontal sinus showing the relation of the nasofrontal outflow tract and nose. The *arrows* represent the flow from the sinuses to the nose. **B,** Lateral view of the normal nasofrontal outflow. The *arrows* represent the flow from the sinuses to the nose. **C,** Superior view of the normal nasofrontal outflow. (**A–C,** Adapted from Zide MF. Nasal and nasoorbital ethmoid fractures. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; pp. 576–577.)

frontal bone outlines the roof of the orbit. The convex outer table is bounded by the scalp and the frontalis, orbicularis, and procerus muscles. The osseous structures that abut the frontal bone are the lacrimal and ethmoid bones inferiorly, the sphenoid inferiorly and posteriorly, the parietal posteriorly and superiorly, the zygoma laterally, the nasal bones anteriorly, and the maxilla anteriorly and inferiorly. The ethmoid air cells and nasal apparatus are situated inferiorly.

The nasal part of the frontal bone extends inferiorly deep to the nasal bones and the frontal process of the maxilla, adding support to the NOE complex. The nasal bones and the maxilla make up the piriform rim. The articulation of the nasal bones forms a crest posteriorly and inferiorly. This crest articulates with the frontal bone, the perpendicular plate of the ethmoid (forming the upper third of the nasal septum), and the septal cartilage. The NOE region is supported structurally by a vertical buttress (the frontal process of the maxilla) and two horizontal buttresses (the supraorbital and infraorbital rims).[37]

The medial walls of the orbit begin behind the frontal process of the maxilla. The thin lacrimal bone and a frail lamina papyracea in the anterior are weak and susceptible to fracture. Higher up, the frontoethmoid suture delineates the level of the cribriform plate and crista galli.

Neurovascular Structures

The arterial blood supply to the frontal sinus is from the supraorbital and anterior ethmoid arteries. Two foramina are present along the suture line: the anterior ethmoid foramen, through which course the nasociliary nerve and the anterior ethmoidal artery, and the posterior ethmoidal foramen, through which pass the vessel and nerve of the same name. Further posterior along the medial orbital wall, the optic nerve exits through the body of the sphenoid bone, 3.5 to 5 mm behind the posterior ethmoidal foramen in a line parallel to the two foramina.

The frontal bone is supplied by the supraorbital, anterior superficial temporal, anterior cerebral, and middle meningeal arteries.[27,35,36] Venous drainage is transosseous through the anastomosis of vessels of the subcutaneous, orbital, and intracranial structures. The primary venous drainage is through the supratrochlear, supraorbital, superficial temporal, frontal diploic (veins of Breschet), superior ophthalmic, and superior sagittal sinuses.[27,35] The relationship of the diploë to the anterior cranial fossae is important to understand because these structures are sites of imbrication of sinus mucosa, which may give rise to mucoceles and can become a conduit for the spread of infection if not removed.

Interorbital Space

The nasofrontal suture is the continuation of the frontoethmoid suture and corresponds to the plane of the base of the skull or frontal sinus. The interorbital space is bounded laterally by the medial wall of the orbits. In the middle is the perpendicular plate of the ethmoid and nasal septum. The anterior wall is composed of the paired nasal bones, the frontal processes of the maxilla, and the nasal processes of the frontal bone.

The ethmoid air cells within the interorbital space occupy the upper half of the wall lateral to the nasal fossa. The dimensions of the anterior end of the ethmoid labyrinth are approximately 2.5 cm vertically and 1 cm transversely. The pyramid-shaped sinus measures between 3.5 and 5 cm from front to back.

The ethmoid air cells drain into the middle meatus, as does the NFOT. The nasofrontal outflow is located in the posterior medial floor of the frontal sinus at the junction of the ethmoid and nasal portions of the floor, and it courses through the anterior ethmoid in the middle meatus, or just anterior to the middle turbinate. The length of the duct, if present, may vary from a few millimeters to a centimeter or more (Figure 23-2).

Medial Canthal Tendon

The orbicularis oculi muscle has three portions: orbital, preseptal, and pretarsal. The pretarsal portions of the upper and lower lids unite at the canthus to form the medial canthal tendon (MCT).

The MCT may be subdivided into a superficial portion and a deeper portion with the lacrimal sac between them. The superficial portion has two "legs" and inserts into the frontal process of the maxilla, providing support to the eyelids and maintaining the integrity of the palpebral fissure.[37,38] The anterior leg attaches to the posterolateral surface of the nasal bones, and the superior leg inserts at the junction of the frontal process of the maxilla and the angular process of the frontal bone. The deeper portion (also known as *Horner's muscle* or the *pars lacrimalis*) attaches to the posterior lacrimal crest.

NOE injuries may cause avulsion of the tendons from the bone or, more commonly, fractures of the bone that contains the attachment of tendons. This portion of the orbital rim is an important anatomic region with regard to reconstruction of NOE fractures.[37]

Lacrimal Apparatus

The lacrimal drainage system is intimately related to the NOE region and can be damaged during trauma to, or reconstruction of, this area. The system removes any excess tears that accumulate after lubrication of the surface of the globe. The superior and inferior lacrimal canaliculi drain the lacrimal lake. The puncta of the canaliculi open just lateral to the lacrimal lake and are surrounded by Horner's muscle. The orifice of the upper punctum faces downward and backward, and the orifice of the lower punctum faces upward and backward. The superior punctum is approximately 3 mm medial to the inferior punctum. The two canaliculi pierce the lacrimal fascia and enter the lacrimal sac at or very near a common point. The canaliculi lie mostly behind the medial palpebral ligament and are surrounded by the pars

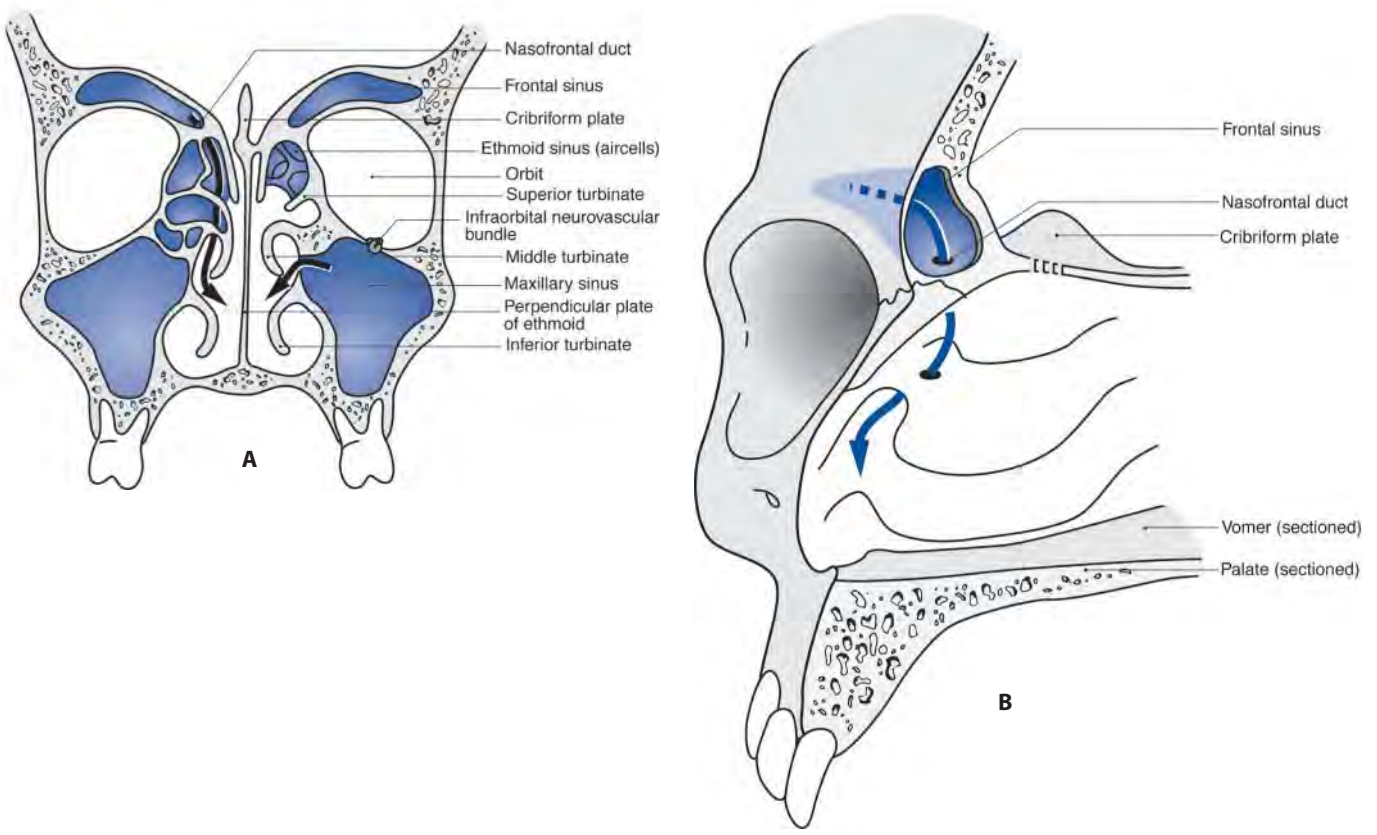


FIGURE 23-2. **A,** Section through the intraorbital space reveals the relationship of the frontal sinus and the ethmoid sinuses to the nose. The *arrows* represent the flow from the sinuses to the nose. **B,** The drainage of the nasofrontal outflow tract into the nose is located in the posterior medial floor of the frontal sinus and at the junction of the ethmoid and nasal portions of the floor. The *arrows* represent the flow from the sinuses to the nose. (**A** and **B,** Adapted from Zide MF. Nasal and naso-orbital ethmoid fractures. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; p. 560.)

lacrimalis.[39,40] The lacrimal canaliculi are lined with nonkeratinized and non–mucin-producing stratified squamous epithelium. The epithelium is 75 to 150 µm thick and consists of a few layers of squamous cells, polyhedral cells, and a basal cell layer.[40]

The lacrimal sac lies in a fossa on the anteromedial wall of the bony orbit. It is lined with pseudostratified columnar epithelium and is approximately 12 mm long.[40] The apex of the sac ends blindly in a superior fundus, and the sac continues inferiorly into the nasolacrimal duct, which is housed in a bony canal. The duct empties into the inferior meatus in the nasal cavity.[39]

PATIENT EVALUATION

Clinical Findings

Periorbital ecchymosis and pain are the most common signs and symptoms associated with fractures of the frontal bone.[5,41–45] Disruption of the periosteum and bleeding from the fracture site allow leakage of blood into the adjacent facial planes, resulting in periorbital ecchymosis. Subconjunctival hemorrhage may occur via the same mechanism. The finding of subconjunctival hemorrhage is sufficient for the diagnosis of frontal bone fracture if the nose and zygomas are unaffected. Fractures of the NOE complex can produce the following signs: nasal deformity, edema and ecchymosis of the eyelids, subconjunctival hemorrhage, cerebrospinal fluid (CSF) leakage, hyposmia, traumatic telecanthus, increased canthal angles, and blindness[20,46] (Figure 23-3).

Soft tissue lacerations in the region of the glabella and the supraorbital rims are also commonly found in association with frontal bone fractures and may be associated with anesthesia or paresthesia in the distribution of the supraorbital and supratrochlear nerves.[5,41–45] Depression of the bone with flatness and cosmetic deformity is noted if the patient is examined soon after injury. Examination of a patient with NOE fractures detects mobility of the nasal bones, traumatic telecanthus, depression of the radix, a wide and flattened nasal dorsum, and an upturned nasal tip (Figure 23-4). From 1 hour to 5 days after injury, there may be enough edema to hide the contour depression. Palpation may reveal crepitation and tenderness over the fracture site.[41–45]

Fractures involving the posterior table of the frontal sinus or the cribriform plate may cause CSF leakage.[41–44,47] Confirmation of the presence of CSF can be made by collecting as

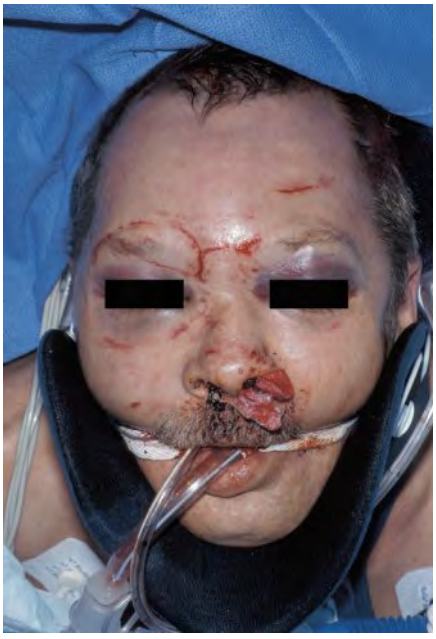


FIGURE 23-3. Initial appearance of a patient with a frontal sinus fracture. Note the bilateral periorbital ecchymosis and forehead laceration.

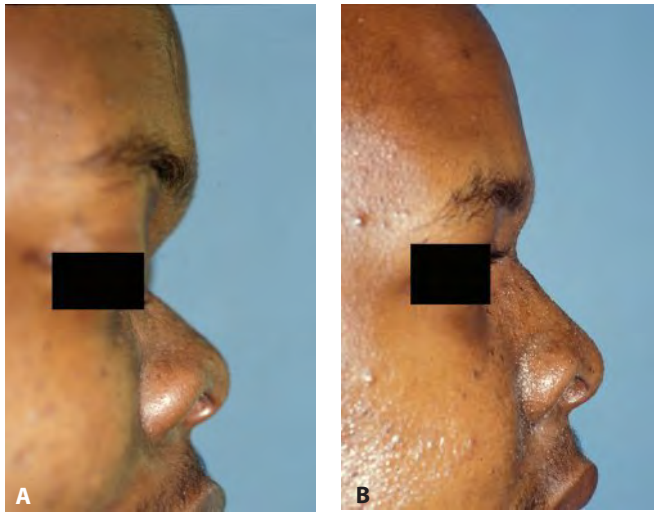


FIGURE 23-4. Preoperative **(A)** and postoperative **(B)** images of a patient with a naso-orbitoethmoid (NOE) fracture show a severely depressed radix.

little as 0.1 mL of this fluid and comparing glucose and chloride levels in the fluid with the patient's serum levels. High chloride and low glucose concentrations in fluid compared with serum indicate the presence of CSF. In addition, the presence of beta 2 transferrin in fluid confirms the presence of CSF[48] (Table 23-1).

The depression of bone fragments into the orbit may cause exophthalmos, proptosis, or ptosis. A depressed injury also causes restricted ocular movement if the superior rectus muscle, the superior oblique muscle, or the trochlea is damaged.[44,45] Medial orbital wall fractures associated with NOE fractures can also cause enophthalmos.

A thorough examination is important to distinguish between a nasal fracture and an unstable NOE fracture. The examiner should place the thumb and index finger over the medial canthus bilaterally. Mobility of these fragments may vary, but any movement implies instability and requires open reduction and stabilization.[37] A ruler or caliper should be used to measure the intercanthal distance. The normal distance is between 28.6 and 33.0 mm for adult women and is between 28.9 and 34.5 mm for adult men. Increased widths suggest an NOE fracture.

Two tests that can aid in the diagnosis of instability of the medial canthus are the "bowstring" test and the bimanual examination. The bowstring test involves pulling the lid laterally while palpating the tendon area to detect movement of fracture segments.[49,50] The bimanual examination requires placing an instrument (e.g., Kelly clamp) high into the nose, with its tip directly beneath the MCT. Gentle lifting with the contralateral finger palpates the canthal tendons and allows an assessment of the instability of the tendon attachment and the necessity for open reduction.[51] The Furnas test may also be performed by grasping the skin overlying the medial canthus with a small tissue forceps[49] (Figure 23-5). A lack of creasing or resistance by the underlying bone is indicative of an underlying fracture.[52] These injuries are associated with

TABLE 23-1. **Normal Values of Constituents of Cerebrospinal Fluid, Serum, and Nasal Secretions**

Constituent	CSF	Serum	Nasal Secretions
Osmolarity (mOsm/L)	295	295	277
Sodium (mEq/L)	140	140	150
Potassium (mEq/L)	2.5–3.5	3.3–4.8	12–41
Chloride (mEq/L)	120–130	100–106	119–125
Glucose (mg/100 mL)	58–90	80–120	14–32
Albumin (%)	50–75	55	57
Total protein (mg/dL; % of total protein)	5–45	6.0–8.4	335–636
Immunoglobulin G (mg/mL)	3.5	1140	51
Beta 2 transferrin (% of total transferrin)	15	0	0

CSF = cerebrospinal fluid.
Adapted from Brandt MT, Jenkins WS, Fattahi TT, Haug RH. Cerebrospinal fluid: implications in oral and maxillofacial surgery. J Oral Maxillofac Surg 2002;60:1049–1056.

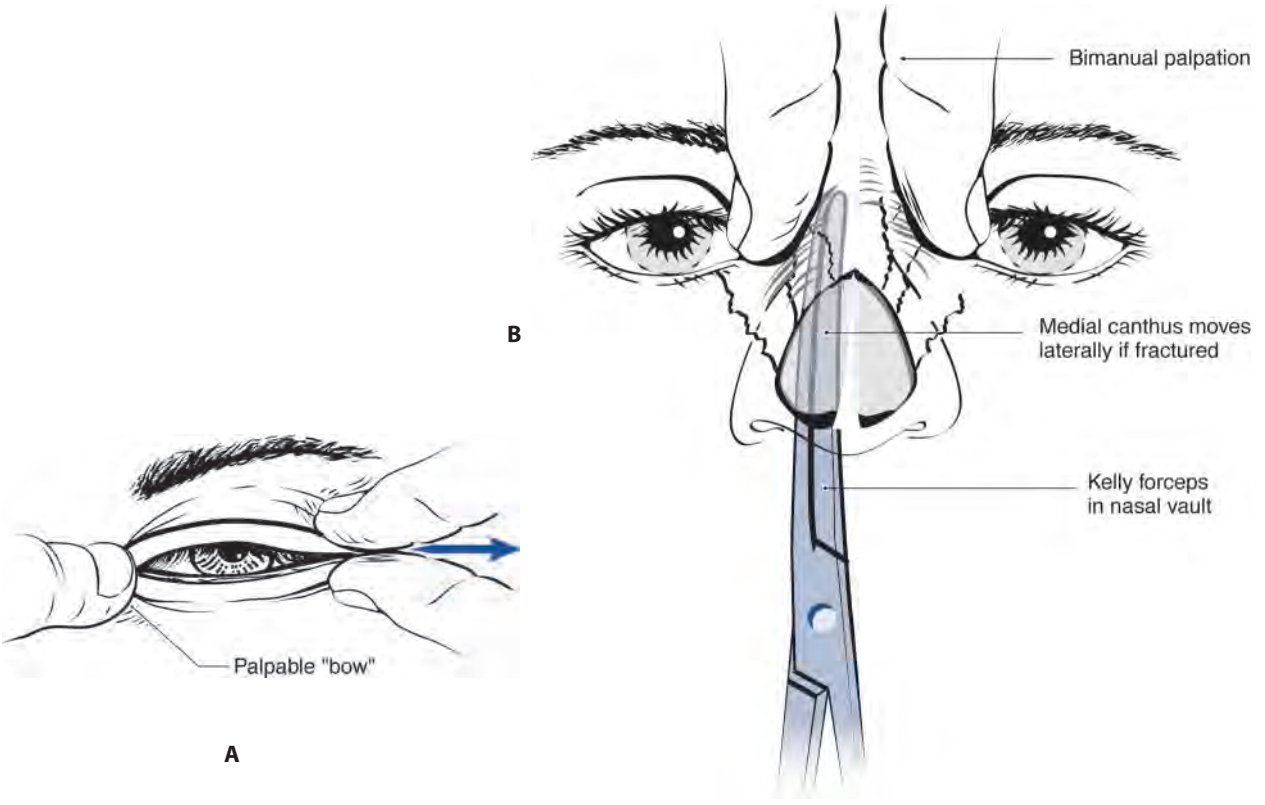


FIGURE 23-5. Illustrations of the bowstring **(A)** and bimanual examination **(B)** for possible NOE fractures. (**A** and **B,** Adapted from Zide MF. Nasal and naso-orbital ethmoid fractures. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; p. 562.)

significant energy transfer and associated neurologic injuries must be looked for and excluded, as must other injuries of the facial skeleton.

Imaging

Adequate imaging is key to assessing NOE and frontal sinus fractures and planning good treatment to prevent poor outcomes after the treatment. Previously,Waters' projections, reverse Towne's projections, lateral skull films, and laminar tomograms were used to visualize midface and upper face fractures. Today, computed tomography (CT) scans are the gold standard for imaging these fractures[5,9,37,53–57] (Figure 23-6).

The plane of choice for frontal sinus imaging is the axial view, preferably with slice thicknesses of 1.0 or 1.5 mm.[19,26,30] The high degree of detail required for imaging NOE fractures necessitates axial and coronal views with slice thicknesses of 1.0 or 1.5 mm.[25,37] Indeed, it has been shown that for severe fractures of the NOE region, two- and three-dimensional CT scans provide the most information about the medial orbital wall, the medial maxillary buttress, and the piriform aperture.[37,58]

Patency of the NFOT

Although the newest CT scanners provide exceptional views and can often provide slices through the NFOT, evidence of their reliability in detecting obstruction of the outflow is scant.[55,56] Obstruction should be suspected with fractures involving the medial supraorbital rim or the frontal bone with nasal ethmoidal component fractures, and it should always be considered when a CSF leak is present.[23] In these situations, an open or intraoperative evaluation of patency is indicated. This evaluation is important because the condition of the nasofrontal outflow has the most influence on the health of the frontal sinus[17,19,23,33,55,59–63] (Figure 23-7).

Classification of NOE Fractures

As with all fractures, NOE fractures are classified as unilateral or bilateral, open or closed, and simple or comminuted. Three types of NOE fractures have been well described.[25,37,39,64] A type I fracture maintains the attachment of the MCT to a large single nasoethmoidal fracture segment. Repairing this type of fracture is straightforward. A type II fracture shows more comminution yet maintains the attachment of the medial canthus to a sizable bony segment. Type III fractures display severe comminution with possible avulsion of the MCT from its bony attachment (Figure 23-8).

Classification of Frontal Sinus Fractures

Traditional fracture classifications can be used with reference to frontal bone fractures (e.g., open or closed). Numerous

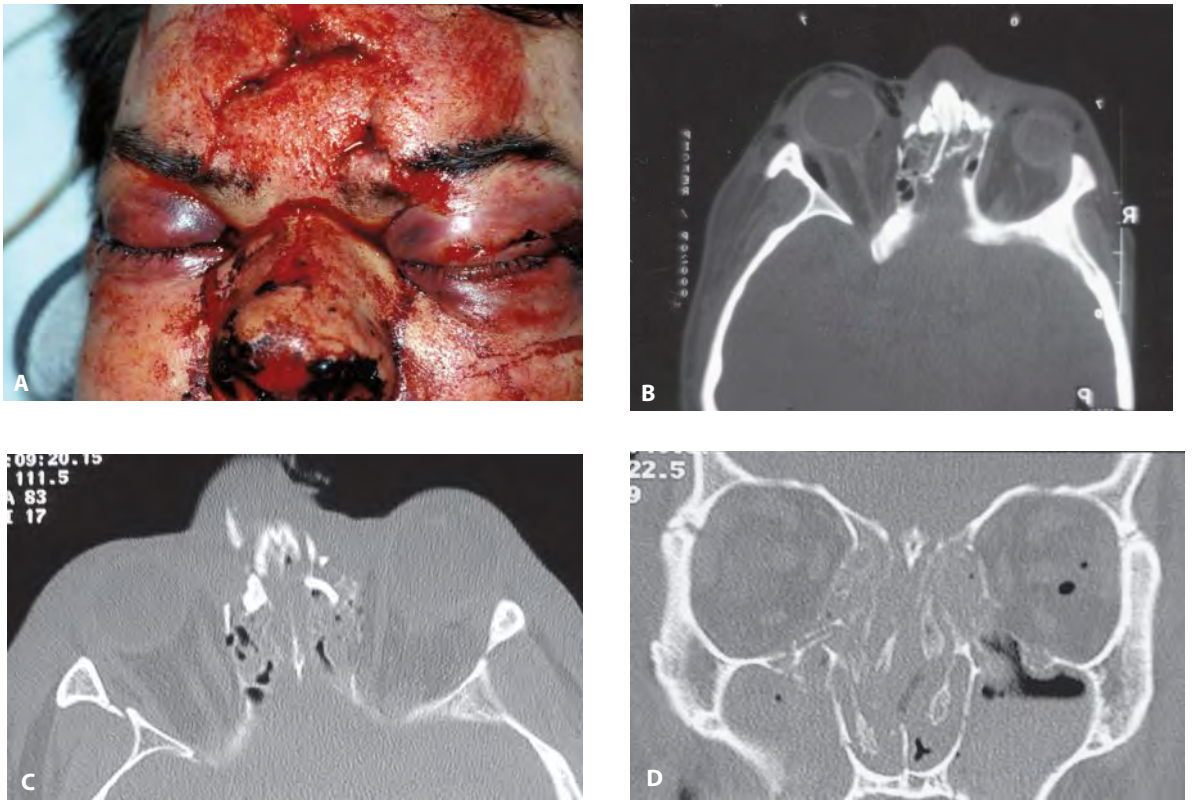


FIGURE 23-6. The detail of fracture anatomy is clearly superior in computed tomography (CT) scans when compared with traditional radiography. **A,** Initial appearance of a patient with an NOE fracture. **B,** Axial CT scan shows the fracture. **C** and **D,** Axial and coronal CT scans of another patient illustrate detailed fracture anatomy.

other classification schemes have been proposed in the surgical literature in an attempt to simplify surgical decision making. Although these schemes are well intended, some are so complex that they actually complicate decision making and are of no value. Consideration must always be given to the condition of the anterior table, the posterior table, and the nasofrontal outflow and the presence of co-morbid intracranial injury and concomitant craniomaxillofacial injuries.[5,8,11,17,65] The simplest and most helpful classification schemes distinguish possible complications and treatments on the basis of types of fractures.

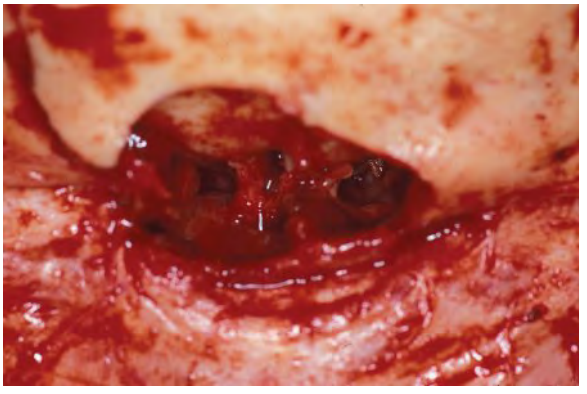


FIGURE 23-7. Intraoperative view of the floor of the frontal sinus with nasofrontal ducts.

Treatment of isolated anterior table fractures is indicated to prevent cosmetic deformities. Posterior table fractures, alone or in combination with anterior table fractures, should be treated so that neurologic sequelae, including meningitis and brain abscess, can be avoided. Combinations of fractures that compromise the NFOT should be treated so that the development of mucoceles and pyoceles can be prevented. These fracture combinations include fractures of the anterior table and the posterior table, fractures of the anterior table and the NOE, and fractures of the anterior table and the medial superior orbital rim.

TREATMENT

Treatment Goals

The primary goals of treating frontal sinus fractures are the creation of a safe sinus so that the risk of long-term complications is minimized and protection of the brain. Cosmesis is a major secondary goal and in NOE fractures involves the restoration of orbital volume, eyelid function, lacrimal drainage, and overall cosmesis. Identification of these goals is important in planning treatment, because it is important to

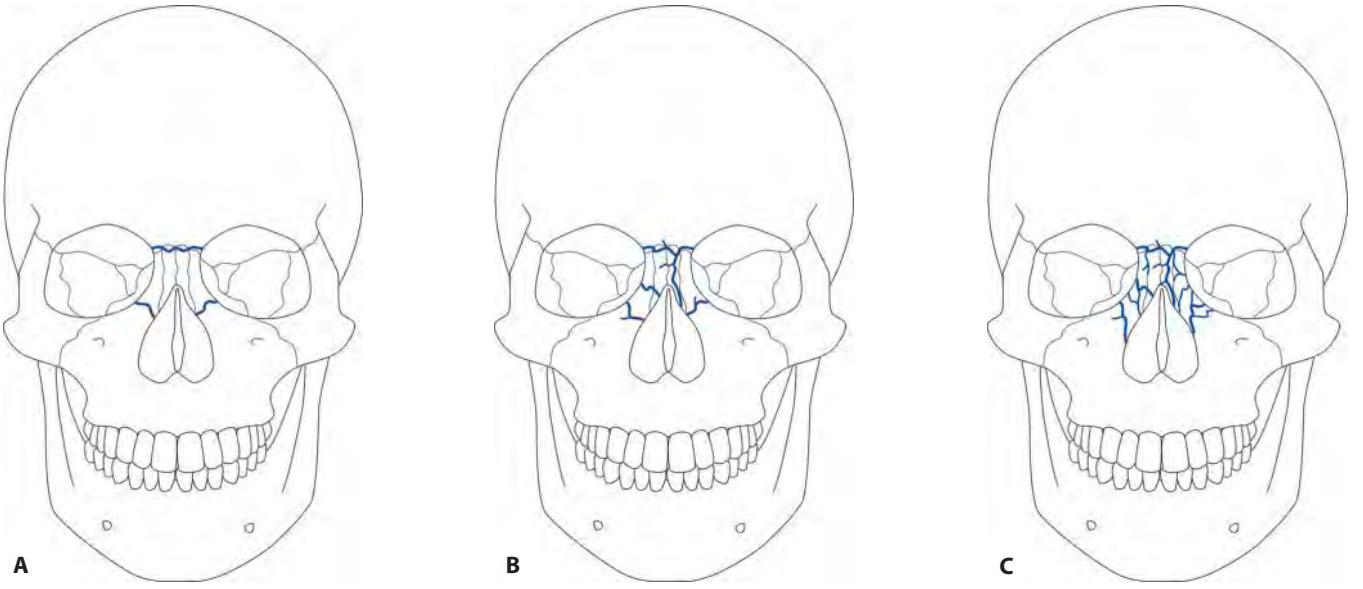


FIGURE 23-8. NOE fracture classification according to Markowitz et al.[64] From left to right: **A:** Type I; **B:** Type II; **C:** Type III.

achieve the functional goals while at the same time ensuring that the cosmetic goals are not compromised.

Surgical Access

Adequate exposure is essential to allow accurate reduction and fixation of fractures as well as any adjuvant procedures such as bone grafting. The coronal approach provides the best access to the frontal bone and sinus and produces the most desirable cosmetic results.[5,9,19,62] Although lacerations may be considered as an approach to access the fracture, their size and shape rarely provide enough access without undue and unsightly extension. Gull-wing or spectacle incisions result in unattractive scars that are highly visible because of their prominence on the brow and the resulting reflection of light. These scars can be camouflaged only with wide-rimmed glasses. The "open sky" approach is equally deforming, leaving an H-shaped scar over the brows and nasion, and like the gull-wing incision, is best avoided.

Although the coronal approach has been well described,[66] the preparation required for a coronal incision varies. If a neurosurgical procedure is anticipated, the hair may be shaved and the skin degreased with alcohol and then prepared using an antimicrobial skin preparation agent, preferably povidone-iodine solution. If a neurosurgical procedure is not anticipated, the hair should be parted coronally from preauricular region to preauricular region. Water-soluble lubricant is helpful in maintaining the part. The hair may then be braided in multiple pigtails and gathered anteriorly and posteriorly on either side of the part.

Local anesthetic with a vasoconstrictor is used to aid in scalp hemostasis. Electrocautery should not be used for the initial incision because it may damage hair follicles. The incision is made to the depth of the loose aponeurotic layer. The flap is undermined along this plane and above the periosteum in an anterior direction. Historically, Raney clips have been used to help with hemostasis. However, hemorrhage may occur when these clips are removed, and electrocautery may need to be used carefully at the end of the procedure. There are also concerns that Raney clips may cause crush injuries to the follicles and compromise scar camouflage.

The flap is elevated to within 2.0 cm of the fracture or within 3.0 cm of the supraorbital rims. The pericranium is then incised, and the reflection of the flap continues deep to the pericranium so that the branches of the facial nerve can be protected. Further reflection can be obtained with greater exposure by extension of the preauricular incision, galeal splitting (if a vascularized galeal flap is not anticipated), or release of the supraorbital nerve from its foramen or notch. If a pericranial flap is likely to be required, the coronal flap should be planned to allow the pericranial flap to be designed and elevated during the exposure.

Osseous Recovery and Access

Recovery of bony fragments in comminuted fractures is best undertaken during the reflection of the coronal flap. Fragments of the anterior table should be released from the periosteum and removed one at a time. Some method of organizing the fragments should be used. For example, the fragments could be numbered and their positions recorded on a map. They should be arranged in the same order on a back table. If contaminated, segments of bone may be cleansed with copious irrigation, scrubbing, and even povidone-iodine solution, before being used for reconstruction as free grafts.[67] Once the anterior table has been removed, access should be adequate for sinus exploration, posterior table inspection, and sinus floor (nasofrontal outflow) evaluation.

If a more extensive neurosurgical procedure is anticipated, osseous recovery may be performed in concert with a craniotomy bone flap. Before small fragments are recovered, the osseous flap design should be mapped out on the frontal bone (with care taken to avoid the sagittal sinus). Bur holes are created at three or four corners of the frontal bone. The tenuous and adherent dura is released through the bur holes, and a craniotome is used to connect the bur holes. The dura is carefully reflected as the bone flap is removed. Recovery of the rest of the osseous fragments can then be completed.

A perimeter-marking technique can be used for removal of the anterior table that is unfractured.[68] The removal of the entire anterior table is important when obliteration of the sinus is anticipated because this procedure requires thorough removal of sinus mucosa. One side of a hemostat or pick-up instrument can be inserted into the sinus, and a small bur hole can be made at the tip of the superficial arm of the instrument. Fixation plates can be adapted before the removal of the remaining anterior table segment.

Intraoperative Evaluation of the NFOT

After access has been obtained and osseous exploration and recovery have been performed, the condition of the frontal sinus floor and the nasofrontal outflow can be assessed by direct visualization (see Figure 23-7). The relative patency of the system can then be evaluated by placing an angiocatheter into the nasofrontal duct and introducing an appropriate fluid medium so that flow can be assessed. A 3.8-cm (1.5-inch) 18-gauge angiocatheter is the best instrument for this purpose. Patency can be confirmed by introducing normal saline and observing its emergence from beneath the medial turbinate or its collection in the posterior pharynx (Figure 23-9).

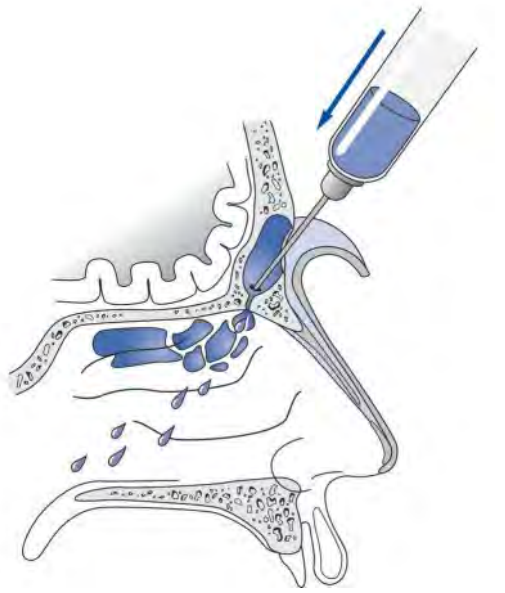


FIGURE 23-9. Technique of identifying a patent nasofrontal outflow tract. (Adapted from Zide MF. Nasal and naso-orbital ethmoid fractures. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; p. 582.)

Because of its dramatic hue, methylene blue dye has been offered as an appropriate fluid for evaluating patency. However, this blue dye can disrupt visualization of the surgical field because completely removing the dye is difficult during a surgical procedure (Figure 23-10). Fluorescein is an excellent alternative because it is clear, colorless, water-soluble, and radiolucent.[69] However, its visualization sometimes requires using an ultraviolet light source and then dimming

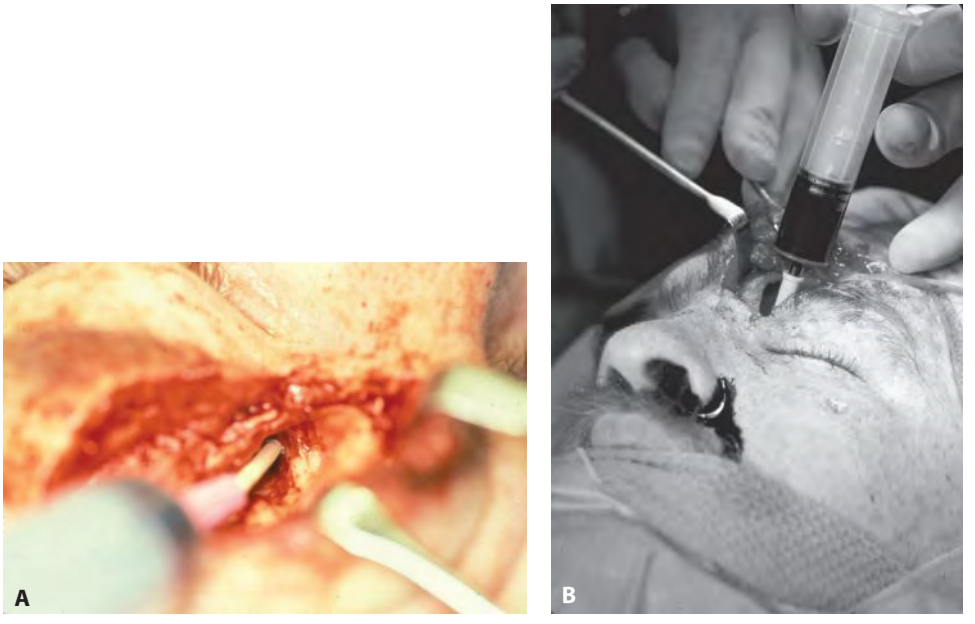


FIGURE 23-10. **A** and **B,** Intraoperative evaluation of nasofrontal duct patency by injection of methylene blue. In **B,** note the methylene blue coming from the patient's nostril. (**A** and **B,** Reproduced with permission from Haug RH, Cunningham LL. Management of fractures of the frontal bone and frontal sinus. Selected readings. Oral Maxillofac Surg 2002;10:1–32.)

the operating room lights. Radiopaque dye has been suggested for use as a diagnostic medium for nasofrontal duct fractures, but its visualization requires a C-arm fluoroscopy unit.[61] Moreover, any spilled radiopaque dye must be completely cleared before additional radiographs or CT images are obtained. Other fluids can be used to check for patency, including propofol, sterile baby formula or indigo carmine dye, but Congo red should not be used as it is neurotoxic.

Anterior Table Fractures

The thinnest area of the frontal bone is the region of the glabella, the anterior wall of the frontal sinus, and this region is highly susceptible to fracture. These fractures may seem straightforward but still deserve careful attention. Simple greenstick or nondisplaced anterior wall fractures do not require operative treatment.[70] Displaced anterior table fractures typically require reduction. Displacement greater than the thickness of the anterior table is an often-used rule of thumb to indicate the need for reduction. The surgeon should closely inspect the sinus floor, the posterior wall, and the patency of the nasofrontal duct. If the posterior wall and the floor are free of injury, the pieces of the anterior wall may be fixated with low-profile bone plates.[34,65,67,71–74] Any void remaining in the anterior wall after reconstruction can be closed by placing titanium mesh, methylmethacrylate, or other bone substitutes. The soft tissue injuries may then be repaired.

Posterior Table Fractures

Fractures to the posterior table of the frontal sinus are more concerning because of the proximity to the anterior cranial fossae (Figure 23-11). Posterior table fractures can be subclassified into three categories: nondisplaced, displaced, and displaced with gross neurologic injury. Each of the subclassifications is invariably associated with anterior wall penetration. Each is treated differently, and each requires neurosurgical consultation or joint management with a neurosurgeon. Antibiotic coverage is particularly important in preventing infection with these fractures.[13]

The surgeon should check carefully for any displacement of the fracture, CSF leak, entrapment of sinus membranes, or dural tears. If the injury is not substantial and the nasofrontal duct is patent, the anterior table is replaced and fixed and the soft tissue injuries are repaired. Comminution of the posterior table, penetrating injury, CSF leak with extensive dural damage, or frontal lobe damage requires frontal sinus cranialization. This involves complete removal of the posterior table, thereby effectively increasing the size of the anterior cranial fossa.[5,17,59,60,63,65] In one review of cases, as many as 16% of patients undergoing frontal sinus surgery required a cranialization procedure.[5] In such a case, the posterior table is gently removed, with either a diamond bur or rongeurs. Care should be taken in the area of the sagittal sinus to avoid violation of it, which can result in severe bleeding. All irregularities of the sinus are smoothed with a bur. After bone removal, the dura should be repaired with primary closure, a fascia or synthetic patch, or a galeal/pericranial flap.[5,65]

The wound is closed in layers. Strict attention must be given to meticulous removal of all of the mucosal elements from the walls, cul-de-sacs, and septa of the sinus and from all bone fragments.[75–77] Failure to remove such elements may result in a mucocele or pyocele. The mucosa is then reflected down into the nasofrontal outflow, and the orifice is obstructed by local bone or muscle. The harvested fat is placed into the sinus and packed until the sinus is full. Finally, the anterior table is reassembled and restored as would be done for a simple anterior wall fracture.

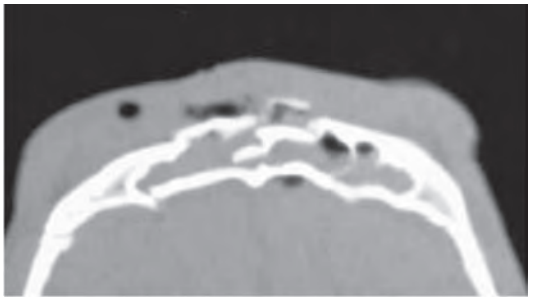


FIGURE 23-11. CT scan demonstrates anterior and posterior table fractures of the frontal sinus.

Orbital Roof and Supraorbital Bar Reconstruction

Once the posterior wall and the sinus floor have been explored, inspected, and evaluated for damage, any orbital roof and supraorbital bar fractures may be reconstructed. After these procedures have been completed, the sinus is ready to be obliterated and the nasofrontal outflow obstructed. The free osseous fragments that have been recovered, mapped, and arranged on a back table should be rigorously curetted for removal of any respiratory epithelium that could become trapped between them during reconstruction. Every remnant of respiratory epithelium should be removed from every crevice and cul-de-sac so that the possibility of future mucocele formation is minimized. This procedure is followed with local ostectomy with a no. 8 round diamond bur and copious amounts of saline. The arranged bone fragments should be consolidated with titanium microscrews (1.0–1.3 mm) and with appropriate plates, mesh, or both.[78,79] Mesh has an advantage in that it provides support and consolidation of the segments in three planes of space[78,79] (Figure 23-12). Titanium mesh has been shown to be compatible with soft tissue, undergoing incorporation with indigenous cells.[80] Resorbable technology continues to show promise, even though resorbable systems currently available are not as versatile as titanium mesh in their ability to be contoured or to stabilize small bone fragments. Before final placement of the consolidated titanium and bone segments, the sinus should be copiously irrigated and hemostasis achieved.[81]

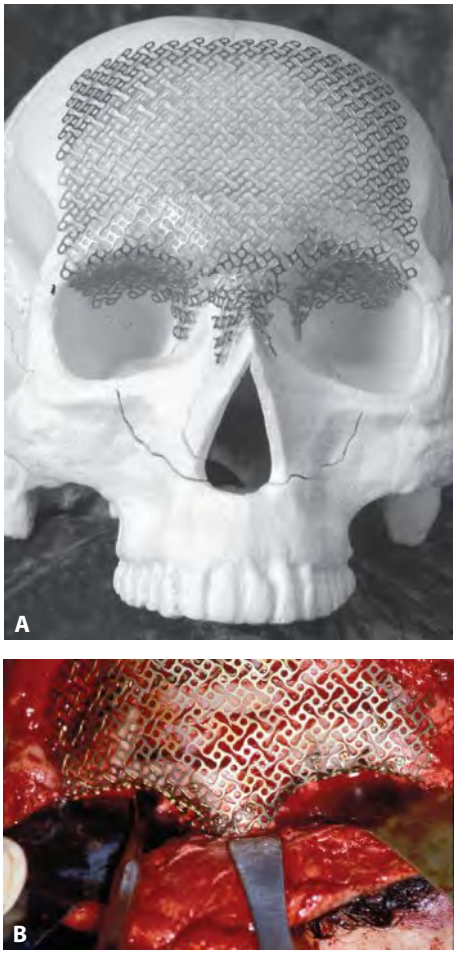


FIGURE 23-12. Reconstruction of the frontal bar and frontal sinus with titanium mesh. **A,** The mesh is adapted to a dried skull and then sterilized before surgery. **B,** Intraoperative view of reconstruction of the frontal bar and nasal dorsum with mesh.

Naso Frontal Outflow Tract (NFOT) Obstruction

NFOT obstruction should not be confused with sinus obliteration. Sinus obliteration is the elimination of dead space by the introduction of another material. Obstruction is one of the methods of isolating the sinus (or brain) from nasal contamination, basically by plugging it with another material.

As stated previously, the condition of the NFOT is the most important factor in maintaining the health of the frontal sinus.[17,19,23,33,55,59–63] This outflow permits the exit of mucin, seroma, or hematoma after injury. If the outflow tract is obstructed, sinusitis, meningitis, or osteomyelitis may develop. The condition of the outflow should be considered in the evaluation of fractures involving the NOE complex, the supraorbital rim, or the sinus floor. If the outflow is not patent, thorough removal of every possible remnant of sinus mucosa is performed by curettage.[17,59,69,76,77] This procedure should be carried out as previously described, using copious amounts of irrigation and with the aid of magnification. Any remaining remnants of the nasofrontal mucosa are then inverted into the nose.

A number of materials can be used to obstruct the nasofrontal outflow. Temporal fascia, temporal muscle, or both can be harvested from the adjacent temporal region. Tensor fascia lata is another alternative, but it may produce morbidity at the second surgical site. Estimating the surface area to be covered is an important technical point. A suture package is a good template for measurement of required size and recording those measurements. Because fascia shrinks, it is important to harvest approximately 20% more graft material than is indicated by the template. Bone graft material can be harvested from the sinus septum, the inner table, or elsewhere on the cranium.[82]

Commercial tissue sealants prepared from human plasma and containing bovine-derived aprotinin are available. These sealants have been shown to be effective tissue adhesives and hemostatic agents.[83–85] Autologous platelet gel and autologous fibrin glue have also been used for similar indications.[86,87] In addition, a new fibrin sealant from the American Red Cross has been reported to show promise as a hemostatic agent without the addition of bovine aprotinin.[88]

Whatever products are chosen, the organization and arrangement of the obstructive media are important. For example, a tissue sealant may be placed after inversion of the sinus mucosa. Fascia or muscle may then be introduced into the remnants of the outflow tract to block passage of nasal contaminants, followed by inertable cranium or remnants of septal bone from the sinus, followed by another layer of tissue sealant. Tissue sealants can be used effectively to seal off the sinus from the nasal cavity when they are applied layer by layer as described previously.

Sinus Obliteration

NFOT obstruction is necessary to seal off the frontal sinus from nasal contaminants. Sinus obliteration adds one more layer to the seal but also eliminates the "dead space" or air within the sinus that may permit fluids to accumulate to cause a seroma or a hematoma. Historically, sinus obliteration has been accomplished in a number of ways, including inserting no substance or object (theoretically permitting bone fill after curettage) or by insertion of hydroxylapatite, glass wool, bone, cartilage, muscle, absorbable gelatin sponge, absorbable knitted fabric, acrylic, or fat.[13,76,77,89–95] In many instances, a pericranial flap or galeal flap can be used for obliteration of the sinus (Figure 23-13). These flaps are raised from the coronal approach and are pedicaled flaps that can be very versatile for isolating intracranial contents. They are robust and have the advantage of not requiring a second surgical site for harvest.[81]

The use of fat has been reported most frequently, and this method historically has provided the most desirable results. Harvesting fat is simple and may be performed by liposuction or an open approach.[92] With the traditional open approach, a transverse semilunar incision is made within the "bikini" line, 5.0 cm superior to the symphysis pubis (Pfannenstiel's incision). The incision is carried through skin and subcuticular

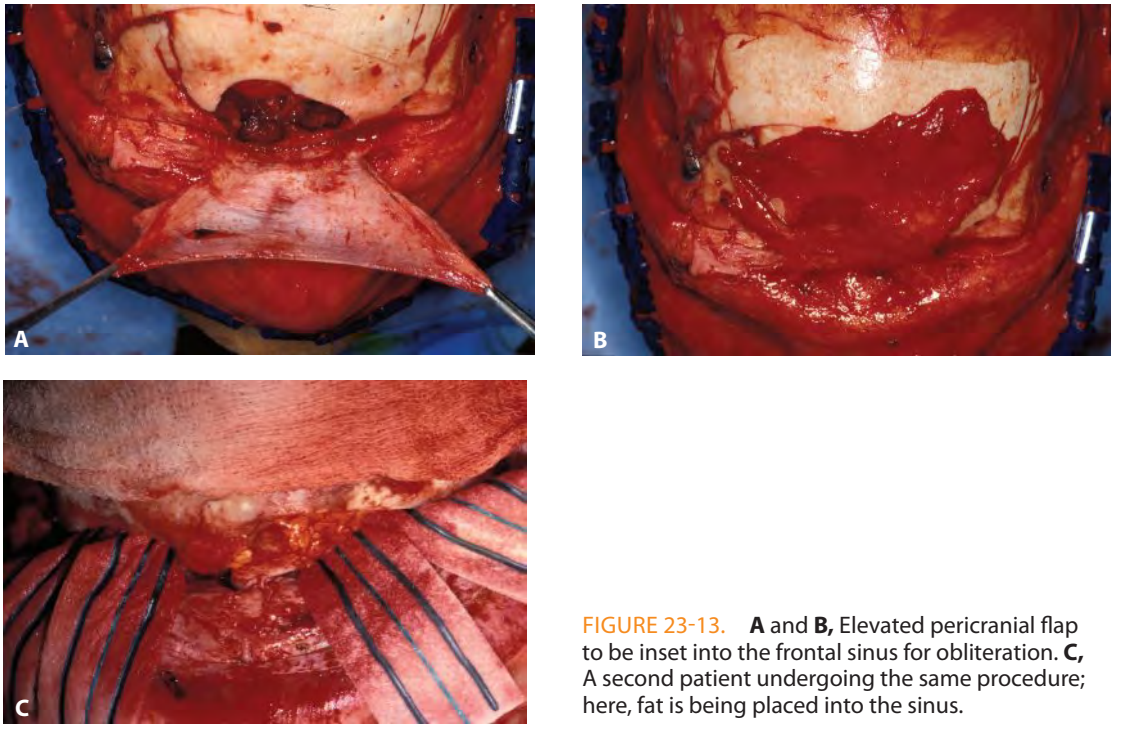


FIGURE 23-13. **A** and **B,** Elevated pericranial flap to be inset into the frontal sinus for obliteration. **C,** A second patient undergoing the same procedure; here, fat is being placed into the sinus.

tissue to the fat. The fat is grasped with an Allis clamp and retracted. Scissors are used to dissect the fat subcutaneously, moving laterally, inferiorly, superiorly, and caudally to the fascia overlying the abdominis rectus muscles, which are then connected, releasing the fat. Irrigation and meticulous attention to hemostasis are important before closure of the incision to avoid hematoma and infection. A periumbilical incision offers the most cosmetic scar for fat graft harvest. A 2-cm incision around the umbilicus followed by gently lifting fat from the incision with forceps and releasing it in a plane 1 cm deep to the skin with small metzenbaum scissors can easily yield 15 to 20 mL of fat for use in the sinus.

Endoscopy in the Management of Frontal Fractures

Increased experience in endoscopy and the desire to optimize cosmesis by minimizing cutaneous scars has led some surgeons to apply the use of endoscopic techniques to the treatment of frontal sinus fractures.[96] Endoscopic techniques have been described for the reduction and fixation of anterior table fractures and for the management of the frontal sinus drainage system. Endoscopy can also be used for the secondary management of contour defects of the frontal bone.[96]

NOE Reconstruction

Early surgical management is important in the reduction of NOE fractures.[25,39,46] The deformities that result from unrepaired NOE fractures are severe and difficult to correct. Correction often requires NOE osteotomies and grafting and satisfactory results are rarely achieved.

In addition to the coronal approach, complete exposure of the NOE area often necessitates lower eyelid incisions (transconjunctival or subciliary) and a maxillary vestibular incision.[37] These approaches aid in the treatment of displaced infraorbital rims and maxillary antrum or piriform rims.

Type I fractures can at times be reduced transnasally and treated without fixation. More often, single-segment NOE fractures are reduced through a coronal incision and secured at the nasofrontal junction, the maxillary buttress, and the infraorbital rims.[37,39]

Transnasal wiring historically has been recommended for fractures graded as Markowitz type II or higher.[19] In spite of the improvements in rigid fixation (bone plates and screws), complete reduction of the NOE area and reattachment of the MCT, or replacement of a small bone segment, never seem to be adequate with microplates alone. For NOE fractures including avulsion of the MCT or in which the MCT is attached to a small bone segment, transnasal wiring should be considered. Recently, techniques have been developed to allow easier capture of the medial cantal apparatus and improved fixation of the medial canthus. The point of fixation of the wires should be directed posterior and superior to the lacrimal fossa so that the medial canthal distance is decreased and widening of the nasal bones and blunting of the medial canthal area can be avoided.[19] Wires must be passed through the medial orbital bone and the superior nasal septum or the perpendicular plate of the ethmoid. Their passage can be facilitated with the use of a spinal needle or a wire-passing awl. Drill holes can also be used to aid in wire passing. Some clinicians have advocated temporary removal of the nasal bone for identification of the "canthal-bearing bone" and for facilitation of the passage of

transnasal wires.[19,37] The MCT and its bony segment can be incorporated into the transnasal wire fixation or an avulsed MCT can be attached to the transnasal wire with sutures. Slight overcorrection of the medial canthal distance is desired. In cases in which fracture comminution prevents adequate fixation of the MCT to a bone segment, stabilization with fixation to a calvarial bone graft has been advocated.[37] In cases in which sufficient medial orbital wall remains, placing a microplate and screw for attachment of the MCT behind the lacrimal crest has been suggested.[39]

Bone grafting may often be necessary in cases of severe comminution of the nasal bones or the medial orbital walls. Onlay of cranial bone grafts to maintain dorsal height and nasal tip projection can be performed through a coronal incision, and these grafts should be fixed rigidly.

Medical Therapy of the Sinus Postoperatively

Saline solution nasal spray can reduce symptoms of rhinosinusitis.[97] This therapy can prevent crusting of the nasolacrimal duct as well as the frontonasal duct and the ostia of the maxillary sinus. Because this treatment is inexpensive and involves little or no risk, it can reasonably be made a part of postoperative care regimens.

There have been no clinical trials related to post-traumatic medical treatment of the sinus. However, for patients in whom the frontal sinus has been left intact, there may be at least a temporary decrease in function of the mucociliary apparatus,[98–100] which may be compounded by postsurgical edema. Mucolytics have been advocated for use in patients with rhinosinusitis to thin the mucus secretions and to improve clearance.[97] During the post-traumatic or postoperative period, the use of mucolytics such as guaifenesin may be beneficial.

Decongestants may also be considered in the immediate postoperative period. Decongestant medications (e.g., pseudoephedrine or oxymetazoline hydrochloride) act by stimulating alpha adrenergic receptors in the mucosa of the upper respiratory tract. This action causes vasoconstriction in the respiratory mucosa, thereby shrinking the mucosa and increasing the size of the airways or ducts.[97,101,102] Topical agents have fewer systemic side effects but are known to have a rebound potential and should be used for no more than 3 days.

Because there is no consensus regarding the use of postoperative antibiotics, their use should be based on the individual patient and type of injury. The extent of soft tissue injury, presence of wound contamination, a concomitant CSF leak, and other associated injuries should all be considered. Current recommendations regarding the use of prophylactic antibiotics for head and neck injuries include a duration of therapy of no more than 24 hours.[103,104] In cases of contamination by a foreign body, antibiotic treatment may be continued for 10 days. In the absence of gross contamination of the wound, a limited number of postoperative antibiotic doses can be considered, or no antibiotics can be given. Antibiotics currently used to treat acute rhinosinusitis include amoxicillin, amoxicillin-clavulanate, azithromycin, cefpodoxime proxetil, clarithromycin, levofloxacin and trimethoprim-sulfamethoxazole.[97] Penicillin is still the drug of choice for treating facial fractures.[105]

COMPLICATIONS

Complications of frontal bone injury vary in severity and may occur many years after the injury. The principal types of complications are those that occur directly at the time of injury, those of an infectious nature, and those that are chronic problems.

The most devastating complications are neurologic problems resulting from displacement or penetration of the frontal bones into the brain. These injuries can result in concussion, severe brain injury, or death. Displacement of the floor of the frontal bone can cause orbital damage and the most frequent ocular complication is diplopia. Damage to the superior oblique muscles or trochlea may result in limited range of motion of the eye globe. Severing of the supraorbital nerve by the injury or during reflection of the osteoplastic flap leaves a permanent anesthesia in the distribution of the forehead.[106] Trauma to the floor of the frontal sinus or displacement of the medial supraorbital rim may cause a CSF leak. Generally, reduction of the fractures corrects this problem. If it is persistent, however, neurosurgical repair is indicated.

Infectious complications most frequently arise from occlusion of the nasofrontal duct or contamination of the sinus by penetrating foreign bodies. The most frequently encountered infection is meningitis.[91] If the nasofrontal duct is occluded, blood may accumulate in the sinus, creating an environment that is conducive to the growth of anaerobic bacteria.[24,107] Frontal sinus abscess is spread by direct extension through small fractures of the frontal bone or transosseous anastomotic vessels.[60] The result of this extension of infection is brain abscess, meningitis, cavernous sinus thrombosis, or (if the abscess is long term) osteomyelitis.

Mucoceles are the most common chronic problems.[108–110] Respiratory mucosa trapped between fracture segments or left behind during obliteration procedures may continue to grow. This continued growth may lead to the formation of mucoceles or pyoceles. The size of the growth determines how much damage occurs to adjacent bone and neurologic tissue. Frontal sinus imaging (CT or magnetic resonance imaging) should be ordered to detect a postoperative mucocele or pyocele. Imaging studies should be performed at 1, 2, and 5 years after surgery or whenever symptoms appear.[111] Complications can occur as late as 20 years postoperatively, and patients should be encouraged to have routine yearly follow-ups.[60]

Pain and headache may be chronic and may persist without an identifiable cause.[24] Cosmetic deformities such as contour deficits and irregularities stem from several causes. Bone loss at the time of injury may not be noticed for

months. Osteomyelitis with subsequent débridement leaves voids in bone. Even if the fractures are properly treated at the time of injury, remodeling may leave irregularities.

Anosmia, the loss of the sense of smell, and hyposmia are known complications of NOE fractures and can occur in as many as 38% of patients with high central midface fractures.[112] In addition, 23% of patients with high midface fractures report a decreased sense of taste (hypogeusia).[34,112]

Dacryocystorhinostomy

Impairment of the lacrimal drainage system after NOE fractures can give rise to epiphora, which may give rise to significant symptoms. Identification of the site of the obstruction can be helpful in treatment planning. Although CT scanning might demonstrate injuries likely to cause impaired drainage, it does not show the functional state of the lacrimal system. Jones tests can be used to assess the patency of the lacrimal system.

There are two components to the Jones tests. In the Jones I (primary dye) test, fluorescein dye is instilled into the inferior cul-de-sac of the eye. After 5 minutes, a cotton-tipped applicator is placed under the inferior turbinate of the nose. The failure to retrieve any fluorescein from the inferior meatus implies that a blockage is present. The site of the blockage can be determined using the Jones II (secondary dye) test. The Jones II test begins by irrigating the inferior cul-de-sac to remove any residual fluorescein. The lacrimal punctum is anesthetized, and an irrigation catheter is inserted to allow some saline to be washed through the system. If it is then possible to detect fluorescein at the inferior meatus, the obstruction is incomplete. If no fluid at all is detected, the obstruction to the lower system is complete. If saline only is collected, this indicates an obstruction of the upper collecting system, because dye is not collected into the system but it possible to irrigate saline through. Impaired drainage may be due to edema and be self-limiting. Persisting epiphora may require a formal repair of the drainage system. Gruss and coworkers[113] reported that the majority of patients with NOE fractures demonstrate adequate drainage with only 17% requiring a formal repair of the drainage system.

Dacryocystorhinostomy (DCR) is the repair of the lacrimal drainage system through the creation of a new "ostomy" or track from the lacrimal canaliculi to the nasal cavity. Techniques that have been described include open (external), endonasal, and soft tissue conjunctivorhinostomy.[114–116]

Perhaps the best-described technique is the open DCR. This procedure is performed through a 10-mm vertical incision placed 10 to 12 mm medial to the medial canthus of the affected eye. Blunt dissection is then used to approach the lacrimal crest. A periosteal incision is followed by careful dissection of the lacrimal sac away from the bony fossa, and an osteotomy is placed posterior to the lacrimal crest. The deep surface of the bone in this region is lined with nasal mucosa, which should remain intact during the osteotomy. Placement of a lacrimal probe can facilitate visualization of the lacrimal sac. After the sac has been freed, it is incised on its medial surface, and superior and inferior releasing incisions are made on the superficial side of the sac (posterior flap). This procedure is followed by a vertical incision of the nasal mucosa and anterior releasing incisions (anterior flap). At this point, Crawford tubes are used to intubate both the superior and the inferior canaliculi. When intubation is complete, the ends of the Crawford tubes are visible in the lacrimal sac and can be inserted through the lacrimal osteotomy and retrieved intranasally inferior to the middle turbinate. These ends are then cut to extend to the nasal vestibule and are sutured in place to the lateral nasal wall[40,115] (Figure 23-14).

Closure is then begun with anastomosis of the lacrimal sac and the nasal mucosa. The anterior flap of the nasal mucosa is closed to the posterior flap of the lacrimal sac. Often, this is technically challenging, and an alternative is to suture the anterior lacrimal sac flap to periosteum to maintain the opening between the lacrimal sac and the nasal mucosa. Care should be taken to avoid suturing the retained polymeric silicone tubing during flap closure. The remainder of the incision is closed in two layers. The tubing is left in place for 4 to 6 months, and patients should use saline nasal sprays to prevent crusting of the tubes (Figure 23-15).

The endonasal approach is conceptually the same procedure, except that the dissection is performed from inside the nose with the aid of endoscopic instruments and a fiberoptic light, which are introduced into the sac through the canaliculi. The nasal mucosa is incised and reflected over an area transilluminated from above. The illuminated area is most commonly seen beneath the middle turbinate, which may need to be displaced medially so that appropriate exposure can be obtained. The transilluminating light can be seen most readily through the lacrimal bone posterior to the frontal process of the maxilla. The frontal process can be removed with a Freer elevator or a 2-mm Kerrison rongeur. The lacrimal sac is then gently lifted free from the lacrimal bone with a Freer elevator. The thin lacrimal bone overlying the sac is then removed. An opening is then made into the lacrimal sac, and the Crawford tubing is inserted as before. Polymeric silicone tubes are left in place for 1 month, and saline spray and lacrimal irrigation are recommended.[116]

CORRECTION OF POST-TRAUMATIC DEFORMITY

Six months to 1 year after the initial surgical correction, secondary deformities of the frontal bone may be addressed. Contour defects result from failure to fully elevate depressed fractures, from voids in bone lost at the time of the trauma, and from infection. A multiplicity of materials has been used to correct contour defects, including bone from the adjacent calvaria, ileum, or rib; cartilage; titanium or stainless steel; polymeric silicone, methylmethacrylate, hydroxylapatite granules, silver, a cobalt-chromium alloy, polytef, polyethylene terephthalate fiber, nylon, polyethylene, and aluminum.[117,118]

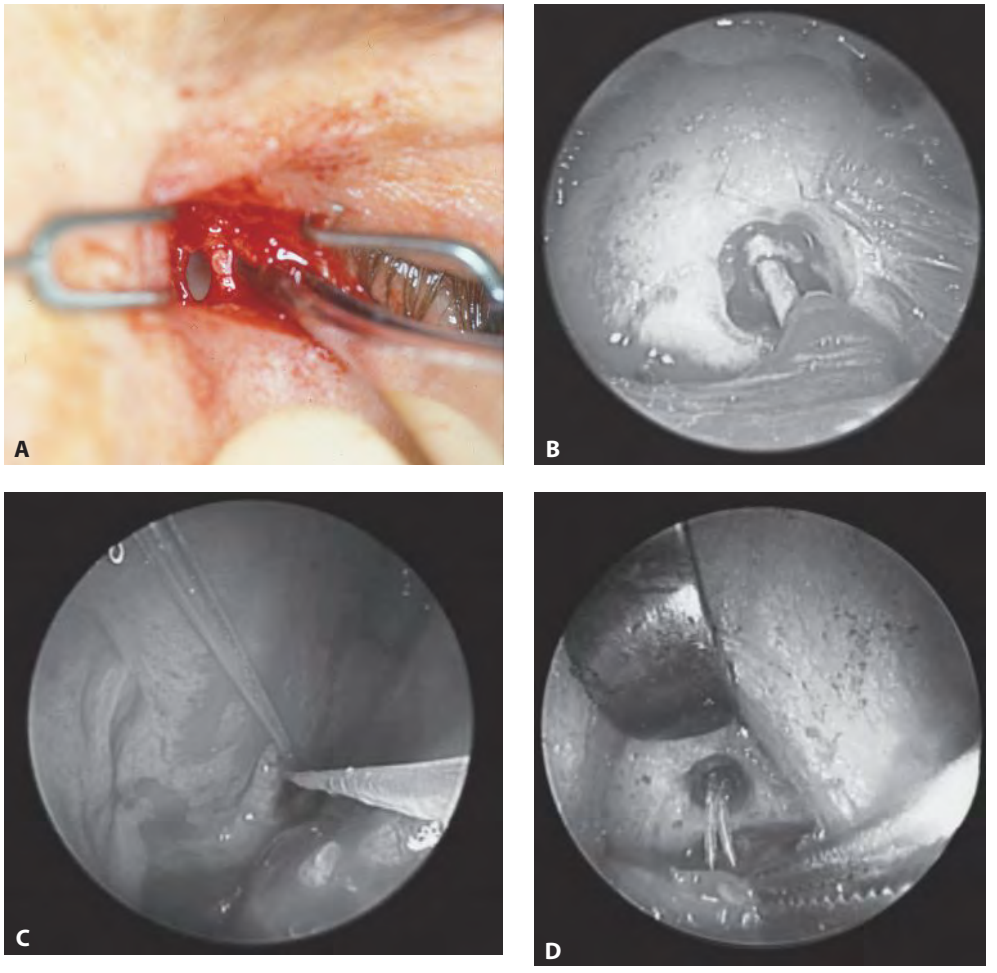


FIGURE 23-14. **A,** Incision of the lacrimal sac. **B,** Osteotomy, made with a round bur, through which the polymeric silicone tubes are placed. **C,** View of the polymeric silicone tubes exiting through the nasal mucosa into the nose. **D,** The lacrimal sac flap is shown being held in the forceps over the polymeric silicone tubing that exits into the nasal cavity.

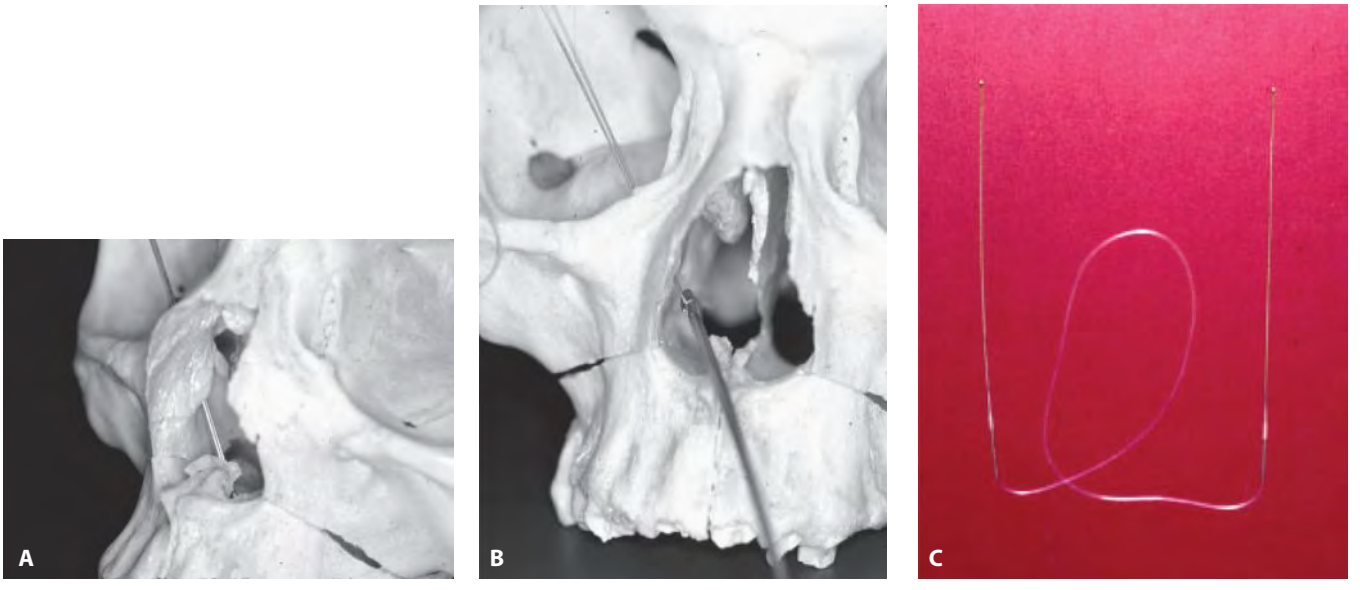


FIGURE 23-15. **A** and **B,** Views of a dried skull demonstrate the path of the nasolacrimal outflow and placement of polymeric silicone tubes. **C,** Crawford lacrimal intubation set.

The procedures for correcting such defects involve one-stage indirect prosthetic techniques, two-stage techniques, single-stage direct techniques, or computer-generated single-stage techniques.[119–121]

The one-stage indirect technique requires that an impression be taken of the defect through the skin. The impression negative is then filled with plaster to form a positive image on which an onlay prosthesis may be fabricated. Acrylic, polyethylene, tantalum, titanium, and cobalt-chromium prostheses may be fabricated with this technique. A full-thickness flap is then reflected, and the prosthesis is secured.

The single-stage direct technique requires that a full-thickness flap be reflected beyond the margins of the defect. Onlay cartilage or bone grafts then may be secured if an autograft is desired. Otherwise, an acrylic resin may be used. The bone is moistened, and acrylic is mixed and placed on a glass or polytef slab and rolled to a uniform thickness. The acrylic is placed directly over the bone and covered with a sheet of separating foil. The full-thickness flap is replaced to ensure proper contour and then is again reflected. A copious amount of saline is used to irrigate the area so that the material does not cause thermal damage to the skull. The flap is then replaced and sutured.

Improvements in computer design and technology now enable the fabrication of prostheses for one-stage reconstructions. The patient undergoes a three-dimensional CT before the operative procedure is performed.[119] A computer-assisted design/computer-assisted manufacturing (CAD/CAM) protocol is then used to create a model of the frontal bone and defect. A prosthesis may be created from polymeric silicone, acrylic, cobalt-chromium alloy, or hydroxylapatite-coated metals. During the operative procedure, the prosthesis is inserted as described previously.

In cases complicated by multiple infective episodes, removal of the infected tissue and recruitment of vascularized tissue using free-tissue transfer can provide an elegant solution.[34]

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CHAPTER 24

Nasal Fractures

Tirbod Fattahi, DDS, MD, FACS

Nasal bones are the most frequently fractured bones of the face in the adult patient. Owing to the nasal complex's location on the face, less force is required to fracture this area than any other facial bone.[1–3] Because of the frequency of injury, as well as the complex topography and different components (bone, cartilage, mucosa, skin) of the nasal complex, appropriate management of the acutely injured nose is imperative in order to prevent adverse sequelae. It is interesting to note that despite the seemingly "simple" approach to the management of nasal bone fractures, the incident of unfavorable results are as high as 62%.[4–7]

The most common etiologic factors for blunt nasal trauma in the United States continue to be motor vehicle collision, interpersonal altercations, and sport-related injuries.[8] Mechanism of injury is certainly important in determining the nature of the deformity as is discussed later. The purpose of this chapter is to review pertinent surgical anatomy, clinical and radiographic diagnostic tools, surgical management, and potential complications associated with nasal complex fractures.

SURGICAL ANATOMY

The nasal complex is composed of underlying nasal mucosa and turbinates, upper and lower cartilages, the cartilaginous and bony septum in the middle, and the paired nasal bones.[9,10] The superficial musculoaponeurotic system of the face composes the overlying soft tissue envelope of the nose and includes all of the perinasal musculature (Figure 24-1). The paired nasal bones are located between the nasofrontal suture cephalically and the upper lateral cartilages caudally. They are laterally bordered by the frontal processes of the maxillary bones. It is important to remember that the nasal bones overlap the cephalic portion of the upper lateral cartilages by 3 to 4 mm (Figure 24-2). The nasal septum is composed of cartilage (anteriorly) and the ethmoid and vomer bones (posteriorly). The quadrangular portion of the nasal septum is the primary support mechanism of the nasal complex and is made of cartilage (Figure 24-3). The cartilage is thick posteriorly along the junction with the vomer and ethmoid bones as well as along the maxillary crest. The vascular supply to the nasal complex is through the internal and external carotid systems via branches of the facial, superior labial, angular, sphenopalatine, ethmoidal, and superior ophthalmic arteries.[11,12] Innervation to the nasal complex is primarily through the second division (maxillary) of the trigeminal nerve.

CLINICAL AND RADIOGRAPHIC DIAGNOSTIC TOOLS

Examination of a patient with nasal trauma is no different than any other acutely injured person. Life-threatening and major organ injuries are addressed primarily before the

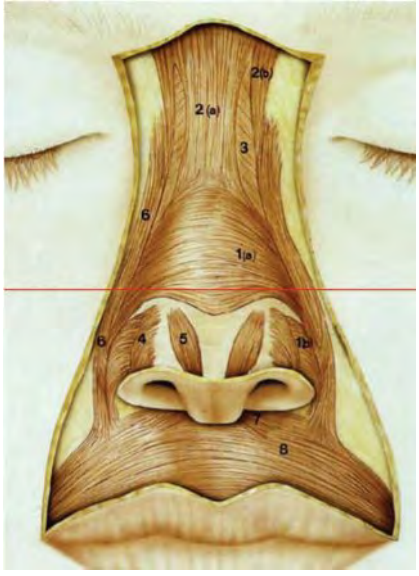


FIGURE 24-1. Perinasal musculature overlying the bony and cartilaginous framework.

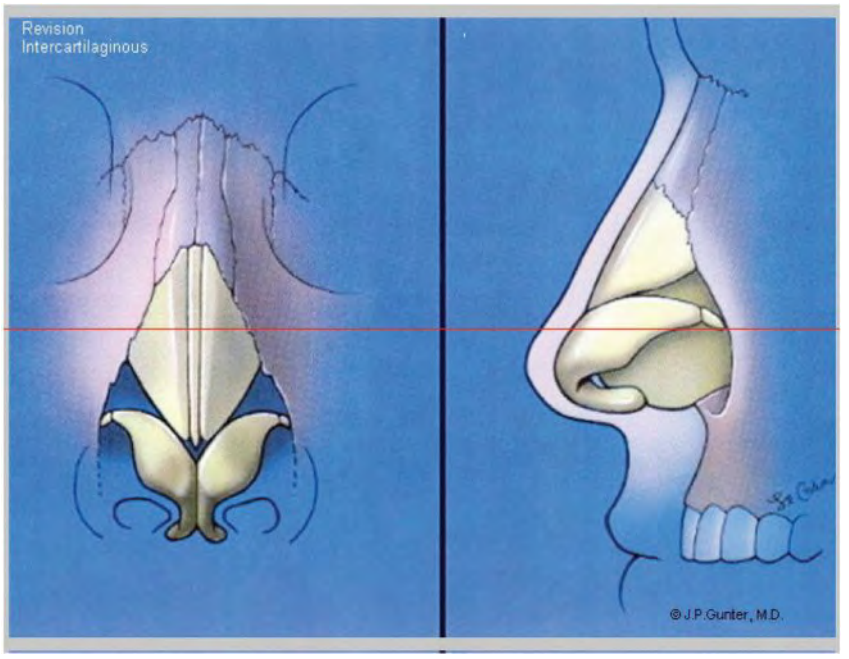


FIGURE 24-2. Cartilaginous and bony framework of the nasal complex.

secondary evaluation of the maxillofacial region. Imperative to the examination of the nose is obtaining a complete history. Mechanism of injury and timing of the event are important because they may assist in predicting a specific type of fracture or injury. For example, if the vector of force contacting the nose is directly perpendicular to the nasal dorsum, it is predictable that the bony complex is typically fractured in a lateral (outward) direction. Conversely, if the force is directed laterally to the nose (a right-handed punch to the left side of the nose), the nasal complex will deviate away from the vector of force with one nasal bone being medially displaced and the other one laterally displaced. Also, acknowledgment of any previous nasal trauma and presence of preexisting nasal deviation is important in determining whether a patient may require surgical intervention.

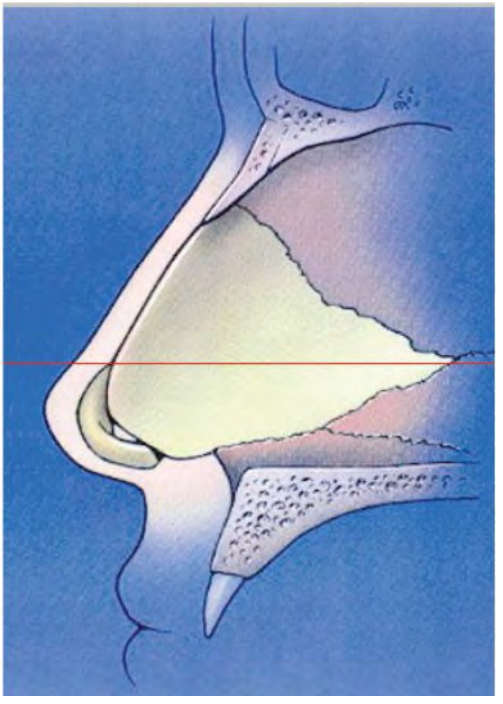


FIGURE 24-3. Cartilaginous and bony septum.

Examination of the nasal complex is done by performing a detailed internal and external evaluation of the nose. All patients with nasal complex trauma will have epistaxis (Figure 24-4). Observation of the facial region may reveal clinical signs of underlying fractures such as periorbital ecchymosis, nasal swelling, nasal complex deviation, cerebrospinal fluid (CSF) rhinorrhea, and lack of nasal projection. Increased intercanthal distance should be noted. Difficulty smelling (anosmia) is a late finding after nasal complex trauma and usually occurs if the cribriform plate of the ethmoid bone is involved. Palpation of the nose may reveal mobility or crepitus indicative of fracture. Internal examination with good lighting, nasal speculum, and suction is imperative to determine the presence of intranasal lacerations, septal deviations off the nasal crest of the maxilla, and the possibility of nasal septal hematoma. Septal hematoma must be drained in order to prevent late vascular complications associated with a devitalized cartilage predisposing to the development of a septal perforation. If a skull base fracture involving the cribriform plate of the ethmoid bone is suspected, a simple double-halo test can be done at bedside to ascertain the presence of CSF.

Today, the standard of care for mid- and upper face maxillofacial trauma radiography is computed tomography (CT) scan without contrast (Figure 24-5).[13–15] Nasal bone injuries and septal deviations and fractures are clearly delineated via CT scan. Although plain nasal radiographs are still utilized in some institutions, their value is limited. Photographs



FIGURE 24-4. **A** and **B,** Significant nasal complex trauma with full-thickness laceration involving the left nasal dome and dorsum.

of the acutely injured nose as well as premorbid photos obtained from the patient or family are helpful for documentation purposes as well as for pre- and postoperative comparison purposes.

Several different classification systems are available for nasal complex fractures.[4,16–18] Unfortunately, no one system is universally accepted. Perhaps the most relevant and accepted classification system is one describing the clinical and radiographic findings: open versus closed, deviated versus nondeviated, and comminuted versus noncomminuted. *Open versus closed* denotes the existence of an open fracture through an external or internal laceration. *Deviated versus nondeviated* refers to the appearance of nasal bones and septum. It is important to determine the status of the septum because failure to address this important structure will increase the potential for secondary post-traumatic rhinoplasty. *Comminuted versus noncomminuted* refers to the status of the nasal bones and the surrounding bony pyramid (frontal process of maxilla, anterior nasal spine, and ethmoidal bones).

SURGICAL MANAGEMENT

The goals of management of nasal bony injuries is to prevent development of a post-traumatic nasal deformity, restoration of proper nasal air flow, prevention of cosmetic deformity, and maintenance of proper nasal complex topography and

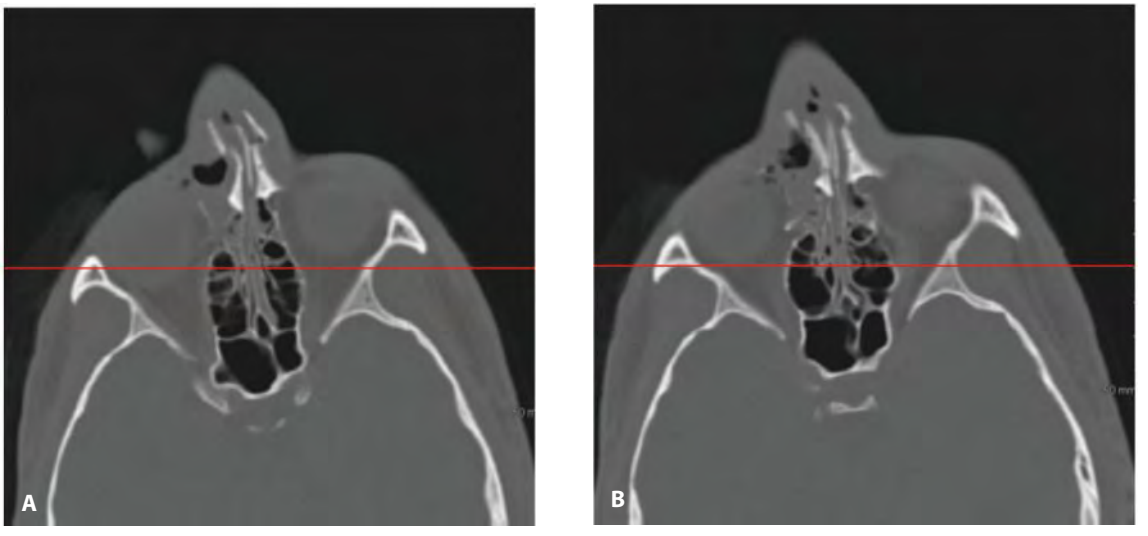


FIGURE 24-5. **A** and **B,** Axial computed tomography (CT) scan images of comminuted nasal bones, septum, and ethmoidal air cells.

projection.[19–21] Restoration of the sense of smell is also important, although anosmia may occur after high-impact nasal and naso-orbital-ethmoidal injuries (covered elsewhere in this book). Repair of nasal bone injuries can be done under local anesthesia, conscious sedation, or general anesthesia.[22–24] However, the most efficient method, and perhaps the most comfortable route, is to perform the repair under general endotracheal intubation. Closed reduction of the nasal complex is still the most commonly performed maneuver for repair of the injured bony nose. However, the incidence of patients requiring a post-traumatic rhinoplasty after closed reduction can range between 9% and 62%.[4,25–27] After the patient is intubated, a thorough manual examination of the nasal complex must be done. Crepitus, mobility, bony depressions, and asymmetries must be assessed and accounted for. Presence and/or absence of perinasal lacerations must also be confirmed. Intranasal examination is performed next. This can be accomplished via endoscopy or using a nasal speculum and good lighting. Once the examination is completed, the nasal cavity is injected with a local anesthetic and a vasoconstrictor. The nasal cavity is then packed with Cottonoid strips soaked in a vasoconstrictive solution. After waiting 10 to 15 minutes, the nasal cavity is examined again. The vasoconstriction significantly improves intranasal visualization. Using appropriate nasal reduction forceps, the nasal bones and the septum must be reduced in the appropriate vector (Figure 24-6). The reduction must be maintained via intranasal packings and/or splints. If conventional methods to repair the nasal complex are not successful, the threshold for performing more extensive procedures such septoplasty (with or without harvest of cartilage) and bony osteotomies in the acute setting in order to "straighten" the nasal complex should be rather low.[8,28–31] The septum can be approached through a hemitransfixion incision and can simply be scored on its convex side in order to remove deviations. Although deviated septal cartilage can be removed at this time, it is more prudent to maintain all septal cartilage at the time of the initial repair in case a post-traumatic nasal deformity develops, at which point cartilage grafts from the septum become rather important reconstructive tools. If the nasal septum continues to deviate off of the anterior nasal spine (ANS), subperichondrial and subperiosteal dissections must be performed in the region of the ANS. The septum must be then manually separated from the ANS and then reattached to it using sutures. This will ensure a midline caudal septum.

Bony vault deviation involving the nasal bones that are not reduced via closed reduction may require open reduction or osteotomies. Time interval from the initial injury to the time of repair (longer than 10–14 days) may impede closed reduction of nasal bones because the adult nasal bone will begin to form a callus after this period of time that may not be easily manipulated. Delay in the initial treatment of the acutely injured nose may require open reduction and bony osteotomy. In this situation, a transoral approach to the nasomaxillary buttresses, frontal processes of the maxilla, and the nasal bones may facilitate an open reduction with or without internal fixation. Endonasal or percutaneous lateral osteotomies to mobilize the bony nose can also be performed.[32,33] Lateral nasal osteotomy is a powerful maneuver in order to straighten the bony nasal vault. Again, consideration should be given to intranasal packing in order to maintain the mobilized bony segments. Use of external splints may also aid in elimination of dead space and maintenance of reduction (Figure 24-7). Approaching the nasal bones through existing perinasal lacerations is also an acceptable approach. Open reduction with or without fixation can be directly performed via this method and can be quite effective (Figure 24-8).

POSTOPERATIVE CARE AND COMPLICATIONS

Consideration should be given to the use of perioperative antibiotics when addressing nasal trauma and repair. Prolonged antibiotic use has been encouraged, especially when internal nasal packings have been used in order to

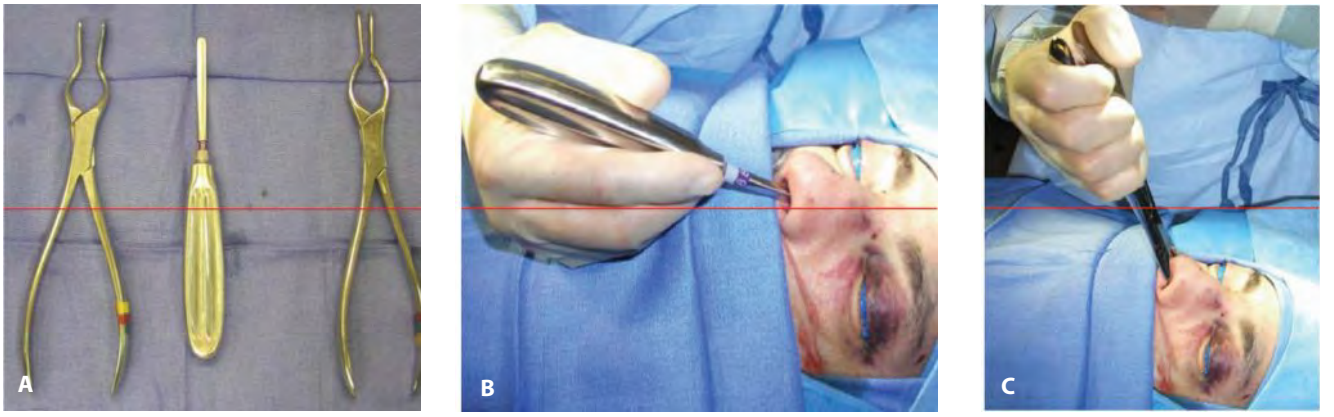


FIGURE 24-6. **A,** Commonly used instruments in the treatment of nasal fractures (*right to left:* Walsham forceps, Boies elevator, Asch forceps). **B** and **C,** Asch forceps in use.



FIGURE 24-7. Nasal splint applied at the completion of reduction.

decrease the possibility of epistaxis. Sinus precautions are also encouraged.

It is interesting to note that the incidence for a residual nasal deformity after closed reduction of nasal fractures can be up to 62%, although several studies have confirmed much better results.[4,19] It is, therefore, imperative to follow the patient's progress in order to ensure that an acceptable functional and cosmetic outcome has been obtained. It is generally prudent to wait up to a year before addressing a residual post-traumatic deformity, although author's opinion is that a *grossly* deviated nose at 1 month after repair of nasal complex fracture will remain a grossly deviated nose at 1 year after surgery. Therefore, in specific cases, earlier intervention via a post-traumatic rhinoplasty is warranted (Figure 24-9).

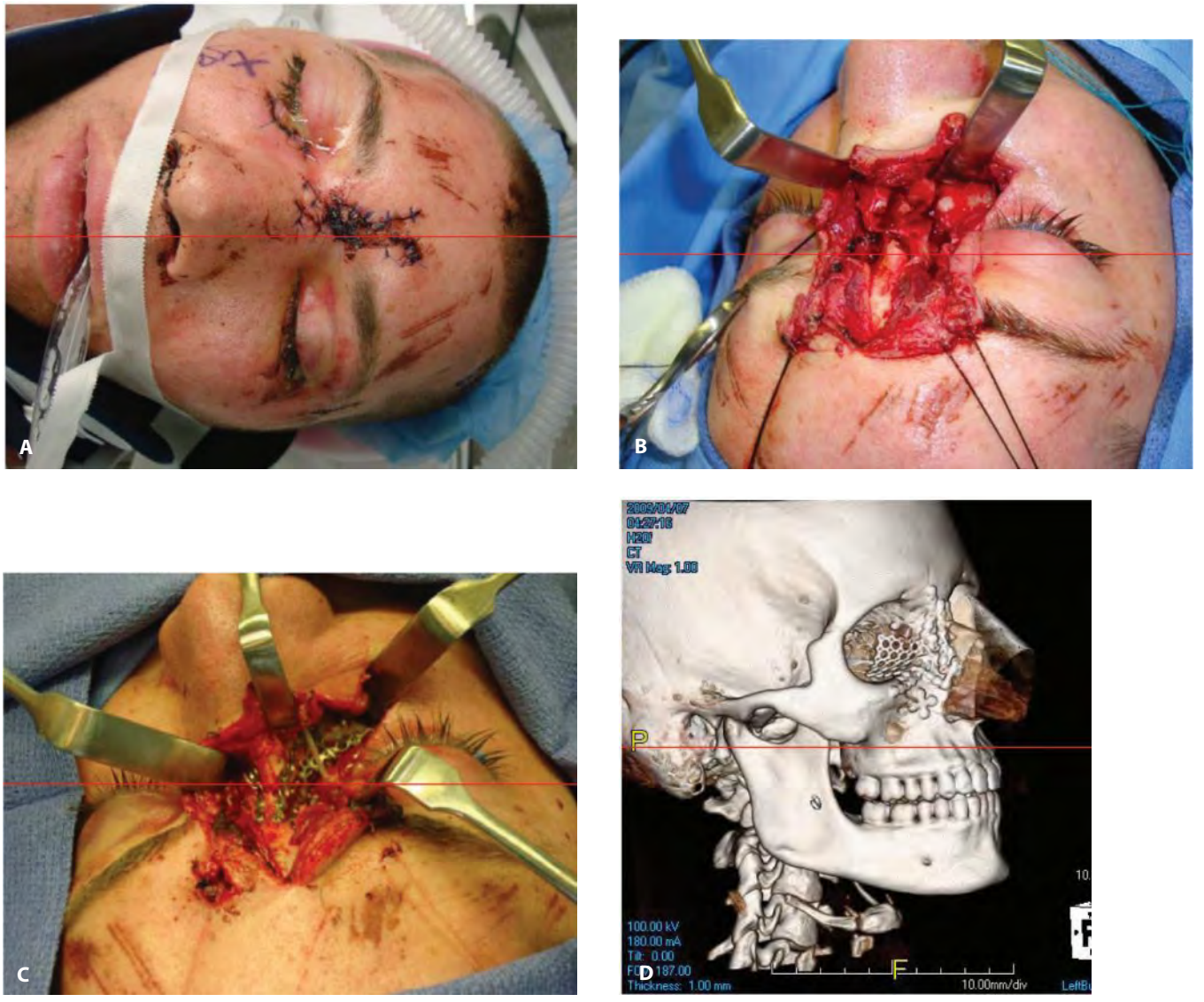


FIGURE 24-8. **A,** Patient with midface fractures and lacerations over the nasal dorsum. **B** and **C,** Utilization of existing laceration to reduce and fixate nasal fractures. **D,** Postoperative CT scan.

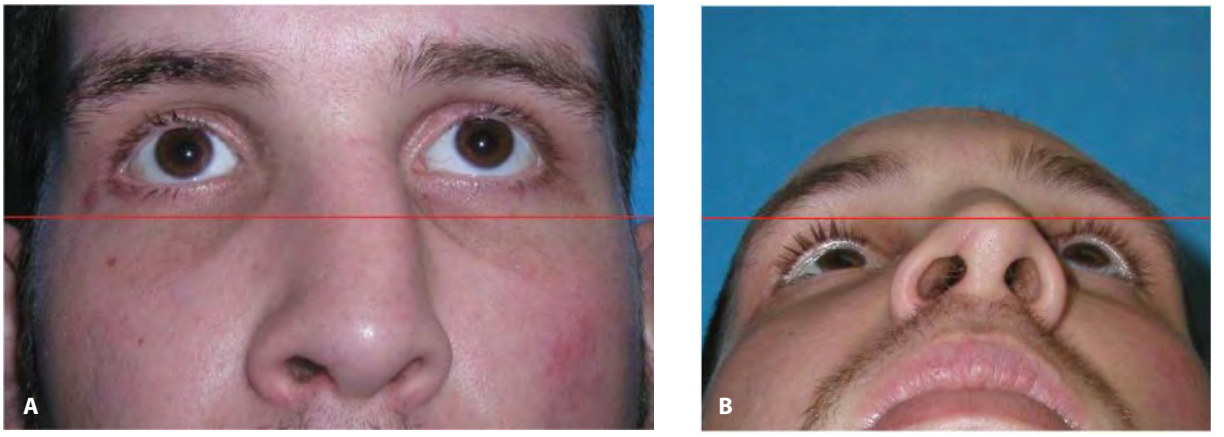


FIGURE 24-9. **A** and **B,** Patient with persistent nasal complex deformity after closed reduction of nasal fractures. This type of deformity can benefit from a post-traumatic rhinoplasty.

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CHAPTER 25

Gunshot Injuries

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Management of gunshot injuries to the face led in many ways to the development of modern maxillofacial surgery, and it remains a cornerstone of the specialty of oral and maxillofacial surgery. There is an aura that surrounds the management of these complex wounds that affects residents as well as experienced clinicians. The mystique that developed in the earliest accounts of management of gunshot wounds (GSWs) persists with the passing along of myths and dogma to subsequent generations of surgeons. Readers are encouraged to use the information in this chapter as a guide, to combine it with their own experience, and hopefully to continue the evolution in treatment of these unique wounds.

HISTORY

The introduction of Chinese gunpowder to Europe around the 13th century was quickly followed by the development of projectile weapons based on its explosive properties. The first recorded use of a cannon was by Edward III against the Scots in 1327, and small arms carried by one or two soldiers began appearing in the 14th century.[1] Early weapons that used modified arrows were replaced with more efficient stone and, ultimately, metallic projectiles.

Improvements in projectiles and firearms led to increasing numbers of more devastating wounds. Surgeons accustomed to dealing with a variety of wounds from blunt, bladed, and pointed weapons were faced with blast and projectile injuries of a completely different nature. Contamination and devitalized tissues led to increasing numbers of infections, which surgeons of the day incorrectly attributed to the gunpowder itself and to the anticipation of "laudable pus."

Subsequent advances in surgical knowledge went on to closely parallel the evolution of firearms. Knowledge gained on the battlefield by famed military surgeons such as Ambroise Paré (1510–1590) elevated the art of surgery to a learned profession.[1] Lessons learned in the management of injuries sustained in combat continues to improve our understanding and management techniques, leading to improved care for the increasing number of civilian gunshot injuries.[2,3]

DEMOGRAPHICS

GSWs are second only to motor vehicle accidents as a source of injury and death, and rank as the eighth leading cause of death in the United States.[4] Recently, the number of firearm-related deaths and injuries in children and adolescents has declined.[5] According to the National Center for Injury Prevention and Control, firearm-related deaths have shown a continual decline from approximately 15 per 100,000 in 1993 to approximately 11 per 100,000 in 1998.[6] Because of past difficulties with surveillance, however, most reports likely underestimate unintentional firearm-related deaths and injuries overall.[7,8]

Interestingly, Patton and Woodward[9] reported that, although GSW admissions decreased at the Henry Ford Hospital in Detroit by 45%, the number of patients who required operations actually increased by 17%. The number of gunshot victims dead on arrival remained steady. A possible explanation is that an increased number of patients are discharged from the emergency department after significant injury requiring admission has been ruled out; these patients are, therefore, not counted as admissions.[9]

The demographics of gunshot injuries are telling. Most victims are young males (<38 yr). Suicides and assaults far outnumber unintentional and accidental shootings. Firearms are implicated in 58% of male suicides and 37% of female

suicides. Importantly, the number of patients surviving and requiring treatment of gunshot injuries outnumber firearm fatalities by approximately 5:1.[8,9]

Currently, an estimated 135,000 GSWs are treated annually in the United States. The incidence of firearm-related injury and death in the United States exceeds that of other developed countries.[10] Although there appears to be a relationship between the rate of household firearm ownership and the homicide rate, most agree that other social factors are required to explain the number of firearm injuries in the United States in comparison with other developed countries.[11] Indeed, in countries in which firearm ownership is required for militia duty, firearm injuries are lower on a per-capita basis than in the United States.[12] The majority of civilian firearm injuries are sustained from handguns (86%), followed by shotguns (8%) and rifles (5%). Approximately 12% to 14% of unintentional and assault gunshot injuries involve the head and neck, whereas 51% of self-inflicted gunshot injuries involve the head and neck.[6] Clark and colleagues[13] reported on their experience at the Maryland Shock Trauma Center and found that of 178 GSWs to the face, 40% involved the frontal bone and cranium, 9% involved the orbits, 14% involved the lower midface (maxilla), 13% involved the mandible, and 24% involved multiple sites. Shotgun injuries more commonly involved the mandible and midface.[13] Demetriades and coworkers[14] reported on the extensive experience of the University of California at Los Angeles. Of 4139 patients admitted with gunshot injuries over a 4-year period, 6% (247) had GSWs to the face. Thirty-eight percent of these had isolated wounds to the face, whereas the remaining 62% had associated injuries to other body areas. They reported that the mandible was the most commonly involved facial bone (54 cases), followed by the maxilla and zygoma (21 cases each). The orbits and nasal bones were involved in 18 and 15 cases, respectively. Thirty-six patients died after admission. All of the deaths were secondary to injuries to the chest, abdomen, or brain. There were no deaths associated with isolated facial injuries.[14]

Aside from the tragedy of firearm-related injuries and the emotional toll such injuries take on victims, their families, and communities, the financial burden to society of firearm-related injuries is significant. This is especially true with regard to the long-term rehabilitation and multiple reconstructive surgeries that many victims of facial GSWs require. Cook and associates[15] reported approximately 115,000 firearm-related injuries in the United States yearly, with an annual cost of treating firearm injuries of approximately & 2.3 billion; of this, taxpayers pay $ 1.1 billion. Although this cost represents only 0.25% of the U.S. health care budget of $950 billion, it is significant considering that the group most affected typically involves younger, healthier patients who usually require very little medical care.[15]

BALLISTICS

Ballistics is the science of projectile motion. A prerequisite to understanding the injuries caused by various firearms is knowledge of the language of ballistics. The potential problems of a wound caused by a projectile can be better anticipated if one has some knowledge of the weapon and projectile type that caused the wound. For example, if the surgeon is aware that a patient suffered a high-energy wound caused by a high-power, high-velocity cartridge, she or he can better appreciate the potential for extensive areas of devitalized tissue that may declare later. In addition, an understanding of firearm nomenclature allows the surgeon some ability to predict the types of weapons that are commonly involved in various types of civilian gunshot injuries. For this reason, the clinician dealing with gunshot injuries should be conversant in the rudiments of ballistics, types of firearms, and projectiles.

Ballistic science seeks to explain the behavior of the projectile and is typically divided into three stages:

1. *Internal (or interior) ballistics* describes the forces that apply to a projectile from the time the propellant is ignited to the time the projectile leaves the barrel. An important consideration is barrel length. In general, longer barrels (rifles) allow the force of the propellant to act on the projectile longer and generate higher velocities than do shorter-barreled weapons. In addition, a longer barrel serves to stabilize the bullet over longer distances.
2. *External ballistics* refers to forces that act on the bullet in flight. The primary factors that govern external ballistics are the weight and shape of the bullet.
3. *Terminal ballistics* is the study of bullet behavior once it impacts the target and is primarily concerned with how much energy is transferred to the target material and the resultant damage. The science of terminal ballistics is most important to the surgeon and is the most common source of controversy when discussing ballistic wounding. Attempts to reproduce the interaction of bullets with living tissue by using various target media such as ballistic gel have led to many myths surrounding wounding and the "stopping power" of various bullets and weapons. Similarly, surgeons have passed on many myths of their own regarding GSWs and the firearms that cause them.

ENERGY AND WOUNDING POWER

Traditionally, kinetic energy has been used as the basis to explain wounds caused by a gunshot. Simple physics can be applied to the projectile using the following formula:

$$KE = mv^2$$

where KE is kinetic energy, m is the mass of the projectile, and v is the velocity of the projectile.

Wounding power is typically related to the amount of kinetic energy transferred to the target:

$$P = m(V_{impact} - V_{exit})^2$$

where P is power and V is velocity.

Based on these formulas, the velocity of a projectile has traditionally been considered far more important than its

TABLE 25-1. **Comparison of Approximate Cartridge Velocities and Muzzle Energy***

Cartridge	Bullet Weight (grains)†	Velocity (ft/sec)	Muzzle Energy (ft/lb)
.22 LR	29	1225	140
.32 auto	71	900	129
.380 auto (9 mm short)	95	955	190
.38 special	145	680	170
.357 magnum	110	1565	535
9 mm	124	1100	345
.45 auto	230	790	370
.44 magnum	240	1420	741
.223 (5.56x45mm or 5.56 NATO)	55	3100	1280
.308 (7.62x51mm or 7.62 NATO)	110	3000	2650
.300 magnum	180	2900	3500
20-gauge shotgun	547	1185	1400
12-gauge shotgun	820	1250	2,600

LR = Long Rifle; NATO= North Atlantic Treaty Organization.
*Velocities and muzzle energy can vary within different cartridges depending on the weight of bullet, powder type, and other variables such as barrel length.
†1 oz.= 437.5 grains.
Adapted from Federal Ammunition Company High-Power Ammunition Handbook. Minneapolis; 1983.

mass in wounding power. Indeed, often guns are classified as low velocity (<350 m/sec), medium velocity (350–600 m/sec), and high velocity (>600 m/sec), or simply into low velocity (<1200 ft/sec) or high velocity (>1200 ft/sec). Considering a typically sized projectile, a velocity of approximately 50 m/sec is required to penetrate the skin, and a velocity of approximately 65 m/sec will fracture bone.[16] Table 25-1 lists a comparison of commonly encountered pistol and rifle cartridges.

In general, there is an inverse relationship between a bullet's diameter (caliber) and its velocity. Unfortunately, the realities of wounding are not as clear-cut, and the emphasis on velocity and kinetic energy of the weapon as they relates to treatment strategies is excessive.[17] In an excellent review, Fackler[18] debunks many of the commonly held beliefs of ballistic injury, including the idolatry of velocity, the exaggeration of the effects of temporary cavitation and pressure, bullet tumbling, the exaggerated role of kinetic energy transfer, and most important, the emphasis on extensive wound débridement. The heterogeneity of the human body, which is composed of tissues of varying densities and elasticities, does not allow formulas to explain all of the nuances of wounding caused by projectiles of different velocities, sizes, and weights. Practically, there is a balance between velocity, projectile mass, and projectile size that governs the amount of energy transferred to the target and resultant tissue wounding. These factors govern the four components of projectile wounding: penetration, permanent cavity formation, temporary cavity formation, and fragmentation.

Penetration allows the projectile to transmit kinetic energy and destroy tissue. A bullet must penetrate to a sufficient depth to cause damage. Likewise, a projectile that overpenetrates or passes completely through nonvital tissue may result in little damage.

The *permanent cavity* describes the space that results from direct tissue disruption and destruction. It is a function of the penetration and size of the projectile. It is generally considered to be the most important factor in the wounding and stopping power of a particular cartridge and bullet.

The *temporary cavity* is produced as the projectile travels through the target tissue. Transfer of kinetic energy results in a stretching of elastic tissues. Although they may remain intact, some of these tissues may be irrecoverably damaged. Arteries may suffer pseudoaneurysm formation and rupture, and nerves may fail to recover function.

Fragmentation, which may not be present in a GSW, refers to the projectile (certain projectiles are designed to fragment; see later) or secondary fragments such as clothing or bone that develop from being struck by the projectile.

Despite claims by many bullet manufacturers, fragmentation of the projectile does not reliably occur in most handgun wounds. Bullets specifically designed as fragmentation rounds typically suffer from low-penetration ability. High-velocity rifle rounds are, however, known for their devastating fragmentation.

The effects of the temporary cavity on wounding are often exaggerated in ballistic literature. Because most tissue has an elastic nature and ability to recover from stretching (certain tissues such as brain are exceptions), damage from temporary cavitation is not as important as many expound. The massive zones of necrotic tissue that were felt to develop from temporary cavitation do not exist in reality. The most important factors in projectile wounding remain penetration and the size of the permanent cavity. A very small projectile traveling at high velocity striking an area of low density (e.g., fat) may impart far less damage than a larger projectile traveling at a lower velocity and striking an area of high density (e.g., bone). The realities of stopping power further call into question many of the claims promulgated through ballistic literature as well as surgical practices. In reality, the power transferred to the victim is the same as what the recoil imparts on the shooter. Again, simple physics explains that the impact of a 9-mm pistol round (see later) is the same as that created by a 0.45-kg weight dropped from a height of

1.82 m or of a 4.53-kg weight dropped from a height of 1.82 cm. In more practical terms, the amount of energy delivered to a body by a bullet is approximately equivalent to that transmitted when one is hit with a baseball.[19]

It is important to understand that the science of wounding power is more than simple physics; it is a complex interplay of projectile and target tissue characteristics that makes each wound unique. For this reason, categorization of wounds based on projectile characteristics such as velocity, although useful, should not promote dogmatic management schemes but instead should serve as guides. Surgeons should be wary of strict categorization schemes and treatment algorithms based only on velocity or another bullet characteristic and should bear in mind Lindsey's statement, "I will keep treating the wound, not the weapon."[17]

FIREARM TERMINOLOGY

As with ballistics, some knowledge of firearms is necessary for surgeons managing GSWs. It is a prerequisite for communicating with law enforcement officers and other clinicians.

Firearms are generally classified as *handguns*, *rifles*, and *shotguns*. Handguns are also referred to as *pistols* and *revolvers*, depending on their mechanical actions. With few exceptions, most are low or medium velocity, typically less than 600 m/sec, and usually cause tissue damage only along the bullet tract. Rifles range from low to high velocities. Shotguns typically are smooth-bore weapons that fire shells filled with lead shot of various sizes. Some shotguns may be modified with rifled barrels to fire shells containing a solid lead projectile referred to as a *slug*. Although they are of low velocity, close-range shotgun injuries are devastating, especially with larger lead shot such as buckshot (see later).

Rifles and handguns are classified by caliber. The *caliber* of a weapon is the diameter of the muzzle bore as measured from the narrowest area (the lands, see later discussion on rifling), which is the same as the diameter of the projectile (bullet). *Cartridge* or *round* refers to the case containing the ignition system (primer), the propellant, and the projectile (bullet). Measurements for American firearms are typically in inches. For example, the .45-caliber pistol bullet is 0.45 inch (1.14 cm) in diameter. Firearms of European origin, such as the 9 mm, have classically used the metric system. The American military round for the M-16 (military version of the AR-15) is usually the 223, which is 0.223 inch (0.57 cm) in diameter, whereas the Soviet AK-47 fires a 30-caliber projectile, or 7.62 × 39 (*39* refers to the length of the case containing the propellant in millimeters; Figure 25-1).

Shotguns were originally designed to be used on small, fast-moving game and typically fired small pellets that dispersed in flight to form a pattern. Typical muzzle velocities range from 335 to 427 m/sec. They are usually referred to by *gauge*, which is an English measurement that describes how many lead balls equaling 1 lb (0.45 kg) would fit into a particular diameter of the barrel. For example, it would take 12 lead balls equal in diameter to the internal diameter of a 12-gauge shotgun barrel to make 1 lb. A 12-gauge shotgun has an internal barrel diameter of 1.85 cm, whereas a 28-gauge shotgun has an internal barrel diameter of 1.41 cm. It is clear that the higher the gauge, the smaller the diameter of the barrel (Figure 25-2A). There are some exceptions to this classification scheme. For example, a 410 shotgun has a barrel whose internal diameter is 0.410 inch (1.04 cm). In general, the lower the gauge, the more powder and shot the shell can contain.

Shot is also classified by size. Commonly encountered shot sizes range from 8 shot (0.23 cm), with approximately 500 pellets in a 12-gauge shell, to number 00 buckshot



FIGURE 25-1. **A,** Representative rifle cartridges. **From left to right:** .300 Winchester magnum, 30-06, .308 (7.62 × 51 NATO), .223 (5.56 × 45 NATO), 7.39 (AK-47 round), and .17 rimfire. **B,** Representative pistol cartridges. **From left to right:** .44 magnum, .357 magnum, .38 special, .45 auto, .40 auto, 9 mm auto, .380 auto (9 mm short), .22 rimfire.

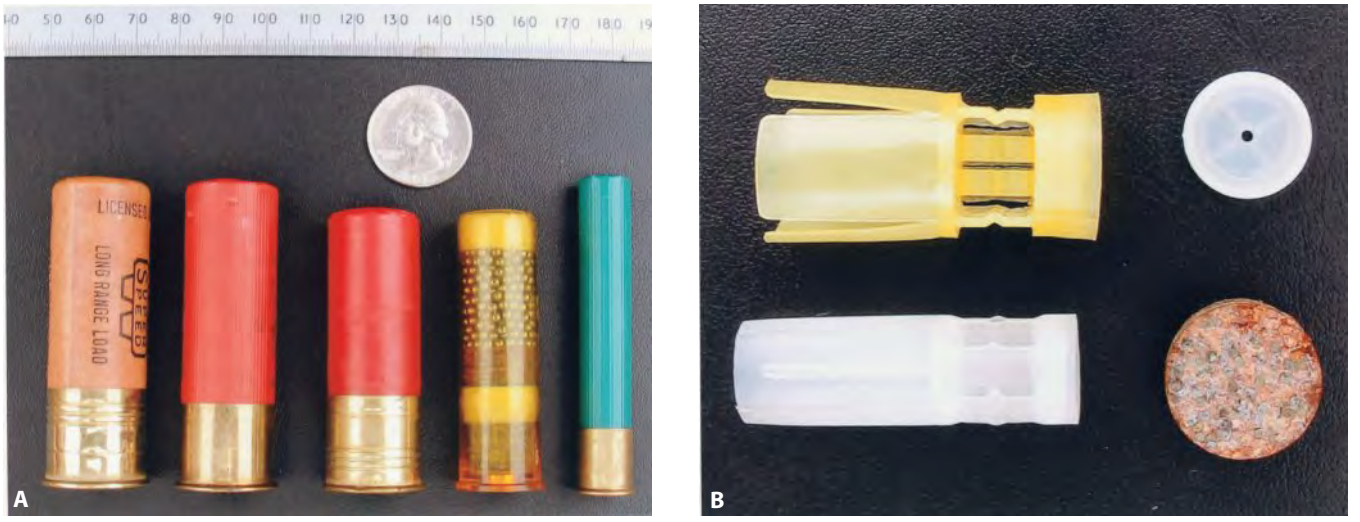


FIGURE 25-2. **A,** Representative shotgun shells. **From left to right:** 10 gauge; 3 in. 12 gauge; $2^{3}/_{4}$ in. 12 gauge; 20 gauge "Demonstrator" shell with shot, wadding, and powder visible; .410 gauge. **B,** Plastic and felt shotgun wadding.

(0.83 cm), with 9 to 15 pellets in a 12-gauge shell. Shells come in different lengths within the same gauge as well. For example, a 12-gauge shell may be a $2^{3}/_{4}$-inch (6.99 cm) or 3-inch (7.62 cm) shell. Longer shells hold a larger charge of powder and shot, which can be used for larger game or game at further distances. As a general rule, longer-barreled shotguns and those with a full *choke* (a constriction of the end of the barrel) keep the pellets in a tighter pattern over longer distances. Finally, some shotguns may be modified with rifled barrels to fire shells containing a solid lead projectile referred to as a *slug*. Shot is usually selected based on the size of game. *Buckshot* refers to larger pellets meant for large game or human targets; it is particularly devastating because its impact is similar to multiple low- to medium-velocity handgun wounds, depending on the range.[20] It is also important to note the different makeup of a shotgun shell (see Figure 25-2A). The pellets are typically separated from the propellant by wadding that helps to contain and transfer the power of the charge to the pellets. This partition can be made of felt or plastic and may be found embedded in close-range wounds (see Figure 25-2B).

Most handguns and rifles have barrels with internal grooves referred to as *rifling* that impart a spin to the bullet. Rifling is made up of lands and grooves, which leave distinctive marks on the fired projectile. The spin imparted by rifling keeps the projectile stable in flight over longer distances. In early firearms that were loaded from the muzzle (muzzle-loaders), the tight fit between the bullet and the barrel that resulted from rifling significantly slowed loading. For this reason, most early military weapons were smooth bore. Sacrifices in long-range accuracy were a tradeoff for rapid rates of fire. This obstacle was overcome in 1847 by Captain Minié, who developed a projectile with a hollow conical base that loaded easily but expanded for a tight fit when the propellant enlarged behind it (Figure 25-3). Ultimately, breech-loading weapons, in which a self-contained round enclosing the ignition system (primer), propellant, and projectile was loaded from the beginning of the barrel instead of the end, overcame these difficulties. The development of rifling, however, allowed high-velocity projectiles that would remain stable in flight over long distances. Eventually, all projectiles become unstable in flight because the center of gravity lies well behind the center of resistance (the bullet tip) causing them to take on various motions during flight.

Oscillation around the long axis of the bullet is referred to as *yaw*. Rifling seeks to stabilize yaw but imparts its own motion, referred to as *precession* (circular yawing), around the center of gravity, creating a decreasing spiral and nutation, which is a rotational movement in small circles.[21] These motions occur during flight through air. Bullets may be modified in an attempt to decrease these motions in flight; an example is a "boat tail" bullet, intended to be stable over longer distances. Upon encountering a denser substance such as tissue, the projectile immediately starts tumbling. Increased tumbling causes more tissue wounding because it presents a larger surface area. Bullets have undergone a variety of



FIGURE 25-3. **Left,** Early round projectile and Minié ball with expanding base. **Right,** Modern full-jacketed and soft point rounds with "boat tail" to improve flight characteristics.

modifications in an attempt to control these motions and increase wounding and stopping power.

The simplest and earliest projectile was a stone or lead ball (see Figure 25-3). Over time, the projectile evolved to the conical-shaped Minié ball. The lead conical bullet remains in use. Modifications are made based on intended use. In general, military rounds are restricted by the Hague convention (1899) to the full-metal jacket. Fragmentation rounds have been outlawed, although some countries continue to use flechette rounds (designed to fire small metal spikes or fragments). Simple lead bullets referred to as *wadcutters* are inexpensive and often used as target rounds. Jacketed bullets with exposed lead tips (soft points or dum dum bullets) are designed to expand on impact for maximum tissue destruction (maximum permanent cavity) and are typically designed for hunting. A variety of modifications have been made to handgun bullets in an attempt to make up for their lack of velocity and to increase wounding (Figure 25-4). Because of their low velocity, handgun bullets have difficulty expanding reliably in tissue. Attempts to overcome this have centered on the creation of bullets with various open ends, so-called hollow points (see Figure 25-4). Some of these are partially covered with a metal jacket in attempt to control expansion. As noted earlier, despite manufacturers' claims to the contrary, reliable expansion is difficult to obtain in low-velocity rounds. Some manufacturers have created +P ammunition, which contains different gunpowder to obtain a higher velocity. Also, some bullets are designed to explode on impact by incorporating an explosive into a hollow cavity in the bullet (devastator rounds).

The ignition of most cartridges is accomplished by a firing pin striking a primer. Some cartridges use a primer built into the case and are referred to as *rimfire* because the firing pin strikes the edge of the cartridge rim to discharge the propellant.

Mention should be made of other projectiles that have been associated with injury. Modern airguns can achieve velocities sufficient to cause tissue damage. The proliferation of paint-ball guns has led to an increase in the number of ophthalmologic injuries.[22] Finally, unorthodox bullets such as wooden, rubber, and "bean bag" projectiles are being used increasingly in crowd-control situations. Although meant to be nonlethal methods of deterrence, these rounds can cause significant tissue damage and even death. They are frequently associated with facial fractures.[23]



FIGURE 25-4. Full-jacketed bullet compared with various hollow-point rounds designed to aid the expansion of lower-velocity bullets.

CLASSIFICATION SCHEMES

Classification of traumatic injuries is helpful in guiding treatment and, more important, tracking outcomes for various treatment modalities. A number of trauma scoring systems and classifications for various injuries have been developed and validated. Similarly, attempts have been made to classify GSWs to assist the surgeon in selecting appropriate management strategies. Many of these classification schemes were developed on the battlefield. Dissimilarities between civilian and military gunshot injuries, such as ammunition, wounding potential of military weapons, and treatment objectives, make these classification schemes of little use in the urban trauma center, which most commonly deals with low- to medium-velocity handgun injuries.[24,25]

Trauma systems have attempted to incorporate gunshot injuries into existing classification and trauma scoring systems. Unfortunately, current schemes have not proved beneficial in guiding treatment and judging outcomes to develop ideal approaches. Attempts to distinguish GSWs as low or high velocity have suffered from the shortcomings noted previously. In addition, velocity is less critical than bullet type, mass, distance to target, and specific vital organs involved because most civilian injuries are caused by low- or medium-velocity weapons.

One of the earliest and simplest classification schemes classifies GSWs as *nonpenetrating* (grazing or blast wound), *penetrating* (bullet does not exit), *perforating* (in and out), and *avulsive.* The International Committee of the Red Cross introduced the armed conflict classification system to improve information gathering and communication regarding war wounds. Because of the diversity of battlefield weaponry, by necessity the system ignores weapon type and instead concentrates on wound severity in terms of tissue damage and anatomic structures involved.[24,25] Gugala and Lindsey[25] suggested a civilian gunshot injury classification scheme. It takes into account energy (high or low), involvement of vital structures (neural and vascular), wound type (nonpenetrating, penetrating, perforating), fracture (intra-articular and extra-articular), and contamination. Primarily used in orthopedics, its usefulness in gunshot injuries to the head and neck is limited.

SHOTGUN WOUNDS

Because of their unique ballistic profile, shotgun injuries are often classified based on the distance to the target. Shotgun pellets have significant aerodynamic resistance and give up

substantial amounts of kinetic energy during flight. In type I shotgun injuries (<5 m), the pellets strike the target as a single mass, resulting in massive kinetic energy transfer, tissue avulsion, and a high mortality rate (85–90%). Patients that survive suicide attempts with shotguns typically survive because, in an attempt to reach the trigger with the muzzle under the chin or in the mouth, the head is hyperextended, which causes the pellets to create devastating injuries to the face but avoid the cranium. Fragments of paper or plastic wadding may be found in the wound. Type II injuries (5–12 m) usually result in much less tissue destruction. At these distances, there is significant dispersal of the pellets and loss of energy. Penetration may occur through deep fascia, but fractures are rare. Ocular injuries can occur as well as embolization of lead pellets, but mortality is less (15–20%). At distances greater than 12 m (type III), usually only the skin is penetrated and mortality is rare (0–5%).[26,27] Because specific information on shooting distances is not often available to the clinician, a system was suggested that evaluated the maximum distance of pellet scatter. Type I injuries had greater than 25 cm of pellet scatter. Type II injuries had 10 to 25 cm of scatter. Type III injuries had less than 10 cm of scatter and would roughly correspond to a type I injury in the classification of Sherman and Parrish.[26] This classification scheme was developed and applied to abdominal shotgun wounds in an attempt to guide therapy.[28] Again, the difficulty lies in applying this scheme, or any scheme, universally to GSWs involving different anatomic sites and weapon types.

It should be noted that rifle and shotgun injuries, although rare in assaults, are frequently encountered in attempted suicide patients. A characteristic wound profile is seen because of the head position assumed when the patient places the barrel of the weapon in the mouth or under the chin and subsequently hyperextends to reach the trigger. Characteristic powder burns are seen at the entrance wound (Figure 25-5). The face frequently takes the full effect of the blast, whereas lethal intracranial involvement is avoided.[29] If a high-energy weapon such as a shotgun or rifle is used, the injury can be devastating with significant tissue loss.

Although classification schemes can serve useful purposes in research as well as clinical practice, strict adherence to treatment algorithms based on wound classification can lead to mismanagement. Importantly, information regarding types of firearm and other details of the shooting are frequently not available, and clinical assessment of the wound remains the most reliable method for determining treatment approaches.

MANAGEMENT

General Principles

On admission, victims of gunshot injuries are best managed by standard advanced trauma life support (ATLS) protocols. Even seemingly innocuous wounds deserve attention, given the erratic nature of the wounds. Specific attention must be given to the possibility of multiple injuries; it is imperative to thoroughly inspect the patient for multiple entrance and exit wounds. Visually disturbing but non–life-threatening facial gunshot injuries can distract medical personnel from other more subtle lethal injuries such as a penetrating thoracic wound that entered through the back. Ophthalmologic and neurosurgical consultations are obtained when indicated. Approximately 17% of patients with a GSW to the face have associated brain injuries, and 8% have associated cervical spine injuries.[14,30] Eye injuries are present in approximately 13% (Figure 25-6).[30] Certain considerations for gunshot injuries should be emphasized.

Airway

Loss of the airway is the single most likely cause of death in an isolated GSW to the face. When confronted with a patient with a facial GSW, surgeons should have a low threshold for establishing a definitive airway through intubation or a surgical airway if intubation is not possible. Intubation either in the field or in the emergency department is required in 25% to 36% of patients. Wounds involving the mandible have the highest rate of intubation (37–53%), followed by those of

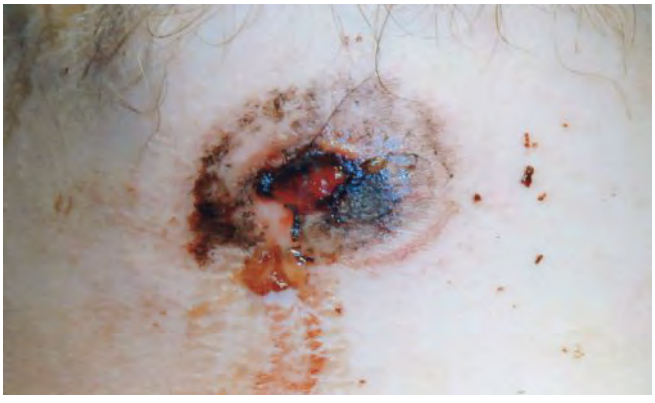


FIGURE 25-5. Submental entrance wound with powder burns characteristic of a suicide attempt by placing a gun under the chin.

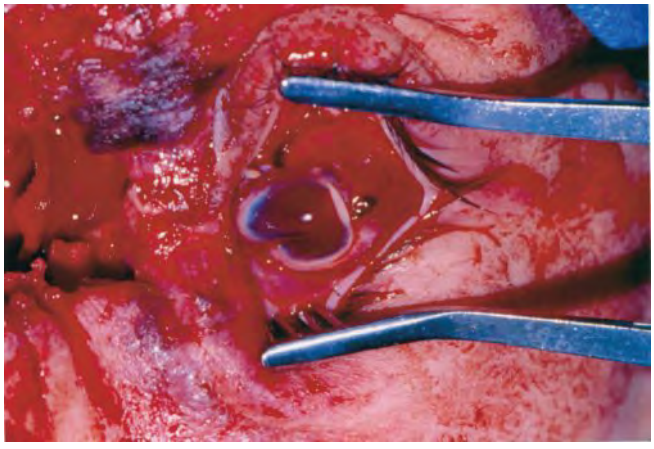


FIGURE 25-6. Perforated globe with shotgun pellet.

SECTION 3

the midface (18–36%).[30–33] Excluding patients that require airway control for associated brain injuries, Demetriades and coworkers[14] found that 17.4% of patients required urgent airway control for facial injuries. Gunshot injuries to the neck may result in tracheal damage and require an emergent surgical airway (cricothyroidotomy). Intubation with fiberoptic assistance is possible, but paralytics should be avoided owing to the risk of expanding hematomas or massive edema. Cricothyroidotomy or an awake tracheostomy is more appropriate in this setting. The need to convert an intubated airway to a tracheostomy depends on several factors. Tracheostomy can make repair of injuries involving the mandible and midface easier. Patients who will require multiple return trips to the operating room for wound débridements and "second looks" will benefit from the decreased risk of multiple intubations. Delayed swelling can be anticipated with trauma to the upper aerodigestive tract including the tongue (Figure 25-7A); this may influence the decision to proceed with tracheostomy. Associated tracheal injuries are another indication for tracheostomy (see Figure 25-7B). Lastly, multiple system injuries with anticipated long-term ventilation are an indication for early tracheostomy. Most experienced surgeons would agree that it is rare to regret having performed a tracheostomy, but tragic to regret not performing one.

Hemorrhage Control

Life-threatening hemorrhage is unusual in civilian gunshot injuries. Low-velocity handgun injuries typically do not involve the great vessels. Demetriades and coworkers[14] in Los Angeles reported only 7.5% of patients with isolated GSWs to the face to be in shock upon admission (systolic blood pressure < 90 mmHg). In their report, 70 patients (28.3% of the total) required angiography, and 10 of these required embolization. Overall, the literature reports angiography in 17% to 63% of patients with a GSW to the face, with positive findings in 15% to 51%. Indications for angiography include expanding hematoma and bleeding that persists despite local measures.[31,34,35] The most commonly involved vessels in these cases were the maxillary and facial arteries. Gunshot injuries associated with high-velocity weapons or fractures, however, can result in significant blood loss. Initial attempts to control hemorrhage in the emergency department center on direct pressure and packing. Blind clamping should be avoided because of the attendant risk of damage to other structures (Figure 25-8). Standard methods for epistaxis control such as Foley catheters or specially designed balloon catheters will control most midface bleeding (Figure 25-9). In cases of mandible fractures, temporary reduction of the fracture may be required. Penetrating injuries can require that the surgeon make difficult choices. Injuries at the skull base may benefit from angiography and embolization (see "Penetrating Neck Injuries," later). Unfortunately, the time necessary to mobilize the angiography suite often makes this an impossible choice for the unstable trauma patient in the middle of the night. For this reason, control of life-threatening hemorrhage is typically best performed in the operating room. Ligation of multiple vessels is required. "Tying off" the carotid is usually ineffectual and dangerous; an attempt should be made to control specific vessels. Lacerations of the internal jugular artery are best controlled with ligation or repair (Figure 25-10). Packing and reduction of fractures should be performed to control bleeding from the midface if possible. There is possibility of late pseudoaneurysm formation and delayed hemorrhage, and selective angiography should be performed as indicated. In addition, the possibility of bullet or fragment embolization should be considered.

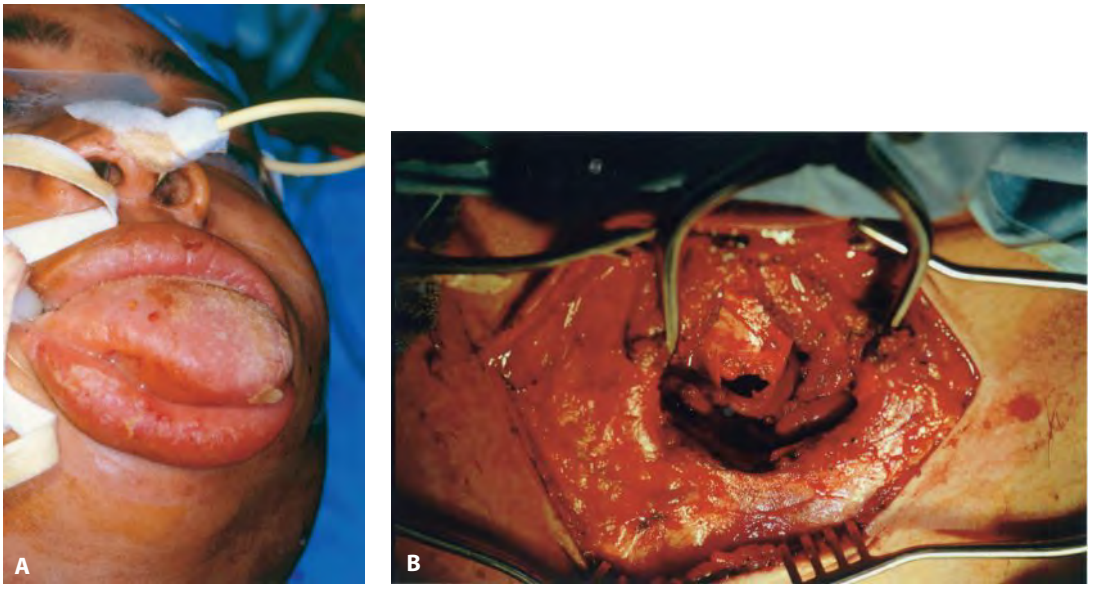


FIGURE 25-7. **A,** Massive late tongue edema. **B,** Tracheal injury from a gunshot wound. (**A** and **B,** Courtesy of Eric J. Dierks, MD, Portland, OR.)



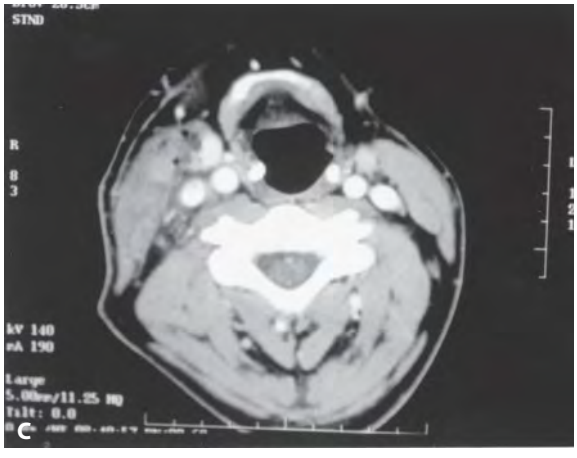
FIGURE 25-8. Blind clamping in the emergency department management of a facial gunshot wound.



FIGURE 25-9. Nasal packing with anterior and posterior balloon catheters.







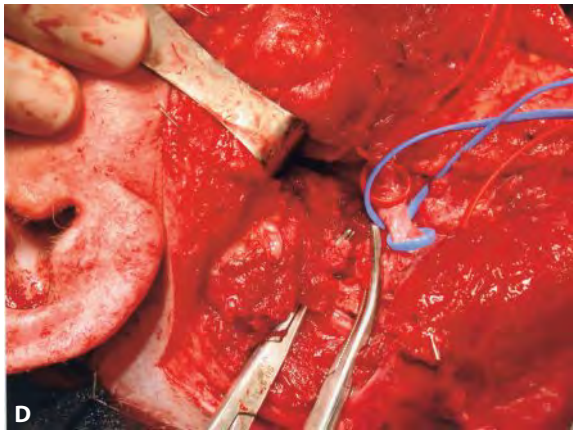


FIGURE 25-10. **A,** Gunshot wound to zone II associated with a mandible fracture. **B,** Plain film demonstrates the bullet in zone II. **C,** Computed tomographic angiography failing to demonstrate tamponaded laceration in the internal jugular vein but showing subcutaneous air and edema of neck injury. **D,** Lacerated internal jugular vein (clamped) found on neck exploration.

Penetrating Neck Injuries

GSWs involving the face may be associated with an entrance or exit wound in the neck, which is divided into three zones originally described by Monson and colleagues from Cook County Hospital[36]:

- Zone I is most commonly defined as the area from the clavicles to the cricoid cartilage. It contains the inferior aspect of the trachea and esophagus along with the major vessels of the thoracic inlet: the common carotid arteries, thyrocervical trunk, internal jugular veins, brachiocephalic trunk, subclavian arteries and veins, thoracic duct, thyroid gland, and spinal cord. Risk of injury to the great vessels is common in this area, and consequently, injuries to zone I carry a high mortality rate (~12%). Some authors place the junction of zones I and II at the cricoid cartilage, whereas others define it as being at the top of the clavicles.
- Zone II represents the area from the cricoid cartilage to the angle of the mandible. It contains the common carotid arteries, internal and external carotid arteries, internal jugular veins, larynx, hypopharynx, and cranial nerves X, XI, and XII. It is the largest area and, therefore, the zone most commonly involved in penetrating neck trauma.
- Zone III spans the region from the skull base to the angle of the mandible. It contains the carotid arteries, the internal jugular veins, and the pharynx along with multiple cranial nerves exiting the skull base. It should be appreciated that GSWs that involve mandibular fractures are accompanied by injuries to zone III.

Van As and coworkers[37] reported on 116 patients shot in the neck in South Africa. Of these, 70 suffered a direct hit to the neck; in 46 patients, the bullet traversed the face or chest first. Of the 116 patients, 85 suffered some vascular injury, although most were minor branches, 61 had some injury to the airway, and 32 had an injury to the pharynx or esophagus.[37] Many patients had more than one injury. Management strategies for penetrating neck injuries are typically based on the zone(s) involved.[38,39] GSWs to the head and neck frequently involve projectiles that traverse or involve more than one zone. For this reason, surgeons may have to modify management plans based on the situation at hand. Although a complete discussion of penetrating neck trauma is beyond the scope of this chapter, general principles should be understood by surgeons managing facial gunshot injuries.

Initially, the patient's stability from an airway and hemodynamic status guides the decision making for penetrating neck injuries (Figure 25-11). In the stable patient, a complete examination is part of the secondary survey of ATLS. Signs of tracheal injury, such as subcutaneous emphysema, stridor, hoarseness, dysphonia, or hemoptysis, require urgent intervention. Hard signs of vascular injury, such as expanding

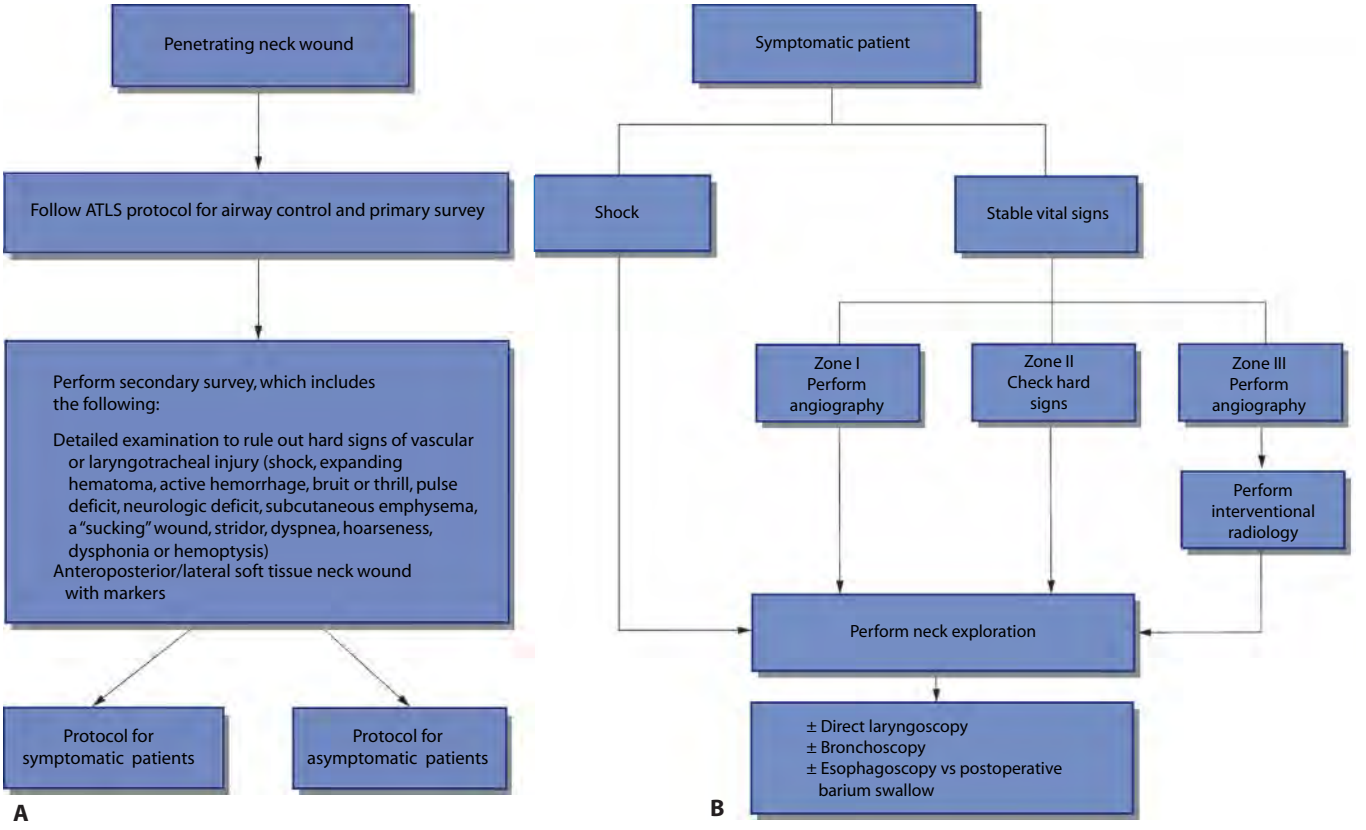


FIGURE 25-11. **A,** Initial decision tree for penetrating neck trauma. **B,** Management of the symptomatic patient with a penetrating neck wound. ATLS = advanced trauma life support.

hematoma and pulse or neurologic deficit, also signal the need for urgent management. In the absence of urgent management needs, the surgeon must rule out occult injuries based on the zones involved.

Injuries to zone I can be associated with significant bleeding because of the large vessels in this area. This is especially true with regard to injuries caused by high-energy weapons. Although serving to protect the vessels, the clavicles are a hindrance to the application of direct pressure to the area and to rapid surgical exposure. In the stable patient, most surgeons advocate routine angiography and an evaluation of the esophagus via rigid esophagoscopy or a barium swallow. The choice between barium swallow and esophagoscopy varies according to the surgeon's preference because both are reasonably accurate at diagnosing injury (90% and 86%, respectively). In addition, there is some controversy regarding the appropriate contrast media. Although meglumine diatrizoate causes less inflammatory response than does barium when it extravasates into tissues owing to an esophageal perforation, it results in a severe chemical pneumonitis if aspirated. For this reason, barium should be used if there is any impairment to the gag and cough reflexes; if there is a leak, early operative intervention allows it to be washed out during surgery. Penetrating injuries to the left neck, and rarely to the right, can result in a chyle leak (Figure 25-12). The surgeon should take care to exclude this at the initial exploration, if possible, and to repair it by oversewing the duct with local tissues. It is useful to have the anesthesiologist apply positive pressure and to place the patient in Trendelenburg's position. Delayed management is much more difficult after the tissues have been exposed to chyle. Conservative management with a diet of medium-chain triglycerides, which are not carried by the gut lymphatics, and drainage should be attempted initially if the leak presents in the postoperative setting. Exploration is indicated for leaks of greater than 400 to 500 mL/day for a week.

Penetrating injuries to zone II are the most common and are most amenable to surgical exploration, if warranted. For asymptomatic patients, computed tomographic angiography is becoming an important tool for screening and can assist in determining whether operative exploration is warranted.[40] Patients can undergo serial examinations over 24 hours if the angiography results are negative. Computed tomographic angiography is faster and less invasive than angiography but is of lower specificity. It should also be noted that injuries that have "tamponaded" themselves can be missed on either (see Figure 25-10C). Some surgeons recommend the use of a barium swallow or rigid esophagoscopy, whereas others recommend observation only if the index of suspicion for injury is low, as with wounds from low-energy guns. If patients have associated mandible fractures, the neck can be explored while the mandible fractures are exposed for fixation.

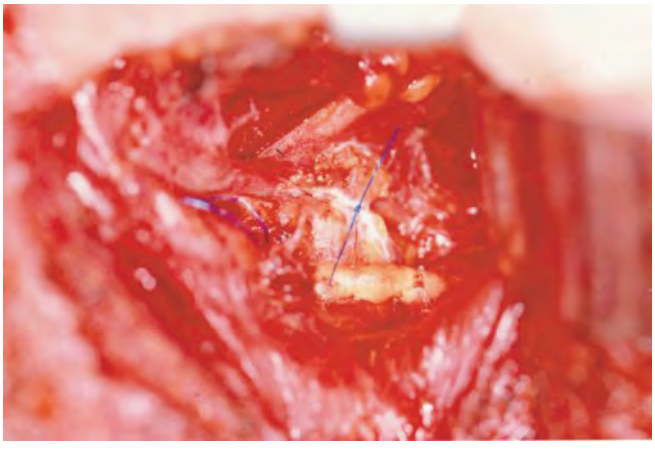


FIGURE 25-12. Chyle leak after penetrating injury to zone I of neck oversewn with nonabsorbable suture and covered with a flap from the sternocleidomastoid muscle.

Imaging is required in zone III injuries if the patient is stable. Diagnosis of vascular injuries at the skull base typically requires angiography, which can also allow intervention if indicated. Injuries to zone III are rarely amenable to surgical intervention.

Overall, angiography remains the gold standard for exploration of vascular injuries of the neck. In Van As and coworkers' report,[37] 89 patients underwent angiography for GSWs to the neck; results were positive in 12 patients, with most lesions occurring in the common carotid followed by the internal and external carotids (3 cases each), the vertebral artery (2 cases), and the subclavian artery (1 case). Currently, ultrasonography is gaining popularity as a rapid noninvasive technique for the evaluation of a variety of traumatic injuries in the emergency department. Ginzburg and associatues[41] investigated the usefulness of duplex ultrasonography to evaluate vascular injuries in a double-blind study using angiography as a control. They reported a 100% true-negative rate, 100% sensitivity, and 85% specificity in detection of arterial injury. Ultrasonography will most likely continue to grow in popularity as a screening tool because of its cost and the speed at which it can be performed.[41] Further improvements in noninvasive vascular evaluation techniques, such as helical computed tomographic angiography and ultrasonography, will reduce the number of patients undergoing traditional angiography and improve patient selection for nonoperative management.

Nutrition

The majority of civilian GSWs affect young, healthy males. Nutritional status becomes an issue only in patients whose injuries preclude oral alimentation for an extended period (>4 or 5 days). Feeding via nasogastric intubation allows bypass of the oral cavity and improved hygiene in the early days after injury. Consideration should be given to percutaneous endoscopic gastrostomy if long-term bypass of the oral cavity is necessary, the patient will be unable to eat, or the patient has a preexisting nutritional deficit.

Imaging

Following the ATLS protocol, standard cervical spine and chest radiographs should be obtained. These can be valuable for visualizing the bullet fragments and in gaining some

insight into the path of the bullet (see Figure 25-10B). It is important to recall, however, that projectiles rarely follow a straight path once they enter tissue.

The ability to obtain accurate three-dimensional images in a rapid fashion has been one of the most important advances in dealing with gunshot injuries to the face. Spiral computed tomography (CT) combined with three-dimensional reconstructions allows the surgeon an unparalleled view of the extent of damage to the maxillofacial skeleton, which lies beneath the skin (Figures 25-13 and 25-14). Although it does not accurately demonstrate the amount of soft tissue damage, clinical inspection combined with three-dimensional imaging allows an accurate assessment. As discussed previously, the importance of temporary cavitation and emphasis on the amount of devitalized tissue distant from the primary wound has probably been overstated in the past. Computed tomographic angiography can also be useful in certain situations for evaluating vascular damage, especially in cases of penetrating neck injuries. It should be remembered, however, that angiography remains the gold standard to evaluate the vasculature. Also, angiography, if available, allows the ability to intervene with embolization of active bleeding vessels that are difficult to approach surgically (see "Penetrating Neck Injuries" earlier). Patients who are not sufficiently stable for imaging should be stabilized in the operating room, and definitive repair should be deferred until appropriate imaging can be obtained.

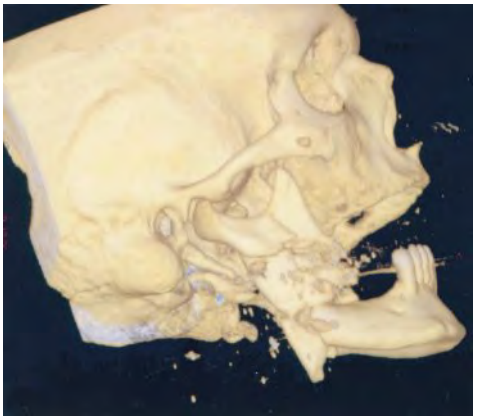
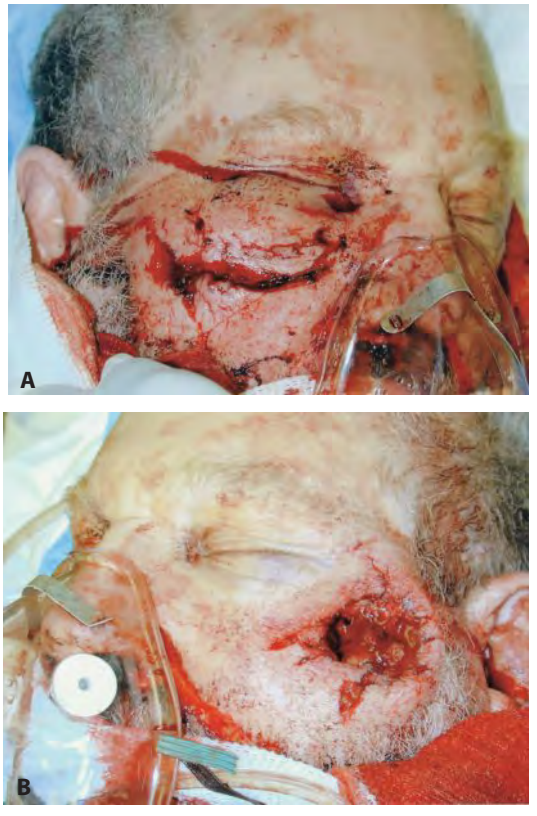


FIGURE 25-13. Three-dimensional computed tomography scan demonstrates fragmentation of the mandible resulting from a gunshot wound. (Courtesy of James R. Koehler, MD, Birmingham, AL.)

Operative Procedure

Paralleling the evolution of firearms has been development in the management of gunshot injuries to the head and neck.



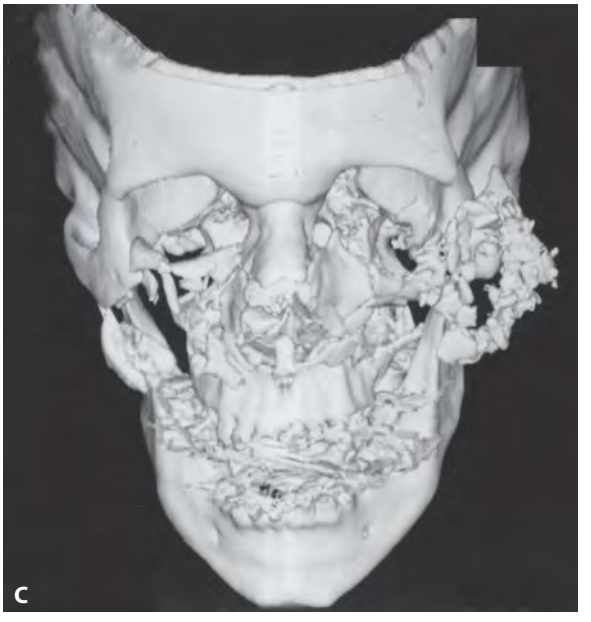


FIGURE 25-14. **A,** High-velocity entrance wound of the right cheek. **B,** High-velocity exit wound of left cheek. **C,** Three-dimensional computed tomography scan demonstrates extensive bony comminution associated with a high-velocity gunshot wound. (**A–C,** Courtesy of James R. Koehler, MD, Birmingham, AL.)

The earliest surgeons dealing with gunshot injuries blamed complications on gunpowder that would later be ascribed to contamination and infections. The mystique that surrounded gunshot injuries persists in some ways to modern times in surgical dogma that is passed down. During World War I, high-energy close-range GSWs and shrapnel wounds to the face necessitated the development of maxillofacial surgery. Kazanjian and Converse[42] described their approach to GSWs as three phases consisting of initial débridement and suturing, immobilization of bony fragments with splints and ligatures, and finally, reconstruction after healing of the soft tissue. Many of the principles developed at that time persist today, with surgeons advocating a phased approach with delayed closure of wounds, débridement of tissue, and secondary reconstruction.[43] Many surgeons still advocate closed reduction and division of care into early (first 10 days), intermediate (10–60 days), and late (>60 days) phases.[30] The different nature of civilian GSWs and improved management techniques have led to a reappraisal of staged approaches, and current management principles should more properly be considered a continuum that is based on the wound and patient profile.[3,13,44,45] The successful application of rigid fixation principles to blunt traumatic injuries resulted in incorporation of these techniques to gunshot injuries. Early surgeons understood the importance of immobilization on the healing of GSWs but lacked the ability to truly immobilize bony structures of the face. The development of rigid fixation techniques and their application to GSWs was an important advance. Early concerns regarding placement of hardware into contaminated sites proved unfounded. By allowing the early stabilization of bone segments, percolation of contaminated oral fluids was prevented, primary bone healing was made possible, and the effects of scar contracture were minimized. This has led most surgeons to advocate early definitive repair of the majority of civilian GSWs, which generally are inflicted with low-velocity weapons.

An operative plan for a gunshot injury to the face is best formulated after characterization of the wound as low or high energy (Figures 25-15 and 25-16). Manson introduced a concept for evaluation of facial GSWs that consisted of four components: soft tissue injury, bone injury, soft tissues loss (true avulsion), and bone loss.[46] After evaluation of the wound, a decision is made regarding early definitive repair versus the need for delayed repair. High-energy GSW and blast injuries to the face are more likely to benefit from serial débridements and delayed closure, especially in the military arena in which delays in medical care are more common. Will and coworkers[2,3] have offered excellent reviews and a treatment protocol based on their experiences in Iraq. Although primarily dealing with higher-energy wounds encountered in a military environment, parallels to high-energy GSWs encountered in the civilian arena are easily made. Their protocol places emphasis on serial débridement of soft tissue injuries with delayed closure after soft tissues have stabilized. This was found to be expecially important in wounds received from improvised explosive devices with their associated contamination.[2,3] The majority of civilian GSWs resulting from assaults can be managed with early definitive repair because these injuries usually result in injury to the soft tissue and bone but rarely loss of these tissues. Impressive soft tissue injuries are usually not avulsive, and most can be closed primarily (see Figure 25-15). Extensive débridement of soft tissue is not indicated. Wound debris should be removed, and wounds should be lavaged with normal saline. Antibiotic solutions such as saline and bacitracin (50,000 U/L) have not been shown to be more effective than normal saline but are still popular. A pulsating irrigator is useful to mechanically agitate debris from the tissue. Obvious devitalized and loose teeth should be removed. Fractures are reduced and fixed rigidly. Teeth should be maintained if possible to aid in restoration of occlusion and proper jaw relations. Drains are often indicated; whether closed suction or Penrose is used depends on the wound. Pressure dressings can also be used to minimize dead space. In cases of true soft tissue avulsion, a decision must be made regarding whether primary flaps or grafting is indicated. In wounds that are relatively clean, local flaps and skin grafts may be appropriate. In grossly contaminated wounds, delayed closure or



FIGURE 25-15. **A,** Gunshot wound resulting from the placement of a low-velocity handgun into the mouth. **B,** Initial closure demonstrates no true tissue loss. **C,** Three-month postoperative photograph demonstrates minimal residual deformity following closure. The facial nerve is intact.

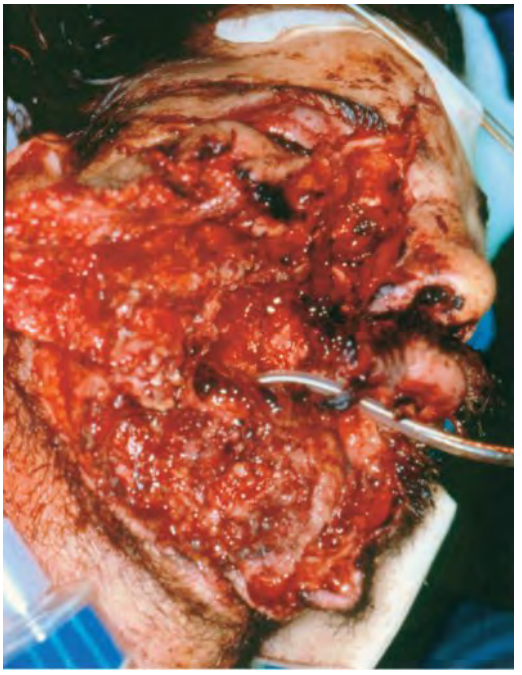


FIGURE 25-16. Extensive wound resulting from a high-velocity weapon.

grafting may be necessary. Closing mucosa to skin can be a useful technique, but many cases can be managed with dressing changes and incorporation of an early flap procedure. Free tissue transfer, although useful, should be delayed until the initial phase of wound healing, when its accompanying vascular spasm and attendant hypercoagulable state have decreased.

In wounds with extensive soft and hard tissue damage and true loss of soft and hard tissue, an approach using early stabilization of bone fragments with maxillomandibular fixation, external fixation, or internal fixation with reconstruction plates combined with conservative management of soft tissue is indicated. In this era of rigid internal fixation, the utility of maxillomandibular fixation should not be overlooked.[14,30] In addition, external fixation devices are still useful in select cases. Second-look operations with conservative wound washouts and débridement of only obviously dead tissue, which have gained popularity in orthopedics, have great utility in injuries to the maxillofacial skeleton. Second débridements should be performed 24 to 48 hours after the initial surgery. This allows for the maintenance of tissue considered "borderline," which can be excised if it truly becomes devitalized. Skin grafts can be used as permanent or temporary replacement for missing tissue to reduce deformity from scar contracture. Once the soft tissues have stabilized, a decision can be made regarding early replacement of lost tissues with free tissue transfer or delayed reconstruction. In general, earlier repair leads to improved outcomes with less scar contracture and resultant deformity. Some authors have advocated the early use of free tissue transfer with multiple skin paddles when two or more facial units are involved to shorten the initial hospital stay.[47] Bone grafts at the time of initial surgery may be indicated in the midface (see later). Again, management strategies should be considered a continuum that is modified as necessary rather than strict distinct stages.

Contamination

It should be remembered that projectiles from firearms are not sterile. This fact is well known to those who have dipped their bullets in feces before assassination attempts but lost on clinicians who have taught that GSWs are indeed sterile. The heat generated by the discharge of the propellant as well as the friction between the bullet and the barrel is not sufficient to sterilize the bullet.[48,49] Contamination can occur from the bullet and also from skin flora and foreign bodies (clothing) carried into the wound. Historically, streptococcal bacteremia was the most important cause of death on the battlefield in the preantibiotic era.[50] Wounds in which the bullet traverses the aerodigestive tract or paranasal sinuses are at particular risk. Devitalized tissue and vascular congestion lead to an ideal environment for bacterial growth. Prophylactic coverage with broad-spectrum antibiotics, typically a second-generation cephalosporin, and tetanus prophylaxis, when indicated, should be initiated in all GSWs. Extensive surgical débridement to prevent infection is rarely indicated in wounds consistent with low-velocity projectiles.

Removal of projectiles, a well-worn tradition in Hollywood, is less commonly indicated in reality. The need for the removal of bullets must be balanced against the real risk of increasing damage. Lead toxicity is a rare complication that does not typically justify the routine removal of bullet fragments.[51] Removal of intra-articular bullet fragments should be considered when the increased risk of lead toxicity is associated with fragments within joint spaces and the potential for long-term deterioration of the joint.[52] Consideration may be given to the removal of brass- or copper-jacketed bullets that are in close proximity to central or major peripheral nerves because of potential neurotoxicity.[53,54] Finally, bullet removal may be indicated in cases in which there is risk of bullet embolization.[55]

It is important to remember that bullet fragments are potential evidence and an appropriate chain of custody is required. Most hospitals have a protocol in place to ensure that this chain is unbroken from the time the bullets are retrieved to when they are logged in as evidence. This usually involves a police officer or other designee taking direct possession of the bullet or fragments in the operating room or nearby. Documentation of injuries with photographs can aid in reconstructing the events leading to the injury and recording where fragments were retrieved. Because some assaults have injury patterns similar to suicides, it is important to consider this chain of custody because subsequent investigations may reveal that an apparent suicide was actually an assault.[56]

SPECIALIZED STRUCTURES

Facial Nerve

Damage to the facial nerve is present in only 3% to 6% of civilian GSWs to the face.[14,30] This is most likely because low-energy weapons are involved in most of these cases. However, such damage is not uncommon in injuries inflicted by higher-velocity firearms. Careful documentation at the earliest possible opportunity is important. If a functioning nerve becomes nonfunctional secondary to swelling, the surgeon can be reasonably confident that function will return. Obvious transection of the nerve requires repair. In heavily contaminated wounds, repair should be delayed for 48 to 72 hours, given the possibility that grafts will be required to span damaged segments. Beyond 72 hours, distal branches of the facial nerve will not respond to a nerve stimulator, making their identification difficult. If possible, tagging the branches with suture at the initial surgery is invaluable. Extensive damage to the proximal nerve may require a temporal bone dissection to identify a viable proximal nerve for grafting. Injuries distal to a line dropped vertically from the lateral canthus (zone of arborization) do not typically require repair because of the multiple interconnections distal to this line and the reasonable expectation of return of function, even if the nerve is temporarily nonfunctioning (see Figure 25-15).

Salivary Ducts

Transected salivary ducts may be repaired or ligated depending on the amount of damage. The parotid duct can be repaired over an intravenous catheter or polymeric silicone tubing, which is then sutured to the buccal mucosa. It is best to avoid bringing the tubing out of the mouth because of the tendency for it to be dislodged. In injuries that penetrate the parotid-masseteric fascia, there is a potential for development of a sialocele or fistula. These typically resolve with drainage and pressure dressings. Aspiration may be required multiple times, and rarely, antisialagogues may be indicated. In addition, removal of any associated foreign bodies may be necessary to resolve the fistula and hasten healing. Dermal grafts can be used at the time of repair (Figure 25-17).

CONTROVERSIES: DELAYED VERSUS EARLY MANAGEMENT AND CLOSED VERSUS OPEN FRACTURE MANAGEMENT

Proponents exist both for closed management of fractures with delayed reconstruction and aggressive early management with open reduction of fractures and replacement of missing tissue as soon as possible. Both groups point to failures and shortcomings of the other to justify their approach. Advocates of delayed repair point to a higher incidence of infection and to benefits of closed treatment.[57] Those who advocate more aggressive primary management report improved functional and aesthetic outcomes.[58] Because neither approach is likely to ever be subjected to a randomized trial measuring outcomes, surgeons must base their treatment decisions on a critical review of the literature and their own experience. As with most arguments in surgical science, the truth most likely lies somewhere in the middle. Certainly, the advantages of aggressive early management are appealing (Figure 25-18). Early return to function and decreased numbers of revision surgeries are laudable goals. Currently, techniques involving open reduction and fixation of fractures resulting from GSWs seem to be gaining in popularity, and patients are less likely to be treated with closed reduction. Given that most of these injuries are low energy, this is acceptable. The main disadvantage of open reduction is infection, which primarily affects the mandible. The reported rate of infection with open reduction and fixation of mandible fractures resulting from a gunshot is around 16% to 17%.[59] However, rigid fixation can frequently be maintained in the event of wound problems and still serves to stabilize mandibular segments. Surgeons should avoid the application of a set protocol to every GSW situation and should instead rely on a careful appraisal of the wound and decide on the amount of early repair that is indicated.

Bone Grafting

Bone grafts are frequently required in the management of GSWs to the face, whether for replacement of true loss of bone (avulsive injuries) or in cases in which comminuted and misplaced fragments need to be replaced or reinforced. Reconstruction with bone grafts gained popularity in World

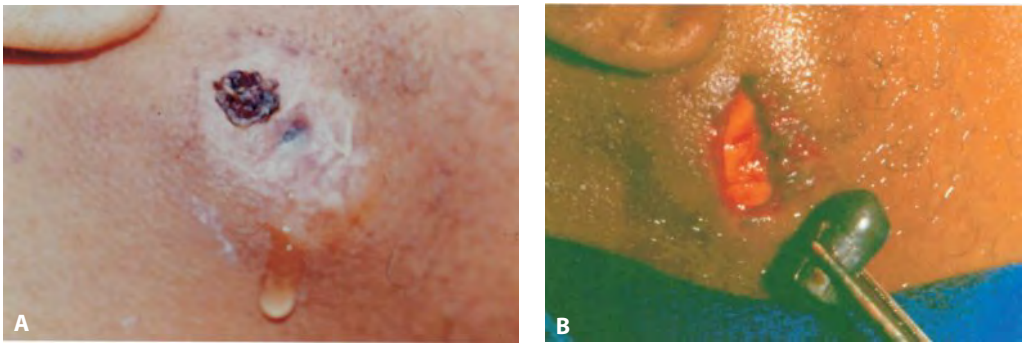


FIGURE 25-17. **A,** Salivary-cutaneous fistula associated with a retained bullet fragment. **B,** The bullet was removed and a dermal graft was placed.

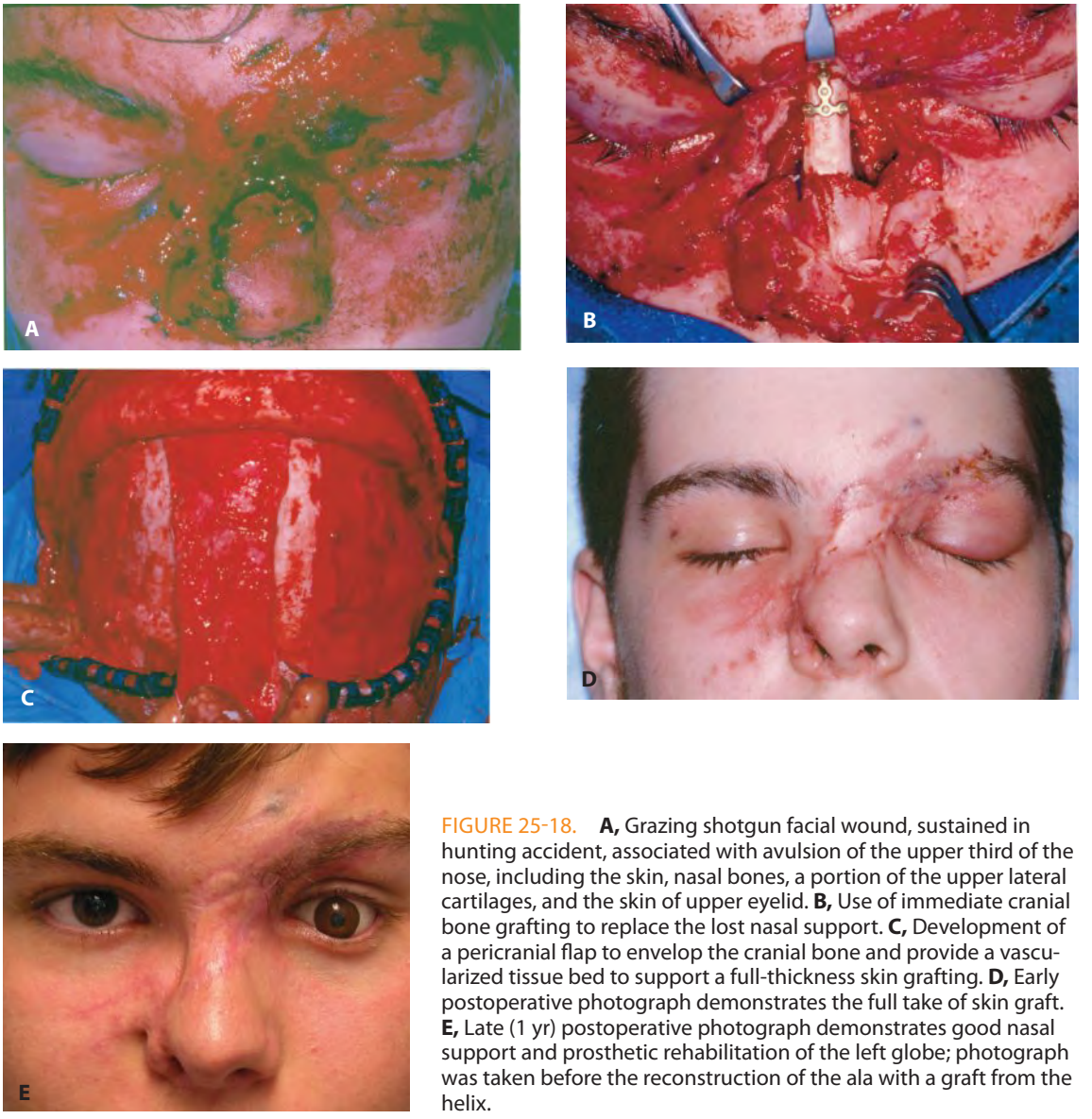


FIGURE 25-18. **A,** Grazing shotgun facial wound, sustained in hunting accident, associated with avulsion of the upper third of the nose, including the skin, nasal bones, a portion of the upper lateral cartilages, and the skin of upper eyelid. **B,** Use of immediate cranial bone grafting to replace the lost nasal support. **C,** Development of a pericranial flap to envelop the cranial bone and provide a vascularized tissue bed to support a full-thickness skin grafting. **D,** Early postoperative photograph demonstrates the full take of skin graft. **E,** Late (1 yr) postoperative photograph demonstrates good nasal support and prosthetic rehabilitation of the left globe; photograph was taken before the reconstruction of the ala with a graft from the helix.

War I, and much of what we know about the healing of free bone grafts was learned after their introduction for late reconstruction of gunshot injuries in wartime. Iliac bone grafts were popular for late reconstruction. Surgical dogma was against early or primary bone grafting and stipulated waiting until soft tissue healing had occurred. More recently, the use of bone grafts in the early setting has gained popularity in an attempt to mitigate soft tissue contracture. Gruss and colleagues[60] have published extensively on their success with early bone grafting to stabilize and support soft tissues and to decrease scar contracture and distortion. The use of cranial bone in blunt injuries was extended to include GSWs with some success. Currently, many surgeons advocate the use of primary bone grafting in the midface. Some surgeons also advocate immediate bone grafting of mandible defects.[61] Most agree, however, that delayed grafting of discontinuity defects of the mandible is still indicated because of the high risk of exposure and loss of bone grafts in this site and that immediate grafting in the mandible should be avoided.[13,57] Clark and colleagues[13] reported a 35% incidence of wound complications in patients undergoing immediate reconstruction of significantly comminuted mandible fractures resulting from GSWs. Conversely, primary bone grafting was uniformly successful in the cranium and midface.[13] Even if bone grafts or plates become exposed in the maxillary or sinonasal area, they should be maintained with wound care until definitive reconstruction. Rigid fixation maintains the mandibular segments. Similar to the midface, if the titanium plate becomes exposed in the mandible, wound care will allow it to be maintained until definitive reconstruction.[46,60] In summary, primary bone grafting in the early phase of GSW management can be useful, but it should be limited to the upper and midface. Maintenance of mandibular segments with rigid reconstruction plates combined with delayed

grafting, distraction osteogenesis or free flap reconstruction offers a more predictable result, and in most cases, primary grafting of the mandible is not indicated.

Late Reconstruction

Once soft tissues injuries have stabilized, definitive reconstruction can occur. Three-dimensional CT scanning can aid in perioperative planning and this can be combined with medical modeling and prefabricated cutting guides to more accurately recapitulate lost anatomy.[62] Delayed bone reconstructions frequently suffer from a scarred hypovascular environment that does not support free bone grafts well. In addition, there is typically a deficiency in soft tissue that becomes more pronounced when wounds are opened. In these cases, vascularized tissue transfer offers the ability to import soft tissue and/or bone into the site. Free soft tissue flaps offer advantages over pedicled flaps, and can be used to create well-vascularized soft tissue beds for future free bone grafts.[63,64] Bone containing free flaps or distraction osteogenesis is more reliable in mandibular continuity defects greater than 2 to 4 cm.[65,66] As noted previously, free tissue transfer is usually delayed until after the acute setting to decrease the incidence of flap loss secondary to clotting of the vascular pedicle. Preoperative angiography often is beneficial to identify appropriate vessels in the neck. Vascularized bone grafts can support osseointegrated implants to complete the reconstruction.[67] Anthony and associates[68] reported on the use of the fibula in patients in whom previous reconstructive attempts for gunshot injuries had failed. Both cases involved secondary reconstructions. Futran[69] has offered a review of the Seattle experience in utilizing free tissue transfer for complex facial trauma in which he outlines the success of free tissue transfer in the management of avulsive injuries when incorporated into a three-phase approach. It is important to note, patients typically required multiple surgical procedures (average of three for oromandibular injuries, five for those with orbital involvement, and eight with nasal involvement).[69] Some surgeons have advocated delayed reconstruction in GSWs that resulted from suicide attempts because of the potential for repeat suicide attempts, arguing that there is a high rate of recidivism and that patients should be stabilized psychologically for some period of time before undertaking an extensive (and expensive) reconstructive effort. However, Cusick and colleagues[70] found an incidence of only 8% confirmed mortality in the follow-up of 91 patients who had attempted suicide. All were patients who had longstanding chronic mental illness. De Leo and coworkers[71] found a higher rate in an elderly European population. In a 1-year follow-up, they found 24% had attempted suicide again, with approximately half being successful in their second attempt. With modern techniques, however, primary reconstruction has become more attractive in most patients who have self-inflicted GSWs.[72] It should be noted, however, that some authors still recommend delayed reconstructive efforts. Siberchicot and associates[58] reviewed 165 patients with self-inflicted gunshot injuries between 1982 and 1996 and suggested that delayed definitive reconstruction was more likely to achieve satisfactory results in appearance and function.

CONCLUSIONS

The development of firearms heralded a new era in surgery as well as warfare. Evolution of more efficient weapons continues to force surgeons to improve techniques. Similarly, improvement in the management of GSWs to the face has paralleled the advancement of oral and maxillofacial surgery. Advances by Varaztad Kazanjian, the "miracle man of the Western front" during World War I, continued through the wars of the 20th century. Improvements in casualty management and triage in the Korean and Vietnam conflicts led to increased survival of those with devastating facial injuries. Techniques and skills developed by oral and maxillofacial surgeons in the management of these injuries translated directly to other areas such as bone grafting and promoted the growth and expanding scope of the specialty. These efforts are continued today by oral and maxillofacial surgeons in urban trauma centers dealing with gunshot injuries to the face and on the battlefields of Afghanistan and Iraq. Improvements in imaging and fixation techniques have resulted in an evolution in management, with an emphasis on earlier repair and a focus on improvement in quality of life.

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CHAPTER

Pediatric Facial Trauma

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Trauma is the leading cause of death in children in North America and is a major cause of disability and financial burden to the health care system. Delays in evaluation and management of major trauma results in more (~30%) early death in children who are seriously injured.[1] For these reasons, the injured child requires very expedient, careful, and thorough evaluation and prompt management to achieve good outcomes. This is often challenging owing to a number of issues, including transport issues, lack of dedicated pediatric emergency care, emergency room environs during evaluation—a hurt, anxious, or noncommunicative child, an absent or distraught caregiver and family, and the urgency of quick assessments and interventions.

The National Trauma Data Bank (NTDB)[2] of the American College of Surgeons collects submitted data from level 1 and dedicated pediatric trauma centers every year in order to follow trends in and identify key epidemiologic factors relative to pediatric injuries in the United States. These data for 2009 suggest the following:

- An *increase in the numbers* of injured children and those requiring emergency and in-hospital management for their injuries.
- Persistent *bimodal increase in trauma* in the early childhood years (age 2–5 yr) with a *peak in adolescence*, reflective of the more mobile, risk-taking, and violent behavior of teenagers, particularly males.
- Most trauma is due to *motor vehicle crashes*, followed by falls, sport and recreation, and interpersonal violence.
- More children *die from head injury* (usually blunt trauma).
- The presence of severe *extracranial injury doubles mortality and morbidity* in head-injured children.
- Most pediatric trauma is considered *preventable.*

HISTORY

The history of an injured child is directed at mechanism, witnesses, and circumstances leading to and surrounding the event[3]. This may give important clues as to the body regions injured, depth or extent of injuries, and important environmental clues, such as the possibility of penetrating injuries, dynamics of impact, acceleration-deceleration or airborne mechanisms, and exposure to drugs or toxins.[3,4] Information must be gleaned quickly as to time since event, approximate blood loss, witnessed seizures or vomiting, lucid intervals followed by somnolence, and regathering information to corroborate and confirm mechanisms of injury.[5] Any suspicion for nonaccidental causes of injury must be pursued to determine possible abuse.[4,5]

PHYSICAL EVALUATION

Assessment of severe trauma in children must take into account anatomic and physiologic differences from those in the adult (Table 26-1). Owing to the small body size with relative increased body surface area, injuries to one region easily involve other regions.[1] Therefore, a high index of suspicion for multiple injuries, which are often unnoticed, must be exercised by the management team during initial trauma evaluation. Also, the disproportion between the head, midface, and neck may result in more airway collapse or compromise in the supine child, and precautionary steps should be taken to address this potential issue. Airway compromise may further lead to worsening hypoxia in children due to increased respiratory rate and oxygen consumption and decreased residual volume.[1,3]

Emergent evaluation of the injured child must follow the established routines as outlined in Advanced Trauma Life Support (ATLS)[6]—A = airway; B = breathing; C = circulation; D = disability; and E = exposure. This is a dynamic process of continual evaluation, intervention, and reevaluation. As stated in the introduction to a text on pediatric trauma[7]: "The ultimate common pathway leading to death in the injured child is profound shock: the inadequate delivery of oxygen to the tissues." Because children may be more profoundly affected by a

TABLE 26-1. **Pediatric Anatomic and Physiologic Distinctives and Clinical Relevance**

Anatomic Distinctive	Clinical Relevance
Large body surface area, small body size	Greater forces of injury dealt to a small body and more organs
Large cranium to body proportion	Head trauma and increased cervical strain
Elastic skeleton	Less external trauma (i.e., fractures) with concealed internal injuries
Short pliable neck, large tongue	Difficult airway, uncuffed endotracheal tubes in very young
Larynx anterior, short, and cephalad with narrowest portion at cricoid	Increased risk of right bronchus intubation
Increased peripheral resistance and reflex tachycardia, less reserve	Rapid response to hypovolemia initially but also rapid cardiovascular decline with continued blood loss

traumatic event owing to more body surface exposure (head, chest, abdomen) to insult and having less reserve in volume status, evaluation and interventions are paramount. As Emeritus Professor Alex Haller[8] observed in the Scudder Oration on Trauma, there is the well-known "golden hour" in adult ATLS, but it is the "platinum half-hour" in children when immediate and meaningful assessments and treatment must take place in order to resuscitate the severely injured child. This initial survey is termed the *primary survey* and often indicates or determines whether the child will survive.

Airway

Airway loss or obstruction is the mechanism that can result in rapid deterioration or death in the injured child. Stepwise evaluation and procedures are as follows:

1. Clear secretions, foreign bodies, loose teeth, or tissue.
2. Assess patency and ability to maintain such.
3. Have a low threshold for intubation.
4. Constant cervical spine protection.

Breathing

It is important to assess the ability to maintain not only the airway but also the quality of respiration. Thorough evaluation of the respiratory drive, effort, and efficacy is key and should also include

1. Oxygen administration to all patients initially, then any with less than 93% arterial oxygen saturation (Sao_2) on room air.
2. Blow-by or hood for infants.
3. *100% oxygen* by high-flow mask or non-rebreathing mask for children not able to maintain patency.
4. Monitor end-tidal CO_2 and exchange (keep < 40).

Circulation

Hypovolemia is key to identify; children compensate well initially in blood or volume loss by compensatory heart rate increase, but once hypotension manifests, the child is on a downward spiral that quickly leads to collapse if not reversed. The following parameters should be noted and met:

1. Total blood volume is roughly 80 mL/kg in the child patient.
2. Place two intravenous lines, pediatric urethral catheter.
3. Initial fluid resuscitation should administer 20 mL/kg of *warmed, isotonic* solution (normal saline or lactated Ringer's) over 15 to 20 minutes with reevaluation of volume status.
4. Repeat administration twice to achieve volume stability as noted by blood pressure increase, return of pulses, capillary refill less than 2 seconds and improved sensorium; reversal of sunken fontanelles and increased skin turgor are late indications of normotension.
5. Obtain cross-matched or O-negative blood for transfusion, initial bolus of 10 to 20 mL/kg.
6. Defer further studies until stabilized.

Disability

Quickly look for and identify potential life-threatening injuries:

1. Fractures, open or with significant deformity.
2. Look for *spontaneous movement of limbs to external stimuli.*
3. What is the *response to voice?* Repeat often.
4. Glasgow Coma Scale (GCS) for children may be utilized with modification for verbal responses:
   a. Appropriate words, smiling, fixes, follows: 5
   b. Cries but is consolable: 4
   c. Persistently irritable: 3
   d. Restless, agitated, nonpurposeful: 2
   e. None: 1
5. AVPU to quickly assess disability; score of P or U suggests GCS less than 8
   a. A = Alert
   b. V = response to Verbal stimuli
   c. P = response to Painful stimuli
   d. U = Unresponsive

Exposure

Careful exposure and examination of the child should be done expeditiously but thoroughly in order to identify any other areas of injury and threat to resuscitation and survival:

1. Examination in a warmed room with overhead warmers.
2. Cover and dress open fractures and wounds, especially burns.
3. A trained person should stabilize the cervical spine while the neck is examined, then place an appropriately sized cervical collar.

The secondary survey then follows and involves a more comprehensive examination looking for other head/neck injuries, cerebrospinal fluid (CSF) leaks, and a quick cranial nerve examination (pupils, eye movement, grimaces, facial symmetry). The neck should be evaluated for tracheal deviation, discoloration (expanding hematoma), or tenderness. Chest rise, symmetry, and bilateral breath sounds should be confirmed. Abdominal injuries are accompanied by guarding or tenderness, a tense or ecchymotic abdomen, and absence of bowel sounds. Vascular integrity should be confirmed by checking peripheral pulses and color or temperature of extremities.

The mnemonic AMPLE is useful for consideration during this phase of data gathering and evaluation:

A = Allergies
M = Medications
P = Past medical history
L = Last meal
E = Environment, events

Generally, this involves having the parent or caregiver present who can reliably respond to such queries.

HEAD INJURIES

Over half of the fatalities associated with trauma in children are due to traumatic brain injury; indeed, 20% to 30% of severe head trauma results in death.[1,9,10] This is usually due to blunt trauma from motor vehicle crashes, followed by falls and sport activities. Diffuse axonal injury often results from the head being violently jerked about, which results in torsional and contrecoup forces applied to and through the brain. This results in a tearing of individual neurons with subsequent edema forming throughout much of the injured brain matter.[9,10] Less common are intracranial hematomas seen more in the adult population. Children have more epidural hematomas (Figure 26-1), which are collections outside of the dura, as opposed to subdural hematomas, which collect under the dura and are associated with venous rupture.[1,4,9] Epidural hematomas result from torn meningeal arteries. These are classically associated with the "lucid" interval after injury followed by rapid neurologic deterioration; it is a neurosurgical emergency that must be quickly confirmed and treated.

Expanding hematomas result in exponential increases in the intracranial pressure (ICP) with initial displacement into the CSF and ventricular chambers and sinuses. It is important to avoid hypotension, hypertension, and hypoxia in pediatric brain injury because these states quickly accelerate forces contributing to increased ICP and less perfusion pressure to the brain, thereby increasing ischemia.[1,3,9] Adequate gas exchange must be assured to avoid hypercarbia, which also will contribute to brain vasodilation and consequent hypertension and hypoxia. The following targets and treatments should be observed in relation to brain injury in children[9]:

- Arterial oxygen tension (Pao_2) > 80 mmHg or Sao_2 > 95%.
- Arterial carbon dioxide tension ($Paco_2$) ~ 35–40 mmHg.

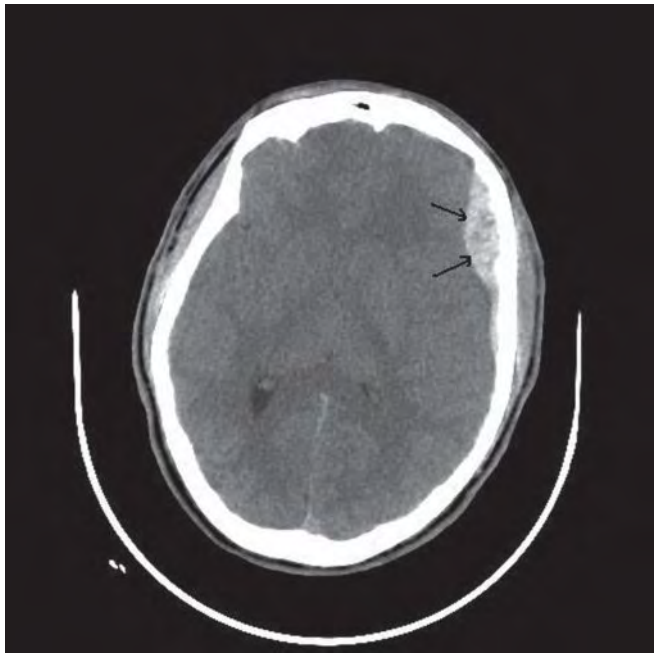


FIGURE 26-1. Computed tomography (CT) scan of epidural hematoma *(arrows)*.

- High-normal blood pressure for age.
- Normothermia.
- Anticonvulsants early in injury.
- ICP ~ 20–25 mmHg.
- Avoid hypoglycemia.
- For *increased ICP,* consider hypertonic 3% NaCl at 3 mL/kg and mannitol 0.5–1.0 g/kg.

The physical assessment is critical in pediatric head injury. Pupillary size and reactivity should be documented initially and often. As noted previously, the GCS is modified in children to obtain the best responses for age. Noting any changes in the established baseline should provoke further inquiry and studies. Brain herniation is accompanied by bradycardia, mydriasis (a critical lateralizing sign), and progressive obtundation.

Neck and Airway

Blunt and penetrating trauma to the neck accounts for approximately 7% of head and neck injuries in children.[11] Approximately 85% are in zone II and mortality increases significantly when injuries are located in zones I and III.[12] Owing to a short, pliable, and small neck in children, access for control of hemorrhage in zones I and III is very limited and represents areas of difficult intervention in the child (Figure 26-2).

Zones I and III injuries of the neck may require angiography to rule out injury to vascular structures because these are difficult to clinically assess in children owing to cooperation (if conscious) issues, anatomy, and reliability of pulsatile characteristics in children.[12] Esophageal injuries are ruled out

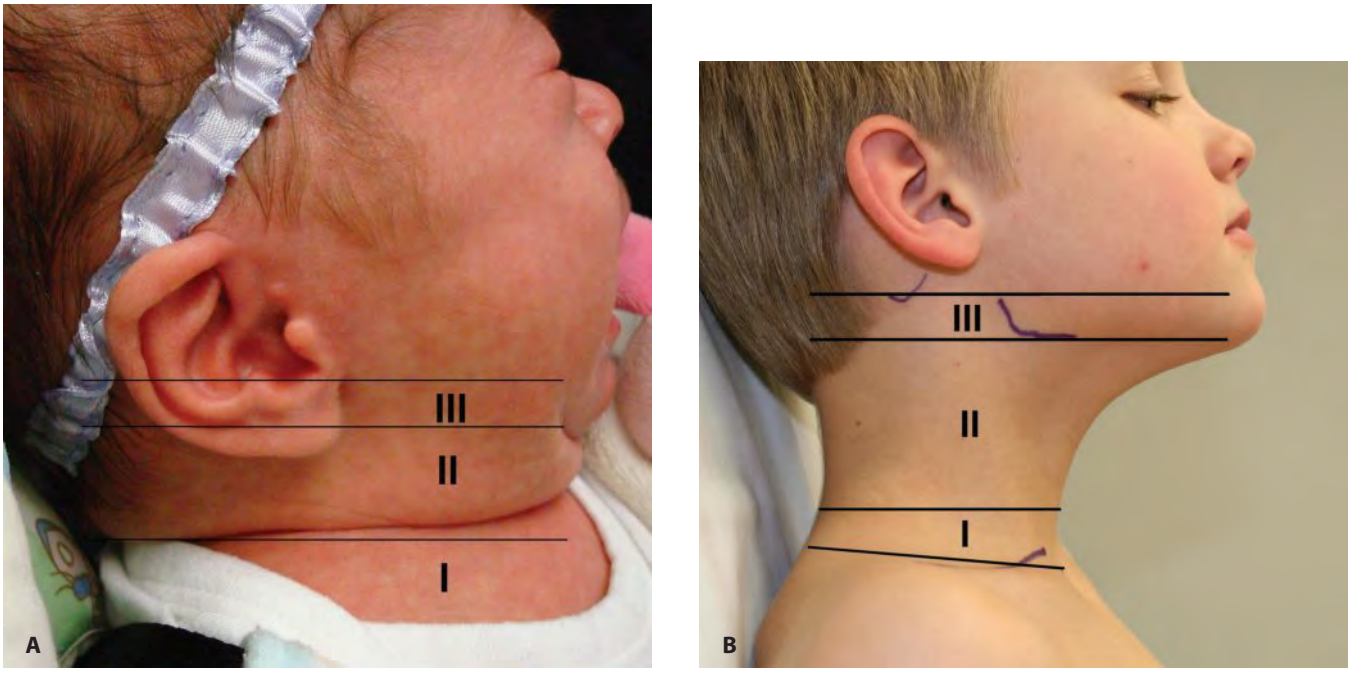


FIGURE 26-2. Zones of the neck in penetrating neck injuries. **A,** Infant neck. **B,** Child neck. Zone I: between the clavicle and the lower cricoid. Zone II: between the lower cricoid and the angle of the jaw. Zone III: between the angle of jaw and the skull base.

by barium swallow studies if there is a strong suspicion of penetrating injury to the throat. An unstable patient may require emergent exploration under controlled conditions.

Signs of laryngeal injury in children include[13,14]

- Dysphonia.
- Hemoptysis.
- Subcutaneous emphysema.
- Laryngeal ecchymosis and tenderness.
- Tracheal deviation.

Cervical spine injuries occur in 1% to 2% of all trauma patients, including children. Most of these injuries result from violent deceleration-acceleration mechanisms often seen in motor vehicle crashes and sports-related activities. Most (~70%) of these injuries occur in C1–4 in the pediatric population.[11] Spinal cord injury without radiographic abnormality (SCIWORA) is higher in pediatric patients; this is the cord injury correlate of diffuse axonal injury of the brain.

Pediatric Perioperative Management

After the primary and secondary surveys have been completed and potential life- and limb-threatening injuries determined and addressed, the child's health status should be confirmed. Usually, this can be determined through a reliable caregiver or access to the pediatric medical record. First, the immunization status needs to be confirmed and addressed if there are questions as to currency; tetanus prophylaxis is the most important status to confirm. Dirty, contaminated wounds should receive a tetanus toxoid booster (0.5 mL) and consideration given to TIG (tetanus immunoglobulin) 250 IU for such wounds in patients who have not been tetanus vaccinated in the last 5 years or when there is uncertainty as to proper immunization status.[15] Any conditions that may adversely impact upon the injured child's health and subsequent management should be identified, such as poorly controlled diabetes, cystic fibrosis, asthma, sickle cell disease, and steps taken to optimize the patient's medical condition.

Antibiotic administration for head and neck injuries in children follows similar recommendations for the adult population. Young children will usually have established skin and mucosa floral populations, the exception being the nasal cavity and sinuses, which harbor more *Haemophilus* (untypeable) and *Moraxella* organisms in the chronically colonized or infected sinus.[16] Injuries that involve the nasosinus region may need to be treated with antibiotics directed at these organisms as well as the typical gram-positive organisms. Clean wounds generally do not require antibiotics after the initial intra- and/or perioperative doses. Exceptions to these wounds would be human and animal bites or avulsive/devascularizing type wounds. Bite wounds should receive 7 to 10 days of antibiotics directed at the mixed flora of the respective bite.

SOFT TISSUE INJURIES

Most craniomaxillofacial injuries in children, particularly the young child, are soft tissue in nature. Trauma forces to the facial region typically result in soft tissue avulsion injuries, lacerations, and contusions; however, the facial skeleton often will be spared owing to its elasticity.[17]

Initial evaluation directed toward soft tissue wounds should take into consideration the viability of injured tissues,

even marginal tissues, and the increased healing potential of children.[18] What may appear to be nonviable tissue in a severely avulsed or macerated wound may survive with good initial care. For these reasons, the surgeon should attempt to preserve tissue as much as possible within the frame of inherent tissue and patient health, particularly in regionally specific tissue zones like the eyelid and nose.[19] After initial resuscitation, these tissues will often respond favorably even after hours of ischemia. All soft tissue avulsion flaps should be carefully returned to and supported in their anatomic position and kept protected and warmed until definitive repair can be undertaken. Care should be exercised to avoid undue pressure or tension on marginal wounds.[17]

Because thorough evaluation of wounds in the injured child is paramount but also difficult to carry out and manage, many wounds will have to be initially addressed under controlled conditions in the operating theater. If the child is conscious and crying, simple observation of facial animation and posturing may indicate depth of soft tissue injury to underlying neurovascular structures. After resuscitation and stabilization of the child, the surgeon can move to the theater to investigate large or penetrating wounds, document nerve and ductal injuries, and determine whether eye injuries require specialist intervention. A stepwise plan for repair can then be made including the need for microreparative procedures, ophthalmologic review, and upper aerodigestive tract evaluation for penetrating neck wounds.

Large, contaminated avulsive wounds should be thoroughly cleansed with pulse irrigation in the theater (Figure 26-3), which dislodges particulate and reduces the bacterial load in the tissues.[17,19] Antibiotic solutions usually are not necessary and have not been shown to be any more beneficial than saline solutions in reducing bacterial infections. Rather, the pulsed mechanical removal of necrotic tissue debris, foreign bodies, and bacterial load is the key for improving the tissue bed and reducing infections. Completely avulsed tissue may be salvaged for use as a free graft and should be utilized for such as an initial reconstructive procedure with a view toward later serial staged revisions as healing and function warrant.



FIGURE 26-3. Pulse irrigation of a contaminated wound.

REGIONAL SOFT TISSUE WOUNDS

The Ear

The ear is a prominent and protrusive facial structure that has limited protection from injury secondary to its functional purpose of hearing. As a result, however, the ear is prone to a spectrum of traumatic injuries ranging from abrasion, simple laceration, complex laceration, partial avulsion to complete avulsion. Also, not only is the injury type important, but the etiology or cause is also critical to the treatment sequence. A clean, linear laceration differs greatly from a macerated dog bite laceration.

The examination must assess for cartilage exposure and hematoma formation. Further, it should include an otoscopic examination of the external auditory canal and the tympanic membrane, so as to address potential canal stenosis and diminished acuity of hearing from tympanic membrane rupture. Fortunately, the ear is resilient secondary to its substantial vascular arcade. The blood supply to the external ear is primarily via the superficial temporal artery and the posterior auricular artery, branches of the external carotid (Figure 26-4). These two main arteries create a vascular ring around the ear and supply an area approximately 6×11 cm, extending from the tragus to 5 cm posterior to the external auditory canal and 6 cm inferior to the mastoid. Additional branches arise from this vascular ring and additional anastomotic networks exist between the posterior auricular artery and the occipital artery (see Figure 26-4). Some arterial twigs from the deep auricular artery (internal maxillary artery) supply the auditory canal.

Treatment of traumatic ear injuries begins with patient management and anesthesia. Delicate repair of a pediatric ear injury requires significant patient compliance, and thus, in most pediatric patients, a general anesthetic is warranted. Also, despite historical concerns regarding the use of local anesthesia with epinephrine in the ear, judicious use via infiltration and nerve blocks is reasonable for the repair of most ear injuries unless there is questionable viability of tissues.

Ear injuries in children require prompt attention, particularly if the cartilaginous units are disrupted or if hematomas are present because these injuries will result in progressive ischemia and deformity during healing.[20] All hematomas should be drained and a bolster dressing applied to prevent hematoma reaccumulation (Figure 26-5). Perioperative antibiotics are not necessary beyond the initial procedure unless there has been contamination or perichondrial and cartilaginous maceration.

Wounds must be profusely irrigated to débride and remove foreign bodies, and hemostasis must be obtained with the judicious use of electrocautery. Irregular or necrotic skin edges are removed with tissue scissors or a blade. Nondisplaced ear cartilage fracture requires no sutures unless

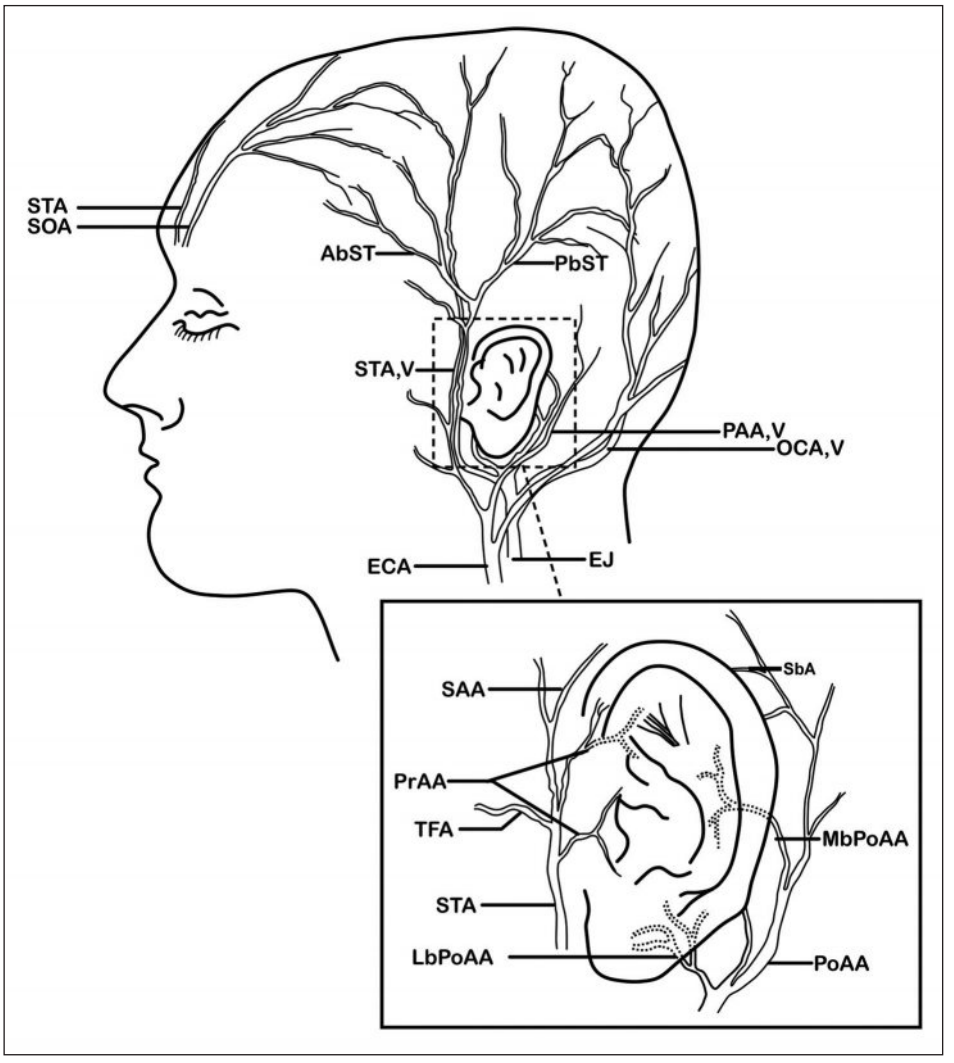


FIGURE 26-4. Blood supply to the scalp and external ear. AbST = anterior branch of superficial temporal; ECA = external carotid artery; EJ = external jugular; LbPoA = lobular branch of postauricular; MbPoA = midbranch of postauricular; OCA,V = occipital artery and vein; PbST = posterior branch of superficial temporal; PoAA,V = postauricular artery, vein; PrAA = preauricular artery; SbA = superior branch of postauricular; SOA = supraorbital artery; StA = supratrochlear artery; STA,V = superficial temporal artery and vein; TFA = transverse facial artery.



FIGURE 26-5. Ear injury with hematoma formation. **A,** Hematoma. **B,** Postdrainage with bolster dressing.

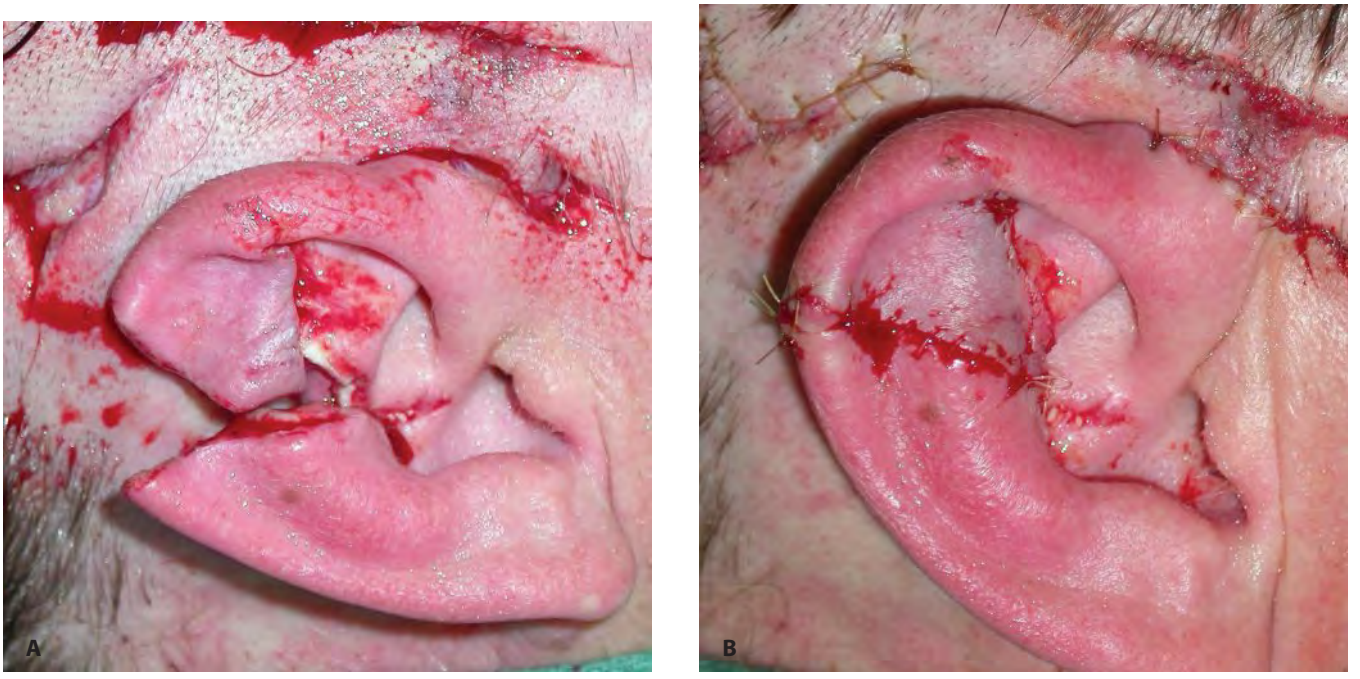


FIGURE 26-6. Complex ear wound. **A,** Full-thickness ear lacerations. **B,** Repair in layers of a full-thickness ear wound.

the ear cartilage is malpositioned or fragmented, in which case a 5-0 or 6-0 absorbable polygalactin suture can be used to place interrupted stitches to reapproximate the cartilaginous framework. The ear skin itself can then be repaired with 5-0 or 6-0 absorbable fast-gut or nonabsorbable sutures (Figure 26-6), but the absorbable suture will result in an appropriate cosmetic outcome and eliminates the trauma, for the child, of suture removal in 5 to 7 days. All completely avulsed segments may be reattached in an attempt to allow for revascularization, but larger avulsions may benefit from microvascular repair or other staged reconstructive modalities.[21] Generally, microrevascularization of one of the branch vessels of the superficial temporal or postauricular systems will suffice to maintain tissue viability, a key element to survival being venous outflow. Wound care should keep the reconstruction site clean and moist and an initial repair should be bolstered for 5 to 7 days with a xeroform gauze (Figure 26-7).

The Eyelid and Lacrimal System

The eyelids are complex and delicate structures and traumatic injuries require a return to both form and function. Examination should first evaluate for globe injuries, because preservation of vision is paramount. Familiarity with eyelid anatomy is key to wound repair (Figure 26-8). Evaluation includes examination for foreign bodies, tissue loss, and

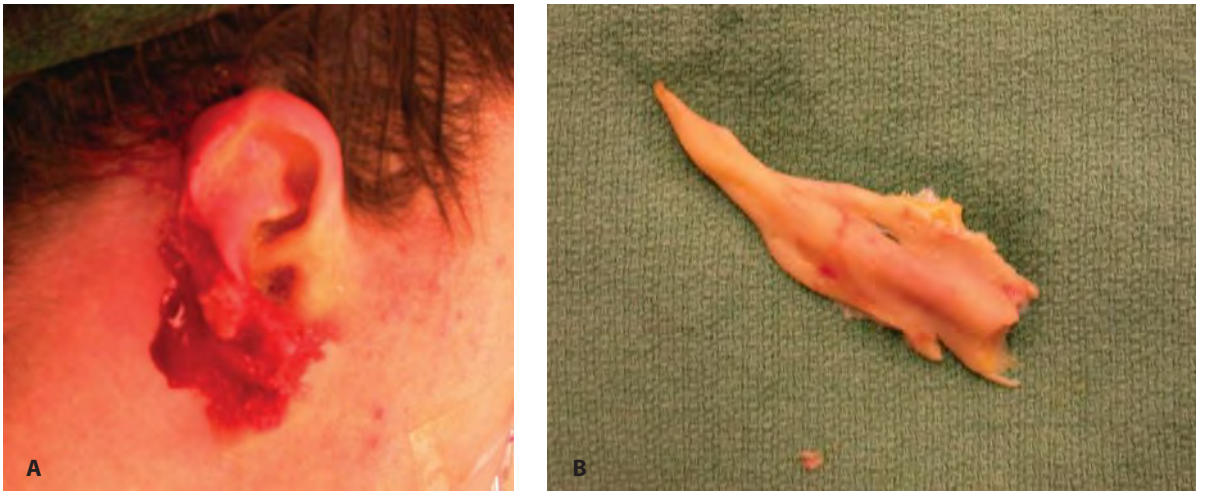


FIGURE 26-7. Ear avulsion wound. **A,** Avulsion wound. **B,** Avulsed helix (skin and cartilage). *(Continued)*



FIGURE 26-7. *(Continued)* **C,** Repair of ear wound with re-implanted avulsed portion. **D,** Six-month postoperative repair of an avulsed ear wound.

corneal abrasions with fluorescein and Wood's lamp (ultraviolet light source). Wound irrigation is then completed with sterile saline. For simple repairs and with cooperative patients, topical anesthetic drops and topical anesthetic ointment may allow for bedside closure in the emergency department. Corneal abrasions are typically treated with a short course of a combination antibiotic/anti-inflammatory/steroid agent.[22]

Anatomic subunits of the eyelid are addressed in a specific order: lid margin followed by extramarginal lacerations.

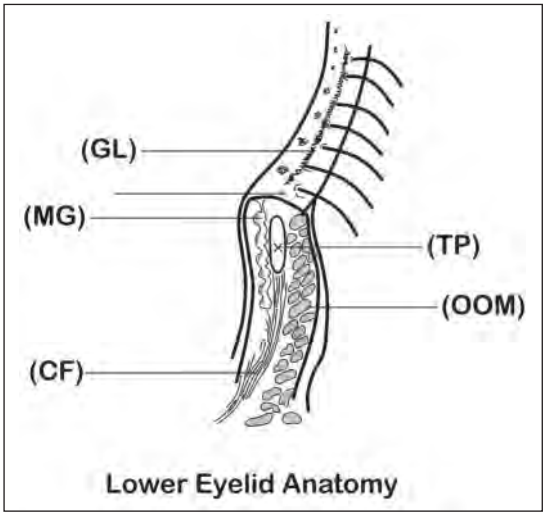


FIGURE 26-8. Lower eyelid anatomy. CF = capsulopalpebral fascia; GL = gray line; MG = meibomian gland; OOM = orbicularis oculi muscle; TP = tarsal plate.

Specifically, a vertical mattress stitch through the meibomian orifices is completed to evert the margins of the eyelid.[23] Coaptation of the tarsal plate is important, followed by anatomic approximation of the orbicularis oculi and skin, as illustrated in Figure 26-9. The wound is then dressed to bolster and protect the site during the healing phase. In cases of avulsive tissue loss to the lids, partial-thickness versus full-thickness and small versus large defect size are critical elements to carefully determine before surgical repair. Partial-thickness defects (<25%) may heal by secondary intention, but owing to eyelid skin laxity, primary closure can usually be achieved after undermining native lid skin. Larger partial-thickness defects and total avulsion of tissues require tissue grafts or local flaps to achieve closure and avoid healing-induced ectropion or lagophthalmos. Full-thickness skin grafts can be obtained from the posterior auricular area or foreskin in uncircumcised boys, resulting in a good tissue match.[24] A bolster should be applied to stabilize the graft and eliminate hematoma formation; a tarsorrhaphy or Frost suture may be necessary to put the lid at rest (Figure 26-10).

Lacrimal Apparatus

An intact and functional lacrimal system is essential to ocular health, but the weakest part of the eyelid is the portion containing the canaliculus. As a result, eyelid trauma is likely to disrupt this system resulting in epiphora and chronic infection.[25] Immediate repair typically results in superior long-term outcomes. A thorough examination should be completed at the time of initial injury and concomitant naso-orbitoethmoid (NOE) fractures should illicit concerns over the need for

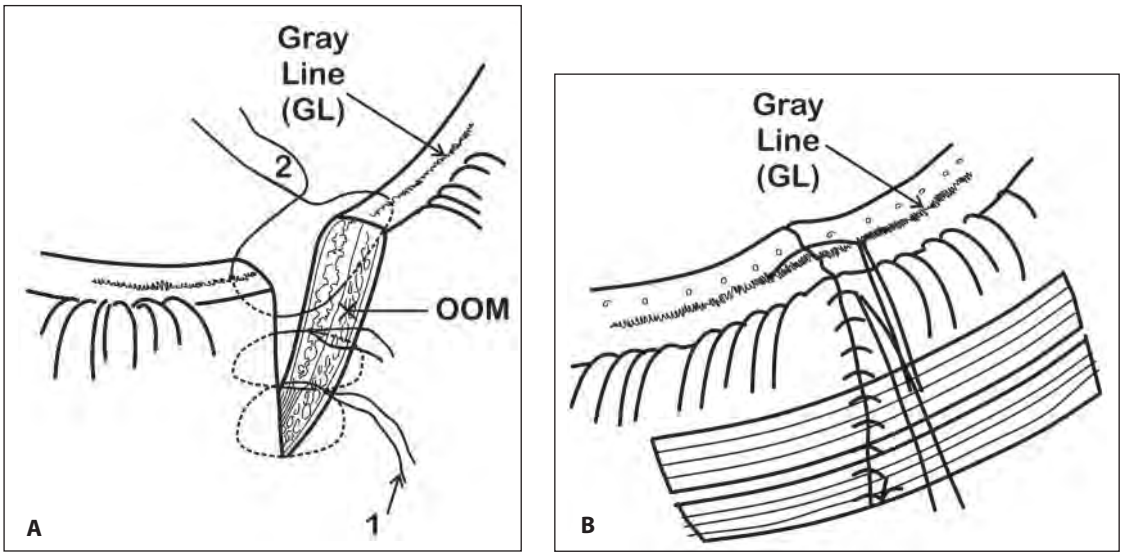


FIGURE 26-9. **A,** Eyelid laceration and anatomic repair. Note alignment of the capsulopalpebral fascia *(1),* gray line *(2),* and orbicularis oculi muscle (OOM). **B,** Eyelid skin and gray line (GL) alignment. Note that the sutures are left long, retracted, and taped inferiorly away from the lashes and conjunctiva.



FIGURE 26-10. Complex upper eyelid avulsion injury in a 14-year-old boy. **A,** Complex eyelid avulsion injury. **B,** Repair of eyelid and local full-thickness skin graft repair. **C,** Dressing support and Frost sutures to immobilize the eyelid during healing. **D,** Six months after eyelid and local flap repair.

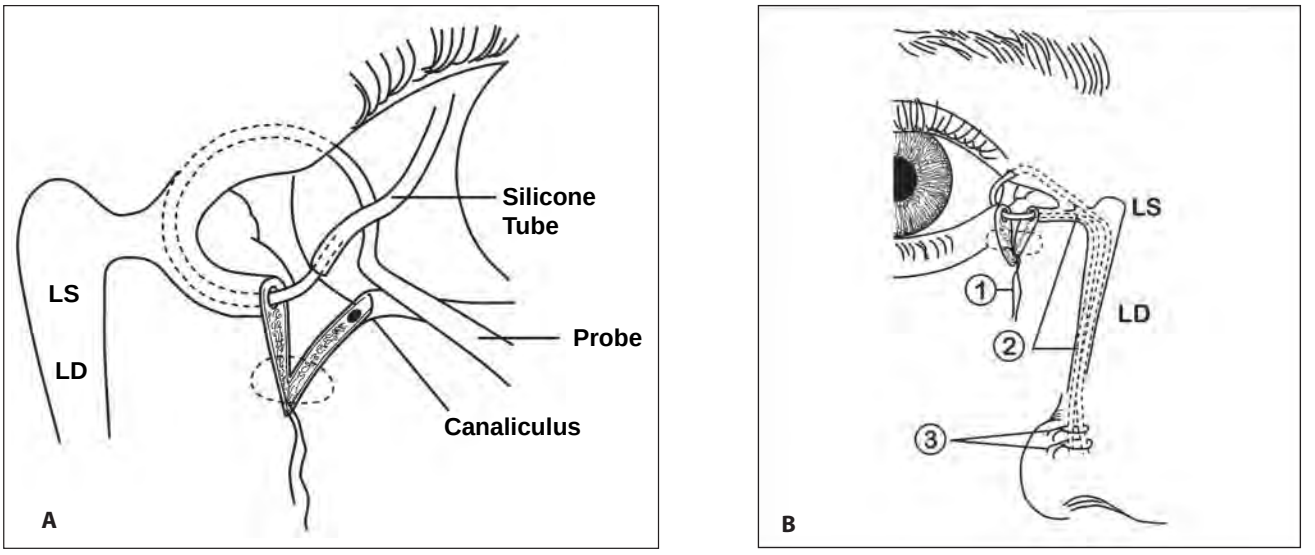


FIGURE 26-11. Eyelid and canalicular injury. LS = lacrimal sac; LD = lacrimal duct. **A,** Silicone lacrimal tube wedged onto pigtail probe and placed through the canalicular and collecting duct system. **B,** Silicone lacrimal tubes placed through the canaliculi and into the lacrimal system *(2)* exiting the nasolacrimal duct in the inferior meatus. Note the sutures are secured intranasally high *(3)* in the vestibule with two nonabsorbable sutures. Closure of the wound *(1)* is commenced once the tubes are in place and secured.

possible lacrimal duct repair. Acute injury will require evaluation in the theater under anesthesia. Generally, lacrimal probe insertion, irrigation of canaliculi, and fluorescein application with detectable egress into the nasal cavity will confirm lacrimal disruption. Traumatic disruption to the canalicular system requires lacrimal intubation with tailored silicone tubes passed through the superior and inferior canaliculi (Figure 26-11) and via the nasolacrimal duct into the nose (Figure 26-12).[26] Lid repair can then proceed once the tubes have been affixed intranasally. The tubing is maintained for a minimum of 6 weeks and then removed. Topical ophthalmologic antibiotics with anti-inflammatories should be used for a period of 5 to 7 days.

The Nose

The pediatric nasal complex is less pronounced and more pliable than the adult nose. However, tangential forces and dog bites can still lead to significant soft tissue injuries. Examination should evaluate for nasal deformity and the presence of all anatomic subunits, including the cartilaginous framework.[27] Intranasal speculum examination should evaluate for mucosal lacerations and septal hematomas. Failure to identify such abnormalities can result in nasal stenosis and a "saddle-nose" deformity as a result of septal collapse, respectively.

The nose is primarily supplied by the angular, infraorbital, and ophthalmic arteries, and internally, the nose is supplied by the anterior and posterior ethmoid arteries and the sphenopalatine arteries. In children with a smaller blood volume, epistaxis can be life-threatening and two intranasal areas need to be addressed: (1) Kiesselbach's plexus, the site of anterior bleeds, and (2) Woodruffs plexus, the location of posterior bleeds.[28] Hemostasis may be obtained with topical vasoconstrictors, followed by packing the nose. A 10- to 14-French Foley catheter may be advanced intranasally and the balloon inflated with saline once it is visualized in the oral cavity. The catheter is then retracted into the nasopharynx and the remainder of the nasal passage is packed with Vaseline or antibiotic ointment–impregnated gauze. Periodic evaluation of the nasal skin and columella should be performed to ensure that there is no undue pressure and ischemia from the tethered catheter, and periodic letdown of the balloon should be done to avoid choanal ischemia. These balloon or tamponade devices should not be used for more than 3 days in young children and 5 days in older children. While the nose is packed, children should be placed on systemic antibiotics to prevent toxic shock phenomenon.[29,30]

Once stabilized, definitive treatment of nasal soft tissue lacerations may commence. Anatomic landmarks are repositioned including the cartilaginous and bony framework. Closure then is achieved in three layers: (1) mucosa and vestibular lining, (2) cartilage, and (3) skin. After drainage of a septal hematoma, the septal mucosa should be coapted with a "quilting" stitch using absorbable suture (4-0 gut) and polyvinyl acetyl sponges or silicone splints should be placed into the nasal passageways to prevent reaccumulation (Figure 26-13).[31] Similarly, a supportive rubber stent wrapped in xeroform or Vaseline gauze may be sutured to the entrance of the nare to prevent stenosis and maintain the aperture (Figure 26-14). Custom-made soft silicone nasal tubes may be used for long-term prevention of stenosis once initial healing has taken place and the rubber stent has been removed. Small, avulsed (usually < 1 cm) nasal tissue segments are likely to survive, once reattached, based on the extensive vascular anastomotic networks and vigorous vascular ingrowth characteristic of wound healing in children.[18]

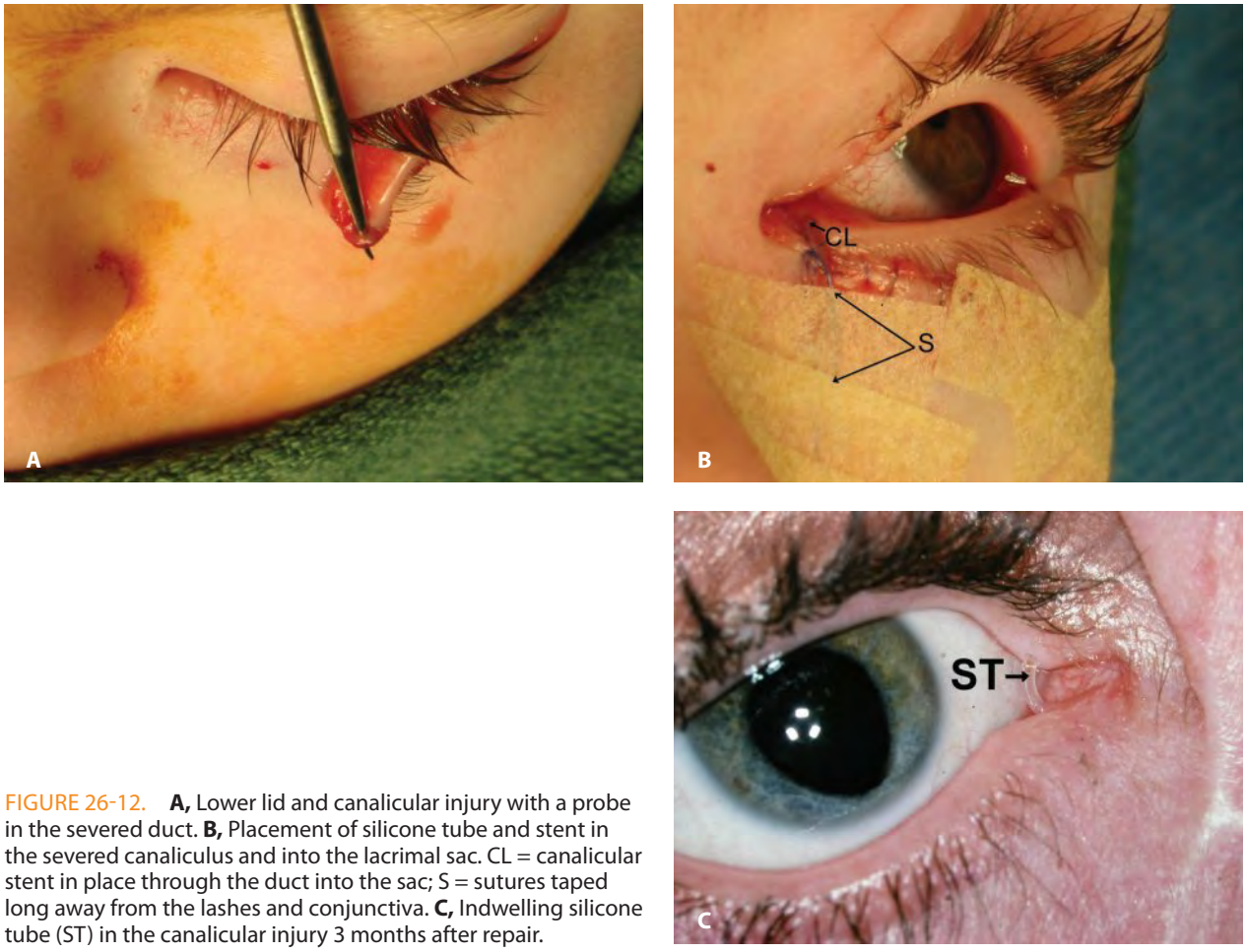


FIGURE 26-12. **A,** Lower lid and canalicular injury with a probe in the severed duct. **B,** Placement of silicone tube and stent in the severed canaliculus and into the lacrimal sac. CL = canalicular stent in place through the duct into the sac; S = sutures taped long away from the lashes and conjunctiva. **C,** Indwelling silicone tube (ST) in the canalicular injury 3 months after repair.

The Scalp

Because the pediatric patient has a disproportionate skull-to-face ratio, the scalp is more exposed to traumatic injury. Further, the scalp is generously vascularized and the blood vessels traverse the connective tissue layer that tents open the lacerated vessels and may result in profuse loss of blood volume and onset of hemorrhagic shock. Initial control of bleeding can be achieved with pressure sutures or hemostatic clips. Specifically, the scalp is supplied by the internal and



FIGURE 26-13. **A,** Nasal wound with exposed bone and cartilage in a 4-year-old boy. **B,** Nasal repair with fracture reduction, supportive dressings, bilateral nasoseptal sponges to obliterate dead space and support the cartilaginous unit.

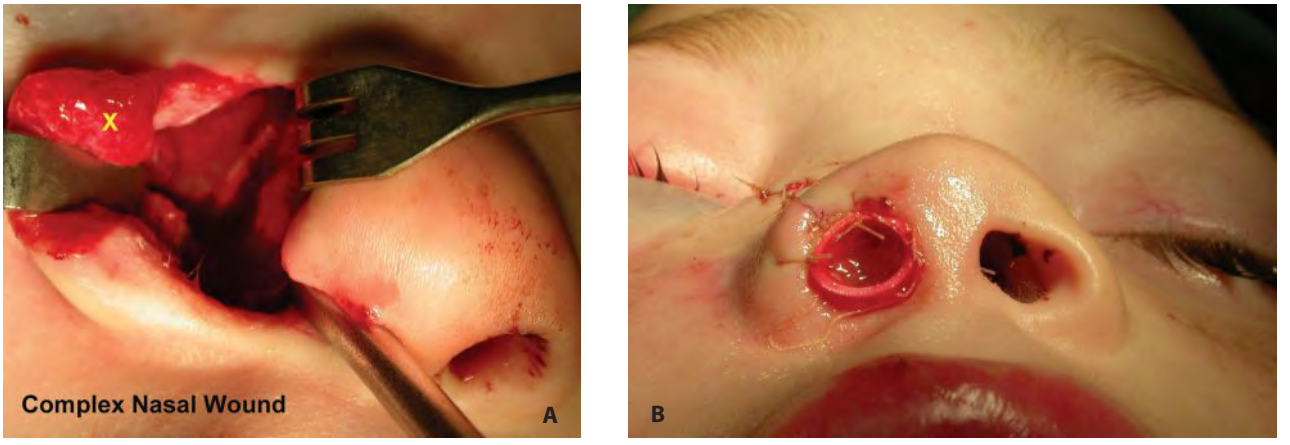


FIGURE 26-14. Nasal wound. **A,** Full-thickness nasal laceration into the passageway. **B,** Repair of a full-thickness wound with placement of a nasal stent to prevent stenosis.

external carotid arteries (see Figure 26-4). The internal carotid, via the ophthalmic artery and the supratrochlear and supraorbital arterial branches, supplies the anterior scalp to the vertex. The superficial temporal, occipital, and posterior auricular arteries of the external carotid supply the remainder of the scalp. Once hemorrhage has been controlled, the wound should be inspected for foreign bodies, gross contamination, and skull fractures. Profuse irrigation should precede closure and may best be accomplished with pulse-vacuum assistance with an antibiotic-impregnated saline solution if gross particulate contamination is present. Nonviable or irregular margins should be conservatively trimmed; suction drain placement is frequently necessary in the scalp to prevent hematoma formation. For significant areas of tissue loss, split-thickness skin or acellular dermis grafts may be employed, for temporary coverage, if the pericranium is intact (Figure 26-15).[32] In cases requiring secondary reconstruction or if significant avulsive injury occurs with

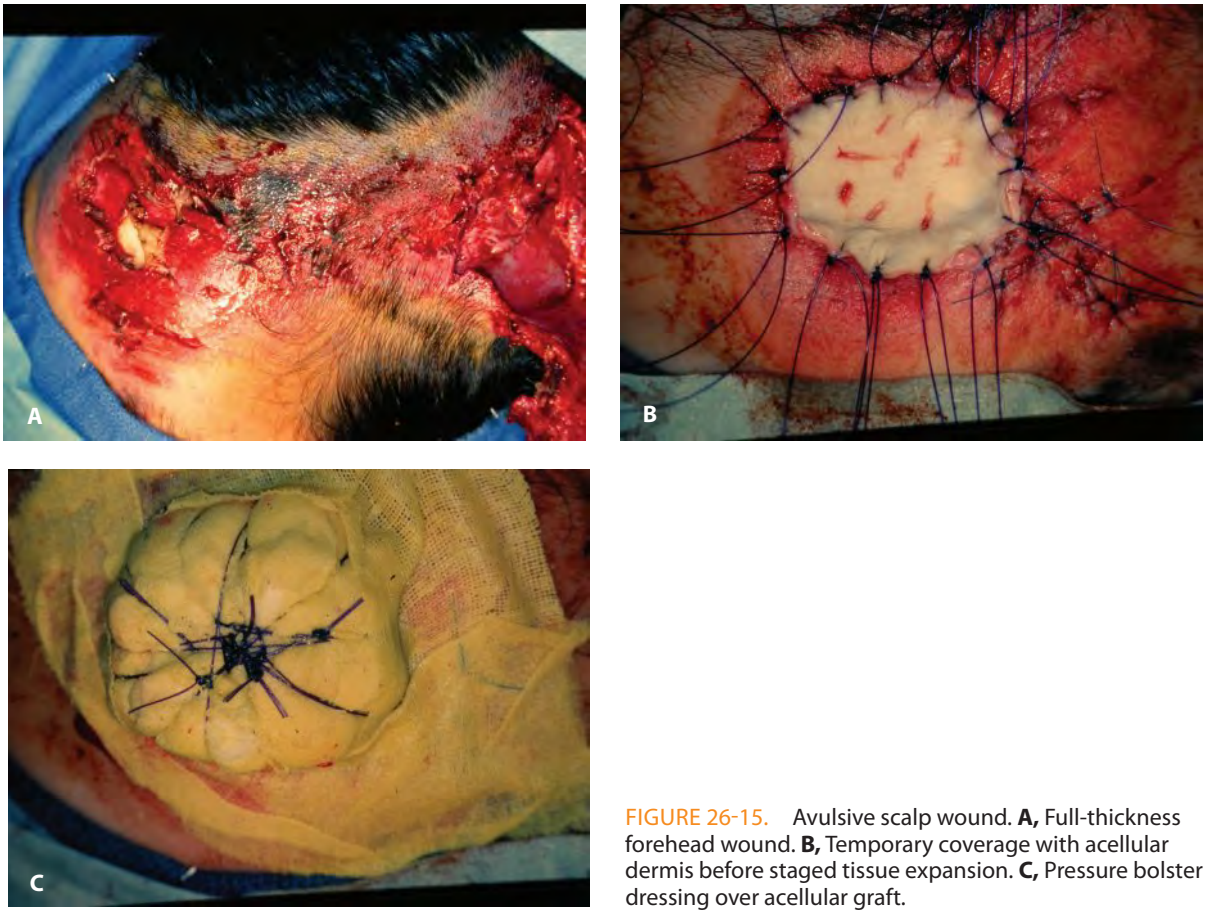


FIGURE 26-15. Avulsive scalp wound. **A,** Full-thickness forehead wound. **B,** Temporary coverage with acellular dermis before staged tissue expansion. **C,** Pressure bolster dressing over acellular graft.

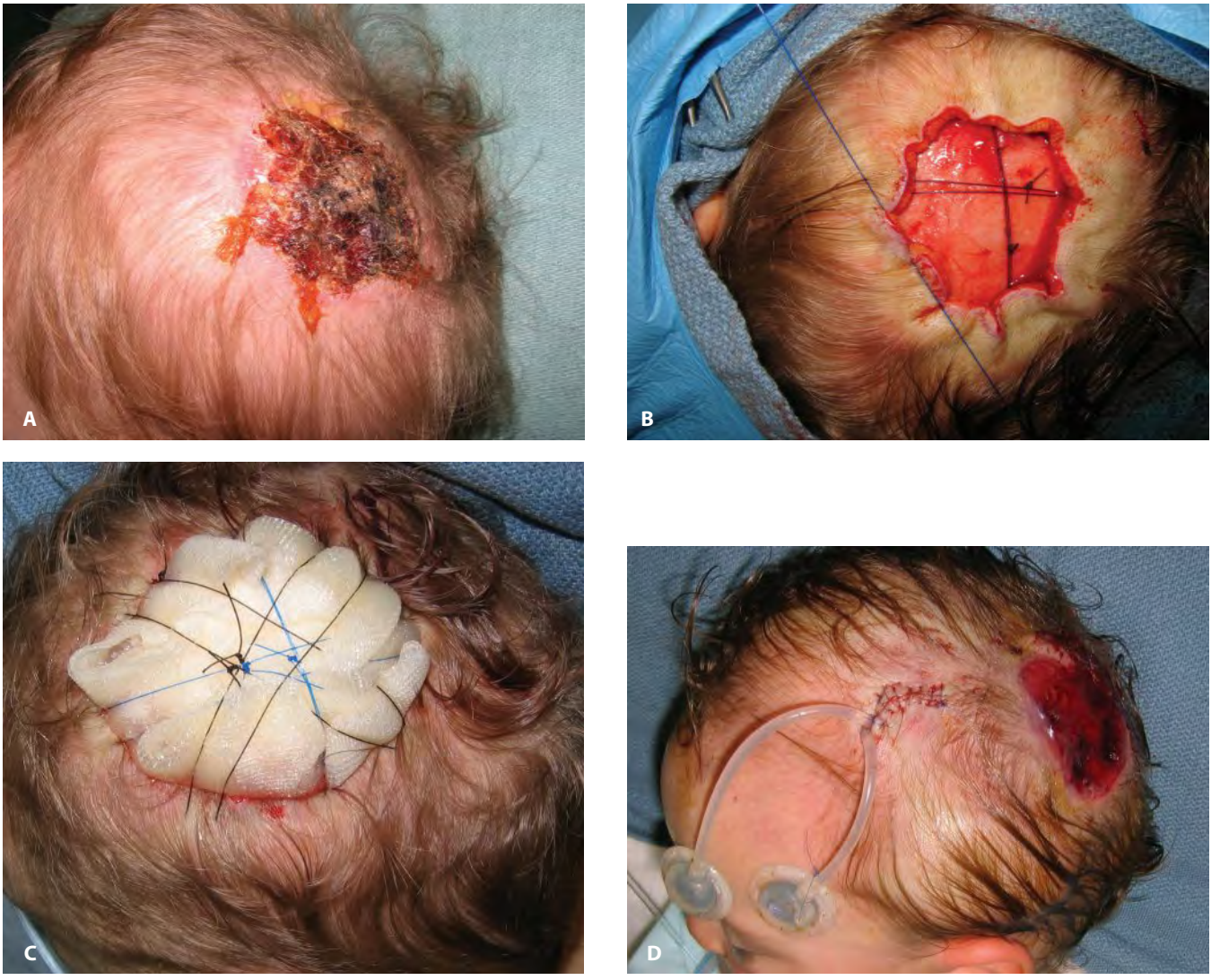


FIGURE 26-16. Scalp wound defect and reconstruction in a 3-year-old boy. **A,** Necrotic, infected full-thickness scalp injury. **B,** Postexcision of necrotic scalp and placement of a purse-string suture. **C,** Pressure dressing over acellular graft and wound. **D,** Staged tissue expansion for a scalp wound.

cranial bone exposure, reconstruction can be achieved with local rotational flaps or via staged tissue expansion and flap procedures (Figure 26-16). In addition, in cases of exposed cranial bone, a wound vacuum-assistance device can develop a vascular bed for grafting as well as for keeping the wound clean.

The Cheek

Injury to the cheeks may involve two critical structures: the parotid gland/duct and the facial nerve. As a general rule, lacerations distal to an imaginary vertical line drawn from the lateral canthus of the eye are less likely to cause critical injury to the facial nerve. Simple closure can then be undertaken without further delay. To evaluate for parotid duct injury, the needleless portion of an angiocatheter is attached to a saline-filled syringe and Stenson's duct is cannulated. Saline is then flushed and, if visualized in the wound site, a duct injury is confirmed. A Silastic stent can then be placed into the wound via the proximal transected end and exiting through Stenson's duct opening into the oral cavity. Repair is completed in the theater under microscopic magnification (Figure 26-17). The Silastic stent is left within the duct and the proximal end is sutured to Stenson's duct orifice or to the buccal mucosa and maintained a minimum of 2 weeks (Figure 26-18).[33] In cases in which the parotid gland parenchyma is lacerated, the parotid capsule should be repaired and a pressure dressing with or without an intrawound drain is important to prevent postinjury sialocele.

Concomitant injury of the facial nerve is likely based on its anatomic proximity. Buccal and zygomatic branch injuries may be less disfiguring long-term owing to extensive cross-innervation and arborization. (Figure 26-19) Temporal-frontal and marginal mandibular branch injuries, however, require microsurgical repair (Figure 26-20) if, as noted in the examination, the injury is proximal to the lateral canthal vertical.[34]

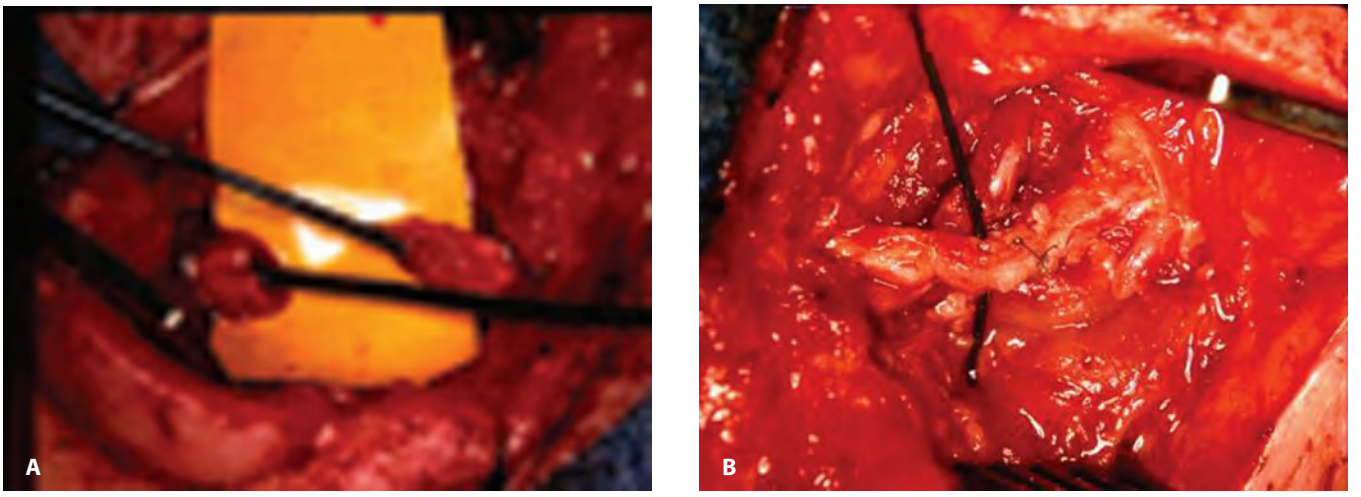


FIGURE 26-17. Complex cheek wound. **A,** Transected Stensen's (parotid) duct with lacrimal probes in severed ducts. **B,** Repaired duct over an indwelling 18-gauge catheter that is sutured at the intraoral papilla.



FIGURE 26-18. Avulsive dog bite wound. **A,** Avulsive cheek wound with evident ischemia and distal facial nerve transaction. **B,** Retracted avulsed cheek segment with evident intact parotid duct (x). **C,** Repaired cheek wound. **D,** Six months postoperative wound repair.



FIGURE 26-19. **A,** Right facial nerve injury involving the buccal and zygomatic branches with obvious weakness **B,** Spontaneous recovery of facial nerve function 6 months after injury.

Deep contamination from embedded foreign bodies should be treated with thorough exploration and vigorous irrigation with a syringe and catheter to the depth of the wound. Owing to the possibility of serosanguineous fluid accumulation, dead space, and contamination particularly from environmental or agricultural substances (Figure 26-21), a drain should be placed and daily irrigation carried out until the wound is clinically clean, after which time formal closure can be undertaken.

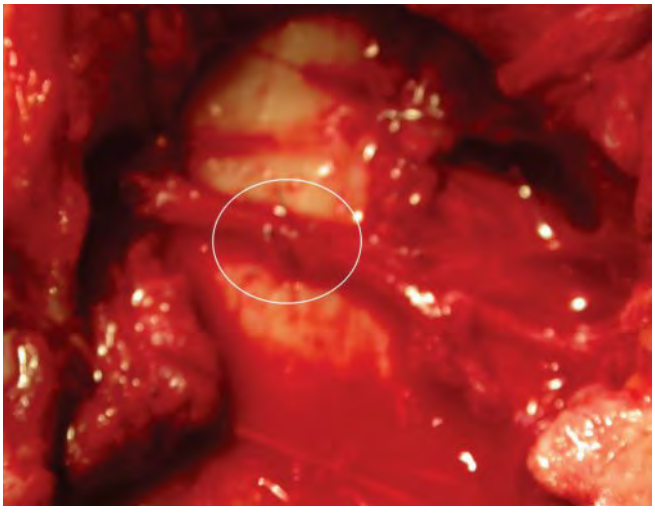


FIGURE 26-20. Temporal-frontal branch injury with microneural repair.

The Lip and Oral Cavity

Lacerations to the lip may occur as a result of a foreign body but often are secondary to the dentition piercing the tissue. A thorough examination must evaluate for embedded teeth within the tissue, and radiography may be necessary to confirm the absence of teeth or the presence of foreign bodies. Washout of lip and oral wounds must be meticulous owing to the potential contamination from saliva. Sequential closure of the lip mucosa, orbicularis oris musculature, and the skin is carried out with precise alignment of the vermilion border and white roll. Tissue adhesives work well in the lip region provided there is good tissue approximation deep to the skin. Parents should be informed of possible late mucocele formation after lip injury.

Dental trauma is a common cause of intraoral injury and examination should rule out embedded or displaced tooth fragments. Plain films are beneficial to evaluate for teeth and foreign bodies, and a chest film may be warranted to evaluate for foreign body aspiration. Floor-of-mouth and tongue



FIGURE 26-21. Penetrating foreign body contamination in facial wound. **A,** Deep wound with a buried foreign body (tree branch). **B,** Removal of a foreign body from a deep cheek wound. **C,** Closure with Penrose drain in a contaminated cheek wound.

lacerations are especially worrisome owing to the potential of delayed edema and airway issues. Also, seemingly benign palatal injuries to the soft palate and oropharynx can result in thrombus propagation through the pharyngeal anastomoses with the internal carotid artery and potential neurologic sequelae. Close observation is recommended for such injuries in the pediatric population.

WOUND CARE ADJUNCTS

Wound Support

Wound support tapes (Steri-Strips) are best utilized in conjunction with suture closure as an additional bolster and pressure dressing. The critical point with Steri-Strips as the primary closure modality is that the area must be kept dry and the strips should be reinforced if any laxity or loosening is noted to prevent increased tension across the wound and more resultant scarring.

Studies have demonstrated similar cosmesis, decreased treatment time, and improved parental perception of pain with the use of tissue adhesives (cyanoacrylate) compared with the traditional use of suture for simple lacerations.[35] As for any wound, it should be appropriately cleansed and débrided and with deep lacerations, the dead space still needs to be obliterated with sutures. Application to the skin of the tissue adhesive should be in a layered fashion with a scant amount used for the initial layer to avoid excess seepage into the wound and an increased likelihood of wound complications. Additional layers should be applied once the skin is sealed.

Although absorbable sutures tend to increase the tissue inflammatory response via enzymatic degradation in natural materials and by hydrolysis in synthetic materials, use in the pediatric population is advantageous. Studies have demonstrated minimal to no significant changes in cosmesis compared with closure of wounds with nonabsorbable suture.[36] Importantly, in the pediatric population, use of absorbable suture eliminates the need for removal and is more time efficient.

Antibiotic Ointments

Topical ointments maintain a moist wound and prevent contamination from common surface bacteria, resulting in

improved wound healing and more rapid reepithelialization. However, allergy can prohibit its use and, in such cases, white petrolatum jelly has been evaluated as an alternative in allergic patients, demonstrating maintenance of a moist wound without a significantly increased risk of infection. Topical corticosteroid-containing ointments may be utilized for short periods of time to decrease the exaggerated hyperemic and inflammatory response that will be observed in many children during wound healing.

Silicone Agents

Topical application of silicone gels or sheets on keloid scars hydrates the tissues (Figure 26-22), applies pressure, and regulates dermal fibroblast proliferation and excess collagen production, preventing or reducing keloid and hypertrophic scar formation.[37] The exact mechanism of action is unknown, however, some studies hypothesize manipulation of local ionic charges or a decrease in production of "pro-inflammatory" substances. These agents are typically used in the late wound healing stages and remodeling phases of scar maturation. Constant pressure results in tissue ischemia, release of metalloproteinase, decreased macroglobulin production, and improved scar hydration, which discourages hypertrophy and induces remodeling, resulting in a softer and flatter scar. Although these adjuncts are beneficial, long-term compliance with children is the limiting factor to efficacy.

Massaging hypertrophic scars has demonstrated marginal effects on the vascularity, pliability, and height of the scars.

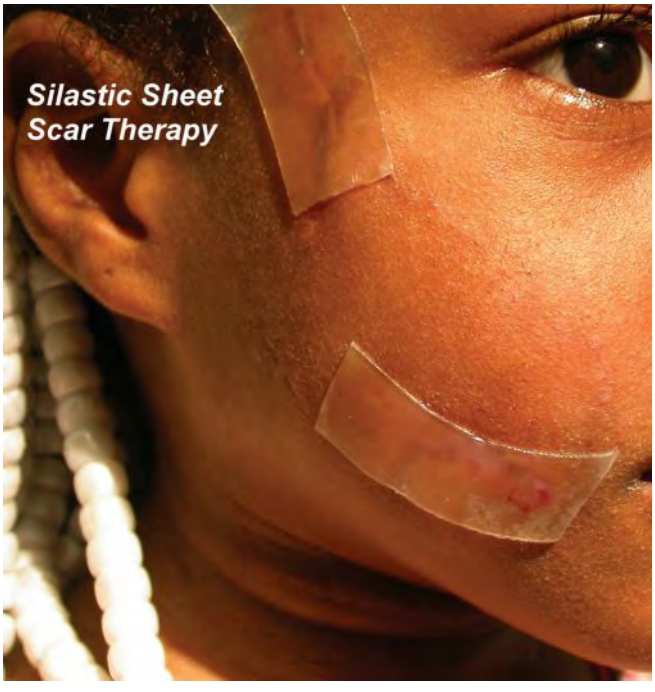


FIGURE 26-22. Self-adhering silicone pressure sheets are useful to decrease hypertrophic scar formation in children, particularly over areas of convexity, as in the malar or cheek area as demonstrated in this young child.

Frictional rubbing should be avoided because it can actually cause hypertrophy but a "kneading" type massage may provide benefit in conjunction with use of a hydrating agent such as lanolin lotion.

DOG BITES

The likelihood of a child sustaining a dog bite during his or her lifetime is reported to be almost 50%, and half of all bites are to the head and neck region. The cause is multifactorial, but in general, the pediatric population is closer to ground level, more inexperienced around animals, and thus, more likely to provoke an attack.[38] Most dog bites in children are from animals known to the family.[39] Because most of the victims are children, the physical and emotional sequelae may be long term.

Profuse pulse irrigation of wounds with conservative débridement is critical to reducing the chance of wound infection and optimizing the aesthetic outcome. Dog bites uniquely contain *Pasteurella multocida* and *Capnocytophaga canimorsus* in about 25% of cases, as well as mixed anaerobes, *Streptococci*, and *Staphylococcus aureus*.[40] Tetanus immunization records and rabies exposure must be considered in the comprehensive treatment of the dog bite patient. Passive immunization with human rabies immunoglobulin provides immediate protection with a serum half-life of 21 days. Immunoglobulin (20 IU/kg) is dispensed with as much of the dose as possible infiltrated in and around the wound, with the remainder given intramuscularly in the gluteal region, using a needle long enough to ensure an intramuscular injection. Active immunization via the vaccine takes 7 to 10 days to induce an active immune response, with immunity lasting approximately 2 years. The vaccine is placed in the deltoid or deep thigh with a dose of 1 mL at 0, 3, 7, 14, and 28 days.[41] Domesticated dogs with a known vaccination status should be quarantined for 10 days, and postexposure prophylaxis can be withheld unless signs of rabies develop in the quarantined dog. Prophylactic antibiotics should be prescribed for 7 days with extended penicillins or doxycycline or erythromycin as alternatives in penicillin-allergic patients. The parents should be informed of the intense inflammatory response that will result after a bite injury. This typically resolves 5 to 7 days after injury with prolonged lymphedema often observed.

PEDIATRIC FACIAL FRACTURES

Estimates suggest that 1% to 15% of facial fractures occur in the pediatric age group. Fractures of the facial skeleton in children are less common than in the adult, and only about 1% of facial fractures occur in patients younger than 5 years. This is due to the elastic nature of youthful bone and the smaller facial unit in children that may be exposed to injurious forces. Also, the anatomic uniqueness of the midface in children—multiple unerupted teeth and small or undeveloped sinuses—buttresses against forces directed at the midfacial region,

TABLE 26-2. **Age and Facial Fracture Order of Incidence by Region**

	Infant	Child	Adolescent
Skull	1	2	5
Midface	5	5	3
Nasal	4	3	2
Orbit	3	4	4
Mandible	2	1	1

distributing these forces to surrounding areas and preventing actual fracture or fragmentation at the point of impact.

At birth, the cranium occupies roughly half of the craniofacial unit and, thus, the skull (frontal region) is more frequently injured or fractured than other facial bones. This tendency continues until approximately 6 to 7 years when a reversal in fracture ratios is seen between the skull and the facial bones. This reversal corresponds to the development of paranasal sinuses, elongation and forward projection of the face beyond the frontal area, and the eruption of adult teeth in the anterior jaws.

Several studies have evaluated facial fracture incidences and patterns in childhood.[42–46] When summarized (Table 26-2), the trends observed previously can be further broken down into subunits of the facial skeleton and age-corresponding fracture patterns.

Fractures of the skull predominate in the very young and drop off significantly as the facial area develops and becomes more prominent (compare the adult and pediatric skulls in Figure 26-23). The mandible, being a relatively more exposed area during most of childhood, appears to be a target of injury during most of the pediatric developmental period.[44] During later childhood and adolescence, fracture patterns are also affected by environmental influences such as increased risk-taking behavior observed in older, particularly male, children, and more recreational or sport-related activities that result in jaw and nasal injuries.[47]



FIGURE 26-23. Adult and child skulls. Note the difference in upper, mid, and lower facial skeletal proportions. Small paranasal sinuses and multiple unerupted teeth in children contribute to midfacial skeletal stability and increased resistance to fracture forces and displacement.

Diagnosis of Facial Fractures

Although fractures are not as common as in adults, a high index of suspicion of underlying skeletal injuries needs to be exercised by the clinician when evaluating the injured child. Mechanism of injury is very important because this may indicate what kind and type of force has been imparted to the craniofacial unit. There will be typically an extensive component of soft tissue injury, both open wounds and edema, that may mask underlying fractures. Also, what may appear as fracture displacement, particularly in the upper nasal region (Figure 26-24), is the voluminous swelling young children develop owing to the rich and responsive lymphatic and vascular beds. After several days, the swelling resolves and the typical greenstick fractures return to their anatomic positions much quicker than in the older adult patient.

Diagnostic imaging is fundamental to properly evaluate facial injuries in children, therefore, computed tomography (CT) scans of the head and maxillofacial units are necessary. The contemporary high-resolution scanners with less radiation exposure afford the kind of thorough imagery necessary for pediatric facial fracture management. One must keep in mind and correlate clinical findings when reviewing CT images in young children because reports of fractures may rather be wide sutures or diastasis in an existing anatomic suture and not a frank fracture.[48] On plain skull films, sutures are not smooth but corrugated and irregular in appearance whereas fractures have smoother margins with clear separation (Figure 26-25). Conversely, there may be traumatically widened sutures, particularly in the skull of young children, that represent true injury and result in hematoma formation or underlying dural tear and must be scrutinized in the early phase of acute management.[49] The underlying dura may become injured or entrapped resulting in the phenomenon of "growing skull



FIGURE 26-24. Extensive swelling over the nasofrontal region often appears to be fractured with laterally displaced nasal-orbital bones.

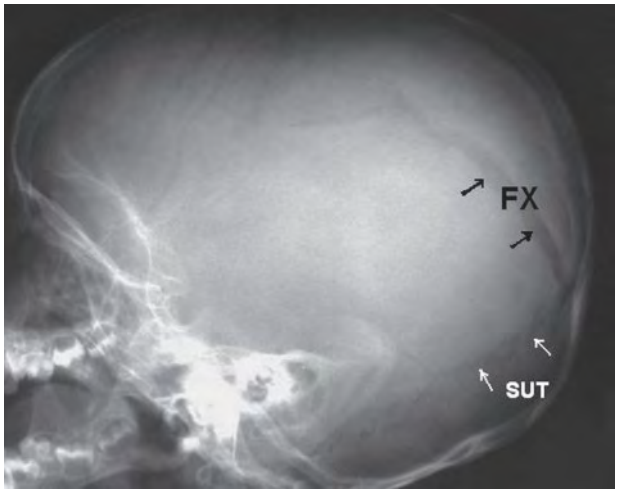


FIGURE 26-25. Plain radiograph demonstrates a linear fracture (FX and *black arrows*) with smooth margins and clear separation as opposed to a normal suture (SUT and *white arrows*) with irregular margins.

fracture" that forms from an underlying leptomeningeal cyst associated with a previous skull fracture.

ANTERIOR CRANIAL FRACTURES

Frontal Sinus

As noted previously, skull fractures are relatively uncommon, particularly as children age. The frontal bone thickens with established cortices and a diploe space that absorbs impact forces quite well before a fracture results. The frontal sinus represents an area more susceptible to fracture and fragmentation as the bone thins around the sinus cavity. The frontal sinus generally becomes radiologically evident at approximately age 7 years in most children, though pneumatization is under way via the ethmoids by 12 to 14 months.[50] Higher kinetic blows to the upper naso-orbital frontal region will sometimes result in frontal sinus and associated cranial or orbital roof fractures. Again, CT scans will document such fractures and indicate whether fragmentation involves vital areas, specifically the drainage system of the nasofrontal duct and orbital roof fractures, which approach the optic canal region.

Frontal sinus fragmentation with displacement of bone fragments into the sinus or extending intracranially are nearly always seen in older children or adolescents because their frontal sinus has been developed to some extent. Neurosurgical consultation should be obtained if there is intracranial extent of injury. Studies have shown that significant frontal sinus fractures in children are often accompanied by intracranial injury or ocular trauma and are also associated with a higher pediatric injury severity scale and poorer outcomes in recovery than what is observed in adults.[51,52]

Generally, contemporary management of frontal sinus fractures in children is treated conservatively with either

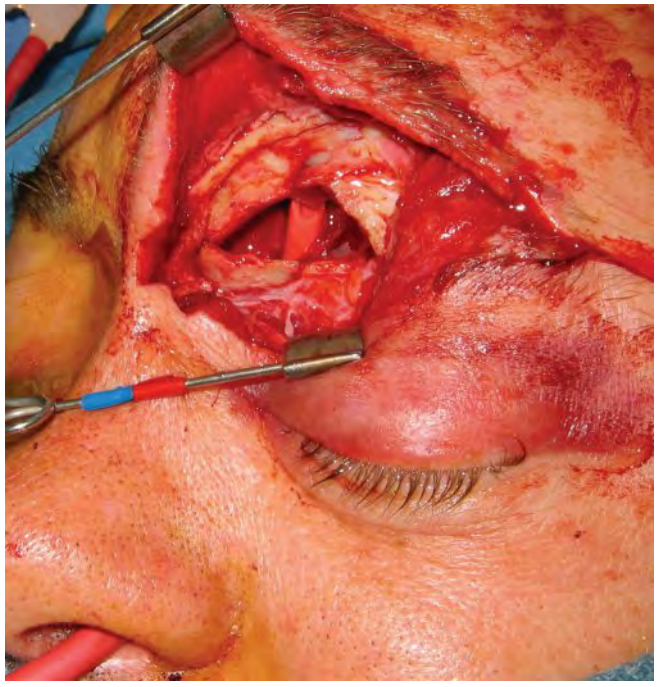


FIGURE 26-26. Frontal sinus preservation with repair and drainage catheter placed via the nasal cavity into the nasofrontal duct.

observation or fracture reduction and sinus drainage procedures.[51,53] Minimal fracture displacement will usually heal uneventfully, but the child should be followed throughout development for onset of sinus disease or worsening conditions in chronically diseased sinuses. Antibiotics are not necessary unless there is a history of chronic sinusitis or surgery is undertaken. Again, current trends maintain sinus cavity integrity with anatomic restoration through fracture reduction and fixation.[54] If sinus drainage is compromised, then endoscopic augmentation of the nasofrontal drainage can be performed[53–55] or a drainage catheter may be placed through the nasofrontal duct for a period of several days or weeks (Figure 26-26). This catheter can be accessed for periodic lavage of the sinus to maintain the physiologic dynamic of nasosinus health.

Frontal sinus comminution occurs less frequently in the pediatric patient than in the adult.[45,51] However, if this occurs (usually in the adolescent) and sinus health integrity is questionable, then obliteration may be undertaken as in the adult patient. In small frontal sinuses, usually stripping the mucosa and obliterating the ductal openings will result in cavity bony fill. If the posterior wall is comminuted, one may remove the wall, repair any dural tears, and seal off the nasofrontal recess with a local pericranial flap (Figure 26-27). This will allow the cranial contents to "herniate" forward and obliterate any retrofrontal space; this will take place over several days. In frontal sinuses larger in volume (typically adolescent males), fat or autogenous bone may be utilized to obliterate the cavity space after the ducts and mucosa have been eliminated.

Again, most frontal sinus fractures can be addressed conservatively in the majority of children; close follow-up with

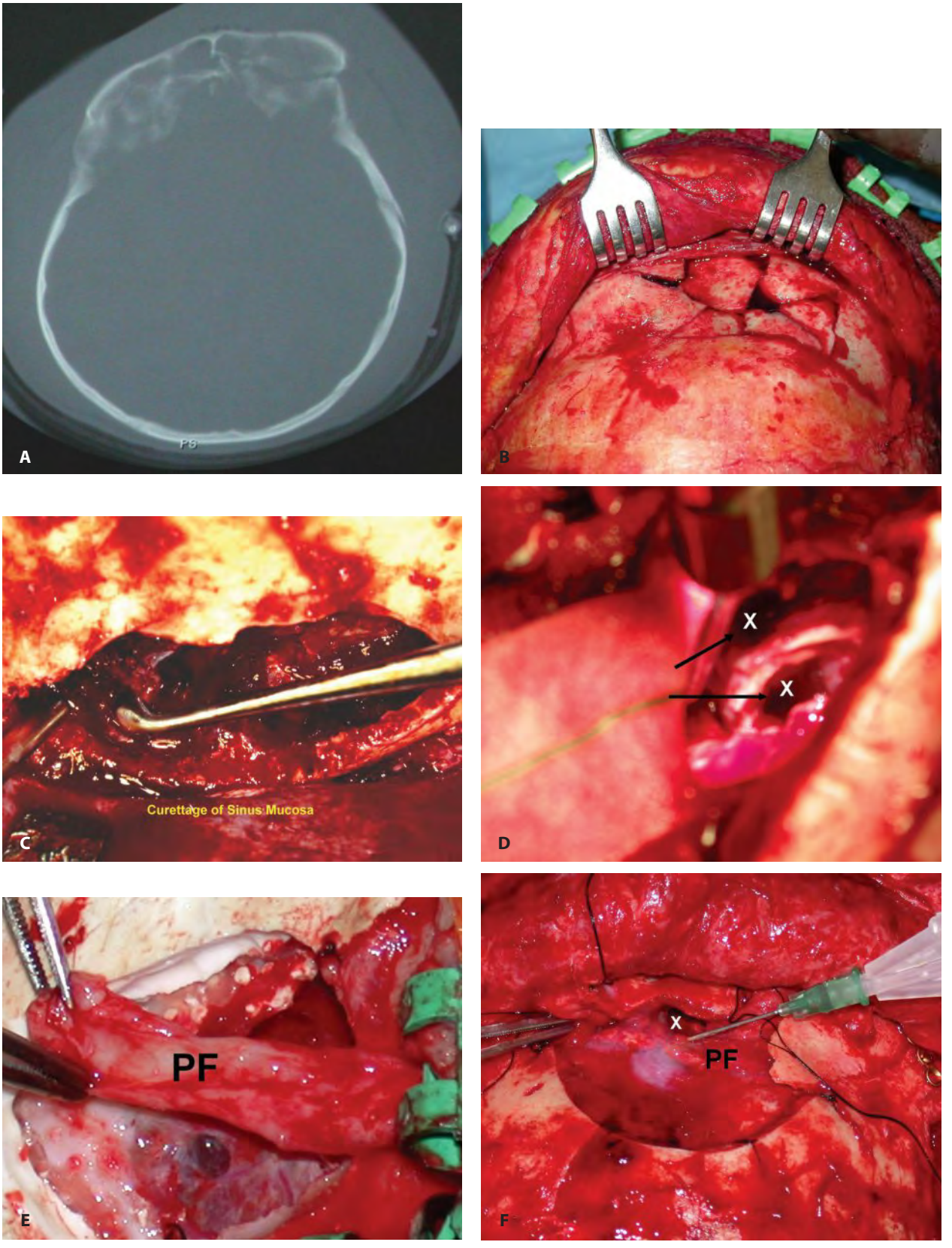


FIGURE 26-27. Frontal sinus fractures in a 13-year-old boy. **A,** CT (axial) scans of frontal sinus fractures. **B,** Frontal bone and sinus comminution. **C,** Curettage of the entire frontal sinus mucosa. **D,** Nasofrontal ducts (X) as they interface with the ethmoidal air cells and upper nasal cavity. **E,** Pericranial flap (PF) is elevated and placed over the frontal sinus floor and nasofrontal duct orifices. **F,** Fibrin glue is applied to the flap (PF) and sinus floor–ductal interface (X) to seal the sinus from the upper nasal cavity and ethmoids.

periodic CT scans to rule out sinus disease, including mucocele formation, is important.

Orbital Roof

Anterior cranial base fractures in children can be very serious with implications of intracranial injury, hematomas and CSF leaks, and vision impairment when the supraorbital rim and orbital roof are involved.[56,57] Studies have indicated that pediatric injury severity scores above 15 are often associated with significant orbital trauma including fractures, ocular adnexa injury, and vision compromise or blindness.[58] When orbital roof fractures are identified, the clinician must radiographically follow the extent of the fracture posteriorly to rule out optic canal involvement. This may necessitate obtaining dedicated coronal views at 1-mm slices at the orbital apex to determine whether the optic canal contents are involved. Subsequent swelling, pressure, and ischemia of the optic nerve or contents may result, which can be confirmed through serial examinations, if possible, by an ophthalmologist.[57] If swelling worsens with decreased vision, then emergent decompression and medical management of papilledema may preserve the visual axis. Often, pediatric patients with entrapment may have none of the typical stigmata of an orbital fracture but will experience associated nausea and vomiting. This is consistent with a "white-eyed" orbital fracture, mostly seen in pediatric patients. In addition, unexplained bradycardia may suggest Aschner's phenomenon or activation of the oculocardiac reflex.[59]

Implosion of orbital bone intracranially or into sinus cavities usually is greenstick in nature in children. For this reason, most orbital wall fractures will improve over several days of observation as the pieces self-reduce to their anatomic positions.[60] In very young children with growing brains, pulsatile mechanisms can result in both displacement of fractures and resorption of free bony pieces; therefore, orbital reconstruction should attempt to separate the intraorbital and intracranial contents when continuity defects occur.[61] Surgical intervention is warranted only with adnexal impairment by fracture fragments. Again, bone fragments displaced superiorly and intracranially may result in dural tears and these require neurosurgical repair. If orbital volume has been lost and there appears to be little physiologic restoration of volume, then reconstruction should be undertaken with resorbable mesh or autogenous materials such as bone or cartilage (Figure 26-28). The diploe space develops by age 3 or 4 years in most children and may be split to use for orbital wall reconstruction. Rib can also be split, shaped, and placed in the orbit for extensive wall defects.

Upper Facial Fractures

NOE Complex

Naso-Orbital-Ethmoid (NOE) fractures are rare in children but have increased in prevalence owing to increased survival after high-velocity injuries seen in motor vehicle collisions, likely as a result of enhanced safety regulations.[46] The NOE complex consists of the medial orbital walls, the nasal bones, and the nasal projection of the frontal bone. Such fractures become more prevalent with the development of the paranasal sinuses, as is the case with frontal sinus fractures and maxillary fractures.

Physical examination demonstrates telecanthus and instability of the medial canthal ligament and central bony fragment. Clinical examination includes the bowstring and Furness tests as well as direct intercanthal measurements. Intercanthal distances are more variable in children, but on average, measurements are approximately 22 mm in infants and increase gradually until adulthood, when approximately 35 mm is the norm. An acute nasofrontal angle may be noted and ophthalmic nerve hypoesthesia seen.

Surgical management is complex and often requires secondary reconstruction. Initial emphasis is placed on restoration of orbital volumes, re-creation of nasal contours, overcorrection of the intercanthal distance, and restoration of midface projection.[62] Access is optimized via a coronal incision, unless a significant laceration preexists in the area. Transnasal fixation with nonabsorbable sutures is a critical step in surgical reconstruction if the medial canthal tendon insertion is disrupted. Overcorrection is suggested and external bolsters are helpful to support the tissues and prevent pseudotelecanthus.

Nasal Complex

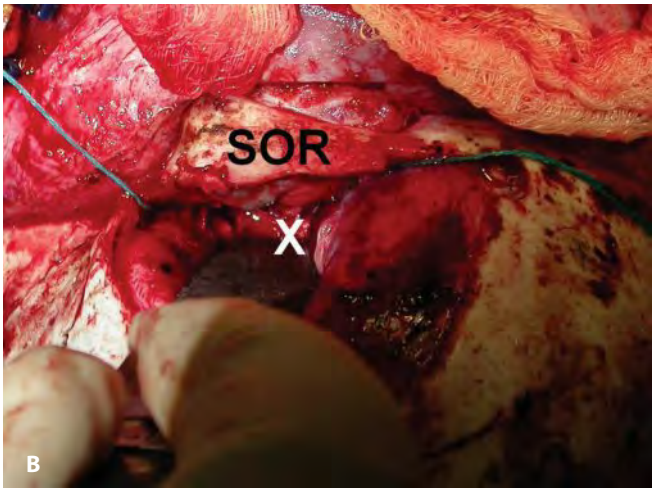
Nasal fractures are the most common facial fracture in both adults and children owing to its prominent exposure.[43,44,46] The mechanism of injury is useful in focusing the clinical examination because a glancing or tangential blow will result in different injuries than a direct frontal force. Patients will often present with considerable nasal bleeding that should be addressed immediately. Clinical examination is usually diagnostic but may be unreliable and difficult in uncooperative children with extensive swelling. Plain radiographs are of minimal assistance and a maxillofacial CT scan should be obtained only if further modalities are required or more complex fractures coexist.

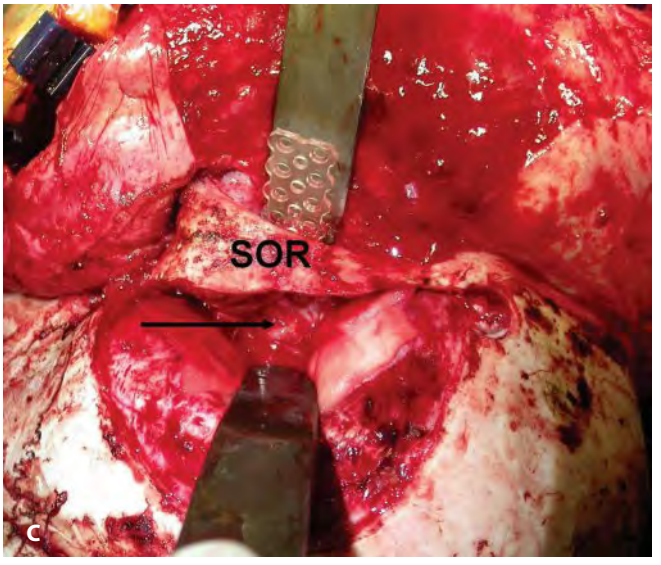
Surgical intervention is required for drainage of a septal hematoma, cosmetic deformity, and functional impairment.[27,63] Generally, less is more as it relates to pediatric nasal complex fractures because more aggressive surgical intervention may adversely affect the nasoseptal growth center. Closed reduction is typically attempted first with open reduction reserved for complex fractures with loss of projection and stability. After surgical correction, extranasal splinting is ideally accomplished with a molded thermoplastic splint and antibiotic-impregnated intranasal packs, absorbable cellulose, or septal splints. Systemic prophylactic antibiotics for prevention against toxic shock syndrome and endocarditis should be utilized during the packing period.[29,30,64]

Orbital Floor

The orbital floor bone is extremely thin, and a direct blow to the globe usually results in dissipation of forces that fractures









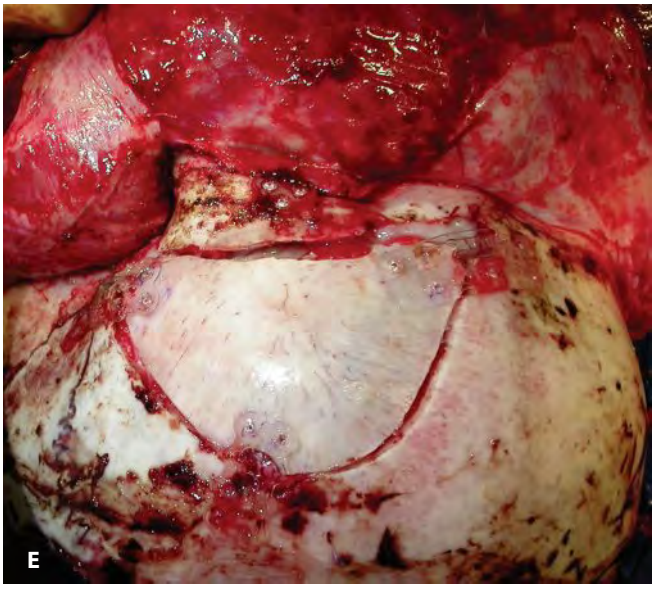


FIGURE 26-28. Severe orbital crush injury in a 4-year-old. **A,** Periorbital disruption and orbital rim fractures. **B,** Superior orbital rim (SOR) and comminuted roof (X) fractures with communication of periorbita and dura. **C,** Reduction of orbital rim (SOR) and roof fractures with resorbable plate *(arrow)* placed across the roof defect and lagged cranial bone (split) to reconstruct the roof. **D,** Split calvarial bone for reconstruction of orbital roof or wall defects. **E,** Reconstructed fronto-orbital fractures with resorbable fixation and autogenous bone grafts.

the floor of the orbit, resulting in a blow-out fracture or, more commonly in children, a blow-in fracture.[56,57,60] Owing to the pliable nature of bone in children, the orbital floor bone often recoils, resulting in a "trap door" phenomenon involving the inferior rectus muscle and associated periorbital tissues. Such a finding requires prompt surgical intervention to relieve the entrapment, allowing for free range of motion of the eye and prevention of long-term functional sequelae. Alcala-Galiano and coworkers[65] reported up to 24% associated ocular injuries with orbital fractures in children; therefore, ophthalmologic consultation should be part of the treatment algorithm. Unless there is vision loss or compromise, permitting edema to dissipate will allow for a more thorough and precise examination.[17,19] Diplopia on upward gaze or restriction of extraocular movements (Figure 26-29) should alert the clinician of entrapment of periorbita, which is usually confirmed on CT coronal imaging. Thin-slice CT imaging is critical to correlate with the clinical examination because subtle findings may be appreciated only on such high-resolution scans.

Surgical repair of orbital floor fractures should be considered for (1) diplopia secondary to entrapment, (2) enophthalmos greater than 2 mm after edema has dissipated, and (3) CT scan–measured orbital floor defects greater than 1 cm^2.[66–68] For cases that do not clearly require surgery, the surgeon must follow the patient closely because pediatric patients heal quickly. Surgical intervention is best performed within 7 to 10 days in pediatric patients.[69] Isolated floor defects in children should be reconstructed with either autogenous materials (split calvarium, rib) or absorbable mesh or membranes.[66]

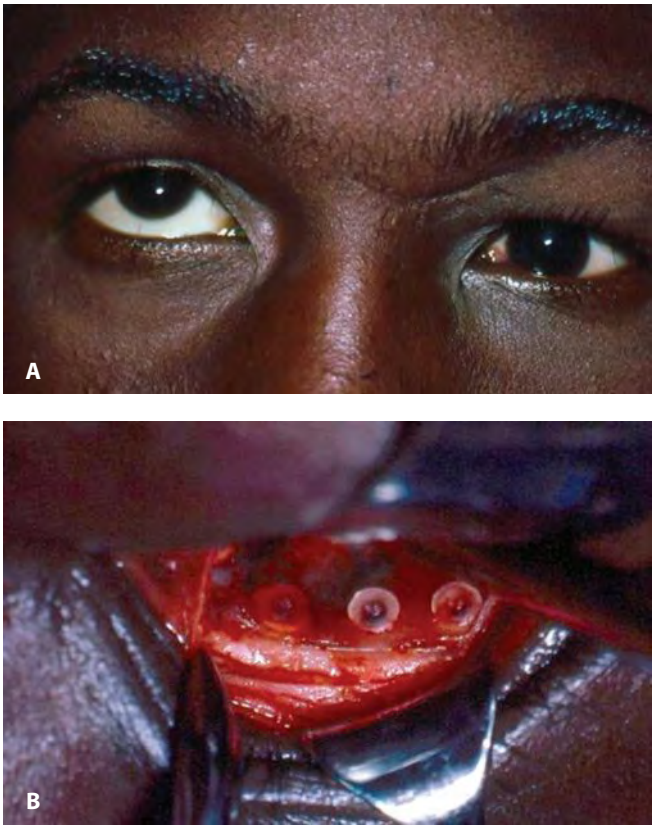


FIGURE 26-29. Orbital floor fracture with entrapment in a 9-year-old child. **A,** Note restricted left eye on upward gaze due to entrapped inferior periorbital soft tissue. **B,** Orbital floor repair with resorbable fixation plate.

Zygomaticomaxillary Complex

The zygomaticomaxillary complex (ZMC) fracture is uncommon in younger children but, as with other pediatric facial fractures, the incidence rises with the development of the paranasal sinuses and the maxillary sinuses in particular as well as the increased prominence of the zygoma with growth. Findings include maxillary nerve hypoesthesia, malocclusion, trismus, subconjunctival hemorrhage, increased transverse facial dimension, and loss of malar projection. Significant displacement results in increased orbital volume and enophthalmos that may initially be camouflaged by edema especially true in children. Ophthalmologic assessment is critical to establish a baseline ocular examination, visual acuity, extraocular movements and evaluate for globe injury (e.g., afferent pupillary defect, hyphema, anterior chamber violation).[60,61] As with isolated orbital fractures, the etiology of visual acuity abnormalities must be determined before facial fracture repair and positive findings require prompt attention.

Nondisplaced or minimally displaced fractures can be managed conservatively, but cases with coronoid impingement, enophthalmos, or significant fracture displacement require intervention.[42,61] Repair must be undertaken within 7 days in young children or healing will ensue, often necessitating refracture (not recommended owing to possible splintering, tearing of maxillary arterial and pterygoid plexus, and arteriovenous fistula phenomenon) or osteotomies to mobilize the zygomatic complex. In pediatric patients, two points of fixation are usually adequate to stabilize the fractures and restore the facial projection.[70] Approaches include an intraoral Keene incision in the maxillary vestibule and an upper eyelid crease incision. Use of the brow incision should be minimized, especially in girls, owing to the potential for postoperative alopecia. A limited transconjunctival approach for concomitant orbital rim and floor repair is well tolerated by children. Conjunctival sutures should not be placed,; rather all sutures (absorbable fast-gut) should be placed well below the conjunctiva in the deeper tissue layers. Fractures are easily repaired with resorbable fixation (Figure 26-30).

Lower Facial Fractures

Maxillary Fractures

Le Fort midface type fractures are rare until aeration of the maxillary sinuses occurs. After the age of 6, maxillary fractures occur more frequently but the elasticity of the bone and the mixed dentition limits displacement. Generally low-midface or maxillary fractures occur as a part of a complex

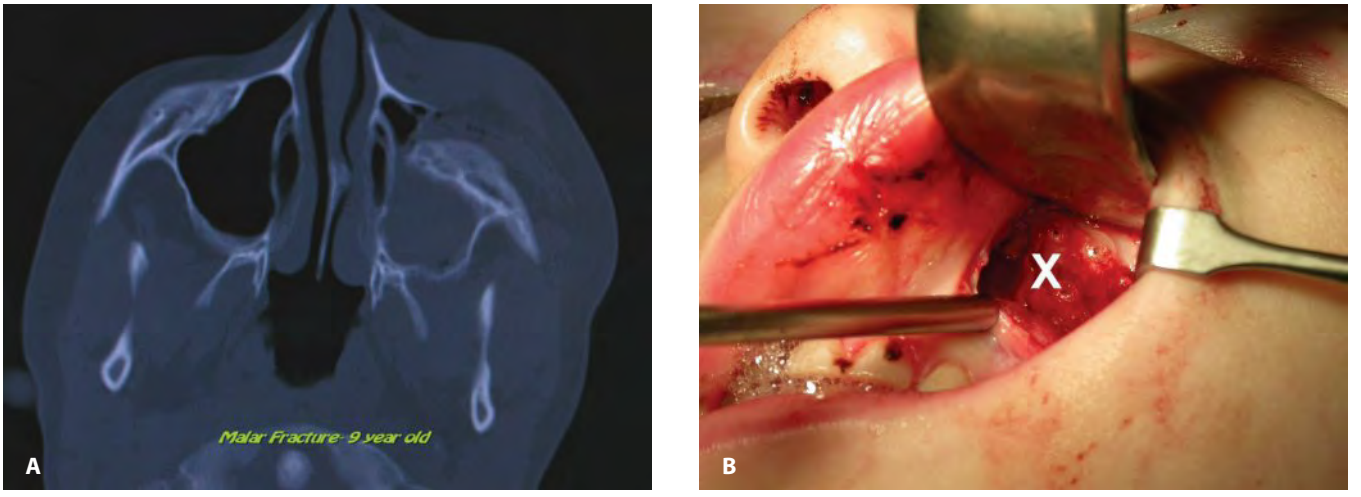


FIGURE 26-30. Zygomatic-maxillary complex (ZMC) fracture in a child. **A,** Axial CT scan demonstrates displaced zygoma. **B,** Intraoral photograph of a resorbable fixation plate (X) at the ZMC buttress.

of fractures in children.[43] Findings include maxillary nerve hypoesthesia, malocclusion, and maxillary mobility. CT imaging demonstrates transmaxillary fractures and separation of the pterygoid process of the sphenoid bone from the maxilla.

Correction of midface projection and restoration of occlusal harmony are the main goals of treatment. Simple maxillary fractures can usually be treated with maxillomandibular fixation (MMF) for 1 to 2 weeks. Vigilance must be exercised to guard against distraction forces placed across a fractured maxilla during MMF that may result in vertical facial increase.[68] Open reduction should be reserved for significant displacement or concomitant facial fractures. In children with unerupted teeth, high Le Fort fractures can be fixated with resorbable fixation once occlusion is established. Low or dental component fractures will necessitate a combination of dental-wire bonding and occlusal splint reduction of the maxillary arch for 2 to 3 weeks.[71]

Mandible Fractures

Lower jaw fractures are the second most common pediatric facial fracture, generally involving the condyle or subcondylar region.[72] The mandible's U-shaped form and mixed dentition have a direct effect on fracture patterns. Mandible fractures in young children are often greenstick-type fractures and tend to be single-site fractures as opposed to multiple sites in the adult.

Findings associated with pediatric mandible fractures are mandibular nerve hypoesthesia, malocclusion, trismus, and chin point deviation toward the side of fracture. Soft tissue swelling associated with jaw fractures in children should be a cause for concern regarding airway patency. Young children will typically swell more in response to injury, and symphyseal or body fractures may result in acute and extensive floor-of-mouth and tongue swelling; therefore, observation and monitoring of the child is important.

Radiographic analysis is essential for the full evaluation of the mandibular components and the bilateral temporomandibular joints. An orthopantogram is particularly helpful in evaluating the position of the inferior alveolar nerve and a composite image of the mixed dentition facilitates surgical fixation. Associated fractures require CT imaging of the maxillomandibular region.

The goals of treatment are to restore facial form, premorbid occlusion, and mandibular function and range of motion. For greenstick, nondisplaced mandibular fractures without malocclusion or other sequelae, a nonchew diet and limitation of activity can be instituted for treatment. Weekly examination to confirm return to normal form and function is important.

As a result of the shape of deciduous teeth and the mixed dentition phase in children, closed reduction via MMF may be challenging. Selective Ivy loops and interdental wiring and bonded brackets or wires may be utilized to accomplish closed reduction. Intermaxillary fixation screws may be used in older children. An acrylic splint also may be fabricated and ligated to the teeth to maintain fracture alignment. As noted previously, any child with MMF requires careful airway observation and astute caregivers who can monitor the child during the fixation phase. Such management should probably include at-home apnea/O_2 monitoring and suction devices.

Condyle

Intracapsular fractures are best treated with a soft diet and continued function and physiotherapy to prevent ankylosis owing to the significant osteogenic potential in children. Extracapsular condylar fractures are best treated with a nonchew diet and closed reduction with elastic guidance in younger children, allowing controlled function and comfort. MMF should be used for only 1 to 2 weeks because extended periods may result in prolonged trismus and ankylosis.[73,74] Open reduction has not been clinically proved to improve outcomes

in children and, thus, should be limited for cases of displacement of the condyle or the presence of bilateral condylar fractures and significant loss of the lower facial third vertical height.[75] Physical therapy should be instituted after brief immobilization to encourage restoration of the functional and physiologic dynamic of the immature condylar-ramal complex. Key parental participation in therapy modalities cannot be overstated.

Body and Symphysis

Brief periods of MMF will suffice for most mandibular body and symphyseal fractures in young children (<6 yr of age). Open reduction and internal fixation is reserved for selected young children and adolescents.[76–78] Again, resorbable plates and screws address most of the fixation needs in these fractures (Figure 26-31). It is felt by some that weakening and ultimate resorption of the fixation plates within 1 year will result in less growth disturbances and normalize the mineralization dynamic of the mandible.[69,77,79] Key principles include (1) a superior border plate or tension band plate is not necessary in the mixed dentition phase, (2) placement of monocortical screws, and (3) limited incision and dissection should be instituted (Figure 26-32).[77–79]

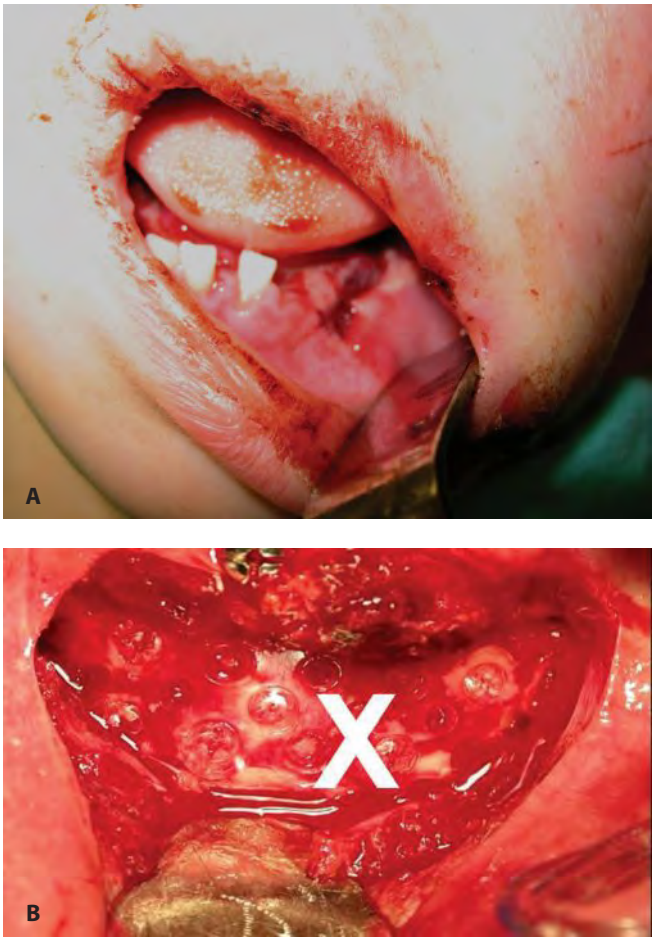


FIGURE 26-31. Mandible fracture in a child with mixed dentition. **A,** Intraoral photograph of a displaced left mandibular body fracture. **B,** Placement of a single resorbable fixation plate across the mandibular body fracture.

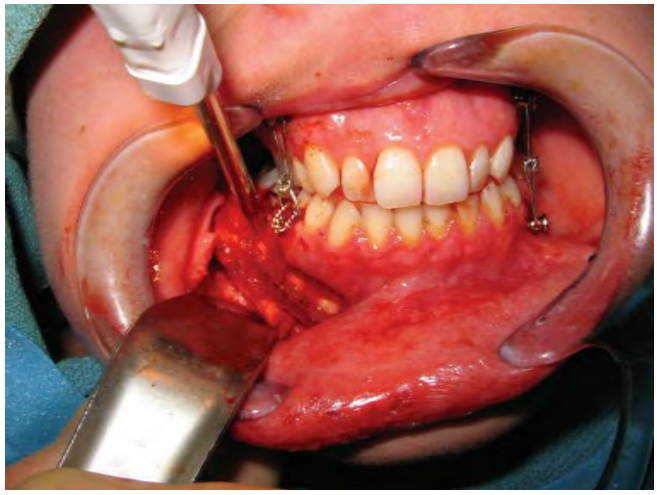


FIGURE 26-32. Right mandibular body fracture in a 14-year-old with temporary intermaxillary fixation and a single resorbable fixation plate.

Dentoalveolar Injuries

Dental trauma is a common occurrence at all ages as a result of dentoalveolar and jaw exposure, falls, assaults, and sporting trauma. The anterior primary dentition has shortened root structure, lending itself to fracture, intrusion, concussion, subluxation, partial avulsion, and complete avulsion injuries.[80] Concomitant alveolar segment bony fractures may coexist with tongue, mucosa, and lip lacerations. Comprehensive evaluation should attempt to account for missing and possibly swallowed or aspirated teeth, especially in obtunded patients. CT imaging is best if associated facial injuries are noted. Plain films are useful to rule out the presence of foreign bodies within wounded soft tissues. Fractured, luxated, or avulsed teeth are optimally treated by a pediatric dentist, but in general, deciduous teeth that are avulsed should not be re-implanted because damage to the underlying adult teeth may occur.[71] Significantly luxated deciduous teeth should be considered for extraction to eliminate any risk of sudden exfoliation and aspiration. Luxated or avulsed adult teeth should be repositioned and re-implanted, ideally within 60 minutes.[81] Stabilization should then be instituted with bonded wires or arch bars, with care taken to avoid extrusion of the unstable teeth. Dentoalveolar segment fractures can usually be reduced and stabilized with arch bars or wires, as in luxated or avulsed teeth injuries.[71,81,82] Relief of occlusion is generally recommended to avoid trauma to the healing periodontal ligament and dental structures. Larger alveolar segments may best be treated by plate and screw fixation, especially if teeth are significantly fractured or absent. A nonchew diet is critical and avoidance of contact activities is prudent. Interval evaluation by a pediatric dentist is suggested to evaluate for the need of space maintenance and longitudinal monitoring of dental development.

GROWTH DISTURBANCE

Fractures in children may affect growth and development. Generally, three main areas of growth may be affected by injuries: the orbital region, the nasomaxillary complex, and the condylar-ramal elements. Fractures of the orbital region in very young children may result in periorbital tissue atrophy and subsequent orbital volume loss, particularly in a crush injury.[83] After the age 5 or 6, this growth disturbance issue of the orbit diminishes.[69,84]

Injuries to the nasomaxillary complex, particularly the cartilaginous unit, may affect midface projection and elongation.[27,63] Surgical intervention at the time of injury probably does not confer physiologic protection from developmental perturbations in the nasomaxillary region. It is important to carefully and thoroughly review this with the parents and advise long-term follow-up during growth.

Condylar fractures involve the mandibular growth center and may result in long-term growth abnormalities via growth restriction and development of a malocclusion and lower facial third facial asymmetry or deformity. Also, degenerative changes are likely to occur, resulting in early-onset arthritis at the temporomandibular joint. Consequently, parental education is especially critical in the treatment process. Unlike adult condylar fractures, children have the capacity for condylar remodeling and complete recovery of form and function.[74] However, as children age, the ability to remodel and spontaneously heal is attenuated.

SUMMARY

Craniomaxillofacial trauma in children is an increasing, debilitating, and preventable entity in pediatric health care in America. There has been a shift of trauma incidence to the younger population owing to increased independence of children and risk-taking behavior among youth, particularly in adolescence, which puts children at risk for significant injury. Children require updated immunization status, and it is vital to have responsible caregivers who can partner with the surgeon and participate in trauma care and recovery.

Children require rapid intervention in trauma care, including facial injuries, to preserve tissue health and optimal results. Facial injuries are generally age-dependent in that cranio-orbital injuries are seen more in the very young whereas midface and lower face injuries are seen more frequently in the older and adolescent child. Particular attention to soft tissue management of the eye, nasal, and ear regions, including neurovascular entities, requires preservation of tissue, focused repair, support of repaired structures, and scar management modalities in order to obtain a good result in form and function. Fracture management in the very young has undergone recent change with the advent of resorbable fixation materials and techniques as well as the increased appreciation for skeletal response to trauma, particularly for long-term facial development. It is important to recognize that jaw function in young children must be preserved and allowed during the healing period and long-term growth assessed by regular follow-up with documentation. Adolescent fracture management mirrors that of adults, but follow-up for growth perturbations should be documented into the early adult years especially for mandibular condylar fractures.

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CHAPTER 27

Management of Panfacial Fractures

Patrick J. Louis, DDS, MD

Management of patients with multiple displaced and comminuted fractures can be extremely challenging not only for those who are inexperienced but also for experienced surgeons. Improper diagnosis, treatment planning, and sequencing produce inadequate results and can lengthen procedure time. However, with the availability of detailed imaging,[1–3] ridged fixation,[4–6] bone grafting techniques,[7–9] and proper sequencing,[4,10,11] outcomes can be optimized.

All facets of facial form and function are important, and one should strive to preserve them. The importance of proper occlusion cannot be underestimated because acute changes in the way teeth come together can be readily detected by the individual.[12] Such alterations can result in myofascial or temporomandibular joint pain.[13] Reestablishing the patency of the nasal cavity is important in the prevention of nasal obstruction and potential problems such as sinusitis and obstructive sleep apnea.[14,15] It is also required to establish the proper quality of speech.[16] Small changes in orbital volume can result in enophthalmos and/or diplopia.[17,18] The reestablishment of facial height, width, and projection is important for the prevention of facial deformities and for the psychological and social well-being of the individual.[19–21] No one of these factors can be considered more important than the other; together, they constitute the face and its associated functions.

In this chapter, discussion is presented on some of the historic perspectives, etiology, anatomic considerations, imaging, bone grafting, soft tissue resuspension, sequencing of treatment, and complications as they relate to the management of panfacial fractures.

HISTORIC PERSPECTIVE

Panfacial fractures are defined as those involving the upper, middle, and lower thirds of the face.[4] These complex injuries are fractures that involve the frontal bones, zygomaticomaxillary complex, naso-orbitoethmoid region, maxilla, and mandible. Complex facial injuries such as these are generally the result of high-velocity trauma.[22] Before the advent of rigid fixation techniques,[23–25] these fractures were treated with wire fixation and head frames.[26–28] With these techniques, it was difficult to establish and maintain the three-dimensional stability of the facial skeleton.

Several important advances in the management of maxillofacial trauma have resulted in improved outcomes. These include the development of high-resolution computed tomography, rigid fixation techniques, soft tissue resuspension, and primary bone grafting. All of these have made a significant impact on the diagnosis and treatment of panfacial injuries; each is discussed later in this chapter.

Etiology

Panfacial fractures result from motor vehicle collisions, assault, sports-related accidents, industrial accidents, and gunshot wounds.[22,29–32] Because gunshot wounds are addressed in Chapter 25, and because there is usually associated soft tissue damage causing them to generally require different principles of management, they are not discussed in this chapter.

ANATOMIC CONSIDERATIONS

Facial Buttresses

Many authors have described the buttresses of the face in both vertical and horizontal planes.[10,32–34] The vertical buttresses include the nasomaxillary, zygomaticomaxillary, and pterygomaxillary buttresses (Figure 27-1). The nasomaxillary buttress includes the maxillary process of the frontal bone and the frontal process of the maxilla, extending lateral to the piriform rim. The zygomaticomaxillary buttress is composed of the zygomatic process of the frontal bone, lateral orbital rim, lateral zygomatic body, and zygomatic process of the maxilla. The pterygomaxillary buttress includes the pterygoid plates of the sphenoid and maxillary tuberosities. Usually, the nasomaxillary and zygomaticomaxillary buttresses are reconstructed, but the pterygomaxillary buttress is not because of inaccessibility. The condyle and posterior mandibular ramus make up yet another buttress establishing posterior facial height.

The horizontal buttresses are also described as *anterior posterior buttresses*.[10] These include the frontal, zygomatic, maxillary, and mandibular buttresses (Figure 27-2). The frontal buttress is composed of the supraorbital rims and the glabellar region. The zygomatic buttress consists of the zygomatic arch, zygomatic body, and infraorbital rim. The maxillary and mandibular buttresses are composed of the basal bone of the maxilla and mandible arches.

None of these buttresses exists in a vacuum. Together, they give the facial skeleton its structural integrity. The bone is generally thicker over these described areas to neutralize the forces of mastication or impact. With the proper reduction of these buttresses, we are able to reconstruct the height, width, and projection of the face.

Key Landmarks

When there are multiple facial fractures involving the upper, middle, and lower face, reconstruction should be approached as a puzzle. Known landmarks and anatomy can be used to reconstruct more precisely those areas that have been damaged. Some key landmarks that may help in establishing the proper positioning of the facial skeleton include the dental arches, mandible, sphenozygomatic suture, maxillary buttress, and intercanthal region.

Dental Arches

When one or both of the dental arches are intact, they can be used as guides. For example, if the patient has suffered a Le

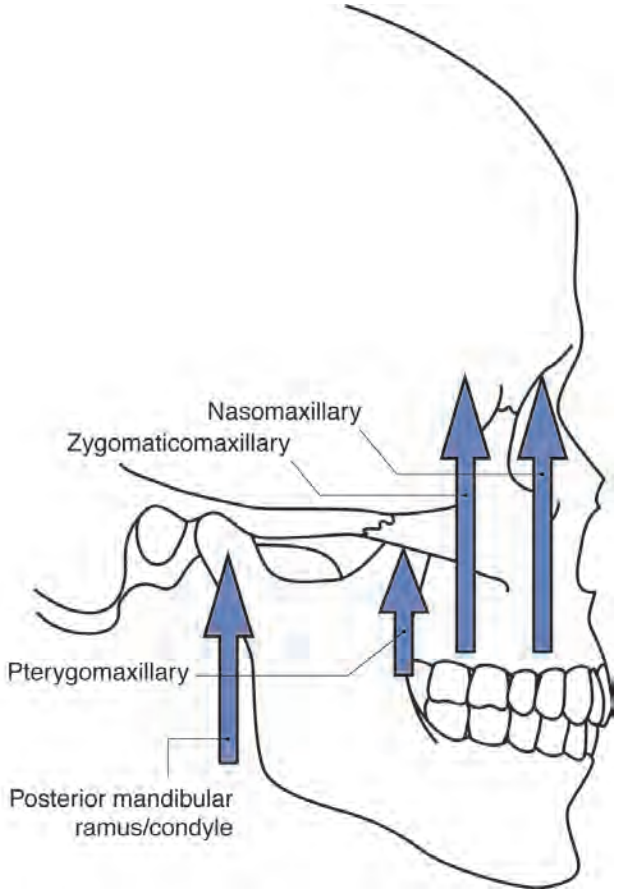


FIGURE 27-1. Vertical buttresses of the face.

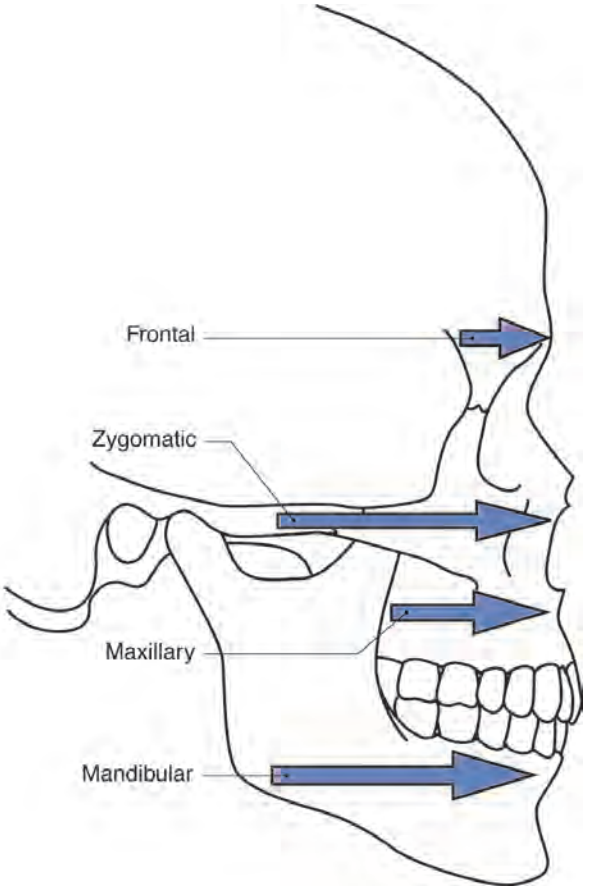


FIGURE 27-2. Horizontal buttresses of the face.

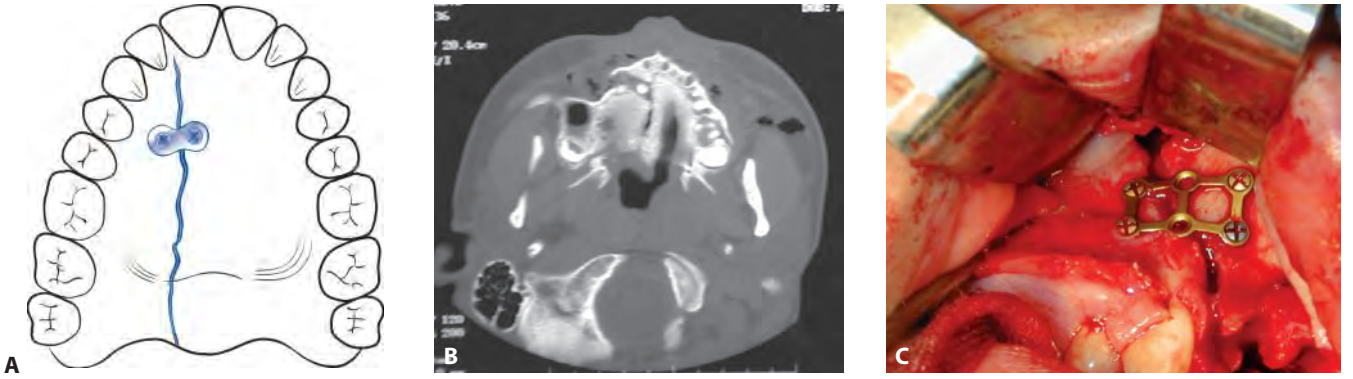


FIGURE 27-3. Fixation of palatal fractures. **A,** Reduction and fixation of a palatal fracture using a miniplate. **B,** Computed tomography (CT) scan, axial view, shows the maxilla with a palatal fracture. **C,** After reduction of the palatal fracture, a miniplate is placed above the alveolus to fixate the fracture.

Fort fracture but no midpalatal split, the maxilla, as an intact arch, can be used to set the mandibular arch and establish proper width. Particularly problematic is the situation in which there is a midpalatal split and the mandible is also fractured along the tooth-bearing region, with associated condyle fractures. This can easily lead to widening of the entire facial complex if these segments are not properly reduced. One approach to this problem is to reestablish the maxillary width by exposing the palatal fracture, then reducing and fixating the region (Figure 27-3).[34–37] This approach should not be considered the first step in the overall sequencing of panfacial injuries, but this technique works well when there is a solitary midpalatal fracture without comminution or avulsion. Owing to concerns about access, many surgeons chose to establish good arch form and fixate the fracture along the facial aspect of the alveolus while avoiding the roots of teeth (see Figure 27-3B and C). A second approach is to obtain impressions for fabrication of dental models. Simulated surgery can then be performed on the upper and lower casts and a surgical splint fabricated (Figure 27-4).[38,39] This is by no means a foolproof method when both the upper and the lower arches are fractured. The more severe the injury (i.e., multiple segments), the more difficult it is to establish a preinjury occlusion. If the patient has dental models of her or his preinjury occlusion from previous orthodontic or prosthetic rehabilitation, these can provide invaluable clues to establishing the proper arch form. A third option is to reconstruct the mandible because this is generally a robust bone that can undergo anatomic reduction if attention is paid to detail.

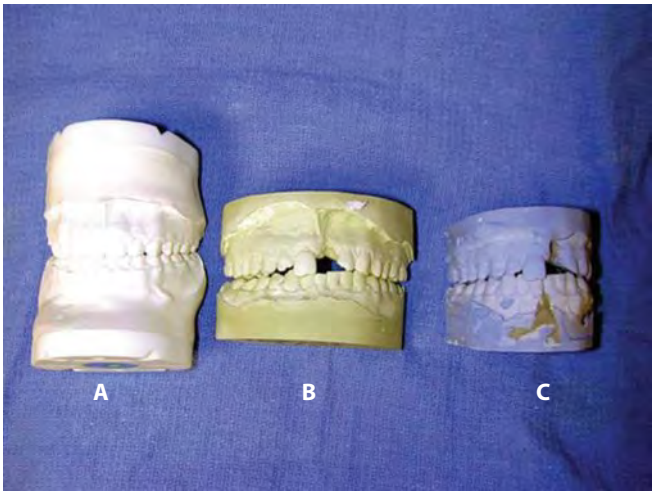


FIGURE 27-4. Dental models from one patient: postorthodontic models **(A),** post-trauma models **(B). C,** Model surgery has been performed on these casts using the postorthodontic models as a guide.

The Mandible

Anatomic reduction at the symphysis and/or body can be achieved with an extraoral exposure of the fracture. Such exposure allows for direct visualization of the inferior border and, to a lesser degree, the lingual cortex. The reduction of both the buccal and the lingual cortical surfaces before fixation yields better results (Figure 27-5).[40,41] When bilateral subcondylar fractures are present, they must be treated to establish the posterior facial height and facial width. When bilateral subcondylar fractures are present and there is an associated fracture along the symphysis and/or body region, the mandible may undergo splaying, with a resultant increase in facial width. The lateral pterygoid muscle attachment at the pterygoid fovea, as well as the lateral capsular ligament of the temporomandibular joint, acts to prevent extremes of movement laterally. The mandibular condyle can be reconstituted to the mandibular ramus to help establish facial height and width.

Sphenozygomatic Suture

The sphenozygomatic suture, along the internal surface of the lateral orbital wall, has been shown in cadaver studies to be a key landmark for both the reduction and the fixation of the zygomaticomaxillary complex.[42–44] If other aspects of the facial skeleton are ignored, use of this suture alone can

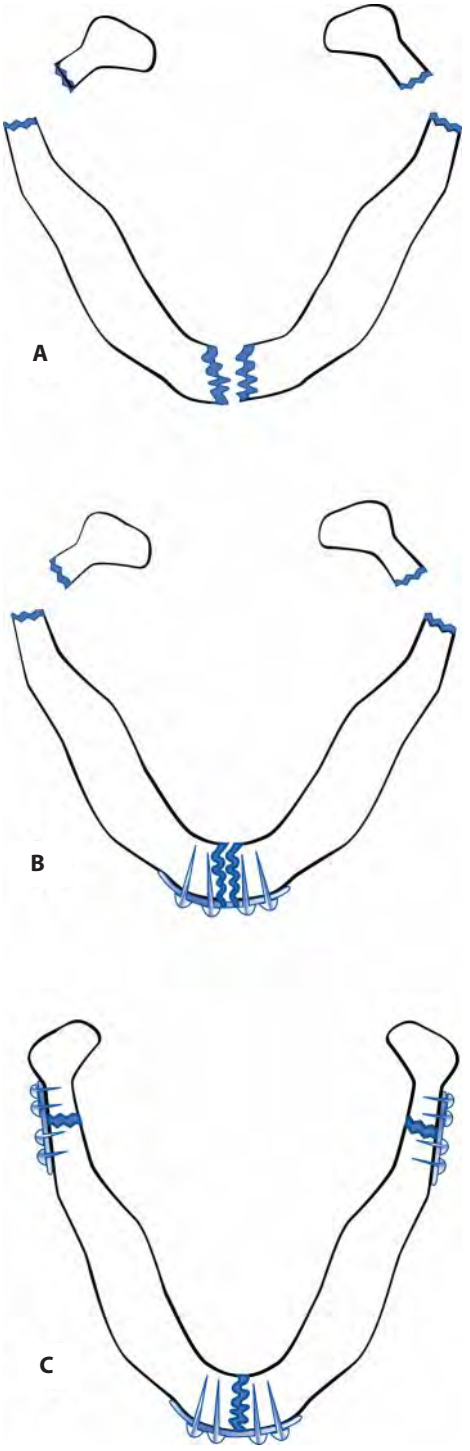


FIGURE 27-5. **A,** Nonreduced mandibular fracture involving the symphysis and condylar process. **B,** Poorly reduced mandibular symphysis fracture with nonreduced lingual cortex and lateral displacement of the mandibular angles. **C,** Well-reduced mandibular symphysis and condylar process fractures. Note the approximation of the lingual cortex in the symphysis region.

result in errors; however, if the orbital roof and superior lateral orbit are intact, this suture can be an important landmark for the proper positioning of the zygoma and zygomatic arch. The sphenozygomatic suture is usually exposed

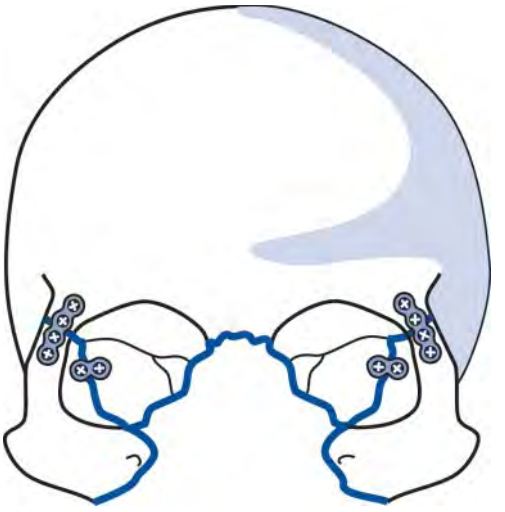


FIGURE 27-6. Reduction and fixation of the sphenozygomatic suture.

along the internal surface of the lateral orbital wall (Figure 27-6).

Once reduced, a small plate can be placed across this fracture for fixation. This area is difficult to place a fixation plate due to the angulation. Most surgeons will reduce the sphenozygomatic suture but not fixate it. Instead, the fixation plate is placed along the frontozygomatic suture. Because the orbital roof and superior lateral orbit are rarely fractured, they are usually accurate landmarks. Likewise, the zygomatic buttress is important in establishing the proper position of the zygoma and/or maxilla. Once the zygoma is in the proper place, the location of the maxilla can be verified. This broad area of surface contact aids in the reduction and fixation process. If there is significant bone loss in this region, consideration should be given to primary grafting to reestablish this buttress.

Intercanthal Region

The intercanthal region may also be used to reestablish midfacial width because the intercanthal distance is fairly constant in the adult facial skeleton.[45] Restoration of the proper intercanthal distance via reduction of the naso-orbitoethmoid complex can help to determine facial width (Figure 27-7).[10] This depends mainly on the fracture type. If there is minimal or no comminution in the region, proper reduction can aid in reestablishment of facial form. Unfortunately, many times this area is severely comminuted and is of little help. Establishing the proper intercanthal distance through measurement is usually performed in patients with severe comminution.

IMAGING

Imaging of the facial skeleton has gone through a gradual evolution in the area of facial trauma. Plain film radiography and linear tomography were the gold standard until the



FIGURE 27-7. **A,** Clinical photograph of patient who has a naso-orbitoethmoid fracture with an intercanthal distance of 43 mm. **B,** Intraoperative photograph shows exposure of the naso-orbitoethmoid fracture.

advent of computed tomography (CT).[46–49] CT has improved our ability to image the facial skeleton and obtain details not possible with plain films (Figure 27-8).[1] It allows the clinicians to determine not only the location of fractures but also the degree and direction of displaced segments.[2,3] Since the introduction of CT, it has undergone an evolution in both the quality of the images and its application. In a previous article, authors reported on "sophisticated CT," in which 5-mm cuts through the facial skeleton were presented.[2] It is now a routine practice at the University of Alabama at Birmingham to obtain 0.75-mm axial cuts with coronal reconstructions. This allows for three-dimensional reconstruction (Figure 27-9), if needed, and decreases the number of repeat scans.[50,51] The scans are loaded onto the hospital information system and can be viewed on computers throughout the medical center and at remote locations. This decreases costs by avoiding the production of multiple hard copies, and it improves efficiency.

With current CT technology, the maxillofacial trauma surgeon can evaluate the fracture pattern by viewing individual cuts or the three-dimensional reconstructions.[3] This allows the surgeon to view necessary details or the overall injury pattern. By manipulating the image windows on a monitor, the surgeon can view hard and soft tissue details. Soft tissue details that can be viewed on CT are not readily apparent on plain films. These include intracranial injuries, injuries to the globe, presence and location of foreign bodies, extraocular muscle entrapment, soft tissue avulsion, displaced teeth, and the airway. If a cervical spine injury is suspected, it may be imaged at the time of cranial and maxillofacial imaging.

The combination of physical examination and current CT imaging allows a clear treatment plan to be generated. This helps greatly with sequencing at the time of surgery.

SURGICAL APPROACHES

Approaches to the facial skeleton in panfacial trauma should permit wide exposure of the fracture to allow for anatomic

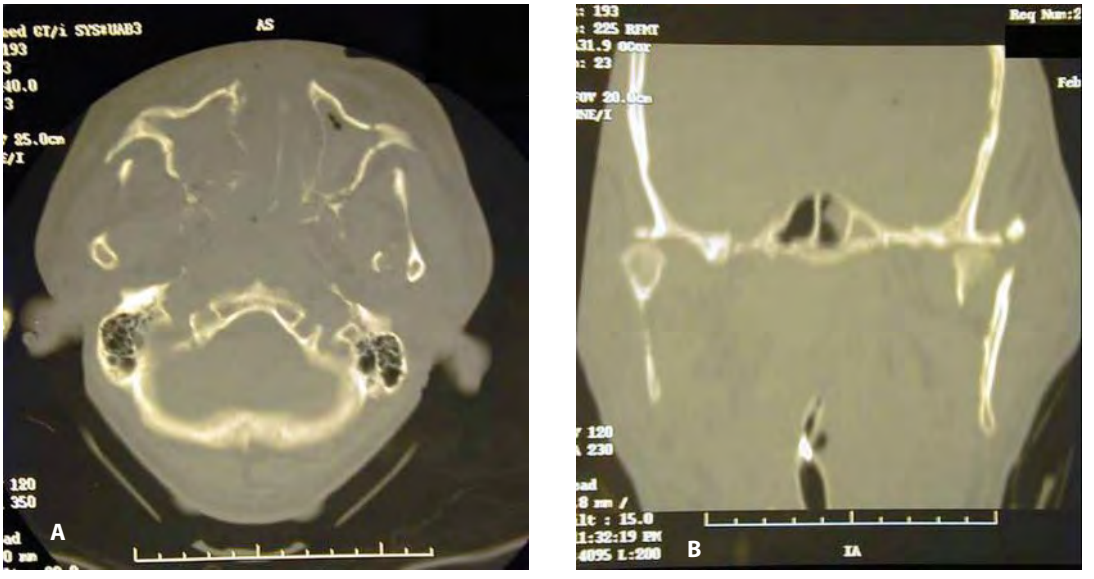


FIGURE 27-8. CT scans show midfacial fractures and a left condylar head fracture on the axial view **(A)** and a left condylar head fracture on the coronal view **(B).**

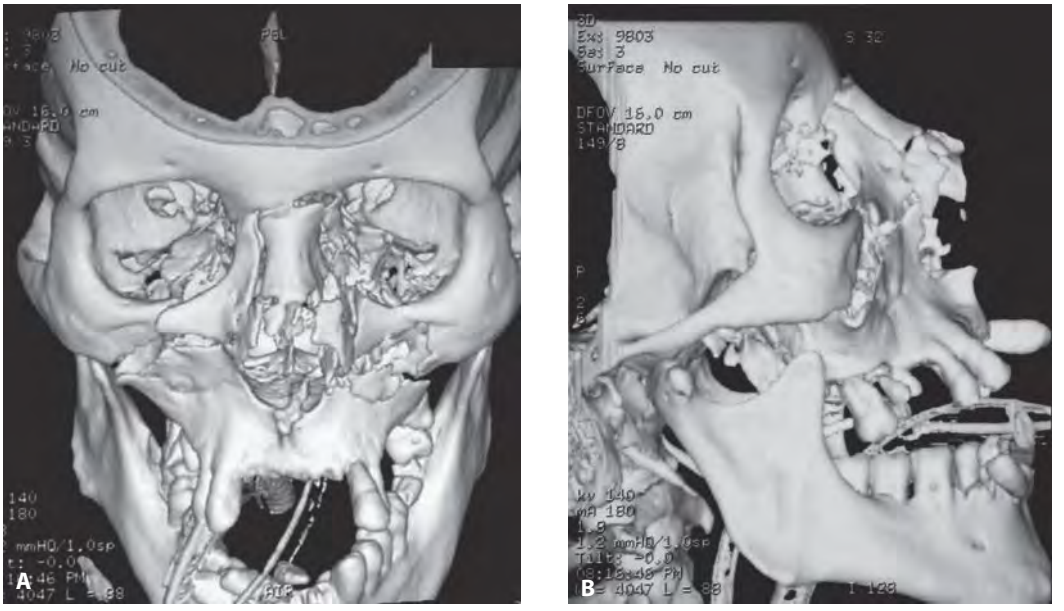


FIGURE 27-9. **A** and **B,** Three-dimensional CT images of patient with extensive midface injuries. Note the detail and quality of the images.

reduction. The location and extent of exposure are dependent on fracture severity and combination. The following describes which fractures can be accessed through the various surgical approaches (Figure 27-10):

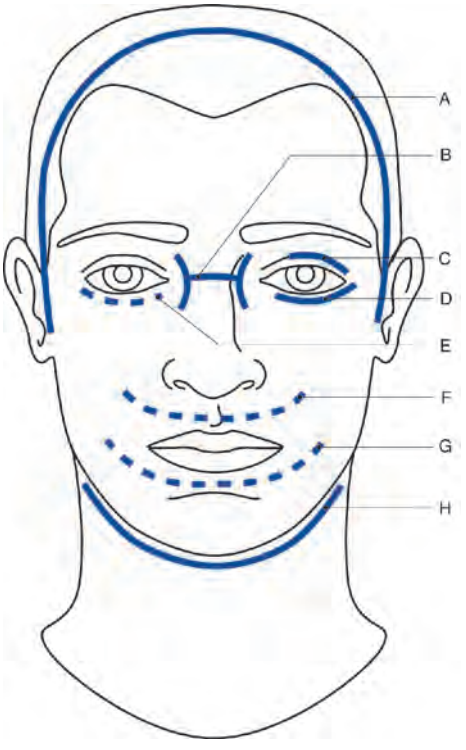


FIGURE 27-10. Surgical approaches to the facial skeleton: coronal with preauricular extension *(a)*, paranasal *(b)*, superior tarsal crease *(c)*, subciliary *(d)*, transconjunctival with lateral canthotomy *(e)*, maxillary vestibule *(f)*, mandibular vestibule *(g)*, and cervical crease *(h)*.

- Coronal flap procedure: frontal sinus, naso-orbitoethmoid (superior aspect), medial canthal tendon, supraorbital rim, orbital roof, superior aspect of the medial and lateral orbital wall, zygomatic arch, and mandibular condyle (with preauricular extension).
- Subciliary and transconjunctival incision with lateral canthotomy: infraorbital rim, medial and lateral orbital wall, and orbital floor. The transconjunctival incision with lateral canthotomy does allow access to the frontozygomatic suture. This requires detachment of the lateral canthal tendon and incision through the orbicularis oculi muscle and periosteum deep to the lateral periorbital skin. The subciliary approach may allow better access to the lateral nasal region.
- Upper eyelid crease incision: superior and lateral regions of the orbit. It is generally used to expose the frontozygomatic suture. This incision is not needed when the bicoronal incision is used.
- Perinasal incisions: naso-orbitoethmoid region, medial canthal tendon, and nasolacrimal sac. These incisions are generally avoided because of the potential for significant scarring. This incision is not needed when the bicoronal incision is used.
- Maxillary vestibular incision: maxilla and zygomaticomaxillary buttress.
- Mandibular vestibular incision: mandible from the ramus to the symphysis. This approach is not usually recommended for comminuted fractures.
- Cervical incisions: mandible, except for when there is a high condylar neck fracture. The approach is generally indicated when anatomic reduction is crucial. It allows the surgeon to visualize the reduction of the lingual cortex. It is also indicated for comminuted and complicated fractures such as a fracture of the atrophic edentulous mandible.

Bone Grafting and Soft Tissue Resuspension

Two procedures have improved outcomes in the management of panfacial trauma: primary bone grafting and resuspension of the soft tissue after extensive exposure of the facial skeleton.[7–9] As previously discussed, the facial buttresses are areas that can serve as guides in the reduction of the facial skeleton and provide stabilization of fractures. With high-velocity trauma, comminution and loss of bony segments can occur in the buttress and "nonbuttress" areas of the face. When these defects are significant, the surgeon may consider the use of bone grafting to prevent soft tissue collapse and to allow for structural support of the facial skeleton. Previous articles have reported on primary bone grafting with few complications.[7–9] Even when the bone graft becomes exposed, secondary wound healing generally occurs. Common areas that may require primary bone grafting include the frontal bone, nasal dorsum, orbital floor, medial orbital wall, and zygomaticomaxillary buttress.

There are many potential sources of bone for a graft, but calvarial bone may be the best. Access is often achieved through a coronal flap that has already been created during the management of the fractures. These grafts have been shown to resist resorption better than endochondral bone.[8] Rigid fixation of these grafts has been shown to decrease resorption (Figure 27-11).[8]

Soft tissue resuspension after surgical access to facial fractures is important for long-term facial aesthetics.[42,52,53] Resuspension may be especially beneficial in the midface region. For repair of midface fractures, the region is usually exposed transorally and from a periorbital approach.[52] The soft tissue attachment over the midface is customarily completely stripped. This frequently results in sagging of the soft tissue, with reattachment at a more inferior position. Manson and colleagues[42] stated that there are two steps to placing the soft tissue back into proper position after exposure of the facial skeleton: refixation of the periosteum or fascia to the skeleton, and closure of the periosteum, muscle fascia, and skin where incisions have been made. The periosteum is inflexible and limits soft tissue lengthening and migration. Its reattachment is usually accomplished by drilling holes in key locations to fix the periosteum to the bone. Areas where periosteal closure should be obtained include the frontozygomatic suture, infraorbital rim, deep temporal fascia, and muscular layers of maxillary and mandibular incisions.[32,42,52,54] Areas where periosteal reattachment should be obtained include the malar eminence and infraorbital rim, temporal fascia over the zygomatic arch, medial and lateral canthi, and mentalis muscle.[42]

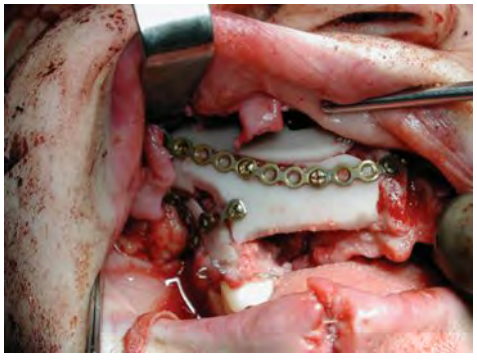


FIGURE 27-11. Primary bone graft rigidly fixed into position to reconstruct the anterior maxillary sinus wall including the naso-maxillary and zygomaticomaxillary buttress. (Courtesy of James Koehler, DDS, MD.)

SEQUENCE OF TREATMENT

Airway Management

How to maintain the airway is a crucial decision in the management of panfacial fractures. There are several options that are dictated by the fracture pattern and extent of other injuries. When there are extensive head injuries and prolonged intubation is anticipated, tracheostomy should be considered.[55–57] Likewise, tracheostomy is an appropriate option to facilitate the management of multiple facial fractures.[10,56,57] In many cases, there are extensive injuries to the naso-orbitoethmoid region, making nasal intubation difficult and hazardous.[58,59] With nasal intubation, access to the frontal sinus and naso-orbitoethmoid region is hindered.

Oral intubation may be an option when maxillomandibular fixation is either not possible or not indicated. When prolonged intubation is not anticipated, options include submental intubation[60,61] or passing the tube behind the dentition, if space permits. If an extraoral approach is indicated to manage a mandibular body/angle fracture or a symphysis fracture, submental intubation may hinder access.

Fracture Management

Much has been written about the proper sequencing of treatment for panfacial fractures.[10,28,42,52,62] Sequences such as "bottom-up and inside-out" or "top-down and outside-in" have been used to describe two of the classic approaches for the management of panfacial fractures. To the author's knowledge, there have been no randomized studies to ascertain whether one approach is superior to the other. The bottom-up and inside-out approach predates the use of rigid fixation but is still a valid approach. It establishes the mandible as a foundation for setting the rest of the face and includes open reduction and internal fixation of subcondylar fractures as well as the remainder of the mandible. The occlusion is set by placing the patient in maxillomandibular fixation; then, the maxilla should be in the proper position. Realignment of the zygomatic buttresses follows in this sequence; however, fixation at this point may lead to inaccuracies in upper midface position. Instead, a break in the sequence is usually preferred here. The zygomaticomaxillary complex is reduced and fixated first. This allows for a more accurate repositioning of the upper midface before fixation at the zygomatic buttress. The maxilla is now fixated along the zygomaticomaxillary buttress.

Last, the naso-orbitoethmoid fracture is reduced and stabilized (Figure 27-12).[62]

The opposite approach, top-down and outside-in, starts at the zygomatic region. The sphenozygomatic suture is reduced and fixated inside the orbit or along the lateral aspect of the orbit by reflect the temporalis muscle. The zygomatic arch is reduced and plated. If the arches are not properly reduced, underprojection of the midface can result. The alignment of the arch can be verified by the proper position of the sphenozygomatic suture. From this point, the zygomas can be further positioned and fixated at the frontozygomatic suture. The naso-orbitoethmoid complex is then positioned to the supraorbital rims, infraorbital rims, and maxillary process of the frontal bones. The maxilla is addressed next, using the position of the zygomaticomaxillary buttress and piriform rim as a guide. Maxillomandibular

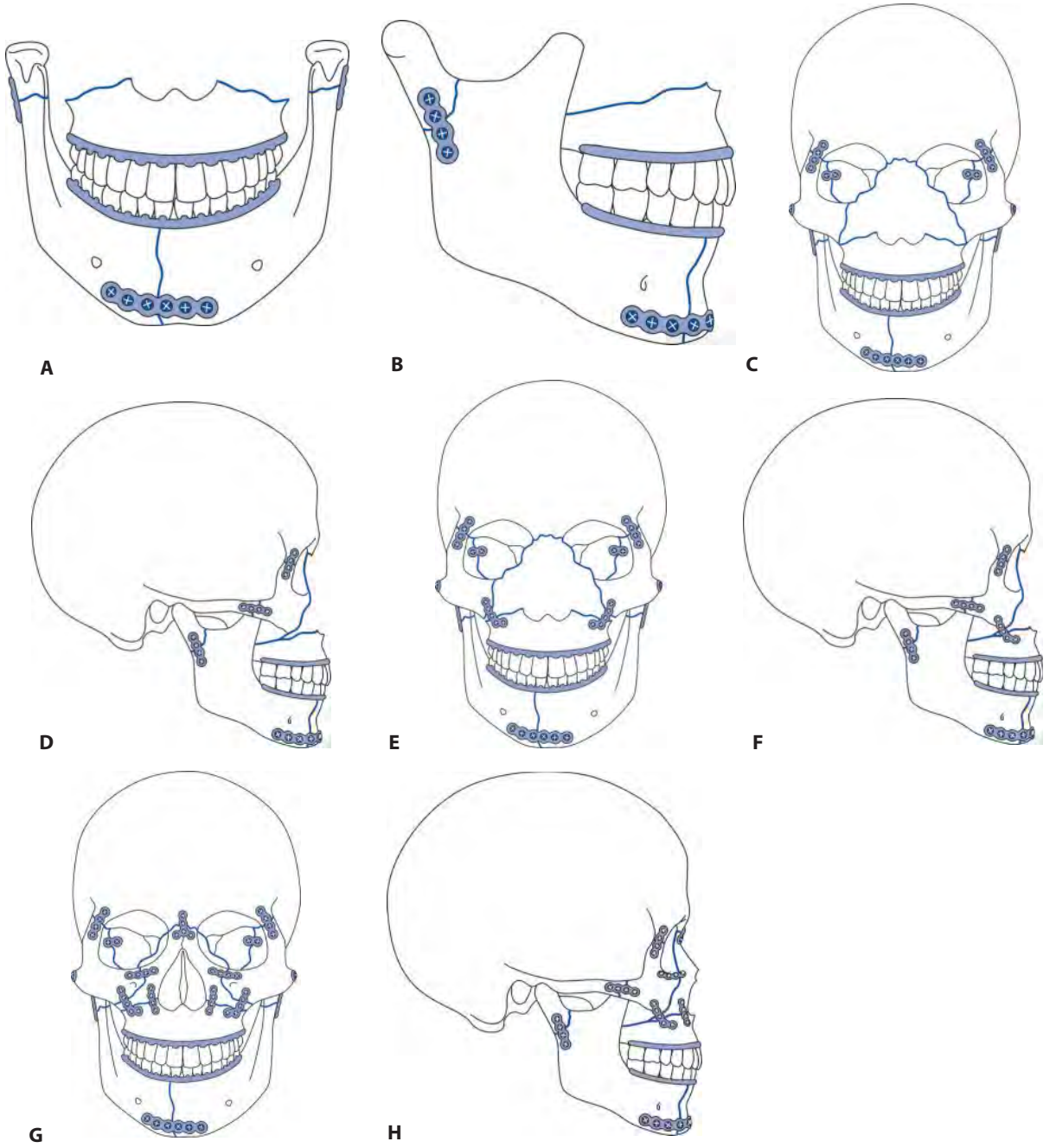


FIGURE 27-12. Bottom-up and inside-out surgical approach. **A** and **B,** Sequencing of panfacial fractures can begin with maxillomandibular fixation. This is followed by reduction and fixation of the subcondylar fractures followed by the symphysis, body, or angle fracture. **C** and **D,** The zygomas are reduced and fixated next using the sphenozygomatic suture, zygomatic arch, and zygomaticomaxillary sutures as guides. The intraorbital plate is usually not necessary except in severe cases of panfacial fractures with comminution. **E** and **F,** The maxilla can now be stabilized in along the zygomaticomaxillary buttress. **G** and **H,** The naso-orbitoethmoid fracture can now be reduced and fixated at the nasofrontal and frontomaxillary sutures and the infraorbital and piriform rims.

fixation can then be established (Figure 27-13).[52] Reduction and fixation of the mandibular condyle and the symphysis/body/angle fractures are then performed.

Some surgeons feel that there is a significant advantage to the top-down and outside-in approach because open treatment of the condyles may not be necessary. The patient is treated with varying periods of maxillomandibular fixation, which may be a valid approach in the case of comminuted intracapsular fractures. Although this is a viable option in some cases, there are two potential complications. One is an unrecognized rotation of the body or ramus of the mandible, resulting in widening. A second complication is temporomandibular joint ankylosis caused by the inability to begin early physical therapy. One author reviewed closed treatment of mandibular condyle fractures and showed compromised results.[63] Early function of patients with condylar head fractures is usually

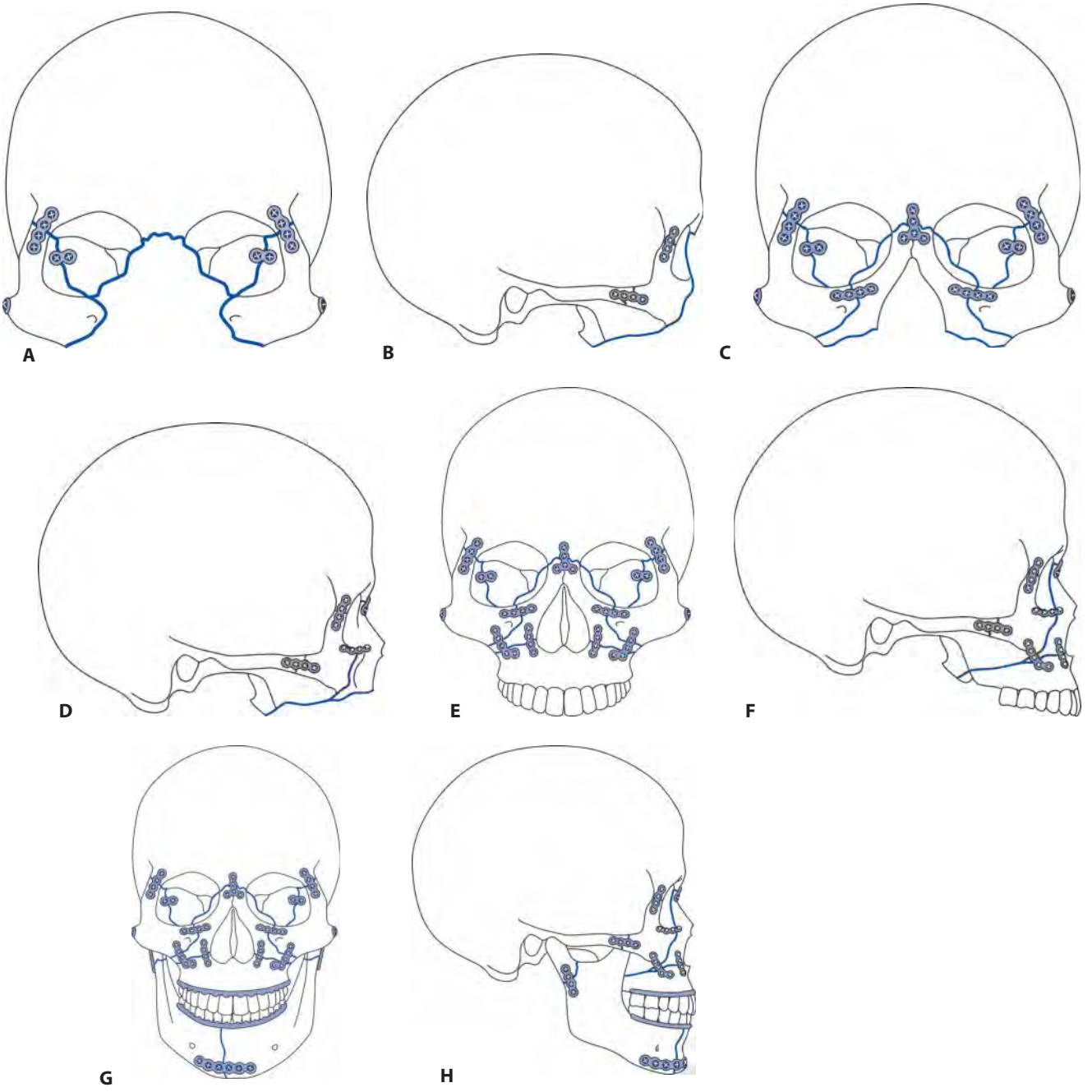


FIGURE 27-13. Top-down and outside-in surgical approach. **A** and **B,** Sequencing of panfacial fractures can begin with the zygomas using the sphenozygomatic suture and the zygomatic arches as guides. The intraorbital plate is usually not necessary except in severe cases of panfacial fractures with comminution. **C** and **D,** The naso-orbitoethmoid fractures can be reduced next and fixated at the nasofrontal suture and maxillofrontal sutures and infraorbital rim. **E** and **F,** The maxilla is reduced and fixated. Stabilization is achieved at the nasomaxillary and zygomaticomaxillary buttresses. **G** and **H,** The mandible is reduced last in this sequence. This is accomplished with the use of maxillomandibular fixation followed by reduction and fixation of the mandibular fractures.

TABLE 27-1. Sequence A: Bottom-Up and Inside-Out*

1. Tracheostomy
2. Repair of palatal fracture
3. Maxillomandibular fixation
4. Repair of condyle fracture
5. Repair of mandibular fractures (body/symphysis/ramus)
6. Repair of zygomaticomaxillary complex fracture (including arches)
7. Repair of frontal sinus fracture
8. Repair of naso-orbitoethmoid complex fracture
9. Repair of maxilla

*See Figure 27-12.

TABLE 27-2. Sequence B: Top-Down and Outside-In*

1. Tracheostomy
2. Repair of frontal sinus fracture
3. Repair of bilateral zygomaticomaxillary complex (including arch) fracture
4. Repair of naso-orbitoethmoid fracture
5. Repair of Le Fort fracture (including midpalatal split)
6. Maxillomandibular fixation
7. Repair of bilateral subcondylar fractures
8. Repair of mandibular fracture (symphysis/body/ramus)

*See Figure 27-13.

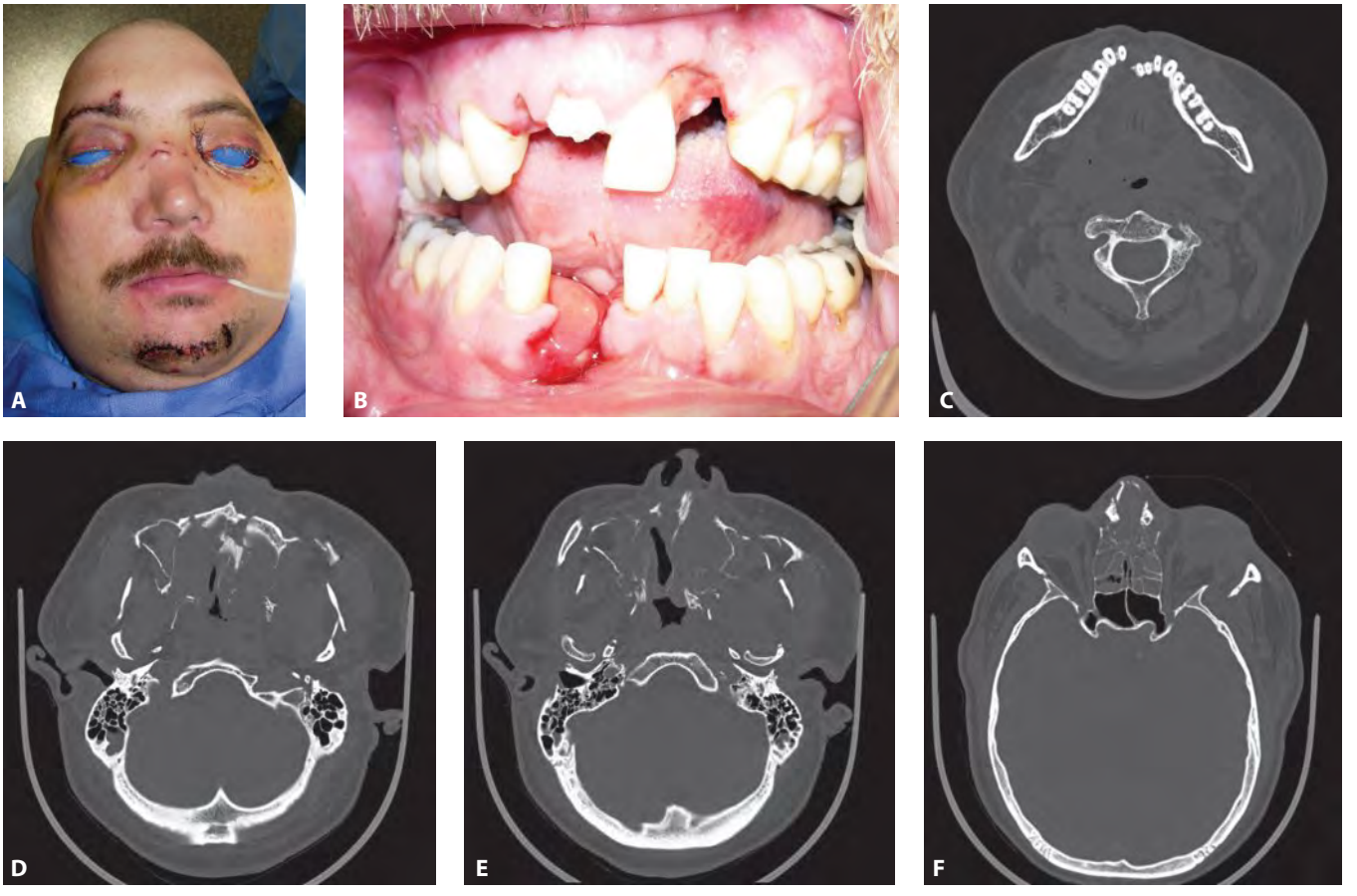
indicated, along with guiding elastics to maintain the range of motion of the temporomandibular joint.

Neither one of these techniques will achieve optimal results in every situation. Instead, an approach that goes from known to unknown is certainly more accurate. For example, if there is a significant calvarial injury, it may be difficult to start from the cranium and proceed caudally. In this case, a sequence that starts caudally and proceeds cranially may achieve more optimal results, allowing the surgeon to reconstruct the damaged cranial portion last. Conversely, if there is significant comminution of the mandible or if key segments are missing, it may be more appropriate to start cranially and proceed caudally. Thus, the maxillofacial trauma surgeon must be comfortable with both approaches and use known landmarks to achieve optimal results.

In Tables 27-1 and 27-2, two common sequences of management of facial fractures are illustrated. Other sequences exist, but they are variations of these two major approaches (Figure 27-14).

COMPLICATIONS

Many complications are associated with various fractures; these are discussed elsewhere in the text, with reference to the specific fracture type. However, a significant complication associated with panfacial fractures discussed here is widening of the facial complex. This occurs when the



(Continued)

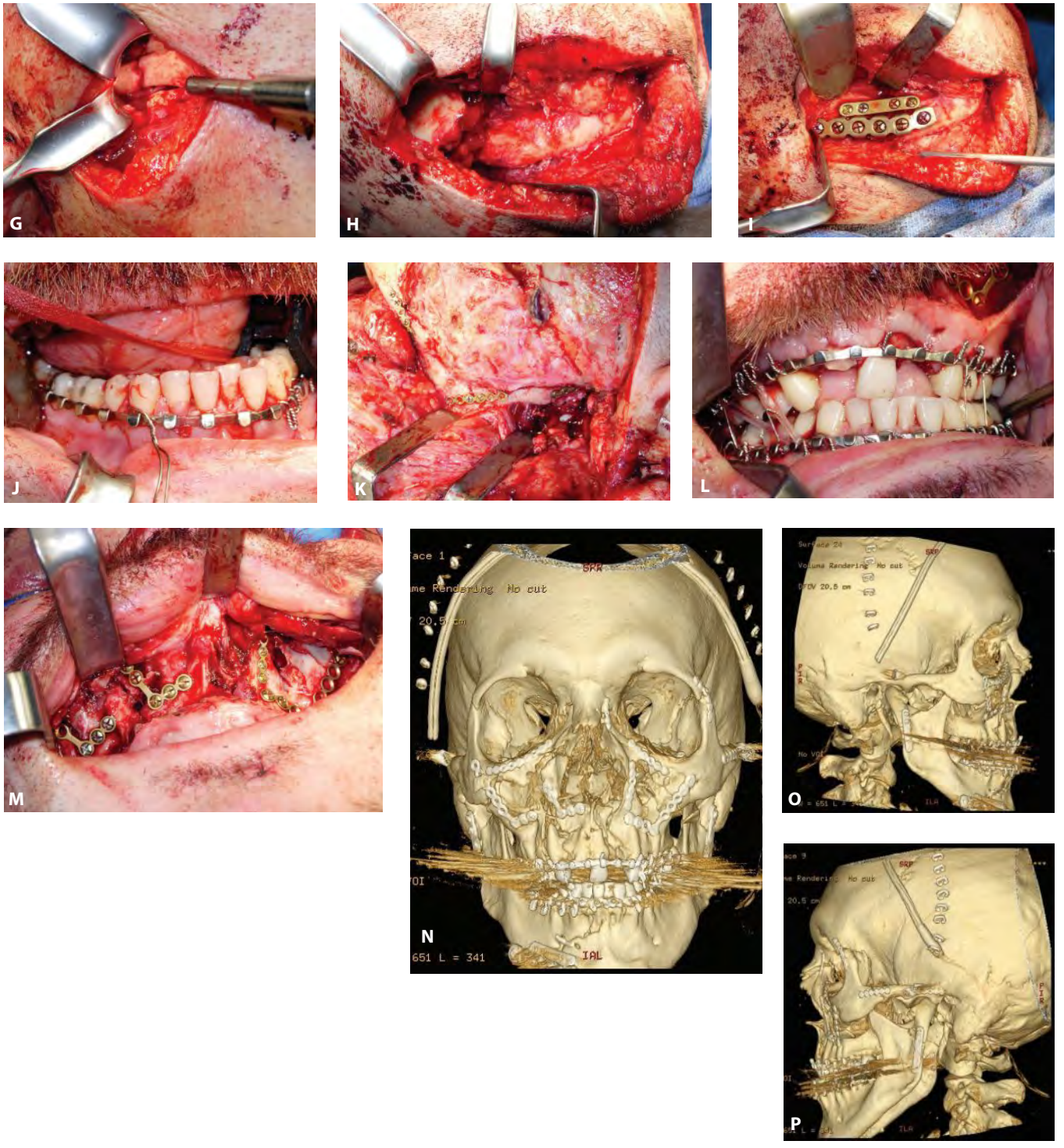


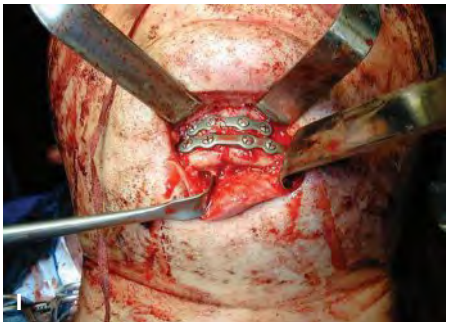
FIGURE 27-14. Patient with panfacial fractures involving the naso-orbitoethmoid (NOE), bilateral zygomaticomaxillary complex (ZMC), Le Fort I level with midpalatal split, mandibular symphysis, and bilateral subcondylar regions. This case illustrates one of the sequencing techniques described in Table 27-1. Note that the palatal fracture was not repaired. Instead, the mandible was repaired first. Next, arch bars were placed. The ZMC fractures were repaired next followed by the NOE fractures. Maxillomandibular fixation (MMF) was established and the maxilla was repaired. **A,** Frontal facial view. Note that the patient is under general anesthesia and has already undergone a tracheotomy. He has facial widening with increased intercanthal distance and a chin laceration. **B,** Intraoral view shows a symphysis fracture, multiple fractured teeth, and anterior open bite. **C–F,** Axial CT scans show the multiple facial fractures as described previously. **G,** Surgical access to the right subcondyle fracture through a retromandibular incision. **H,** Surgical access to the right symphysis fracture through the chin laceration. **I,** After the subcondylar fractures were repaired, the symphysis fracture was stabilized with two plates. **J,** Upper and lower arch bars are placed next. **K,** The ZMC and NOE fractures were accessed through a coronal incision, reduced, and repaired. **L,** After the maxilla was mobilized, MMF was established. **M,** The maxilla was plated with four miniplates. **N–P,** Postreduction three-dimensional CT scans show good alignment of the fractures and good facial form.

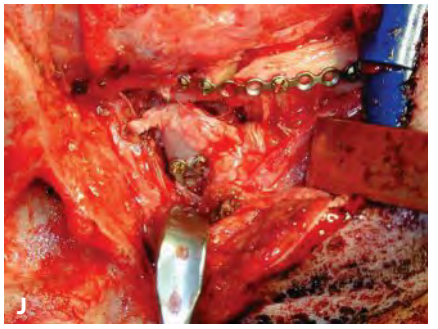
surgeon fails to properly reduce key areas that guide in establishing facial width.[42] If the first area approached is fixated in an improper location, subsequent fragments will be reduced and fixed in an improper spatial arrangement, resulting in a series of errors and, usually, a widened facial complex. To prevent this, the surgeon must use stable segments, known landmarks, and anatomic reduction in the management of panfacial fractures.

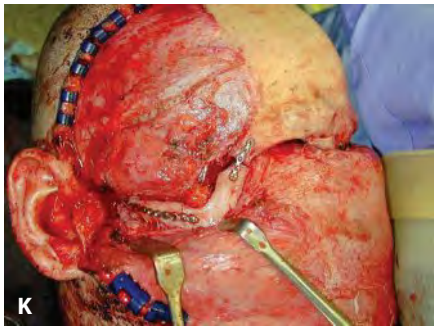
If the complication does occur, the surgeon must assess the patient and determine the severity and location of the problem. This is done through physical examination and CT imaging (Figure 27-15). In severe cases, three-dimensional CT



FIGURE 27-15. **A** and **B,** A 21-year-old male who fell from a height of two stories. Facial fractures included the frontal sinus, naso-orbitoethmoid, bilateral zygomaticomaxillary complex, Le Fort I with midpalatal split and avulsion of tooth no. 9, mandibular symphysis, and bilateral intracapsular condyle fractures. In this photograph, it is evident that the patient has significant facial widening owing to a failure to establish proper facial width. He also has bilateral bony ankylosis of the condyles secondary to a closed reduction of the condyle fractures. **C** and **D,** Three-dimensional stereolithographic models generated from CT imaging. Note the significant widening of the mandible and midface. **E** and **F,** Simulated surgery was performed on this model and mandibular plates were prebent. Note the significant narrowing of the model. Mandibular condyles are now positioned in the fossae. **G** and **H,** Model surgery was performed on the dental cast, based on the preorthodontic models that were brought in by the family. A surgical splint was fabricated. *(Continued)*







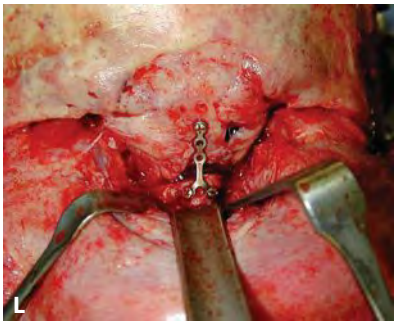








FIGURE 27-15. *(Continued)* **I** and **J,** During the surgical management, the old fractures are exposed via a bicoronal incision with preauricular extension, transconjunctival incisions with lateral canthotomies, a maxillary vestibular incision, and the use of the previous chin scar. The hardware was removed. The previous fractures were recreated by performing bilateral condylar process osteotomies, a symphysis osteotomy, and a Le Fort I with left paramidline split. With the aid of the presurgical splint, the patient was placed in MMF. The mandible was reconstructed first by reducing and fixating the condyles and with the aid of the prebent plates, and by reducing and fixating the symphysis. The *arrow* points to the condylar process osteotomy and fixation plate. **K** and **L,** A Le Fort III osteotomy is created to imitate the initial fractures. This portion of the upper midface is mobilized and advanced. Greenstick fractures of the zygomatic components of the upper midface are also performed to rotate the posterior aspect medially. Once reduced, these fractures are fixated with miniplates. **M,** Last, the maxilla is fixated at the piriform rims and the zygomaticomaxillary buttress with miniplates. The patient is taken out of fixation to verify the occlusion and begin early function. **N,** Early postoperative result. Note the decrease in facial width and increase in facial height. Patient also had zygomatic and recontouring nasal augmentation, bone grafting to the orbits, lateral canthopexy, midface resuspension, and genioplasty. **O,** 1 year follow-up (**A–O,** Courtesy of Dr. Patrick Louis and Dr. John Grant.)

reconstruction of the entire facial skeleton can be obtained and, if indicated, a three-dimensional stereolithographic model can be made.[64,65] The model allows the surgeon to identify and re-create the fractures during model surgery. The fracture may be reduced anatomically and stabilized with plates, which can then be sterilized and used at the time of surgery. This technique and the use of proper landmarks can aid in the proper reduction and fixation of the fractures.

CONCLUSIONS

The management of panfacial fractures is extremely complex. There are, however, many technologic advances that can aid the surgeon in the proper management of these fractures. The most important of these advancements is imaging. With the advent of high-resolution scanners, the surgeon has a more accurate picture of the fracture pattern. Once the

proper diagnosis is established, the surgeon should be able to institute an appropriate sequence of treatment.

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SECTION 4

Maxillofacial Pathology/Infections

28

CHAPTER

Differential Diagnosis of Oral Disease

John R. Kalmar, DMD, PhD, and Carl M. Allen, DDS, MSD

The practice of oral and maxillofacial (OMF) surgery is based upon diagnosis. From small private practices in rural communities to large quaternary care medical centers, the OMF surgeon is called upon to evaluate and diagnose a wide variety of conditions affecting the face, jaws, head, and neck as well as the tissues of the oral cavity. The term *diagnose* comes from the Greek words *dia* (through, apart) and *gnosis* (knowledge), meaning literally "to know apart" or "to distinguish." Clearly, the ability to correctly diagnose is important to virtually all professions, yet it is perhaps most strongly linked to the clinical practice of medicine and dentistry. For health care practitioners, a *diagnosis* is defined as the determination of the nature of a disease or pathologic condition. An accurate diagnosis is important and occasionally critical to the patient because it enables the most appropriate treatment to be initiated as soon as possible. The sooner the correct diagnosis is obtained, the more likely the patient will be able to avoid further expensive and possibly unnecessary laboratory studies, use of ineffective or improper medications, and the inconvenience of additional costly consultation(s).

Several terms are frequently associated with the diagnostic process. Occasionally, the diagnosis of a lesion or condition is relatively straightforward with few, if any, reasonable alternative explanations or diagnoses. More often a variety of conditions with similar clinical features need to be considered. In this situation, a *differential* or *differential diagnosis* (DDx) is prepared. The DDx represents a listing of the likely diagnostic considerations for a particular pathologic finding or condition, ranked in descending order of probability. The top consideration is termed the *clinical diagnosis* (i.e., *working* or *tentative diagnosis*, *clinical impression*) and is the culmination of the clinician's evaluation and training. Although initially based upon clinical signs, symptoms, and history, this list of diagnoses is subject to modification or refinement following additional studies such as radiographic imaging and hematologic or serum analysis (Figure 28-1).[1,2] As is discussed later, the DDx will likely vary depending upon the experience and knowledge base of the treating clinician. Finally, when the clinician believes that the nature of the disease has been identified to a reasonable degree of certainty, the term *final diagnosis* is used. This progression from information (history and clinical presentation) to possible diagnoses to final diagnosis is known as the *diagnostic process* or *method*. A case example is provided later. Although the assignment of a final diagnosis often represents the end of the diagnostic phase of patient care, experienced clinicians recognize that the "final" diagnosis is not always correct. Follow-up evaluation of the patient's response to therapy and careful observation of the subsequent disease course are essential aspects of comprehensive patient management. Should a lesion or condition not behave in the expected manner, revision of the final diagnosis may be indicated and may ultimately require additional studies, including rebiopsy.

THE DIAGNOSTIC PROCESS

The diagnostic process begins with the gathering or accumulating of information. In some instances, this will include a significant historic component, whereas in other cases (e.g., asymptomatic lesions discovered upon routine examination), the information may be limited strictly to the clinical findings, together with any necessary diagnostic studies or tests. Depending upon the experience and expertise of the practitioner, a confident final diagnosis may require nothing more than clinical inspection. In many cases, however, even the most experienced diagnostician requires additional information, ranging from appropriate imaging to laboratory studies.

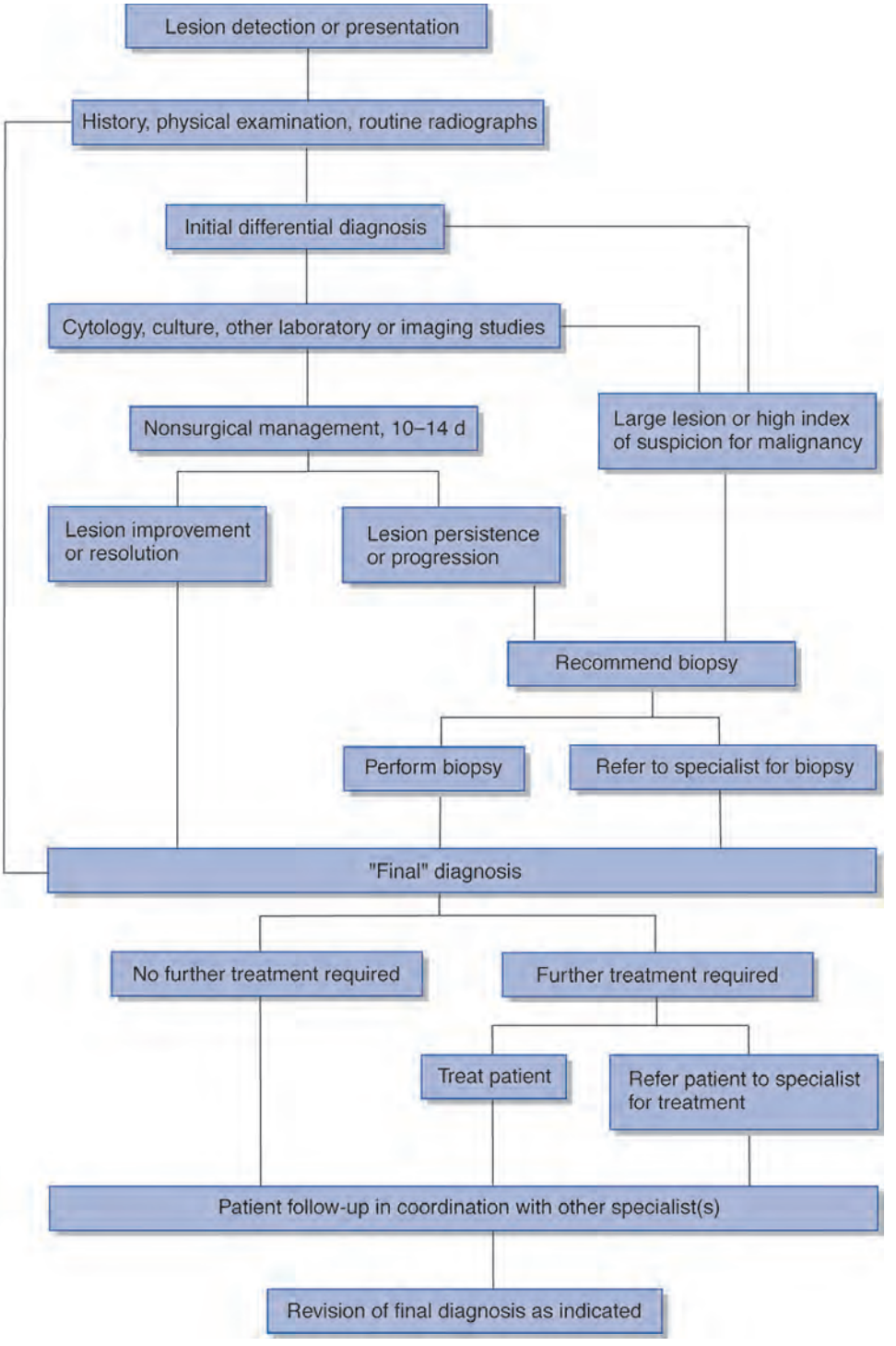


FIGURE 28-1. The diagnostic process. (Adapted from Ellis E III. Principles of differential diagnosis and biopsy. In Peterson LJ, Ellis E, Hupp JR, Tucker MR, editors. Contemporary Oral and Maxillofacial Surgery. 4th ed. St. Louis: Mosby; 2003; pp. 458–478.)

Case Study: From DDx to Final Diagnosis

A 37-year-old man presents with a history of gradual painless enlargement of the left anterior upper jaw over the past several years. His medical history is unremarkable, and he denies trauma to the area. Clinical examination reveals a 2.5-cm bony firm swelling of the right maxillary facial alveolus in the area of teeth nos. 10 and 11 with an unremarkable overlying mucosa (Figure 28-2). Slight mesioangulation of the lateral incisor is observed and the mass is nontender to palpation. The adjacent teeth are unrestored and vital.

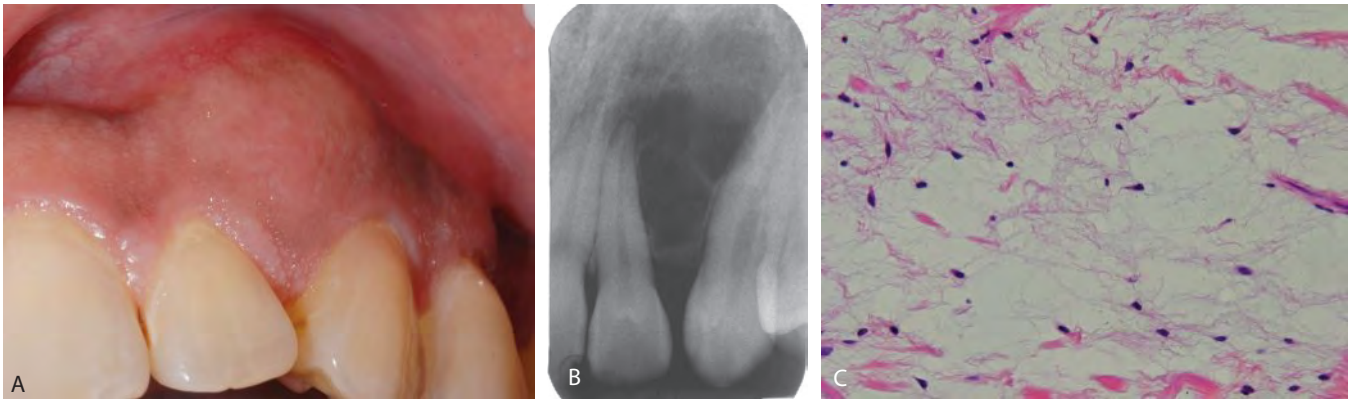


FIGURE 28-2. **A,** Sessile 2.5-cm bony swelling of the maxillary left anterior facial alveolus with history of gradual enlargement over several years. Note displacement of the adjacent lateral incisor tooth. **B,** Periapical radiograph of the maxillary left anterior alveolus. **C,** Photomicrograph of a hematoxylin and eosin–stained incisional intra-alveolar biopsy specimen reveals a proliferation of paucicellular, myxoid connective tissue (original magnification, ×40). Diagnosis: odontogenic myxoma.

Initially, the DDx would focus on the most common conditions that might present clinically in a fashion similar to the lesion in this patient. Likely considerations could include central giant cell granuloma, central ossifying fibroma, calcifying odontogenic cyst, ameloblastoma, and odontogenic keratocyst.

Important additional information is easily obtained in this case through routine dental radiography. An intraoral film reveals a poorly corticated triangular multilocular radiolucent lesion of the right maxilla that appears related to the mesioangulation of the lateral incisor tooth. The radiographic findings, in turn, warrant revision of the DDx to favor conditions associated with multilocular radiolucent presentations: central giant cell granuloma, ameloblastoma, odontogenic keratocyst, odontogenic myxoma, and glandular odontogenic cyst.

An incisional biopsy of the lesional tissue reveals a myxomatous, paucicellular proliferation of small benign-appearing spindle cells. The final diagnosis is odontogenic myxoma.

History

Development of a DDx often begins with data gathering, including a history of the specific problem being investigated as well as the patient's medical, dental, and social history. The patient's perception of lesion duration can be important because longstanding lesions may suggest a developmental or benign process, whereas rapidly evolving lesions often represent reactive, infectious, or malignant conditions. Exceptions to these generalizations are numerous, however, because mycobacterial infections may develop slowly, as do some neoplasms that are considered malignant (e.g., basal cell carcinoma). Furthermore, the reliability of the patient to provide an accurate history is occasionally compromised owing to the patient's inattention, limited mental capacity, or denial of disease.

Symptoms, particularly related to pain or tenderness, are important in formulating a DDx. Pain and tenderness (pain on palpation) are often signs of an inflammatory or infectious process. Adenoid cystic carcinoma is a salvary gland malignancy well known for the early onset of low-grade intractable pain. Other symptoms such as dysthesia or paresthesia can be associated with inflammatory conditions such as osteomyelitis or malignancies such as metastatic disease or lymphoma.

Reported changes in the physical features of a lesion may also provide important insights. A mass that demonstrated gradual enlargement would suggest a possible neoplasm whereas a mass that reportedly fluctuated in size would more likely represent a reactive process. Similarly, changes in symptoms may be significant. Decreasing pain or tenderness likely represents a resolving inflammatory or infectious process, whereas pain that develops within a longstanding previously asymptomatic mass may indicate malignant transformation of a benign tumor (e.g., carcinoma arising in pleomorphic adenoma).

Clinical Examination

Following a review of the patient's medical history and history (if any) of the present lesion or condition, the clinician typically proceeds to gather objective data through careful clinical examination. A variety of lesional attributes should be recorded, including (1) site, (2) size, (3) character (e.g., flat [macule], depressed [ulcer], raised [mass]), (4) color, including an assessment of homogeneity versus heterogenity, (5) surface morphology (e.g., smooth, pebbly, granular, verrucous), (6) consistency on palpation, (7), the border (e.g., smooth, irregular, indistinct, sharply defined), (8) local symptoms, and (9) the distribution (with multiple or confluent lesions).

Site

The precise anatomic location of a lesion with reference to normal anatomic landmarks can provide essential diagnostic information and is discussed later in greater detail.

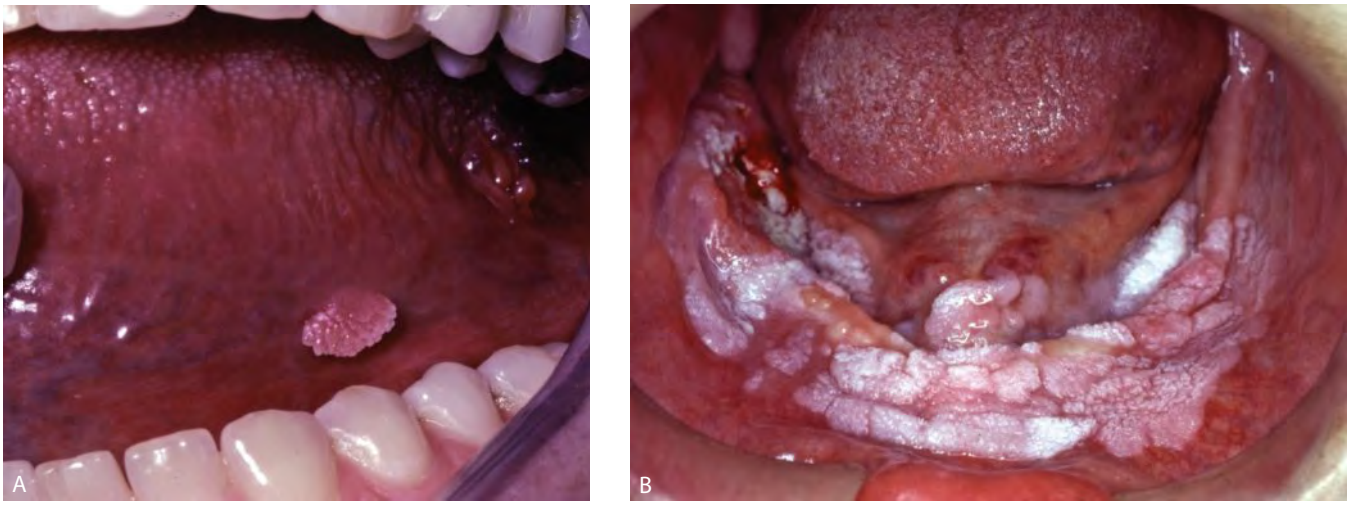


FIGURE 28-3. **A,** Asymptomatic papillary epithelial lesion in a 50-year-old male that has been present for approximately 2 years without apparent increase in size. Clinical diagnosis: squamous papilloma. **B,** Asymptomatic epithelial lesion in a 65-year-old male with a papillary or granular surface that has been present for approximately 2 years with slow progressive enlargement. Clinical diagnosis: verrucous carcinoma.

Size

Lesion size can have diagnostic implications, particularly when combined with an estimate of lesion duration to give an approximate rate of growth or enlargement. The finding of a large lesion may indicate a locally aggressive or malignant neoplasm if the history suggests a relatively recent onset. Yet, even when abnormal tissue has been noted for several months or years, a history of progressive increase in the size of the affected area should be viewed suspiciously (Figure 28-3). As mentioned previously, relying on the accuracy (or veracity) of the patient history can be problematic and should be weighted accordingly in the DDx. Confirmation of the clinical history through other health care practitioners can be helpful in this regard.

Character

Establishing the character of the lesion is an essential aspect of the clinical evaluation. Ulcers can be seen with traumatic, infectious, or neoplastic conditions, whereas masses or swellings more commonly indicate neoplasms, reactive proliferations, cysts, or enlarged lymph nodes. A history or evidence of vesicle or bulla formation might be suggestive of a viral condition, an immunobullous disorder, or possibly an inherited mucocutaneous disease.

Macular lesions, which are completely flat by definition, usually represent an area of color change. A brown or black macule is often the result of intraepithelial melanin pigment; a red or purple macule usually represents hemoglobin in either its oxygenated or its reduced form, respectively. A dull flat white implies keratin production, an area of translucent whitish change may mean increased epithelial edema, and a shiny creamy yellow-white appearance is usually a sign of an ulcer's fibrinous pseudomembrane. A blue or grayish macule is frequently associated with exogenous (amalgam, foreign body) or endogenous (melanin) pigmented material that is deposited within the connective tissue below the epithelium (Figure 28-4). Although additional information is provided later, the border of most pigmented lesions in the oral cavity is relatively homogeneous in color with a smooth well-defined margin. By contrast, a pigmented lesion with significant border irregularity and color variegation should be considered suspicious for melanoma (see Figure 28-4).

Morphology

The surface morphology of a lesion can be virtually diagnostic for certain conditions. Examples include the "tapioca pudding" appearance of a lymphangioma or the papillary epithelial fronds of squamous papilloma (see Figure 28-3). In other settings, a papillary or granular surface architecture may indicate a malignant epithelial neoplasm such as verrucous carcinoma or granulomatous conditions ranging from deep fungal infections to foreign body reactions to immune-mediated conditions such as Crohn's disease or sarcoidosis.

Consistency

Palpation is used to assess the consistency of a lesion, its lateral or deep margins, and the presence or absence of tenderness. When assessing consistency, detection of a doughy-soft mass suggests a cystic lesion or a benign fatty tumor. A rubbery-firm character may be detected with a variety of benign or neoplastic disorders, including metastatic disease within a lymph node. A hard or bony consistency naturally indicates a mineralized or calcified component to the lesion.

Border

The border of a potentially malignant (premalignant, dysplastic) surface mucosal epithelial lesion may vary from

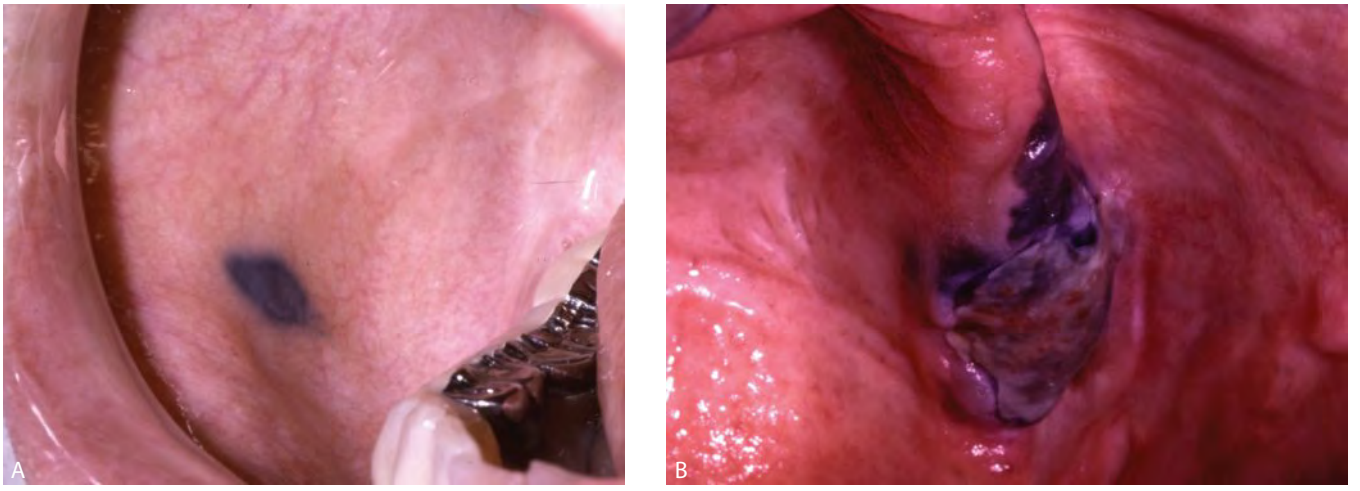


FIGURE 28-4. **A,** Macular grayish 0.8-cm pigmentation with well-defined borders of the right posterior buccal mucosa in a 52-year-old female. The patient reported that it had been present for years. Clinical diagnosis: amalgam tattoo. **B,** Ulcerated area of dark macular pigmentation affecting the posterior maxillary left alveolar mucosa in a 65-year-old male. Note the border irregularity and color variegation. Clinical diagnosis: melanoma.

smooth to markedly irregular, but its outline is usually sharply or well defined compared with the adjacent mucosa (see Figure 28-3). The border of a primarily submucosal or subcutaneous lesion can be described as encapsulated, well-demarcated, or poorly demarcated (infiltrative). An encapsulated process is often freely movable within the deep soft tissues, a finding common to a variety of benign neoplasms and cysts. The margins of some benign lesions (e.g., neurofibroma) and some low-grade malignancies (e.g., acinic cell carcinoma) may be well demarcated, but they are generally less mobile than those of encapsulated lesions. The margins of many malignancies are indistinct, because the tumor invades and blends with the surrounding host tissues.

Symptoms

As noted earlier, local symptoms such as pain or tenderness are usually associated with an inflammatory process, especially acute inflammation. Although benign or malignant neoplasms may also present with tenderness, this feature is often related to ulceration of the surface mucosa and is usually mild to moderate in intensity. With the possible exception of adenoid cystic carcinoma or osteosarcoma, severe pain or dysthesia is usually a late-stage development with oral malignancies, often related to tumor invasion of local nerves. Tenderness may also be a prominent clinical feature of certain benign tumors such as traumatic neuroma.

Distribution

Finally, the presence of multiple identical or similar lesions can suggest a number of conditions, depending upon their particular character (e.g., ulcerations, papules, vesicles) and distribution. Multiple small painful recurrent ulcerations bilaterally on the ventrolateral surface of the tongue in a young adult female patient are most suggestive of the herpetiform variant of recurrent aphthous stomatitis. Conversely, the finding of a unilateral focus of several small relatively painless ulcerations on the left hard palate would be more consistent with a recurrent intraoral herpes infection.

DEVELOPING THE DDX

After collecting the historic and clinical information, the final diagnosis may be obvious; however, in many instances, the diagnosis is not readily apparent and a DDx is appropriate. Several approaches have evolved over the centuries of medical and dental practice to assist in the categorization or grouping of diseases. These grouping strategies permit the vast number of *possible* diagnostic considerations for a given lesion to be reduced to the more *probable* conditions. The focus provided by the DDx helps the clinician determine initial patient management, ranging from conservative measures and follow-up evaluation to selection of additional laboratory or imaging tests that are most useful in determining a final diagnosis.

The major diagnostic strategies or approaches that have been used to group or organize the DDx are based on:

1. The history and clinical presentation.
2. The potential disease pathogenesis.
3. The disease location (more specifically, the frequency of a given condition in a particular location).

In actual practice, clinicians with specialty training or greater experience typically employ all of the strategies simultaneously. As a consequence, the specialist will typically produce a narrower, more precise DDx that, in turn, leads more directly to the correct final diagnosis (see the Case Study later).

History and Clinical Presentation

The physical characteristics of a given lesion in the context of the history and clinical setting often permit the clinician to arrive at a reasonable list of diagnostic possibilities, if not a likely final diagnosis. For example, the solitary, 1-cm pedunculated papillary epithelial lesion depicted in Figure 28-3A was clinically thought to be a squamous papilloma with few alternative considerations. The lesion depicted in Figure 28-3B received a clinical diagnosis of verrucous carcinoma, with a DDx that included proliferative verrucous leukoplakia and squamous cell carcinoma. Multiple incisional biopsies would be appropriate to determine a final diagnosis.

Disease Pathogenesis

Another useful approach to developing a DDx is to consider whether the clinical and historic aspects of the lesion can be explained by any, some, or all of the broad categories of disease pathogenesis. These categories include developmental, inflammatory/immune-mediated, infectious, neoplastic, and metabolic conditions. This is a time-honored systematic method of diagnosis, and many clinicians find it useful to critically consider diagnostic possibilities from each category. For example, an asymptomatic lesion that has been present for several years and feels encapsulated upon clinical palpation would be most consistent with a developmental or benign neoplastic process. Although inflammatory conditions, malignant neoplasms, and metabolic conditions might not be excluded completely, they would not receive primary consideration in the initial DDx. Similarly, although a developmental etiology would be unlikely for a chronic ulceration of the lateral tongue in an adult patient, disorders from the neoplastic (especially malignancies), infectious (e.g., mycobacterial or deep fungal infections), and immune-mediated (e.g., Wegener's granulomatosis or regional enteritis) categories would have to be considered.

Disease Location

The third diagnostic grouping strategy relies on the identification of lesions that most commonly present in a particular anatomic location. The tendency for certain conditions to occur with increased frequency at certain sites is well recognized. For example, a nontender bluish fluctuant mass of recent onset involving the lower labial mucosa likely represents a mucocele. By contrast, mucocele would not be included in the DDx of a painless persistent bluish mass of the attached gingiva because salivary gland tissue is not normally present at that site. This latter setting would, however, be completely consistent with a gingival cyst of the adult. A nonhealing relatively insensitive ulceration of the lateral tongue in an adult patient with no history of trauma and no identifiable source of irritation would be highly suspicious for squamous cell carcinoma. Salivary gland neoplasia would be a likely consideration for a rubbery-firm mass of the posterior lateral hard palate.

Case Study: Neophyte versus Expert Clinician

An otherwise healthy 72-year-old woman complains of sores in her mouth for the past year. Her medical history is unremarkable and she is not taking any medications. She has not been aware of any blisters, and she feels the problem is getting worse. The lesions tend to wax and wane in severity and have affected several areas of the mouth, including the hard and soft palates, the labial mucosa, and the ventral tongue.

Examination shows several shallow erosions and ulcerations with ragged margins. The lesions range from 0.5 to 1.0 cm in diameter and involve the lower labial mucosa, the ventral tongue bilaterally, and the anterior soft palate. No vesicles or bullae are seen, and no white striae are evident.

The inexperienced diagnostician who is not very familiar with oral lesions might provide a DDx based on conditions that are primarily ulcerative: herpesvirus infection, aphthous ulcers, erosive lichen planus, squamous cell carcinoma, and candidiasis. On the basis of this list, the patient would likely be placed on one or possibly more courses of antiviral medication. The patient's condition would not improve, and she might then be switched to antifungal medication(s). After that approach has failed to resolve the problem, topical corticosteroids might be prescribed. After several weeks of topical corticosteroid use with little or no impact on the patient's oral sores, the diagnostician may recommend that a biopsy be performed. In this situation, the patient has invested several months' time and spent hundreds of dollars on inappropriate or ineffective medications—all in the absence of a clear diagnosis.

For the experienced diagnostician who is more familiar with oral conditions, the DDx would be much smaller: cicatricial pemphigoid or pemphigus vulgaris. With a greater understanding of oral disease, the specialist should be able to eliminate many of the considerations that the first clinician entertained. For example, recurrent herpesvirus infection does not typically affect nonkeratinized mucosa in an immunocompetent patient and would not wax and wane in severity. Although aphthous ulcers often exhibit a waxing-and-waning course, the lesional margins are usually smooth, not ragged. Erosive lichen planus would be considered unlikely owing to the lack of radiating white striae at the periphery of the oral lesions, as well as the lack of buccal mucosa involvement. Squamous cell carcinoma would not be reasonable because of the multifocal presentation and the history of waxing and waning. Finally, although candidiasis is occasionally associated with tenderness or irritation of the oral mucosa, it does not induce true ulceration and would, therefore, have a low probability of representing the actual diagnosis.

Based on the patient's age, the distribution of the lesions, the history of the process, and the clinical appearance of the lesions, a DDx that centers on immune-mediated disease would be most appropriate. In this situation, biopsies for examination with both light microscopy and direct immunofluorescence (DIF) would be requested or

performed after the initial consultation. Histopathologic evidence of acantholysis and DIF findings of interepithelial deposits of immunoglobulin G (IgG) and complement component 3 (C3) would establish the final diagnosis of pemphigus vulgaris in a relatively rapid and cost-effective manner. Besides the monetary savings, a more timely and correct diagnosis often saves the patient from unnecessary suffering and mental anguish, both by initiating effective treatment earlier and by relieving the anxiety that many patients experience when they do not know the nature of their disease. Early diagnosis and treatment of conditions such as pemphigus vulgaris may also reduce disease progression or the need for more aggressive therapy.

DETERMINING THE FINAL DIAGNOSIS: ADJUNCTIVE DIAGNOSTIC METHODS

If the final diagnosis cannot be determined based on history and physical examination alone, a variety of procedures and tests are available to assist in the diagnostic process. Generally, diagnostic tests should be used to either confirm or eliminate the most likely (clinical) diagnosis. A methodic approach together with a proper rationale for selecting each test typically leads to the correct diagnosis in the most rapid, cost-effective manner. Tests that fail to address the most likely diagnostic possibilities should be delayed because the probability that they will provide useful information is small yet they can dramatically increase costs to the patient. An exception to this statement would be a situation in which a particular test can rule out a rare or unusual condition of serious clinical significance. Finally, diagnostic tests should be interpreted by individuals with specialty training to that specific test or area, whenever possible, to ensure the most timely and accurate result or final diagnosis.

Diagnostic studies or adjuncts are not necessarily complex or expensive. For example, a putative vascular lesion can be readily evaluated by pressing it with a glass slide to test for possible blanching (diascopy). The bruit of a vascular malformation may be heard upon auscultation using a stethoscope. Operative findings at the time of surgery occasionally provide important diagnostic clues, such as the presence of cheesy keratotic debris within a cystic lesion associated with an impacted tooth, suggestive of an odontogenic keratocyst, or the empty intraosseous cavity seen with traumatic bone cyst. Finally, simple follow-up evaluation of a lesion can provide important diagnostic insight with respect to biologic behavior. A lesion or condition that persists or progresses 2 or 3 weeks after initial inspection will often require an additional test(s) to establish the diagnosis.

Light-Based Adjuncts to Conventional Mucosal Examination

Since the early 2000s, several devices have become commercially available to potentially assist in the screening examination of oral mucosa in healthy asymptomatic patients for potentially malignant or malignant epithelial lesions. Some of these products employ a disposable chemiluminescent light source whereas others are battery-operated or use an alternating-current (AC) power source to produce illumination. Numerous clinical studies have been published regarding these devices and summarized in several recent review articles.[3–5] Readers are directed to these reviews for more detailed summary information regarding each device. Overall, their conclusions were quite similar: there is currently insufficient evidence to suggest that light-based adjuncts provide any benefit over conventional examination alone for routine evaluation of the oral mucosa. Conversely, there is limited data with one light-based system to suggest an alternative beneficial role, the surgical management of potentially malignant oral epithelial lesions or squamous cell carcinoma, by improving real-time margin delineation of previously biopsy-proven disease. Additional studies appear warranted.

Diagnostic Imaging

Depending on the clinical setting, imaging studies may be both appropriate and necessary to the workup of an oral lesion. Additional information on this topic is available in an excellent radiology text edited by White and Pharoah.[6] Briefly, imaging studies can include plain radiographic films, sialography, ultrasonography, computed tomography (CT), cone beam computed tomography (CBCT), magnetic resonance imaging (MRI), radionuclide imaging, and positron-emission tomography (PET).

Plain Films

For evaluation of bone lesions, plain films are the most commonly employed imaging modality and continue to be both cost-effective and extremely useful. Panoramic radiographs are now commonly used a screening study in many dental practices, and it is not unusual for these films to detect a previously unidentified skeletal abnormality. Evaluation of such a lesion includes an assessment of features such as localization (single, multifocal, generalized), margins (well defined, poorly defined), internal structure (radiolucent, radiopaque, mixed), effects on surrounding structures (teeth, inferior alveolar canal, cortical bone), and whether there have been any associated symptoms. For example, a corticated, unilocular radiolucent lesion at the apex of a nonvital tooth likely represents a periapical cyst or granuloma whereas a similar-appearing radiolucency below the level of the inferior alveolar canal in the posterior mandible more likely represents a Stafne defect. Sharp margins indicate a benign process in most instances, whereas poorly defined margins can signify a malignancy. Notable exceptions to this rule include osteomyelitis and fibrous dysplasia, which typically have borders that blend with the surrounding bone. Radiolucent lesions reflect conditions that do not generate a calcified product. Radiopaque and mixed lesions represent conditions that can produce a mineralized product, such as bone, cementum, dentin, or enamel. It is generally safe to assume that the

vast majority of lesions associated with the crown of an impacted tooth are odontogenic in origin. If the teeth are erupted, however, determining whether a lesion is of odontogenic origin can be problematic because there are few areas in the jaws in which a 2-cm lesion does not appear to be tooth-related. Symptoms such as pain or paresthesia may suggest infection or malignancy, but benign conditions can occasionally present in this fashion.

Sialography

Sialography has almost become a lost art. This technique relies on retrograde injection of a radiopaque fluid, also known as *contrast medium*, into the duct system of either the parotid or the submandibular salivary gland. A plain radiograph is made, and the pattern of distribution of the contrast medium is assessed. Many of the previous indications for sialography such as evaluation of salivary gland neoplasia have been supplanted by newer imaging modalities such as MRI. Nonetheless, sialography can be useful in assessing chronic obstructive salivary gland disease and gland function. The characteristic sialographic finding of punctate sialectasis ("blossoms on a branchless tree" pattern) seen in patients affected by Sjögren's syndrome is helpful in supporting that diagnosis.

Ultrasonography

Ultrasonography is an excellent tool for the evaluation of superficial soft tissue masses of the head and neck, including possible salivary gland, thyroid, and parathyroid tumors. It can help distinguish a solid mass from one that is cystic and has been used in the analysis of regional lymph nodes, to provide guidance for fine-needle aspiration (FNA), to assess salivary gland stones and atherosclerotic plaques in the carotid. Ultrasonography uses a transducer to create high-frequency sound waves that pass into and interact with the subcutaneous tissues. Different tissue structures and densities result in different degrees of reflection or echo production that are detected by the unit and displayed visually on a monitor in real time. Although ultrasonography does not expose the patient to ionizing radiation, tissue resolution is typically less than that achieved with CT or MRI technology. In addition, tissues subjacent to bone or adjacent to air-filled structures are not well visualized by ultrasonography.[7]

Computed Tomography

CT is a cross-sectional radiologic imaging technique that is particularly useful in the analysis of bone lesions. As with all three-dimensional imaging, CT eliminates image superimposition from structures outside of the field of interest. By permitting three-dimensional reconstruction of the tissues, the density and margins of a given lesion can be assessed with finer detail than with plain films, lesion localization is more precise, and changes such as cortical erosion or expansion are more readily apparent. Use of contrast media has extended the utility of this technique in areas of soft tissue pathology. Furthermore, more recent designs such as multidetector helical units or electron beam CT have made data acquisition more rapid with reduced doses of ionizing radiation to the patient while maintaining or improving image resolution. Limitations of CT in the head and neck (and CBCT, below) can include metal artifacts associated with metallic dental restorations and metal implants, resulting in variable image distortion and degradation.[6]

Cone Beam Computed Tomography

CBCT is a modified form of CT that uses a divergent (cone-shaped) radiation source and detectors to produce three-dimensional images.[8] Units dedicated to the OMF region became commercially available in 2001, and with the introduction of smaller and less expensive versions, CBCT has become increasingly popular in private practice dental and dental specialty offices.[9] Additional advantages of CBCT include rapid image acquisition (usually < 30 sec), superior spatial resolution (compared with conventional CT), and reduced radiation dose compared with conventional CT (an estimated 1.5–12.3 times lower when comparing medium CBCT scan volumes) that can be further lowered by selective collimation of the primary beam with resultant smaller scan volumes.[8,10,11] In addition, office-based units often include integrated software that facilitates image assessment and measurement for applications such as orthodontics or implant placement. Limitations include greater radiation exposure compared with routine panoramic radiography (ranging from 4- to 44-fold or more), reduced spatial resolution compared with routine intraoral radiographs, metal artifacts and greater image noise and reduced image contrast compared with routine head CT.[8,11,12] In addition, concerns and potential legal issues have been raised by some authors, including a selection of scan volume or field of view, responsibility for interpretation of the entire scan volume, and interstate referral of CBCT scans for the purpose of interpretation.[13,14]

Magnetic Resonance Imaging

MRI is a form of cross-sectional imaging that uses a powerful magnet rather than ionizing radiation to produce three-dimensional views of the patient. In simple terms, the magnetic field it produces is differentially absorbed and released by atoms, especially hydrogen atoms, within the body. The released energy can then be detected and converted into an image of the patient's tissues. Although primarily used in the evaluation of soft tissue lesions, it can also provide diagnostic information about hard tissues as well. Two distinct views or images are typically generated: T_1 and T_2. In the T_1-weighted image, adipose tissue has the highest signal and this view is often used for identifying anatomic structures. By comparison, the T_2 image highlights tissues with high water content and is especially useful in depicting inflammatory processes and neoplasms. Limitations of MRI include relatively long scan times and problems with metals, especially those that are influenced by magnetic fields.

Radionuclide Imaging

Radionuclide imaging relies on the specific uptake of any one of several isotopes by various types of tissues or cells.

Localization of the isotope is determined by examining the patient with a gamma scintillation camera. The most commonly used isotope, technetium 99m pertechnetate, can demonstrate areas of high metabolic activity. It is useful in identifying areas of active skeletal growth or metabolism such as condylar hyperplasia, fibrous dysplasia, or osteitis deformans as well as metastatic disease. Radionuclide imaging can also be used to identify areas of inflammation, such as osteomyelitis, but studies employing both PET and CT are increasingly popular for this purpose.

PET and PET/CT

PET is a recently developed form of cross-sectional imaging technology that relies on the identification of metabolically active cells, such as metastatic deposits of squamous cell carcinoma, through their preferential uptake of radionuclide-labeled substrate such as glucose. Positrons emitted from the radionuclide interact with local electrons, releasing energetic photons that are analyzed through a ring of detectors. While PET can be used alone, combined use of PET and CT (PET/CT) is considered by some authorities to be more accurate than either technology alone in detecting malignancy of the head and neck.[15,16] By contrast, an expert panel recently concluded that PET offered no benefit over conventional anatomic imaging methods for primary head and neck tumors.[17] Others report no significant improvement in detection of recurrent head and neck cancer with PET/CT over PET alone.[18–20] Similarly, although PET is viewed by many authors as essential to the staging of patients with head and neck cancer, a recent meta-analysis found that it provided only marginal improvements in sensitivity (from 75% to 80%) and specificity (from 79% to 86%) over conventional diagnostic tests (CT, MRI, or ultrasonography with FNA).[21] PET has been a useful supplement to endoscopy and biopsy for identification of occult primary tumors in patients presenting with metastatic neck disease.[15,17,22] Both PET and PET/CT have also proved accurate in post-treatment surveillance by helping to distinguish altered anatomic landmarks or areas of fibrosis from recurrent tumor as well as the detection of second head and neck primaries.[15,23] Most recently, PET and PET/CT have been used to monitor patient response to cancer therapy and, when necessary, as a guide for adaptive treatment regimens.[15,24] PET is not recommended for neoplasms that are relatively inactive metabolically (e.g., low-grade mucoepidermoid carcinoma). Drawbacks include the increased cost of PET or PET/CT and potential false-positives, especially due to inflammatory changes.

Analysis of Lesional Tissue: Histologic, Immunohistochemical, and Molecular Evaluation

A final diagnosis is often based on the results of histopathologic examination of lesional tissue. In most cases, the diagnosis is achieved through routine processing and staining measures. In others, a definitive diagnosis cannot be made without additional staining techniques or sophisticated immunohistochemical or complementary DNA studies. Several sampling techniques are available to the clinician and a variety of analytical methods are available to the pathologist. Their selective use depends on the clinical setting and the diagnostic challenges posed by the individual patient specimen.

Exfoliative Cytology

Exfoliative cytology is a relatively inexpensive noninvasive technique that may be used to provide additional information related to lesions of surface origin. Its utility in the diagnosis of conditions such as candidiasis, herpesvirus (herpes simplex virus, varicella zoster virus) infections, and pemphigus vulgaris is well documented.

A modified form of cytologic sampling that employs an oral brush instrument to collect epithelial cells followed by automated histopathologic evaluation was introduced in 1999 and is now commercially available as BrushTest. Suggested advantages over conventional oral cytology include improved sampling of all epithelial layers and increased sensitivity and specificity in the detection of precancerous and cancerous lesions versus results with routine exfoliative cytology. The technique does not provide a definitive diagnosis, however, and cannot be used as a substitute for scalpel biopsy and routine histopathologic examination (see later). Given the potential to delay diagnosis and subsequent treatment, therefore, brush cytology is not indicated in clinical settings in which the index of suspicion for potentially malignant or cancerous change is high. Examples of these settings include a lesion located in a high-risk area for oral cancer (i.e., ventrolateral tongue, floor of mouth, tonsillar pillars, soft palate), a lesion with suspicious physical features (e.g., large size, color variegation, persistent ulceration), or a lesion in a patient with significant risk factors (i.e., heavy smoking, heavy alcohol use, or both). When a mucosal lesion persists but the index of suspicion for malignancy is low, the brush cytology technique may be useful by excluding the presence of potentially malignant epithelial cell changes. For such innocuous lesions, a finding of abnormal cells could trigger scalpel biopsy (and definitive diagnosis) before a surgical biopsy might otherwise have been deemed necessary.

Fine-Needle Aspiration

FNA is a useful method for evaluating subcutaneous or more deeply situated mass lesions, although obtaining a diagnostic sample and interpreting the results accurately require specialized training. This type of procedure is most widely used in determining the nature of salivary gland or neck masses. Currently, FNA is available in most large urban areas throughout the United States, often in conjunction with major medical centers.

Incisional Biopsy

Incisional biopsy is generally indicated for large lesions (>2 cm) and those that could represent unencapsulated or potentially malignant neoplasms. By definition, an *incisional biopsy* is a diagnostic surgical procedure in which a sample

or portion of a lesion is removed for histopathologic review, leaving the remainder of the lesion at the biopsy site. In cases of suspected malignancy, an incisional biopsy is usually the procedure of choice unless the clinician performing the biopsy will also be involved in definitive treatment of the cancer (see later).

Excisional Biopsy

Excisional biopsy is typically used to manage clinically benign lesions that are less than 2 cm in diameter. An *excisional biopsy* is defined as a diagnostic surgical procedure in which all clinically abnormal tissue is removed for microscopic analysis. Excisional biopsy should not be performed on a suspected malignant lesion unless the performing clinician will also be involved in definitive treatment. Otherwise, the surface mucosa may be completely healed by the time the patient is referred to the oncologist, obscuring the extent of the original lesion and unnecessarily hindering definitive treatment planning.

Specimen Orientation

Specimen orientation is recommended whenever a clinician suspects that a lesion may have recurrent or malignant potential, including conditions such as epithelial carcinoma in situ or pleomorphic adenoma. This can be accomplished through the use of suture(s), an accompanying sketch of the specimen with orientation to the surrounding tissues, or both. Such anatomic orientation of the tissue sample allows the pathologist to properly subdivide and process the specimen so that the adequacy of excision can be assessed at all surgical margins. The terms *negative* or *clear margins* are used when the surgical margins appear free from tumor involvement. When tumor is transected or lies immediately adjacent to the surgical margin without evidence of a capsule, proper specimen orientation permits the location of the *positive margin(s)* to be determined as precisely as possible. With this information, the surgeon can then plan the most conservative surgical approach that will also accomplish the primary goal of therapy: complete removal of residual pathologic tissue.

Specimen Information

Although obtaining an adequate biopsy specimen is an important result of proper surgical technique, proper diagnostic technique requires that the surgeon also transmit adequate clinical information to the pathologist through use of the specimen or biopsy data sheet. Inflammatory, reactive, and even neoplastic conditions can have overlapping histopathologic features that are difficult (if not impossible) to distinguish without an adequate description of the clinical setting. Lacking this information, the pathologist may not be able to provide a completely accurate or specific diagnosis. Pertinent details from the medical or dental history, the history of the lesion, the location and physical characteristics of the lesional tissue, and when applicable, the radiographic features can assist with the histopathologic analysis. Clinical findings at the time of biopsy can also provide essential information. A good example is the discovery of an empty cavity during surgical exploration of a radiolucent bone lesion. This situation often means that only minimal tissue can be submitted for review; however, the operative finding is virtually pathognomonic for traumatic bone cyst. Quality close-up clinical photographs or digital images can be helpful, particularly for specialists who have dental backgrounds or clinical training in orofacial diseases such as OMF pathologists. Biopsies of bony pathology should be accompanied by good-quality plain films or other imaging whenever possible, because correlation may help distinguish conditions such as fibrous dysplasia, ossifying fibroma and focal cemento-osseous dysplasia.

Importance of the Clinical Diagnosis

A final piece of information that should always accompany the biopsy specimen is the clinical diagnosis. The clinical diagnosis is important at two levels. First, it provides the pathologist with an educated, chairside "best guess" of the clinician as to what the lesional tissue most likely represented. Should the initial histopathology of the submitted specimen differ substantially from the clinical diagnosis, the pathologist may request deeper sections, rotation of the specimen, or special studies to ensure that all aspects of the biopsy material have been thoroughly examined. Second, in cases where the final histopathologic diagnosis varies significantly from the clinical diagnosis, it is the clinician who should proceed cautiously. A direct conversation with the sign-out pathologist can help ensure that no erroneous information or implications about the case had been received by either party. After discussing the case, the surgeon may be satisfied with the unexpected diagnosis and plan accordingly. If not, the clinician may request a second opinion on the original biopsy material or choose to perform a second biopsy procedure. In essence, the clinical diagnosis serves as a "litmus test" for both the pathologist and the surgeon, an important function that ultimately benefits the patient as well.

For the OMF surgeon, such "diagnostic discord" can be minimized if the tissue specimen is initially reviewed by an OMF pathologist. The OMF pathologist receives highly specialized training in both clinical and microscopic pathology of the head and neck, including odontogenic cysts and tumors and salivary gland diseases. The typical general surgical pathologist, by comparison, has a modest degree of experience with respect to oral conditions and may be unfamiliar with the unique microscopic features of lesions from this area. To provide perspective, individuals trained in OMF pathology programs review tens of thousands of oral biopsy specimens before graduation. By contrast, it is unusual for general surgical (anatomic) pathology residents to examine more than a few hundred specimens from the maxillofacial region during their training. Furthermore, the OMF pathologist has a command of the terminology used by the dental profession and can more readily correlate the clinical and radiographic features with the microscopic findings of oral

biopsy specimens. Just as a general surgeon may be able to remove a set of impacted third molars, the general pathologist may be able to provide an adequate diagnosis for an oral biopsy. In most situations, however, professionals who are trained specifically to diagnose and manage problems related to the OMF region are able to accomplish their respective tasks with greater efficiency, accuracy, and consistency.

The Microscopic DDx

A final diagnosis cannot always be made on the basis of routine hematoxylin and eosin–stained sections alone. In such a situation, the pathologist is faced with a *microscopic* or *histopathologic DDx*. For some cases, special chemical stains may be useful in the detection of suspected microorganisms or the identification of tissue products such as mucin or amyloid. In other cases, particularly spindle cell malignancies and a group of undifferentiated neoplasms termed *small blue cell tumors*, the final diagnosis can be even more challenging. Thankfully, even though these tumors may be indistinguishable at the light microscopic level, they often continue to produce molecules that relate either to their cellular origin or to their newly acquired form of differentiation. To more accurately classify such tumors, these molecular products of origin or differentiation are routinely assessed through the use of *immunohistochemical (IHC)* studies. These techniques employ a wide variety of monoclonal and polyclonal antibodies that are directed against specific cellular or integrated viral antigens (e.g., those produced by the Epstein-Barr virus) that are usually expressed even in otherwise "undifferentiated" neoplasms. The antibodies are linked to an enzyme that is capable of cleaving a selected chemical substrate. This activity produces a pigmented product (often brown; hence, the term "brown stains") that is deposited in the tissues wherever the target antigens are expressed. The diagnosis of a particular tumor often requires the analysis of several antigens to fully explore the histopathologic differential. In cases of malignant lymphoma, for example, it is not uncommon for a panel of 10 or more "antigen probes" to be used to fully classify the neoplastic process. Complete subclassification, in turn, allows the oncology team to design optimal treatment for that particular tumor.

Although routine formalin-fixed paraffin-embedded tissue sections can be used to perform most IHC studies, an important exception involves tumors that require analysis by *flow cytometry*. Flow cytometry permits rapid, highly specific subclassification of lymphomas and leukemias by IHC probe analysis, but the tissue samples must not be fixed and are best analyzed immediately after collection. Another exception to this rule concerns the definitive diagnosis of immunobullous disorders such as mucous membrane (cicatricial) pemphigoid. When these conditions are considered within the DDx, perilesional tissue should be obtained and submitted in a special holding medium known as *Michel's solution (Michel's medium)*. A holding medium is necessary because the molecular structure of the diagnostic antigens in these conditions (e.g., immunoglobulins, complement, and fibrinogen) is typically degraded by formalin fixation. These specimens are processed as frozen sections and are evaluated by *direct immunofluorescence (DIF)*, a special form of IHC that employs antibodies tagged with fluorescent markers. When a special ultraviolet-capable microscope is used, these markers reveal the presence and pattern of immunoreactants necessary to confirm or refute a potential autoimmune disease process. *Indirect immunofluorescence (IIF)* is used for conditions such as pemphigus vulgaris, in which elevated levels of circulating autoantibody are often seen. For IIF studies, patient serum is first incubated with a segment of control substrate (typically monkey esophagus). The serum is removed and the substrate is then incubated with antibody probes similar to those used in DIF studies. As with DIF, ultraviolet microscopy is used to examine the substrate for evidence of antibodies derived from patient serum that bind to epithelial or basement membrane components.

Although novel, more selective IHC probes continue to be developed, immunohistochemical techniques have been increasingly supplemented and even replaced by newer *molecular techniques*. These techniques include sophisticated cytogenetic studies such as *fluorescence in situ hybridization (FISH)* as well as probes that use complementary DNA (cDNA) to identify disease-specific DNA sequences in human tissue samples. Examples include restriction fragment length polymorphism analysis with Southern blot or antigen receptor gene rearrangement analysis by polymerase chain reaction for the determination of clonality in B- or T-cell proliferations.

PATIENT FOLLOW-UP

One of the most important aspects in the diagnosis and management of a given oral lesion or condition is the follow-up evaluation. This appointment permits the clinician to assess the abnormality for physical or symptomatic changes, gain insight into the kinetics of growth or rate of resolution, and assess the impact of initial conservative treatment measures or recommendations to the patient. These additional pieces of information may support the working diagnosis, and no further workup may be required (see Figure 28-1). Alternatively, the follow-up findings may indicate that further investigation of the differential considerations, such as biopsy and histopathologic analysis, is warranted. Finally, careful follow-up should be considered mandatory for patients who have been previously diagnosed with or treated for oral epithelial dysplasia, carcinoma in situ, or carcinoma.

Formal guidelines have also been suggested for the follow-up and management of oral lesions that are not clearly cancerous or potentially cancerous.[21] Such guidelines provide a systematic protocol for the management of persistent oral pathologic conditions and serve to reduce the medicolegal risk associated with this critically important aspect of patient care (Table 28-1).

After initial evaluation and documentation of an oral lesion, a follow-up examination should be scheduled in 7 to

TABLE 28-1. **Recommendations for the Follow-Up of Oral Pathology**

1. Initial reevaluation: 7–14 days after lesion detection/examination.
2. If no evidence of lesional progression or suspicious clinical alterations, reevaluate at 1-, 3-, 6-, and 12-mo intervals; thereafter, reexamine in conjunction with normal recall visits (every 6–12 mo).
3. If lesional progression or suspicious clinical changes are noted, incisional or excisional biopsy should be performed as soon as possible, and the specimen should be reviewed by an oral and maxillofacial pathologist.
4. If no evidence of preneoplastic change (dysplasia) or malignancy (carcinoma or sarcoma) is reported, schedule follow-up as in step 2 and document subsequent findings in the patient's record.
5. When diagnosis of dysplasia (premalignancy) or malignancy is reported, refer or schedule immediately for appropriate work-up and therapy; following definitive treatment, begin follow-up evaluations as in step 2 or similar protocol.

Adapted from Alexander RE, Wright JM, Thiebaud S. Evaluating, documenting and following up oral pathological conditions: a suggested protocol. J Am Dent Assoc 2001;132:329–335.

14 days, with or without treatment. In the absence of normal healing or signs of resolution or if the lesional tissue appears to have enlarged, biopsy would be indicated. If the lesion has remained essentially unchanged and the clinical suspicion of malignancy is low, the clinician should inform and advise the patient regarding the next course of action. Patient referral to an OMF pathologist for clinical consultation and second opinion may be one alternative. If specialty consultation is not readily available and the patient decides to defer biopsy, the informed decision should be recorded in the progress notes and reevaluation of the area should be scheduled at 1, 3, 6, and 12 months after the initial examination. During the follow-up period, diagnostic options include the brush cytology technique (to identify evidence of atypical epithelial cells in surface lesions) or surgical biopsy (to establish a firm diagnosis). The need for these options varies depending on the concerns or desires of the patient or the experience of the clinician. At any time point, however, evidence of significant lesional change should immediately trigger a recommendation of surgical biopsy. After a year, most unchanged lesions can be monitored at routine semiannual or annual dental visits.

Finally, it should be recognized that these recommendations, although sound, do not represent rigid guidelines or medicolegal standards of care that cover every clinical scenario. Each patient and abnormality deserves individual attention and management that may vary from the protocol herein, based upon training, experience, and the clinical judgment of the practitioner.

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29

CHAPTER

Odontogenic Cysts and Tumors

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Odontogenic cysts and tumors are relatively uncommon lesions of the oral and maxillofacial region that must be considered whenever examining and formulating a differential diagnosis of an expansile process of the jaws. The clinical presentation, radiographic appearance, and natural history of these lesions varies considerably, such that odontogenic cysts and tumors represent a diverse group of lesions of the jaws and overlying soft tissues. Collectively speaking, their occurrence is frequent enough to warrant a thorough discussion. As a whole, these pathologic entities have been studied and reported on extensively.

Purely defined, *odontogenic* refers to derivation from a tooth-related apparatus. Tooth formation is a complex process that involves both connective tissues and epithelium. Three major tissues are involved in odontogenesis including the enamel organ, the dental follicle, and the dental papilla. The enamel organ is an epithelial structure that is derived from oral ectoderm. The dental follicle and dental papilla are considered ectomesenchymal in nature because they are, in part, derived from neural crest cells.

For each tooth, odontogenesis begins with the apical proliferation from the oral mucosa of epithelium known as the *dental lamina* (Figure 29-1). The dental lamina, in turn, gives rise to the enamel organ, a cap-shaped structure that subsequently evolves into a bell shape. After forming the enamel organ, the cord of dental lamina normally fragments and degenerates; however, small islands of the dental lamina may remain after tooth formation and are believed to be responsible for the development of several of the odontogenic cysts and tumors.

The enamel organ has four types of epithelium. The innermost lining is referred to as the *inner enamel epithelium* and becomes the ameloblastic layer that forms tooth enamel. The second layer of cells adjacent to the inner enamel epithelium is the *stratum intermedium.* Adjacent to this layer is the *stellate reticulum,* followed by the *outer enamel epithelium.* Surrounding the enamel organ is loose connective tissue known as the *dental papilla.* Contact with the enamel organ epithelium induces the dental papilla to make odontoblasts

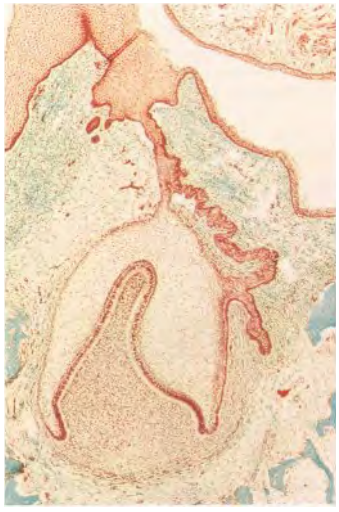


FIGURE 29-1. The enamel organ is seen emanating from the dental lamina (hematoxylin and eosin [H&E], original magnification ×20). (Reproduced with permission from Cawson RA, Eveson JW, editors. Oral Pathology and Diagnosis. Color Atlas with Integrated Text. Philadelphia: WB Saunders; 1987.)

that form dentin. As the odontoblasts deposit dentin, they induce the ameloblasts to begin forming enamel.

After the initial formation of the crown, a thin layer of the enamel organ epithelium known as *Hertwig's root sheath* proliferates apically to provide the stimulus for odontoblastic differentiation in the root portion of the developing tooth. This epithelial extension later becomes fragmented but leaves behind small nests of epithelial cells known as *rests of Malassez* in the periodontal ligament space. The rests of Malassez are believed to be the source of epithelium for most periapical cysts but generally are not believed to give rise to any of the odontogenic neoplasms, with the possible exception of the squamous odontogenic tumor.

In the development of a tooth, after completion of enamel formation, the enamel organ epithelium atrophies to form a

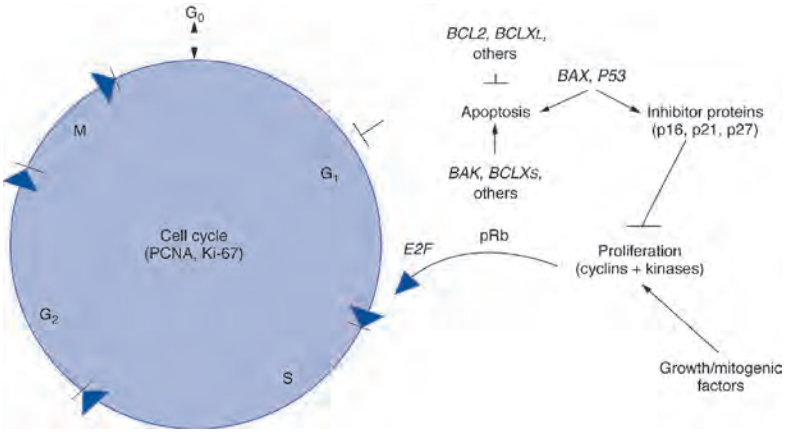


FIGURE 29-2. The cell cycle—a concept of proliferation versus apoptosis. PCNA = proliferating cell nuclear antigen.

thin flattened layer of cells that covers the enamel of the unerupted tooth. This layer of epithelium is known as the *reduced enamel epithelium.* In the normal sequence of events, this reduced enamel epithelium later merges with the surface epithelium and forms the initial gingival crevicular epithelium of the newly erupted tooth. However, if fluid accumulates between the reduced enamel epithelium and the crown of the tooth before tooth eruption, a cyst is formed that is known as a *dentigerous* or *follicular cyst.*

An understanding of the progression of odontogenic cysts and tumors within the oral and maxillofacial region requires a thorough knowledge of the cell cycle of these lesions and an appreciation of the concept of proliferation versus apoptosis (programmed cell death). Most of the pathogenetic mechanisms of odontogenic cysts and tumors can be explained via the cell cycle (Figure 29-2). Normally cell division is divided into four phases: G_1 (gap 1), S (deoxyribonucleic acid synthesis), G_2 (gap 2), and M (mitosis). A key event is the progression from G_1 to the S phase. Genetic alterations, if unrepaired in the G_1 phase, may be carried into the S phase and perpetuated in subsequent cell divisions. The G_1-S checkpoint is normally regulated by a well-coordinated and complex system of protein interactions whose balance and function are critical to normal cell division.[1] As can be seen in Figure 29-2, once genetic change occurs that encourages the development of an odontogenic cyst or tumor, a series of events mediated by the odontogenic lesion occur that may promote proliferation. Such events support the pathogenetic mechanism involved in the progression of the cyst or tumor.

The purpose of this chapter is to review the clinically significant and more commonly encountered odontogenic cysts and tumors. In so doing, salient clinical and radiographic features are discussed, as are the pathogenetic mechanisms supporting proliferation of some of the more aggressive odontogenic cysts and tumors. Recommendations for treatment and prognostic information are also offered.

ODONTOGENIC CYSTS

With rare exceptions, epithelium-lined cysts in bone are seen only in the jaws.[2] Other than a few cysts that may result from the inclusion of epithelium along embryonic lines of fusion, most jaw cysts are lined by epithelium that is derived from odontogenic epithelium, hence, the term *odontogenic cysts.* These cysts are subclassified as developmental or inflammatory in nature. Although the cell type is often known, developmental cysts are of unknown origin; however, they do not seem to be the result of an inflammatory reaction. Inflammatory cysts, conversely, are the result of inflammation (Table 29-1).

Dentigerous Cyst

By definition, a *dentigerous cyst* occurs in association with an unerupted tooth, most commonly, mandibular third molars. Other common associations are with maxillary third molars, maxillary canines, and mandibular second premolars.[2] They may also occur around supernumerary teeth and in association with odontomas; however, they are only rarely associated with primary teeth.[2,3] Although dentigerous cysts occur over a wide age range, they are most commonly seen in 10- to 30-year-olds. There is a slight male predilection, and their prevalence

TABLE 29-1. **Classification of Odontogenic Cysts**

Developmental
Dentigerous cyst
Eruption cyst
Odontogenic keratocyst*
Orthokeratinized odontogenic cyst
Gingival (alveolar cyst of the newborn)
Gingival cyst of the adult
Lateral periodontal cyst
Calcifying odontogenic cyst†
Glandular odontogenic cyst
Inflammatory
Periapical (radicular cyst)
Residual periapical (radicular cyst)
Buccal bifurcation cyst

*Although the odontogenic keratocyst is included with the odontogenic tumors according to the 2005 World Health Organization (WHO) classification (keratocystic odontogenic tumor), the author prefers to include it as an odontogenic cyst.
†The term *calcifying odontogenic cyst* includes both nonneoplastic cysts and true neoplasms. Although the calcifying odontogenic cyst is included with odontogenic tumors in the 2005 WHO classification, it is discussed with the odontogenic cysts in this chapter.



FIGURE 29-3. This unilocular radiolucency of the right mandible associated with impacted tooth no. 32 was discovered when the patient noted a swelling of the upper aspect of his right neck.

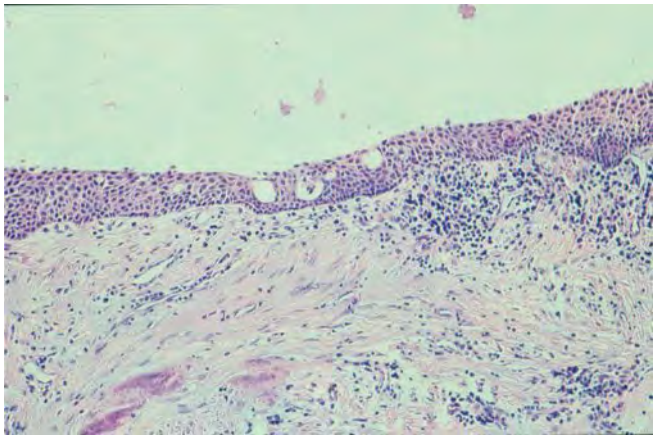


FIGURE 29-4. The incisional biopsy of the radiolucency in Figure 29-3 shows an atrophic stratified squamous epithelium without significant associated inflammation. The diagnosis is dentigerous cyst (H&E, original magnification ×40).

appears to be higher in whites than in blacks. Many dentigerous cysts are small asymptomatic lesions that are discovered serendipitously on routine radiographs, although some may grow to considerable size, causing bony expansion that is usually painless until secondary infection occurs.

Radiographically, the dentigerous cyst presents as a well-defined unilocular radiolucency, often with a sclerotic border (Figure 29-3). Because the epithelial lining is derived from the reduced enamel epithelium, this radiolucency typically and preferentially surrounds the crown of the tooth. A large dentigerous cyst may give the impression of a multilocular process because of the persistence of bone trabecule within the radiolucency. However, dentigerous cysts are grossly and histopathologically unilocular processes and probably are never truly multilocular lesions.[2] Three types of dentigerous cyst have been described radiographically, including the *central* variety, in which the radiolucency surrounds just the crown of the tooth, with the crown projecting into the cyst lumen. In the *lateral* variety, the cyst develops laterally along the tooth root and partially surrounds the crown. The *circumferential* variant of the dentigerous cyst exists when the cyst surrounds the crown but also extends down along the root surface, as if the entire tooth were located within the cyst.

One diagnostic dilemma for oral and maxillofacial surgeons is distinguishing between a dentigerous cyst and an enlarged dental follicle. This distinction becomes clinically significant when the surgeon considers whether to submit tissue removed with an impacted third molar for histopathologic examination as opposed to clinical designation as a follicle, with simple disposal of the tissue. The radiographic distinction becomes somewhat arbitrary; however, any pericoronal radiolucency that is greater than 4 or 5 mm is considered suggestive of cyst formation and should be submitted for microscopic examination. It is noteworthy that pathologists also struggle with the distinction between dental follicles associated with developing teeth and odontogenic lesions.[4,5] It seems that odontogenic cysts, odontogenic fibroma, and odontogenic myxoma are the lesions most often inappropriately diagnosed by surgical pathologists owing to a general unfamiliarity with the normal process of odontogenesis.[4]

Of perhaps even greater concern is the large unilocular radiolucency. Although most commonly classified radiographically as dentigerous cysts, it is incumbent upon the surgeon to section these excised specimens in the operating room and to consider frozen-section analysis. In fact, some specimens may contain a focus of unicystic ameloblastoma and, therefore, require consideration of more extensive treatment.

The histologic features of dentigerous cysts may vary greatly depending mainly on whether or not the cyst is inflamed. In the noninflamed dentigerous cyst, a thin epithelial lining may be present with the fibrous connective tissue wall loosely arranged (Figure 29-4). In the inflamed dentigerous cyst, the epithelium commonly demonstrates hyperplastic rete ridges, and the fibrous cyst wall shows an inflammatory infiltrate.

Treatment and Prognosis

Most dentigerous cysts are treated with enucleation of the cyst and removal of the associated tooth, often without a preceding incisional biopsy (Figure 29-5). Larger cysts that are treated in the operating room should probably undergo frozen-section diagnosis and appropriate treatment that might be dictated by other diagnoses. Curettage of the cyst cavity is usually advisable at the time of removal of the cyst in the event that a more aggressive cyst or an odontogenic tumor is diagnosed histopathologically after removal in an office setting. Such diagnoses would include the odontogenic keratocyst and the unicystic ameloblastoma.

Large dentigerous cysts may be treated with marsupialization (Figure 29-6) when enucleation and curettage might otherwise result in neurosensory dysfunction or predispose the patient to an increased chance of pathologic fracture. Some patients who are not candidates for general anesthesia may also be treated with a marsupialization procedure in an office setting under local anesthesia. This permits decompression of the large dentigerous cyst with a resultant reduction in the size of the cyst and bony defect. At a later date, the reduced cyst can be removed in a smaller-scale surgery.



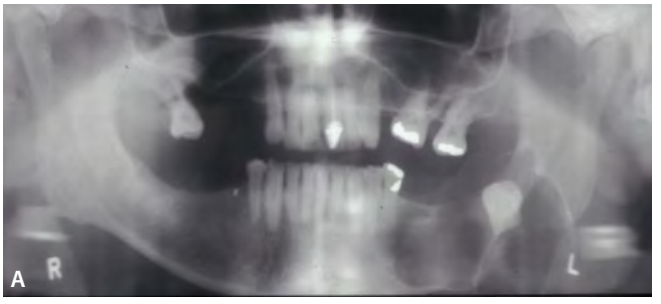


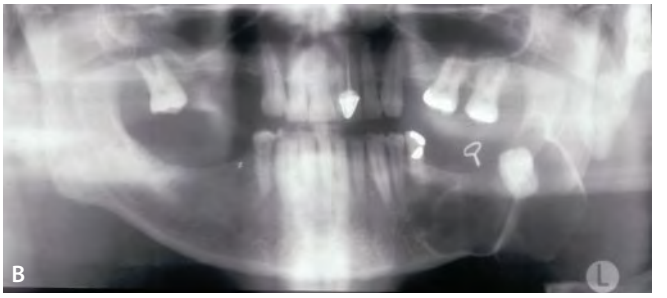
FIGURE 29-5. **A,** The dentigerous cyst in Figure 29-3 is treated with enucleation and curettage of the cyst and removal of the etiologic tooth. **B,** The 1-year postoperative radiograph shows an acceptable bony fill.

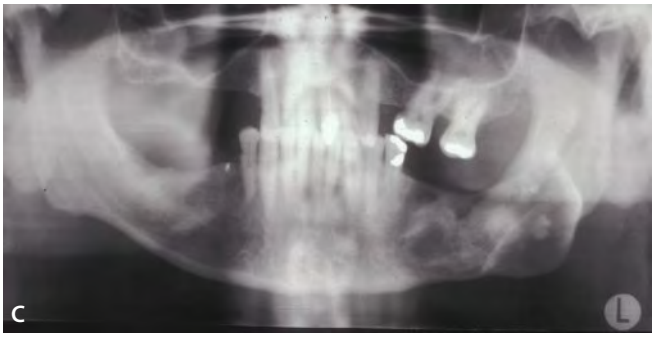
The author emphasizes the need for histopathologic examination of all radiolucencies that are empirically diagnosed as dentigerous cysts. This includes those that are enucleated as well as those that undergo marsupialization, during which it is important to inspect the cyst lumen and submit a representative portion of the lesion for histopathologic examination. Support of this statement stems from the occasional formation of a squamous cell carcinoma, mucoepidermoid carcinoma, or ameloblastoma from or in association with a dentigerous cyst.[6–8] The prognosis for most histopathologically diagnosed dentigerous cysts is excellent, with recurrence being a rare finding.

Odontogenic Keratocyst

The odontogenic keratocyst is a distinctive form of developmental odontogenic cyst that deserves special consideration because of its specific histopathologic features and aggressive clinical behavior. Some investigators have proclaimed the odontogenic keratocyst as a benign cystic neoplasm rather than a cyst, and some textbooks have applied the nomenclature "keratocystic odontogenic tumor" including the World Health Organization classification of odontogenic tumors.[9] The rationale for adopting this nomenclature stems from the results of a few studies that have shown certain molecular







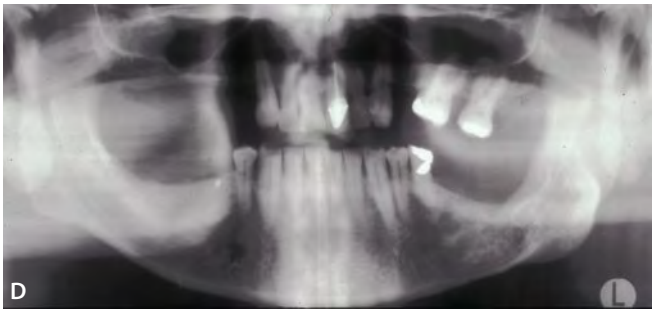


FIGURE 29-6. **A,** This large biopsy-proven dentigerous cyst occurred in an elderly patient who had coronary artery disease. Owing to the size of the cyst and the compromised cardiac status of the patient, a relatively noninvasive marsupialization was performed. **B,** An acrylic plug with a wire handle was placed in a small surgical entrance into the cyst cavity. The cyst shrunk considerably, after which time the etiologic impacted tooth was removed with a small remnant of dentigerous cyst. The 1-year postmarsupialization radiograph **(C)** shows the initiation of bone fill, and the 5-year postmarsupialization radiograph **(D)** shows an excellent fill of bone without evidence of recurrent cyst.

genetic alterations that are also present in some neoplasms.[10] Nonetheless, these studies did not examine other cystic lesions of the jaws such that it is not currently known whether these alterations are unique to the odontogenic keratocyst or more globally expressed. Most oral and maxillofacial pathologists feel that insufficient evidence exists to support renaming this pathologic entity such that the odontogenic keratocyst remains within the section of odontogenic cysts in this chapter.

Two variants of the odontogenic keratocyst are well known; the sporadic cyst and the syndromic cyst associated with the nevoid basal cell carcinoma syndrome. Both variants of the odontogenic keratocyst are believed to be derived from remnants of the dental lamina. This cyst shows a different growth mechanism and biologic behavior from the previously described dentigerous cyst. Most authors believe that dentigerous cysts continue to enlarge as a result of increased osmotic pressure within the lumen of the cyst. This mechanism does not appear to hold true for odontogenic keratocysts, and their growth may be related to unknown factors inherent in the epithelium itself or enzymatic activity in the fibrous wall.[11]

Adequate diagnosis and treatment of the odontogenic keratocyst is important for three reasons: (1) this cyst is recognized as being more aggressive than other odontogenic cysts,[12] (2) the odontogenic keratocyst has a higher rate of recurrence than other odontogenic cysts,[13] and (3) the association with nevoid basal cell carcinoma syndrome requires that the clinician examine a patient with multiple cysts of the jaws for physical findings that might diagnose this syndrome.[14–16]

Odontogenic keratocysts may be found in patients ranging in age from infancy to old age; however, 60% of cases are seen in people between 10 and 40 years old.[17] In his series of 312 cases, Brannon[18] found a mean age of nearly 38 years. The peak prevalence was in the second and third decades of life, with only 15% occurring past the age of 60 years. Woolgar and colleagues[19] reviewed 682 odontogenic keratocysts from 522 patients and found a mean age of 40 years for patients with single nonrecurrent cysts and 26.2 years for patients with multiple cysts of the nevoid basal cell carcinoma syndrome. A slight male predilection is usually seen, and 60% to 80% of cases involve the mandible, particularly in the posterior body and ascending ramus.[2] Although it is rare for a dentigerous cyst to appear multilocular on radiographs, it is most common for odontogenic keratocysts to appear multilocular (Figure 29-7). Many appear unilocular and can, therefore, be confused with dentigerous cysts. It is clear, therefore, that the differential diagnosis of a unilocular radiolucency must include both entities and that treatment should include curettage in the event that the diagnosis is later determined to be odontogenic keratocyst. When multiple multilocular radiolucencies are noted on a panoramic radiograph, the clinician must perform an incisional biopsy and investigate the possibility of nevoid basal cell carcinoma syndrome (Table 29-2).

Histologically, the odontogenic keratocyst is readily recognized. A uniform layer of stratified squamous epithelium, usually six to eight cells in thickness, is present (Figure 29-8). The parakeratotic surface is characteristically corrugated. The wall is usually thin and friable, which can pose problems for removal in one piece intraoperatively. Epithelial budding and the presence of daughter cysts may be noted in the connective tissue wall. It is generally advisable to ask the pathologist to examine the sections carefully for these two features because they generally impart a more aggressive character to the cyst. The presence of daughter cysts is more commonly noted in syndromic cysts than in sporadic cysts.



FIGURE 29-7. This multilocular radiolucency, present in a 54-year-old man, should suggest an odontogenic keratocyst when formulating a differential diagnosis.

TABLE 29-2. **Clinical Features of the Nevoid Basal Cell Carcinoma Syndrome**

≥50% Frequency
Multiple basal cell carcinomas
Odontogenic keratocysts
Epidermal cysts of the skin
Palmar/plantar pits
Calcified falx cerebri
Enlarged head circumference
Rib anomalies (splayed, fused, partially missing, bifid)
Mild ocular hypertelorism
Spina bifida occulta of cervical or thoracic vertebrae
15–49% Frequency
Calcified ovarian fibromas
Short fourth metacarpals
Kyphoscoliosis or other vertebral anomalies
Pectus excavatum or carinatum
Strabismus (exotropia)
<15% Frequency (But not Random)
Medulloblastoma
Meningioma
Lymphomesenteric cysts
Cardiac fibroma
Fetal rhabdomyoma
Marfanoid build
Cleft lip and/or palate
Hypogonadism in males
Mental retardation

Adapted from Gorlin FJ. Nevoid basal cell carcinoma syndrome. Medicine 1987; 66:98–113.

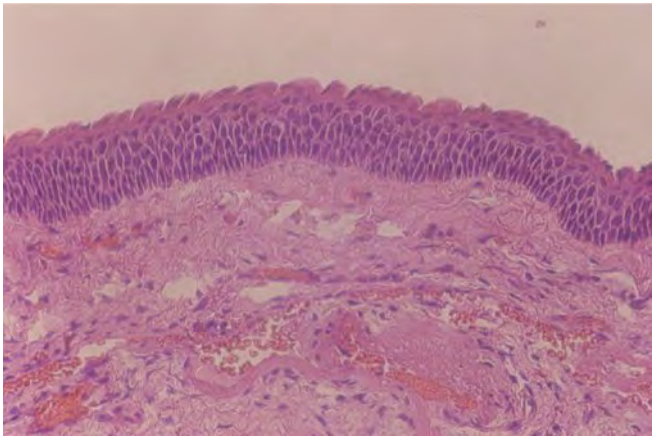


FIGURE 29-8. The classic histologic appearance of an odontogenic keratocyst from the incisional biopsy of the lesion in Figure 29-7 (H&E, original magnification ×40).

Treatment and Prognosis

Like the treatment of most odontogenic cysts, the odontogenic keratocyst may be treated with enucleation and curettage and is ideally removed in one piece, which requires acceptable access and lighting (Figure 29-9). As such, many patients are suitably treated in an operating room setting under general anesthesia. This is particularly helpful when removing large cysts. It is the author's experience and that of others that a large majority of sporadic odontogenic keratocysts may be effectively managed with a thorough enucleation and curettage surgery.[20,21] MacIntosh[22] once advocated the resection of odontogenic keratocysts with 5-mm linear margins as the preferred primary method of treatment and reported on 37 patients with 43 lesions emphasizing the efficacy and superior results of resection over all other therapeutic undertakings.

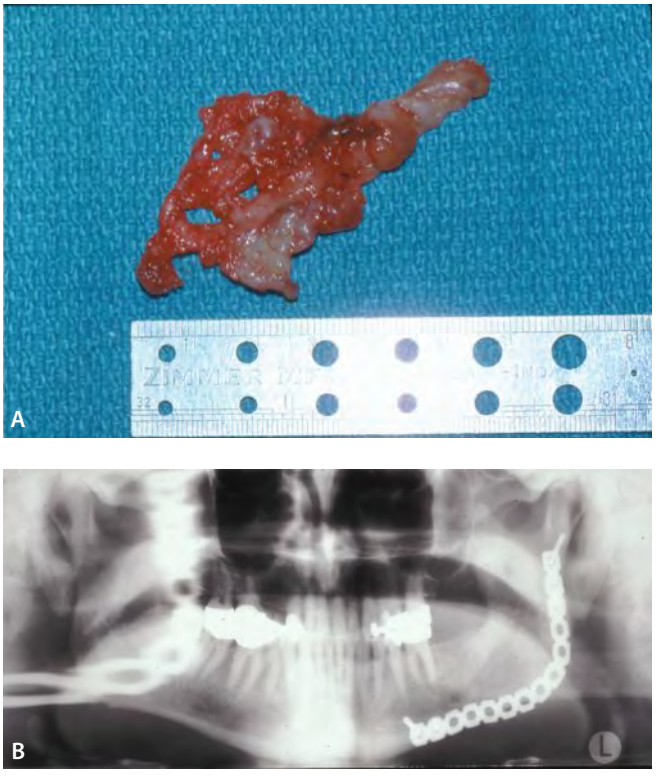


FIGURE 29-9. **A,** A very thin cyst lining was encountered when performing the enucleation and curettage of the odontogenic keratocyst in Figure 29-7. **B,** The 7-year postoperative radiograph shows an excellent fill of bone. A reconstruction bone plate was placed at the time of the enucleation and curettage to prevent a pathologic fracture of the mandible.

The reported frequency of recurrence of the odontogenic keratocyst ranges from 2.5% to 62.5% in various studies.[13] This wide variation may be related to the total number of cases studied, the length of follow-up periods, and the inclusion or exclusion of orthokeratinized cysts in the study group. Several reports that include large numbers of cases indicate a recurrence rate of approximately 30%.[2] Regezi and coworkers[23] point out that the recurrence rate for solitary odontogenic keratocysts is 10% to 30%. They indicated that approximately 5% of patients with odontogenic keratocysts have multiple sporadic jaw cysts (nonsyndromic) and that their recurrence rate is greater than that for solitary lesions.[23] Brannon[24] has suggested three mechanisms responsible for recurrence: (1) remnants of dental lamina within the jaws not associated with the original odontogenic keratocyst being responsible for de novo cyst formation; (2) incomplete removal (persistence) of the original cyst secondary to a thin friable lining and cortical perforation with adherence to adjacent soft tissue; and (3) remaining rests of dental lamina and satellite cysts after enucleation. Vedtofte and Praetorius[25] reviewed 72 patients with 75 odontogenic keratocysts and observed remnants of dental lamina between the cyst membrane and the overlying mucosa. As such, they advocated the excision of overlying mucosa in conjunction with removal of the cyst. Williams and Connor[13] recommended a primary enucleation and curettage surgery for odontogenic keratocysts, including the use of methylene blue as a marking agent, followed by a 3-minute application of Carnoy's solution. They indicated that resection should be considered for the treatment of a recurrent odontogenic keratocyst, with inclusion of appropriate bone and soft tissue margins.

Pathogenetically, the odontogenic keratocyst expresses cell cycle phenomena that support its proliferation.[23] These include the release of the cytokines interleukin 1a (IL-1a) and IL-6 as well as parathyroid hormone–related protein that encourage resorption of bone.[23,26] Moreover, the expression of proliferating cell nuclear antigen (PCNA) in odontogenic cysts has been assessed. It is hypothesized that the identification of the proliferative activity in odontogenic cysts and tumors may be useful to predict their biologic behavior. The same may be true of the Ki-67 antigen. In fact, two studies have been performed that have quantified these parameters.[27,28] The conclusion of both studies is that an increased proliferative activity for the odontogenic keratocyst in comparison with the dentigerous cyst is noted consistently. These results are in agreement with the more aggressive behavior

seen with the odontogenic keratocyst compared with the dentigerous cyst.

The orthokeratinized odontogenic cyst, once thought to be a variant of the odontogenic keratocyst, is now generally well accepted as being a different clinicopathologic entity from the more common parakeratinized odontogenic keratocyst. The orthokeratinized odontogenic cyst should, therefore, be placed in a different category. These cysts usually appear as unilocular radiolucencies, but occasional examples have been multilocular. A majority of these cysts are encountered in a lesion that appears clinically and radiographically to represent a dentigerous cyst, most often involving an unerupted mandibular third molar tooth. Histologically, the epithelium is thin and orthokeratinized, and a prominent palisaded basal layer, characteristic of the odontogenic keratocyst, is not present. Enucleation and curettage of the orthokeratinized cyst is curative in most cases. The reported rate of recurrence of 2% is far lower than the previously quoted statistics for recurrence of the odontogenic keratocyst.[2]



FIGURE 29-10. This 12-year-old shows some of the clinical features of the nevoid basal cell carcinoma syndrome including mandibular prognathism.

Nevoid Basal Cell Carcinoma Syndrome

The nevoid basal cell carcinoma syndrome (basal cell nevus syndrome, Gorlin's syndrome) is an autosomal dominant inherited condition that exhibits high penetrance and variable expressivity. It is caused by mutations in the *PTCH* tumor suppressor gene, mapped to chromosome 9q22.3-q31. Affected patients (Figure 29-10) may demonstrate frontal and temporoparietal bossing, hypertelorism, and mandibular prognathism (see Table 29-2).[16] Other frequent skeletal anomalies include bifid ribs, lamellar calcification of the falx cerebri, and multiple odontogenic keratocysts (Figure 29-11).[16] The most significant clinical feature is the tendency to develop multiple basal cell carcinomas that may affect both exposed and non–sun-exposed areas of the skin (Figure 29-12). Pitting defects on the palms and soles can be found in nearly two thirds of affected patients (Figure 29-13). The discovery of multiple odontogenic keratocysts is usually the first manifestation of the syndrome that leads to the diagnosis. For this reason, any patient with an odontogenic keratocyst should be evaluated for this condition. Although the

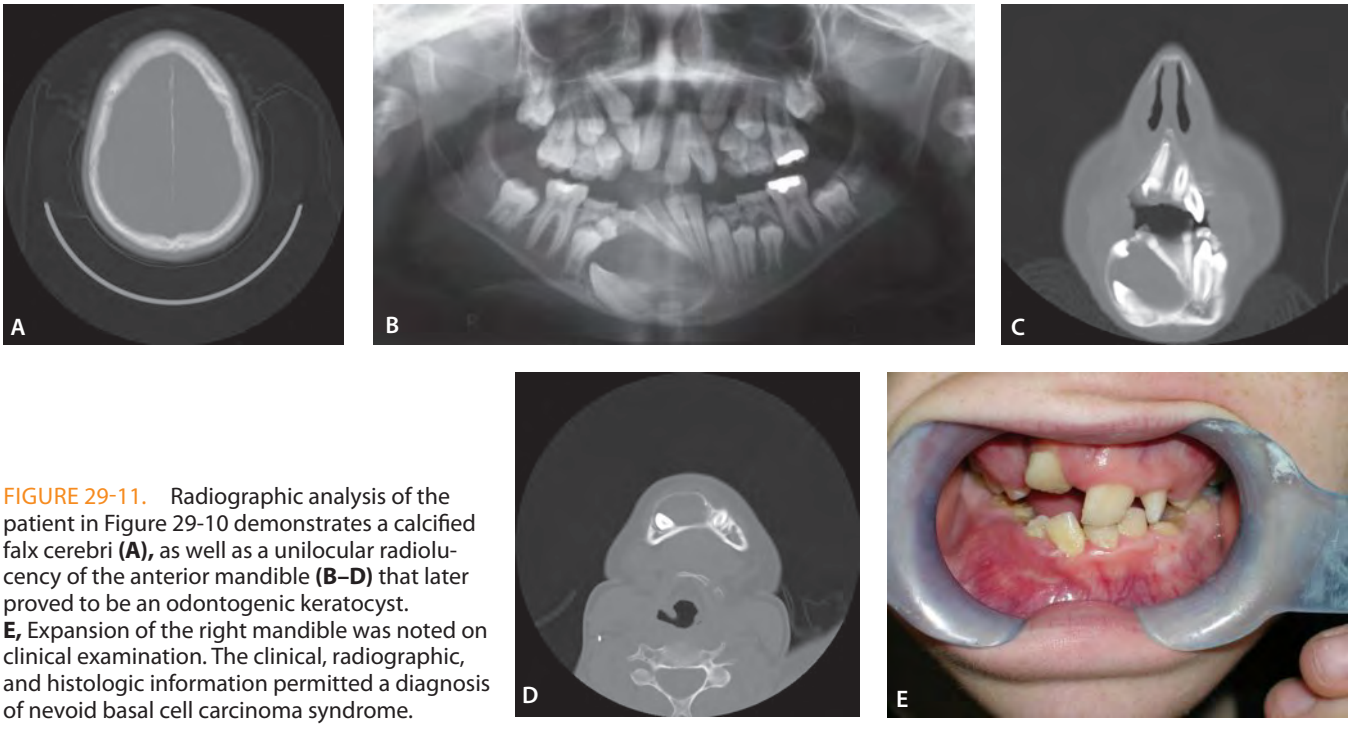


FIGURE 29-11. Radiographic analysis of the patient in Figure 29-10 demonstrates a calcified falx cerebri **(A),** as well as a unilocular radiolucency of the anterior mandible **(B–D)** that later proved to be an odontogenic keratocyst. **E,** Expansion of the right mandible was noted on clinical examination. The clinical, radiographic, and histologic information permitted a diagnosis of nevoid basal cell carcinoma syndrome.



FIGURE 29-12. The father of the patient in Figure 29-10 shows evidence of facial disfigurement due to the removal of approximately 50 basal cell carcinomas of the face related to his diagnosis of nevoid basal cell carcinoma syndrome. The father had previously undergone the removal of one odontogenic keratocyst of the anterior maxilla.

cysts in patients with nevoid basal cell carcinoma syndrome cannot definitely be distinguished microscopically from those not associated with the syndrome, they often demonstrate more epithelial proliferation and daughter cyst formation in the cyst wall.

The treatment of the odontogenic keratocyst in patients with nevoid basal cell carcinoma syndrome can be difficult owing to the large number of "recurrences" in these patients.



FIGURE 29-13. Plantar pitting can be observed by immersing the foot in povidone-iodine solution followed by a conservative wash of the foot with saline. The solution is taken up in the pits present in the plantar surface of the foot.

As a matter of point, the author chooses to refer to these "recurrences" as new primary cysts owing to the autosomal dominant penetrance of the syndrome and cyst development. It is certainly possible that many of these cysts are persistent, particularly when considering how common it can be to retain rests of the dental lamina when enucleating an odontogenic keratocyst. Whatever the mechanism, a resection hardly seems to be warranted when managing a syndromic cyst. Marsupialization is a more desirable procedure (Figure 29-14) and has been shown to result in complete resolution of the sporadic cyst, with no histologic signs of cystic remnants, daughter cysts, or budding of the basal layer of the epithelium.[29] Although all of the eight cases in the series by Pogrel and Jordan[29] were sporadic cysts, a similar approach to syndrome patients with odontogenic keratocysts that had been operated on multiple times has been performed with success in a small sample size.[20] Practically speaking, the marsupialization procedure should be undertaken with the intention of ultimately removing a smaller odontogenic keratocyst once radiographs demonstrate a significant decrease in the size of the cyst (see Figure 29-14).

Glandular Odontogenic Cyst

The glandular odontogenic cyst (sialo-odontogenic cyst) is a rare and recently described cyst of the jaws that is capable of aggressive behavior and recurrence. Although it is generally accepted as being of odontogenic origin, it shows glandular or salivary features that seem to point to the pluripotentiality of odontogenic epithelium because cuboidal/columnar cells, mucin production, and cilia are noted in these cysts. Glandular odontogenic cysts occur most commonly in middle-aged adults, with a mean age of 49 years at the time of diagnosis.[2]

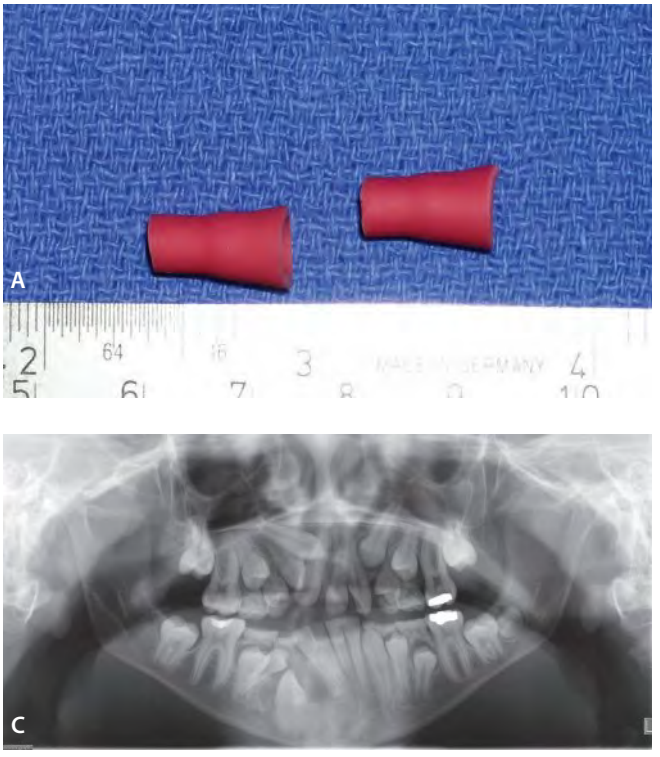


FIGURE 29-14. The patient in Figure 29-10 underwent marsupialization of his anterior mandibular odontogenic keratocyst. A red rubber catheter was fashioned **(A)** and placed in the cyst cavity after suturing the lining of the cyst to the oral mucosa **(B). C** and **D,** The marsupialization procedure permitted effective decrease in the size of the cyst so as to permit retention of the teeth and removal of the residual cyst after 9 months.

Eighty percent of cases occur in the mandible,[23] and a strong predilection for the anterior region of the jaws has been reported, with many mandibular lesions crossing the midline (Figure 29-15). These cysts may appear either unilocular or multilocular radiographically.

There is a histologic similarity between the glandular odontogenic cyst and the predominantly cystic intraosseous mucoepidermoid carcinoma. However, the epithelial lining of the glandular odontogenic cyst is typically thinner and does not show evidence of the more solid or microcystic epithelial proliferations seen in mucoepidermoid carcinoma (Figure 29-16). Waldron and Koh[30] reviewed the similarities between the two lesions and concluded that it is entirely possible that some cases previously diagnosed as central mucoepidermoid tumors may be reclassified as examples of glandular odontogenic cysts.

Treatment and Prognosis

Most glandular odontogenic cysts are treated with enucleation and curettage (Figure 29-17). Some authors, however, point to a recurrence rate of approximately 30% and, therefore, recommend resection.[31]

Calcifying Odontogenic Cyst

The calcifying odontogenic cyst (COC), or Gorlin's cyst, is an uncommon lesion that demonstrates considerable histopathologic diversity and variable clinical behavior. Although designated as a cyst, some investigators provide evidence for subclassification as a neoplasm as well.[32,33] In addition, the COC may be associated with other recognized odontogenic tumors, most commonly the odontoma. Adenomatoid odontogenic tumors and ameloblastomas have also been associated with the COC. Ghost cell keratinization, the characteristic microscopic feature of this cyst, is also a defining feature of the cutaneous lesion known as the *calcifying epithelioma of Malherbe* or *pilomatrixoma.* The World Health Organization's classification of odontogenic tumors groups the COC with all its variants as an odontogenic tumor rather than an odontogenic cyst. Specifically, this lesion is



FIGURE 29-15. **A** and **B,** This glandular odontogenic cyst presented as a unilocular radiolucency of the anterior mandible crossing the midline.

referred to as a *calcifying cystic odontogenic tumor* (Table 29-3). The commentary on the second edition by Kramer and associates[34] points out that some COCs appear to be nonneoplastic, but others show an infiltrative pattern of growth.

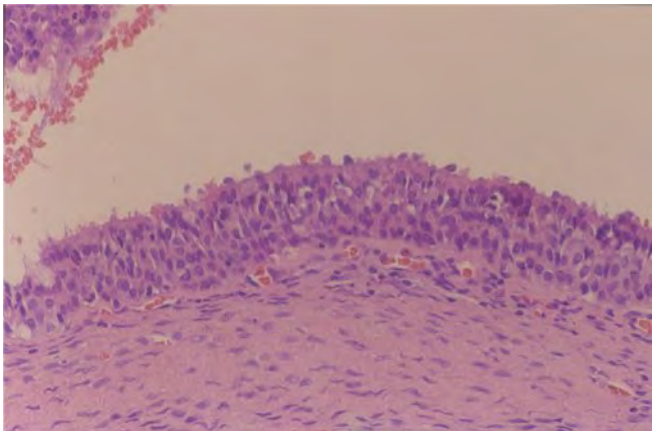


FIGURE 29-16. Histopathology of the lesion in Figure 29-15 shows a nonkeratinized stratified squamous epithelium with intraepithelial mucous cells and cilia (H&E, original magnification ×40).

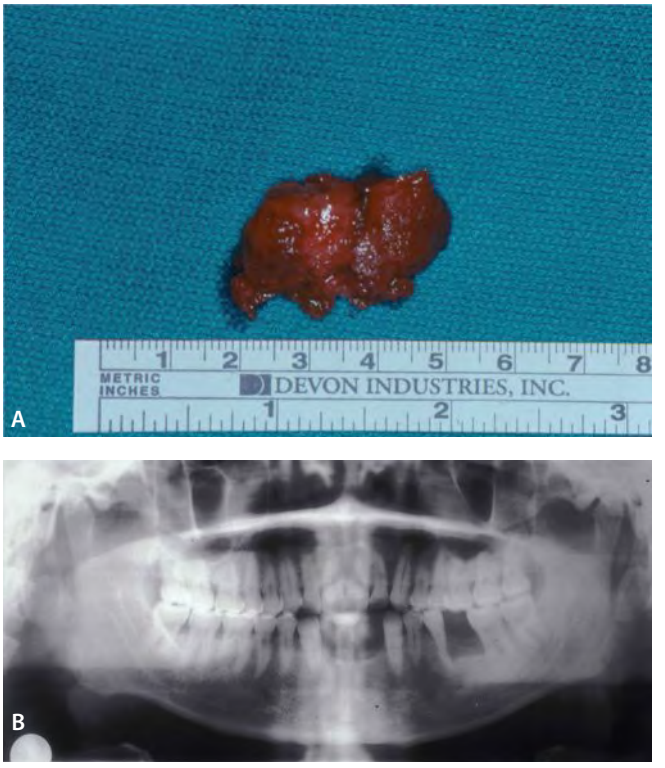


FIGURE 29-17. **A,** The patient whose radiograph appears in Figure 29-15 underwent an enucleation and curettage of the cyst as well as removal of the anterior mandibular teeth. **B,** The 3-year postoperative radiograph shows acceptable bony fill.

They further indicate that more experience with the COC may provide reliable criteria for their reclassification. The review by Hong and colleagues[32] designated 79 of 92 cases of COC as cysts, with the remaining 13 cases being neoplastic in nature.

The COC is predominantly an intraosseous lesion, although 13% to 30% of reported cases occur as peripheral lesions.[2] Both the peripheral and the central lesions occur with about equal frequency in the maxilla and mandible. There appears to be a predilection for the incisor and canine areas. Patients range in age from infant to elderly, with a mean age of occurrence of approximately 30 years. COCs that are associated with odontomas tend to occur in younger patients, with a mean age of 17 years.[2] The more rare neoplastic variant of the COC appears to occur in elderly patients. Most COCs appear radiographically as unilocular well-defined lesions (Figure 29-18). The radiopaque structures within the lesions have been described as either irregular calcifications or toothlike densities.

Treatment and Prognosis

The standard treatment for the COC is enucleation and curettage (Figure 29-19). A limited number of recurrences have been reported after such treatment. When a COC is associated with another recognized odontogenic tumor such as an ameloblastoma, the treatment and prognosis are likely to be

TABLE 29-3. **Classification of Benign Odontogenic Tumors**

Tumors of Odontogenic Epithelium with Mature, Fibrous Stroma without Odontogenic Ectomesenchyme
Ameloblastoma, solid/multicystic type
Ameloblastoma, extraosseous/peripheral type
Ameloblastoma, desmoplastic type
Ameloblastoma, unicystic type
Squamous odontogenic tumor
Calcifying epithelial odontogenic tumor
Adenomatoid odontogenic tumor
Keratocystic odontogenic tumor*

Tumors of Odontogenic Epithelium with Odontogenic Ectomesenchyme with or without Hard Tissue Formation
Ameloblastic fibroma
Ameloblastic fibro-odontoma
Ameloblastic fibrodentinoma
Odontoameloblastoma
Odontoma
- Compound type
- Complex type

Calcifiying cystic odontogenic tumor
Dentinogenic ghost cell tumor

Tumors with Mesenchyme and/or Odontogenic Ectomesenchyme with or without Odontogenic Epithelium
Odontogenic fibroma
Odontogenic myxoma/myxofibroma
Cementoblastoma

*As noted in the section of odontogenic cysts, the odontogenic keratocyst has been considered recently to represent a benign cystic odontogenic tumor. Owing to the lack of universal acceptance of this theory, the odontogenic keratocyst is included in the classification of odontogenic cysts as well as in this table of benign odontogenic tumors.
From Odontogenic tumours. Barnes L, Eveson JW, Reichart P, Sidransky D, editors. World Health Organization Classification of Tumours—Pathology and Genetics of Head and Neck Tumours. Lyon, France: International Agency for Research on Cancer (IARC); 2005; pp. 283–327.

the same as for the associated tumor. Although only a few cases have been reported,[33] a carcinoma arising in a COC may occur. One such reported case resulted in multiple pulmonary metastases and was referred to as an "odontogenic ghost cell carcinoma" by the authors.[35] It has not been demonstrated whether the malignant COC arose from previously benign lesions and, if so, whether that precursor was the cystic or neoplastic type. The rare neoplastic variant of the COC should be treated with resection (Figure 29-20).

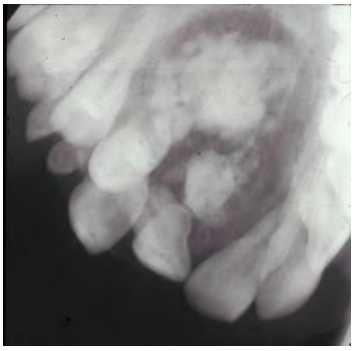


FIGURE 29-18. This calcifying odontogenic cyst appears as a mixed radiolucent/radiopaque lesion on the occlusal radiograph.

ODONTOGENIC TUMORS

Odontogenic tumors make up a complex group of lesions of great importance to oral and maxillofacial surgeons. Many of these lesions are true tumors, whereas some are hamartomas. Like normal odontogenesis, odontogenic tumors demonstrate varying inductive interactions between odontogenic epithelium and odontogenic ectomesenchyme. This ectomesenchyme was formerly referred to as *mesenchyme* because it was thought to be derived from the mesodermal layer of the embryo. It is now accepted that this tissue differentiates from the ectodermal layer in the cephalic portion of the embryo; hence, the designation *ectomesenchyme*. Odontogenic tumors are typically subclassified by their tissues of origin (see Table 29-3). Tumors of odontogenic epithelium are composed only of odontogenic epithelium without any participation of the odontogenic ectomesenchyme. Other odontogenic neoplasms, referred to as *mixed odontogenic tumors*, are

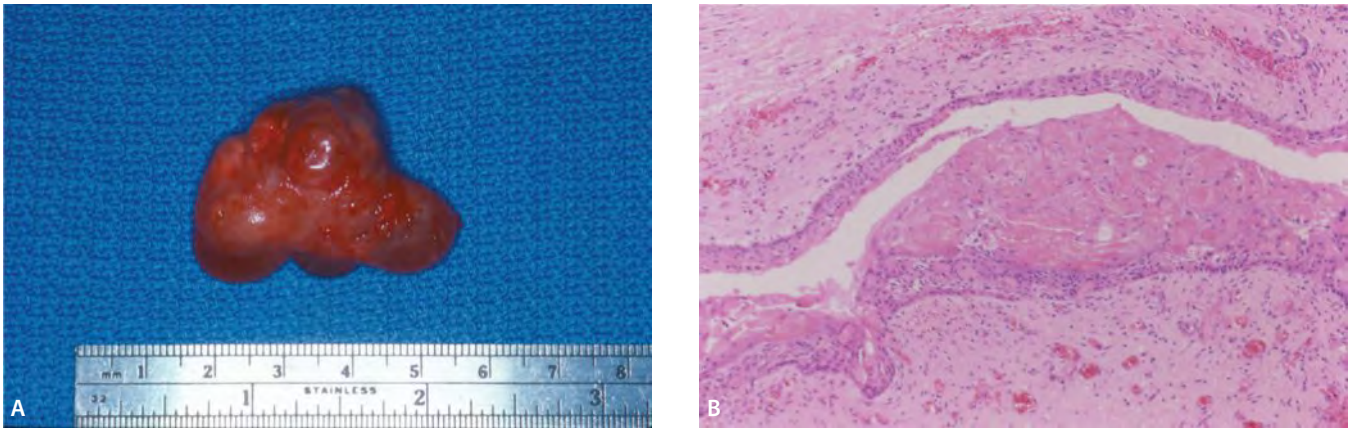


FIGURE 29-19. **A,** The patient whose radiograph is seen in Figure 29-18 underwent enucleation and curettage of the lesion. **B,** The histopathology shows characteristic ghost cells (H&E, original magnification ×40).

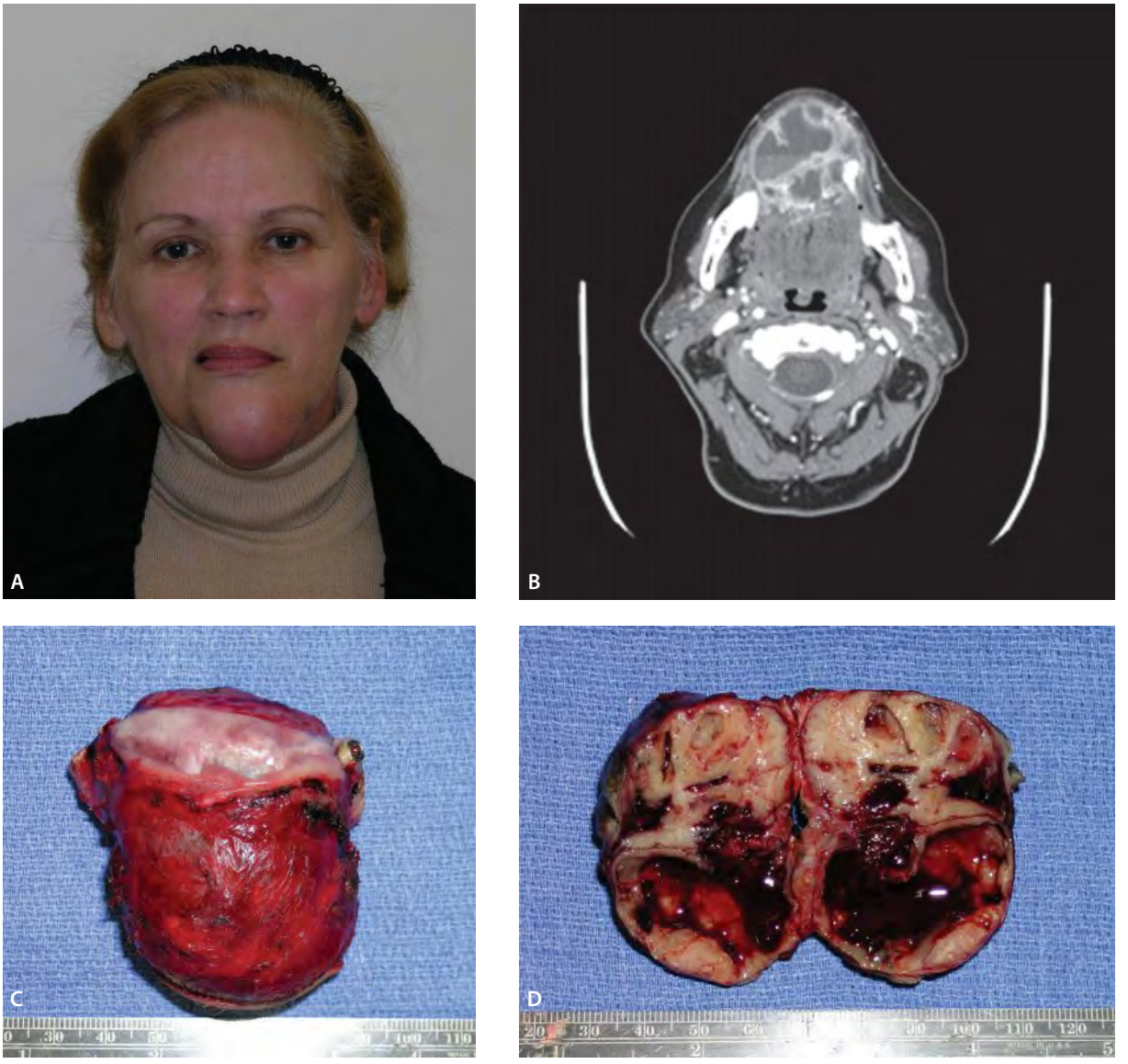


FIGURE 29-20. **A,** A 65-year-old woman with a large mass of the anterior mandible that showed a neoplastic (solid) calcifying odontogenic cyst on incisional biopsy. **B,** The computed tomography (CT) appearance of the tumor. **C,** Specimen from the segmental resection. **D,** The bivalved specimen identifies solid and cystic areas of the tumor.

composed of odontogenic epithelium and ectomesenchymal elements. A third group, tumors of odontogenic ectomesenchyme, includes those tumors composed principally of ectomesenchymal elements. Although some odontogenic epithelium may be included within these lesions, it does not appear to play an essential role in their pathogenesis.

The frequency of odontogenic tumors seems to be geographically determined (Table 29-4). Studies from North America seem to indicate that odontogenic tumors represent approximately 1% of all accessions in oral pathology laboratories,[36,37] whereas African studies have a much higher incidence of odontogenic tumors.[38–43] Moreover, the ameloblastoma is more commonly encountered in African and other underdeveloped countries than in North America.

TABLE 29-4. **Incidence of Odontogenic Tumors**

Specimens	STUDY (YR)		
	Regezi et al.[36] (1978)	Odukoya[38] (1995)	Daley et al.[37] (1994)
Total	54,534	1511	40,000
Total odontogenic tumors	706 (1.3%)*	289 (19.1%)*	445 (1.1%)*
Ameloblastoma	78 (11.0%)†	169 (58.5%)†	79 (17.8%)†
Adenomatoid odontogenic tumor	22 (3.1%)†	18 (6.2%)†	14 (3.1%)†
Odontoma	473 (67.0%)†	12 (4.2%)†	204 (45.8%)†
Myxoma	20 (2.8%)†	34 (11.8%)†	24 (5.4%)†

*Percentage of total specimens in respective study.
†Percentage of total odontogenic tumors in respective study specimens.

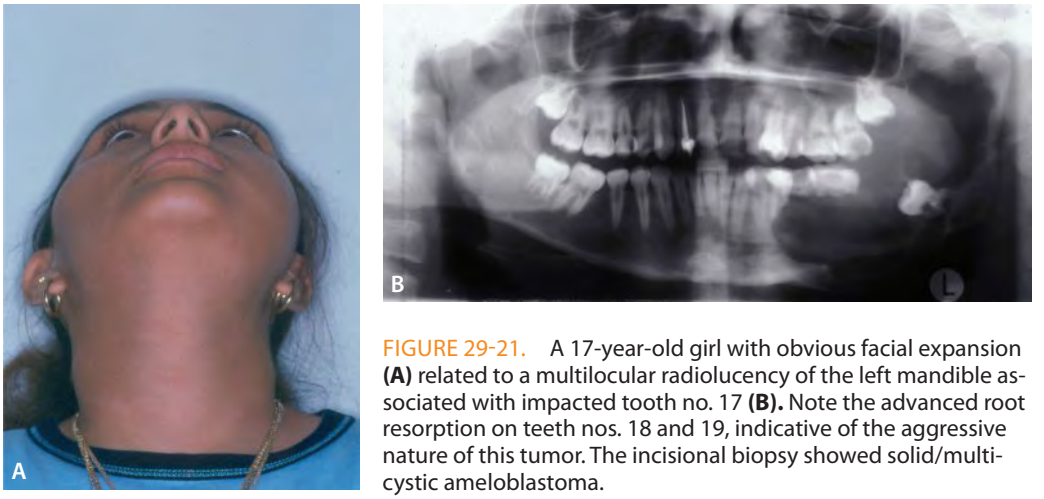


FIGURE 29-21. A 17-year-old girl with obvious facial expansion **(A)** related to a multilocular radiolucency of the left mandible associated with impacted tooth no. 17 **(B).** Note the advanced root resorption on teeth nos. 18 and 19, indicative of the aggressive nature of this tumor. The incisional biopsy showed solid/multicystic ameloblastoma.

Ameloblastoma

The ameloblastoma is the most common clinically significant and potentially lethal odontogenic tumor. Excluding odontomas, its incidence equals or exceeds the combined total of all other odontogenic tumors. These tumors may arise from rests of the dental lamina, a developing enamel organ, the epithelial lining of an odontogenic cyst, or the basal cells of the oral mucosa.[2] The ameloblastoma occurs in three different variants, each with specific implications for treatment and a unique prognosis: solid or multicystic, unicystic, and peripheral. In an analysis of the international literature, 3677 cases of ameloblastoma were reviewed, of which 92% were solid or multicystic, 6% were unicystic, and 2% were peripheral.[44]

Solid or Multicystic Ameloblastoma

This variant of the ameloblastoma is encountered in patients over a wide age range.[45] It is rare in children in their first decade of life and relatively uncommon in the second decade.[46] The tumor shows a relatively equal rate of occurrence in the third through seventh decades. There is no gender predilection, and racial predilection is most controversial. About 85% of this variant of the ameloblastoma occur in the mandible, most commonly in the molar/ramus region.[47] About 15% of multicystic ameloblastomas occur in the maxilla, usually in the posterior regions.[48–51] A painless expansion of the jaws is the most common clinical presentation; neurosensory changes are uncommon, even with large tumors (Figure 29-21). Slow growth is the rule, with untreated tumors leading to tremendous facial disfigurement (Figure 29-22).[52]

The most common radiographic feature is that of a multilocular radiolucency. Buccal and lingual cortical expansion is common, frequently to the point of perforation. Resorption of adjacent tooth roots is common. Histologic patterns include *follicular*, in which the stellate reticulum is located within the center of the odontogenic island (Figure 29-23); *plexiform*, in which the stellate reticulum is located outside



FIGURE 29-22. Twenty years of undisturbed growth of a solid/multicystic ameloblastoma led to significant facial disfigurement **(A),** with an impressive radiographic appearance **(B). C,** A segmental resection of the right mandible was performed.

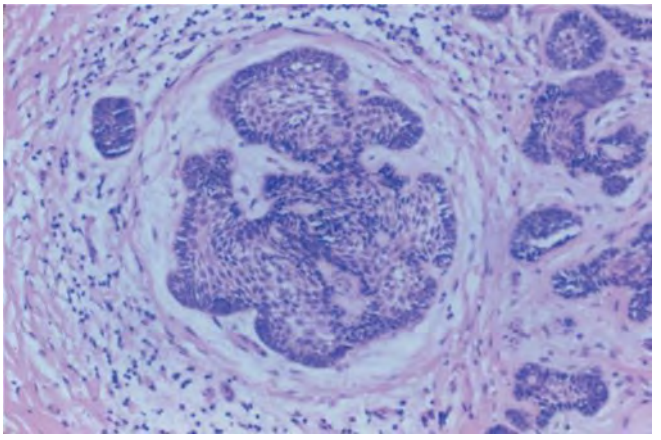


FIGURE 29-23. The incisional biopsy of the patient in Figure 29-20 shows follicular variant of the solid/multicystic ameloblastoma (H&E, original magnification ×60).

of the odontogenic rest; *acanthomatous*, in which squamous differentiation of the odontogenic epithelium is present; *granular cell*, in which the tumor islands exhibit cells that demonstrate abundant granular eosinophilic cytoplasm; *desmoplastic* owing to extremely dense collagenized stroma that supports the tumor; and the least common *basal cell* variant, in which nests of uniform basaloid cells are present, with a strong resemblance to basal cell carcinoma. In this latter tumor, stellate reticulum is not present in the central portions of the nests. One additional exception surrounds the desmoplastic variant, which is generally not a radiolucent tumor radiographically owing to its high content of collagenized stroma.

Pathogenetically, the proliferative capacity of ameloblastomas has been studied. As might be conjectured, the recurrent ameloblastoma is associated with the highest number of PCNA-positive cells, followed by the previously unoperated ameloblastomas.[28] The nuclear PCNA positivity of the unicystic ameloblastoma was notably lower than the positivity of the solid/multicystic ameloblastoma.[28] Other cell cycle features supporting the aggressive behavior of the ameloblastoma include the overexpression of *BCL2* and *BCLX*, as well as the expression of IL-1 and IL-6.[53]

Treatment and Prognosis

The ameloblastoma continues to be a subject of fascination in the international literature. Unfortunately, although most agree that aggressive treatment is essential for cure of this tumor, the fact remains that a consensus has not been reached on the biologic behavior of this neoplasm and how best to treat it.[54] The literature is, therefore, paradoxically a source of both information and misinformation. Conflicting opinion, extending backward in time, has served both to educate and to confuse, and it has been left to generations of surgeons to sift and interpret what they consider to be clinically valid. It is the author's strong opinion that this neoplasm is both highly aggressive and curable.[55,56] This notwithstanding, numerous methods of treatment have been recommended, ranging from simple enucleation and curettage to resection.[57–63] The solid or multicystic ameloblastoma tends to infiltrate between intact cancellous bone trabeculae at the periphery of the tumor before bone resorption becomes radiographically evident. Therefore, the actual margin of the tumor often extends beyond its apparent radiographic or clinical margin.[64] Attempts to remove the tumor by curettage, therefore, predictably leave behind small islands of tumor within the bone, which are later determined to be recurrent disease. These must be realized as *persistent disease* because the tumor was never controlled from the outset. When a small burden of tumor is left behind, it may be decades before this persistent disease becomes clinically and radiographically evident, and long after a surgeon falsely proclaimed the patient to be cured.

Owing to the highly infiltrative and aggressive nature of the solid or multicystic ameloblastoma, the author recommends resection of the tumor with 1.0 cm linear bony margins (Figure 29-24). This linear bony margin should be confirmed by intraoperative specimen radiographs (see Figure 29-24B).

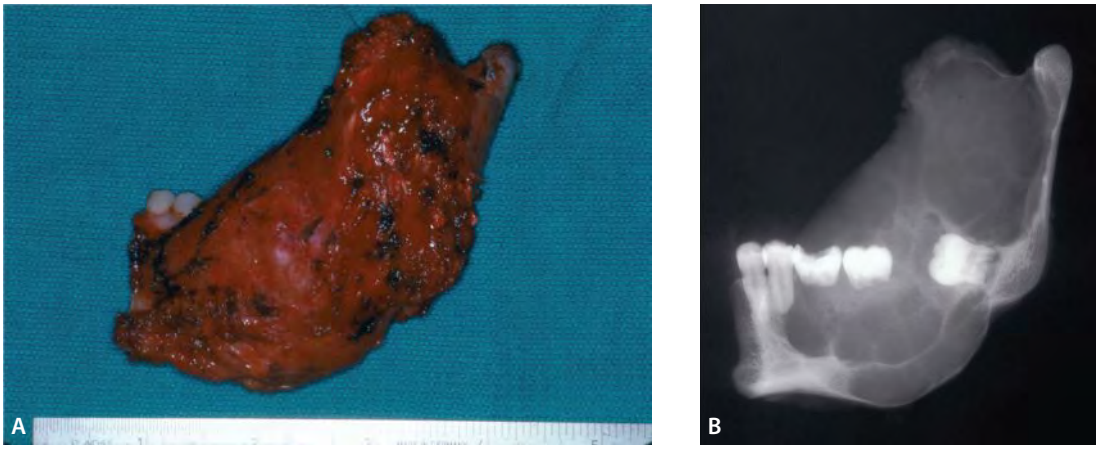


FIGURE 29-24. **A,** Treatment of the ameloblastoma of the patient in Figure 29-21 required a disarticulation resection of the left mandible. **B,** The effectiveness of the bony linear margin should always be evaluated by intraoperative specimen radiographs.

Soft tissue margins are best managed according to the anatomic barrier margin principles whereby one uninvolved surrounding anatomic barrier is sacrificed on the periphery of the specimen.[65] When all soft and hard tissue margins are histologically negative, the patient is likely to be cured of this neoplasm. Unfortunately, any less aggressive treatment modality may be fraught with inevitable persistence discovered at variable times postoperatively.[66] Moreover, although the persistent and occasionally nonresectable ameloblastoma is radiosensitive, once this tumor defies curative surgical therapy, radiation is of questionable use in salvaging these patients.[67,68]

One very important question that has surfaced in the literature is whether or not the ameloblastoma is malignant. Discussions by Willis[69] and Carr and Halperin[70] exemplify this debate. Willis[69] stated that attempts to distinguish between benign and malignant ameloblatoma are futile in that they are all malignant because they are locally invasive and prone to recur. Carr and Halperin[70] stated that malignant and benign are poor terms when applied generally to the ameloblastoma. Furthermore, they stated that the use of one term versus the other seems secondary to an understanding of how the tumor may behave and to an approach to treatment based upon this understanding. Gold and Williams[71] justified their belief that all ameloblastomas are malignant in drawing an analogy between basal cell carcinoma of skin and the ameloblastoma. They indicated that the basal cell carcinoma is slow growing, infiltrative, capable of great destruction of soft tissue and bone, recurrent when not completely eradicated, capable of invading vital structures, seldom metastatic but capable of metastasis, presenting a number of histologic patterns, and arising from the epithelial skin surface and skin adnexa. They rationalized that if one substitutes oral epithelium for skin surface and dental lamina/enamel organ for skin adnexa, one would be describing the ameloblastoma. Despite their argument, Gold and Williams lamented that the long-held traditional belief that the ameloblastoma is benign will be difficult to reverse. Finally, as agreed by most surgeons and pathologists, the ameloblastoma is distinctly aggressive, infiltrative, and unpredictable in its behavior.

Unicystic Ameloblastoma

In 1970, Vickers and Gorlin[72] published their findings regarding the histologic alterations associated with neoplastic transformation of ameloblastomatous epithelium. These histologic changes were (1) hyperchromatism of basal cell nuclei of the epithelium lining the cystic cavities, (2) palisading and polarization of basal cell nuclei of the epithelium lining the cystic cavities, and (3) cytoplasmic vacuolization, particularly of basal cells of cystic linings. They referred to these changes as early histopathologic features of neoplasia. *Unicystic ameloblastoma* refers to a pattern of epithelial proliferation that has been described in dentigerous cysts of the jaws[73–76] that does not exhibit the histologic criteria for ameloblastoma published by Vickers and Gorlin. This entity deserves separate consideration based on its clinical, radiographic, and pathologic features. Moreover, in many cases, it may be treated more conservatively than the solid or multicystic ameloblastoma with the same degree of cure.[77]



FIGURE 29-25. This unilocular radiolucency associated with tooth no. 17 should generate a differential diagnosis of dentigerous and other odontogenic cysts as well as a unicystic ameloblastoma.

Unicystic ameloblastomas are most commonly seen in young patients, with approximately 50% of these tumors being diagnosed during the second decade of life. The average age of patients with unicystic ameloblastomas has been reported as 22.1 years, compared with 40.2 years for the solid or multicystic variant.[44] More than 90% of these tumors are found in the mandible, usually in the molar/ramus region.[78] A unilocular radiolucency, mimicking a dentigerous cyst, is the most common radiographic presentation for the unicystic ameloblastoma (Figure 29-25). Most, if not all, unicystic ameloblastomas are unilocular radiolucencies.[2] Three histopathologic variants of unicystic ameloblastoma have been described that impact treatment and prognosis. In the *luminal unicystic ameloblastoma*, the tumor is confined to the luminal surface of the cyst (Figure 29-26). The lesion consists of a fibrous cyst wall with a lining that consists totally or partially of ameloblastic epithelium. The *intraluminal unicystic ameloblastoma* contains one or more nodules of

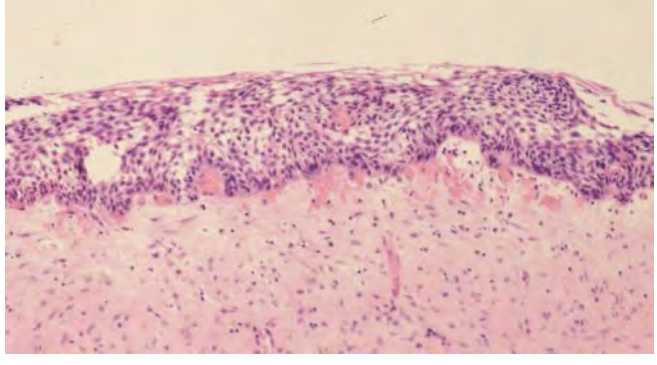


FIGURE 29-26. The histopathology of the lesion in Figure 29-25 shows luminal unicystic ameloblastoma (H&E, original magnification ×40).

ameloblastoma projecting from the cystic lining into the lumen of the cyst. These nodules may be relatively small or largely fill the cystic lumen and are noted to show a plexiform pattern that resembles the plexiform pattern seen in conventional ameloblastomas. As such, these tumors are referred to as *plexiform unicystic ameloblastomas.* In the third variant, known as *mural unicystic ameloblastoma*, the fibrous wall of the cyst is infiltrated by typical follicular or plexiform ameloblastoma. The extent and depth of the ameloblastic infiltration may vary considerably.

Pathogenetically, the unicystic ameloblastoma seems to have a proliferative capacity between that of the odontogenic keratocyst and that of the solid or multicystic ameloblastoma.[28]

Treatment and Prognosis

The clinical and radiographic findings in most cases of unicystic ameloblastoma suggest that the lesion is an odontogenic cyst, most commonly a dentigerous cyst. Under the circumstances, the surgeon should routinely open a "cystic" lesion and look for luminal proliferation of tumor. When able, histopathologic examination of such a process should occur with frozen sections. This is particularly important when dealing with large cysts. With a histologic diagnosis of unicystic ameloblastoma, the surgeon should request the pathologist to obtain multiple sections through many levels of the specimen to properly subclassify the variant of unicystic ameloblastoma. When the ameloblastic elements are confined to the lumen of the cyst with or without intraluminal tumor extension, the enucleation has probably been curative treatment. When the cyst wall has been violated by the tumor as in a mural variant of unicystic ameloblastoma, the most appropriate surgical management is quite controversial. If this diagnosis is made postoperatively, the surgeon may wish to adopt close indefinite follow-up examinations of the patient. If a preoperative incisional biopsy provides a diagnosis of mural unicystic ameloblastoma, the surgeon might recommend a resection of the tumor owing to the fact that this variant of the unicystic ameloblastoma has a higher rate of persistence than do the luminal or intraluminal unicystic ameloblastomas.

The treatment of a luminal or intraluminal variant of the unicystic ameloblastoma is enucleation and curettage (Figure 29-27). In a collective sense, the "recurrence" rate of all unicystic ameloblastomas has been reported as 10% to 20% after enucleation and curettage.[77] This is significantly lower than that of enucleation and curettage of the solid or multicystic ameloblastoma. The question then arises as to when to resect a unicystic ameloblastoma. Three instances are likely to require such treatment. The first is the recurrent unicystic ameloblastoma. A tumor that recurs after a well-performed enucleation and curettage should probably be approached with the more aggressive resection. Second is the mural ameloblastoma. This variant of the unicystic ameloblastoma is probably more aggressive than the luminal and intraluminal variants of the unicystic ameloblastoma owing to the presence of tumor in the cyst wall and, therefore, closer to the surrounding bone. It seems logical to approach these tumors with a surgery similar to that for the solid or multicystic ameloblastoma (Figure 29-28). The final indication for resection of a unicystic ameloblastoma is in the management of very large tumors (see Figure 29-28) with significant expansion such that an enucleation and curettage surgery would effectively result in a resection of the involved jaw.

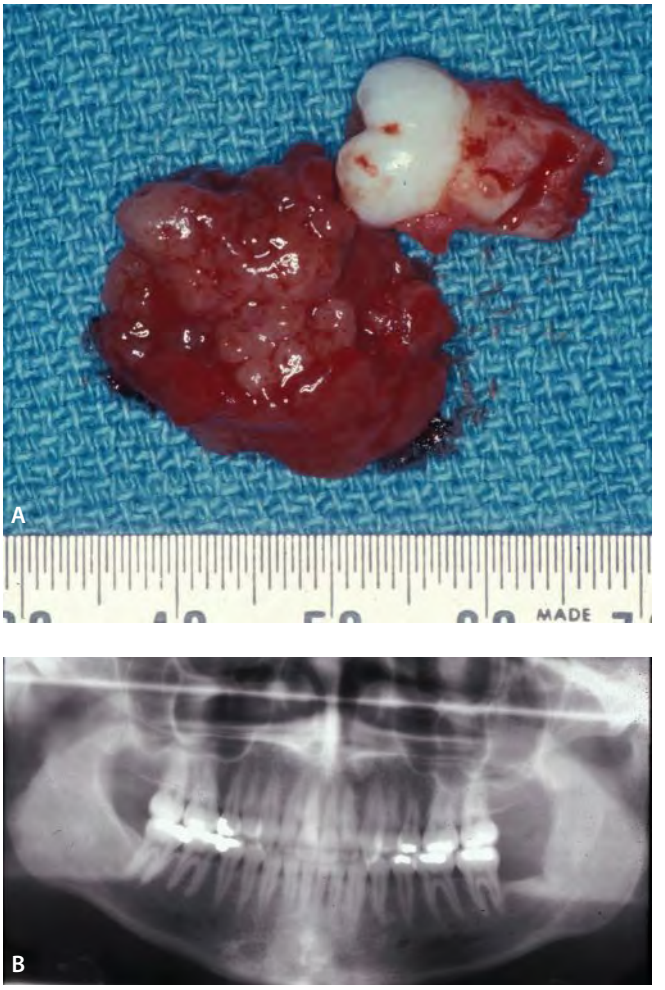


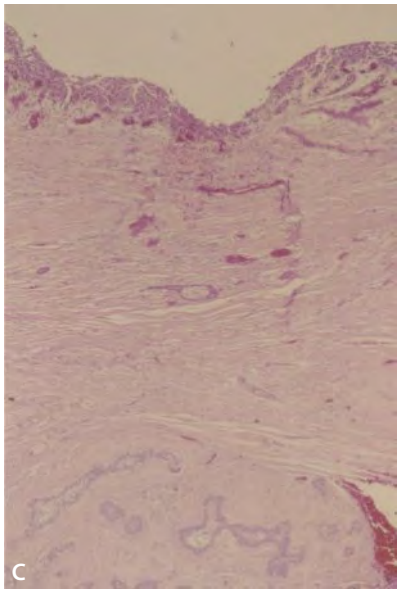
FIGURE 29-27. **A,** The luminal unicystic ameloblastoma in Figure 29-25 is treated with an enucleation and curettage surgery. **B,** The 5-year postoperative radiograph shows an acceptable bony fill.

Peripheral Ameloblastoma

The peripheral or extraosseous ameloblastoma is the most rare variant of the ameloblastoma. This tumor probably arises from rests of dental lamina or the basal epithelial cells of the surface epithelium and shows the same features of the intraosseous form of the tumor.[79] Clinically, these tumors present as nonulcerated sessile or pedunculated gingival lesions (Figure 29-29). Most examples are smaller than 1.5 cm and usually occur over a wide age range, with an average reported age of 52 years. Although these tumors do not infiltrate bone, they may be seen to "cup out" bone in the jaws (Figure 29-30).







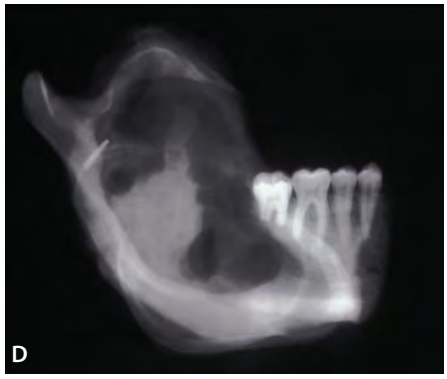


FIGURE 29-28. This 18-year-old male presented with significant right facial expansion **(A)** associated with the destructive radiolucency of the right mandible noted on the panoramic radiograph **(B). C,** The incisional biopsy documented the mural variant of unicystic ameloblastoma (H&E, original magnification ×20). **D,** A disarticulation resection was performed and a specimen radiograph was obtained.

SECTION 4

Treatment and Prognosis

The peripheral ameloblastoma is most appropriately treated with a wide local excision. When surgical margins are negative for tumor, cure is the likely consequence. Malignant transformation of a peripheral ameloblastoma is very rare.[80]

Malignant Odontogenic Tumors

Malignant odontogenic tumors are very rare. They may arise from the epithelial components of the odontogenic apparatus. The rests of Malassez, the reduced enamel epithelium surrounding the crown of an impacted tooth, the rests of Serres



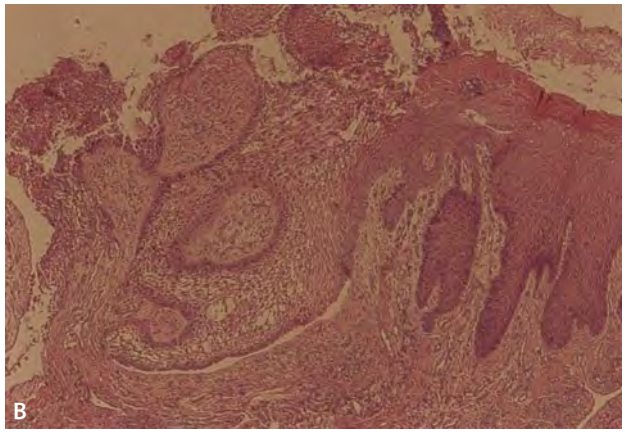


FIGURE 29-29. This lesion of the right palatal mucosa **(A)** showed peripheral ameloblastoma on incisional biopsy (H&E, original magnification ×40) **(B).**



FIGURE 29-30. A "cupped-out" lesion in bone at tooth no. 4 is noted in the patient in Figure 29-29.

in the gingiva, and the linings of odontogenic cysts represent the precursor cells for malignant transformation. Malignant odontogenic tumors are classified in Table 29-5. In general, all of these tumors exhibit typical microscopic features of malignancy, with the exception of the metastasizing (malignant) ameloblastoma and the clear cell odontogenic carcinoma. Behaviorally, all of these tumors have the potential for either regional nodal or distant metastases.

TABLE 29-5. **Classification of Malignant Odontogenic Tumors**

Odontogenic Carcinomas
Metastasizing (malignant) ameloblastoma
Ameloblastic carcinoma—primary type
Ameloblastic carcinoma—secondary type (dedifferentiated), intraosseous
Ameloblastic carcinoma—secondary type (dedifferentiated), peripheral
Primary intraosseous squamous cell carcinoma—solid type
Primary intraosseous squamous cell carcinoma derived from keratocystic odontogenic tumor
Primary intraosseous squamous cell carcinoma derived from odontogenic cysts
Clear cell odontogenic carcinoma
Ghost cell odontogenic carcinoma
Odontogenic Sarcomas
Ameloblastic fibrosarcoma
Ameloblastic fibrodentino- and fibro-odontosarcoma

From Odontogenic tumours. Barnes L, Eveson JW, Reichart P, Sidransky D, editors. World Health Organization Classification of Tumours—Pathology and Genetics of Head and Neck Tumours. Lyon, France: International Agency for Research on Cancer (IARC); 2005; pp. 283–327.

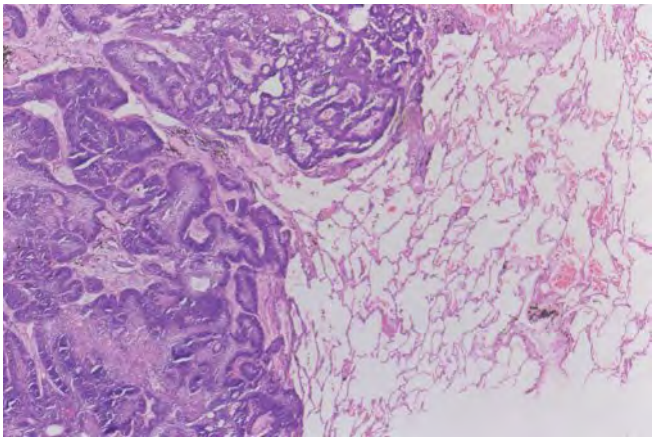


FIGURE 29-31. Histologically benign ameloblastoma is noted in the lung. This finding satisfies the definition of metastasizing ameloblastoma (H&E, original magnification ×20).

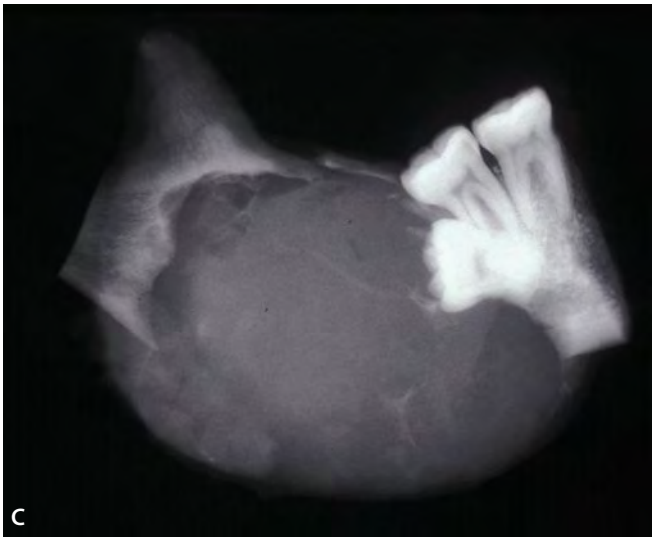
Metastasizing (Malignant) Ameloblastoma

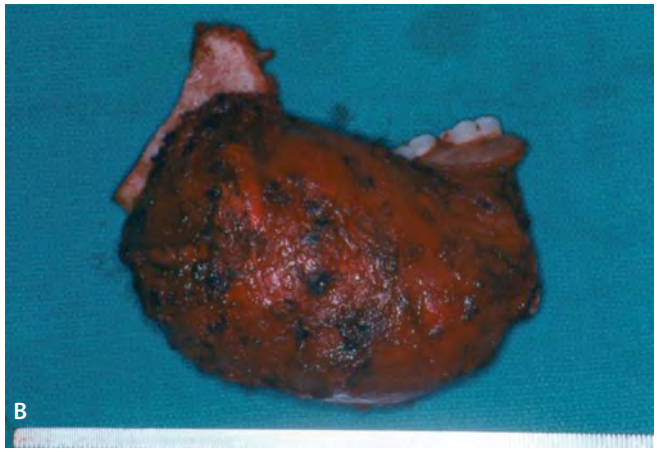
Metastasizing ameloblastomas are best described as neoplasms that have the histologic features of benign ameloblastoma as shown by the primary growth in the jaws and by any metastatic growth.[81] The most common sites of metastatic disease are the lungs (Figure 29-31), followed by the cervical lymph nodes and visceral organs.[82–84] Lung metastases have sometimes been regarded as aspiration phenomena, yet the peripheral location of many of these deposits supports hematogenous spread. MacIntosh[85] has pointed out that the aspiration hypothesis leaves unanswered the questions of why other aggressive oral lesions generally considered to be benign, such as the odontogenic myxoma or calcifying epithelial odontogenic tumor, have not been encountered in the lungs, particularly because many of these tumors have also been treated with conservative surgeries such as the enucleation and curretage. In addition, he stated that the aspiration hypothesis should be supported by the observation of a greater probability of tumor growth in the middle or lower lobe of the right lung, which is not the case. Eversole[86] points out that instances of metastasis have arisen from solid or multicystic ameloblastomas rather than unicystic tumors.

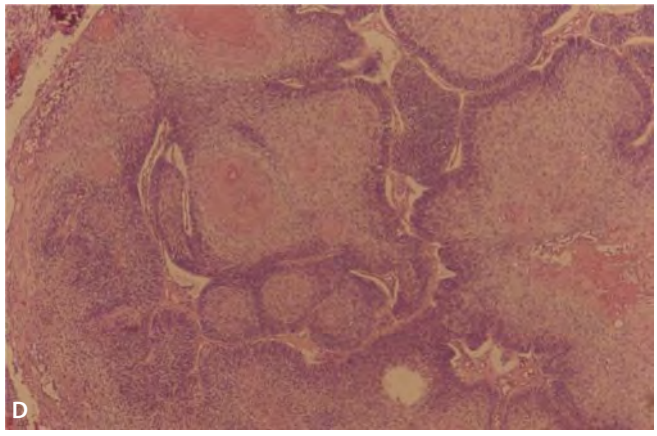
Ameloblastic Carcinoma

Ameloblastic carcinomas are malignant epithelial odontogenic tumors that exist in the background of benign ameloblastomas. This designation is reserved for an ameloblastoma that has cytologic features of malignancy in the primary tumor (Figure 29-32), in a recurrence, or in any metastatic deposit. Although ameloblastic carcinomas have been reported to metastasize to the lungs and distant organs,[87,88] many cases do not metastasize. In Corio and coworkers' series of eight cases of ameloblastic carcinoma,[89] rapid growth and pain were common symptoms. These symptoms are recognized as being uncommon in patients with benign ameloblastomas.









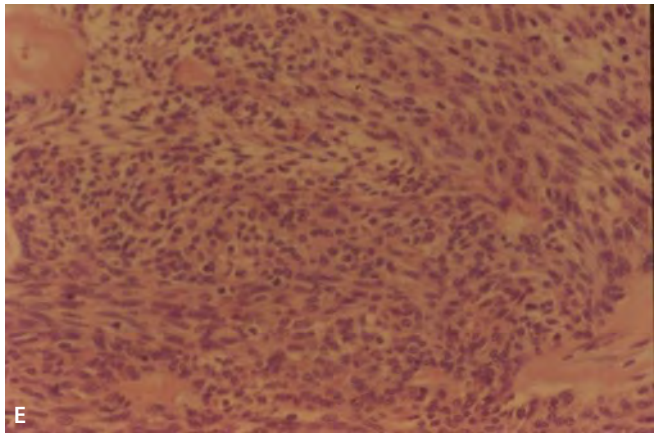


FIGURE 29-32. **A,** The large destructive radiolucency of the right mandible was present in a 22-year-old man who complained of precipitous growth and pain. The incisional biopsy showed benign solid/multicystic ameloblastoma. **B** and **C,** A segmental resection was performed. **D** and **E,** Final histopathology of the resection specimen showed ameloblastic carcinoma in a background of benign ameloblastoma (H&E, original magnification ×20 **[D]** and ×100 **[E]**).

Primary Intraosseous Squamous Cell Carcinoma

Squamous cell carcinomas that are encountered in the jaws, lack any continuity with the oral or antral mucosa, and occur in the absence of a primary carcinoma located elsewhere are termed *primary intraosseous squamous cell carcinomas.* These cases are assumed to arise from odontogenic epithelium. They typically occur in elderly patients and tend to occur in the mandibular body region. The 5-year survival rate is 30% to 40%.[86] Squamous cell carcinomas may also arise from the linings of odontogenic cysts. Cystogenic carcinomas are seen in patients older than 50 years of age and typically occur in the mandible. Finally, dentigerous cysts can undergo glandular metaplasia, and there are rare instances of central mucoepidermoid carcinomas reported to arise from odontogenic cyst lining.

Clear Cell Odontogenic Carcinoma

Although the clear cell odontogenic carcinoma is of putative odontogenic origin, histologic similarities to the developing tooth germ are lacking in many instances.[86] The differential diagnosis includes metastasis from a distant site, especially the kidney. The clear cell variant of renal cell carcinoma is the chief entity to consider. The clear cell odontogenic carcinoma is generally seen in elderly women, with the maxilla and mandible being affected equally.

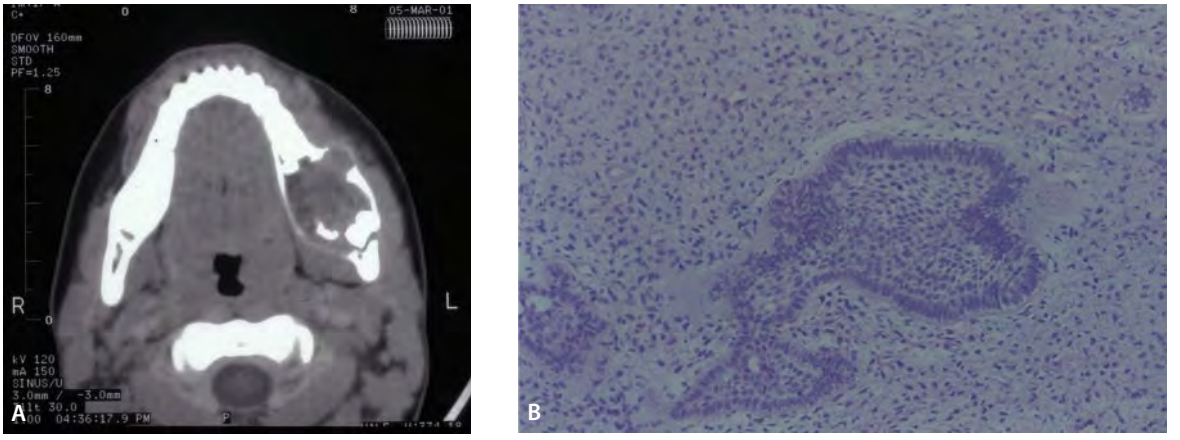


FIGURE 29-33. **A,** A destructive unilocular radiolucency is present in a 15-year-old boy. **B,** Incisional biopsy confirmed ameloblastic fibroma (H&E, original magnification ×40).

Malignant Epithelial Odontogenic Ghost Cell Tumor

The epithelial odontogenic ghost cell tumor, also known as dentinogenic ghost cell tumor, is the solid variant of the calcifying odontogenic cyst. Both epithelial and ectomesenchymal odontogenic elements are present; however, only the epithelial component shows cytologic features of malignancy.

Ameloblastic Fibroma

The ameloblastic fibroma is considered to be a true tumor in which the epithelial and mesenchymal tissues are both neoplastic. This is in distinction to the ameloblastic fibro-odontoma and odontoma that represent developmental stages of the same hamartomatous lesion.[90,91] The ameloblastic fibroma tends to occur in young patients in the first two decades of life. The posterior mandible is affected in 70% of cases (Figure 29-33). Radiographically, either a unilocular or a multilocular lesion is observed.

Treatment and Prognosis

The ameloblastic fibroma is recognized as an indolent tumor that is effectively treated by an enucleation and curettage surgery (Figure 29-34). Although recurrence is rare under the circumstances, resection should be reserved for recurrent lesions. Approximately 45% of ameloblastic fibrosarcomas develop in the setting of a recurrent ameloblastic fibroma.[2]

Ameloblastic Fibro-odontoma

The ameloblastic fibro-odontoma, as previously discussed, probably represents a hamartoma. Moreover, some investigators believe that this lesion is only a stage in the development of an odontoma and does not represent a separate entity. Slootweg[90] points out that when one considers the data on age, site, and sex, it seems that the ameloblastic fibro-odontoma is an immature complex odontoma. As with ameloblastic fibromas, the ameloblastic fibro-odontoma occurs more frequently in the posterior regions of the jaws. This lesion is commonly asymptomatic and is discovered serendipitously or when radiographs are exposed to provide a diagnosis for asymmetrical eruption of the dentition in children (Figure 29-35). These lesions are distinctly well circumscribed and appear as mixed radiopaque/radiolucent masses.

Treatment and Prognosis

The ameloblastic fibro-odontoma is treated effectively with an enucleation and curettage surgery (Figure 29-36). Recurrence after this approach is very rare. Malignant transformation of ameloblastic fibro-odontoma has been reported but is exceedingly rare.[92]

Odontoma

Odontomas are the most frequently occurring odontogenic tumors, with prevalence exceeding that of all other odontogenic tumors combined. As stated previously, these lesions are generally well accepted as representing hamartomas. Odontomas present centrally within the jaws in one of two



FIGURE 29-34. An enucleation and curettage surgery is performed in the patient in Figure 29-33. The associated permanent teeth are removed with the tumor.



FIGURE 29-35. A panoramic radiograph of a 9-year-old boy shows a mixed radiolucent/radiopaque lesion of the left posterior mandible. Ameloblastic fibro-odontoma is a likely diagnosis owing to the patient's age as well as the radiographic character of the lesion.

forms: compound, in which multiple small toothlike structures exist; and complex, in which irregular masses of dentin and enamel are present with no anatomic resemblance to a tooth. Compound odontomas are predominantly seen in the anterior maxilla (Figure 29-37), whereas complex odontomas are typically seen in the posterior maxilla or mandible (Figure 29-38).

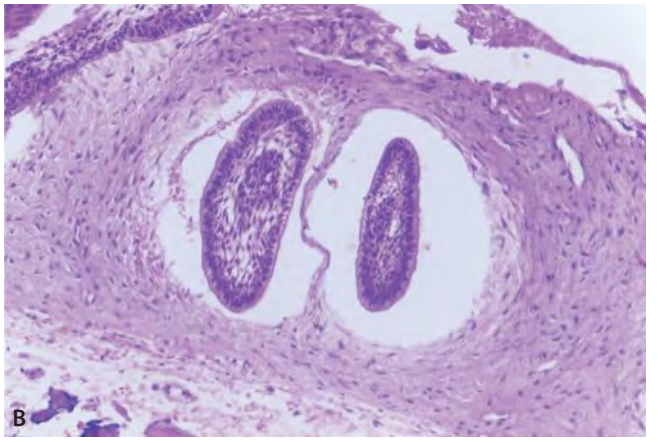
Treatment and Prognosis

Odontomas are treated with simple enucleation and curettage and are not known to recur.

Odontogenic Myxoma

The odontogenic myxoma is an uncommon benign neoplasm of the jaws that is thought to be derived from ectomesenchyme and histologically resembles the dental papilla of the developing tooth. These tumors are slow growing with a potential for aggressive behavior and a high recurrence rate after subtherapeutic removal.[93] They occur over a wide age range but seem to occur most commonly in the third decade of life. Although the tumor can occur anywhere in the jaws, the posterior mandible is most common location (Figure 29-39). Histologically, the tumor is composed of haphazardly arranged stellate, spindle-shaped, and round cells in an abundant loose myxoid stroma that contains only a few collagen fibrils (Figure 29-40). Radiographically, the odontogenic myxoma appears as a unilocular or multilocular radiolucency that may displace or cause root resorption of teeth in the area





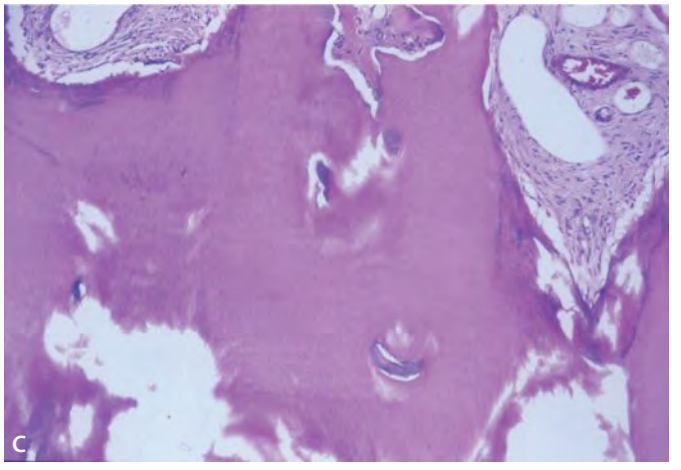


FIGURE 29-36. **A,** Enucleation and curettage is performed of the lesion in Figure 29-35. The permanent tooth is removed with the lesion. **B** and **C,** The histopathology shows ameloblastic fibro-odontoma (H&E, original magnification ×20).





FIGURE 29-37. **A,** An expansile lesion of the right maxilla. **B,** Multiple small toothlike calcified structures are removed that represent compound odontoma.

of the tumor. Although not pathognomonic of the odontogenic myxoma, the radiolucent defect may contain thin wispy trabeculae of residual bone, which are often arranged at right angles to one another in a "stepladder" pattern (see Figure 29-39B). In some patients, the tumor may have a greater tendency to form collagen fibers; such lesions are designated *fibromyxomas.*

Pathogenetically, the proliferation and aggressive behavior of the odontogenic myxoma may be related to overexpression of antiapoptotic cytokines *BCL2* and *BCLX*.[94]



FIGURE 29-38. A complex odontoma of the left posterior mandible.

Treatment and Prognosis

Odontogenic myxomas should be treated with resection with 1.0-cm bony linear margins as confirmed with a specimen radiograph (Figure 29-41). These tumors are not encapsulated and tend to infiltrate the surrounding bone such that complete removal by curettage is nearly impossible. Resection of the tumor with a normal surrounding margin of bone and soft tissue that shows negative margins should be curative.[56]





FIGURE 29-39. A large soft tissue mass of the left posterior mandibular gingiva **(A)** associated with an underlying radiolucent lesion of the mandible **(B).**

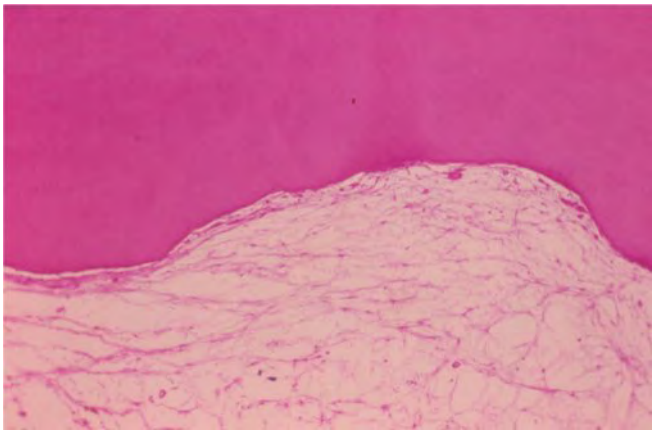


FIGURE 29-40. The biopsy of the tumor in Figure 29-39 that shows odontogenic myxoma with a loose myxoid stroma, in this case, eroding into the cementum of a tooth root (H&E, original magnification ×40).



FIGURE 29-42. A 40-year-old woman with a 5-year history of an expansile mass of the left maxilla.

Calcifying Epithelial Odontogenic Tumor

The calcifying epithelial odontogenic tumor, also known as the *Pindborg tumor*, is an uncommon lesion that accounts for less than 1% of all odontogenic tumors. It is particularly noteworthy that the three studies depicted in Table 29-4 reported only 15 cases of this odontogenic tumor among a collective series of 1440 odontogenic tumors. Fewer than 200 cases have been reported in the international literature. Although this tumor has been reported over a wide age range, it is most often encountered in patients between 30 and 50 years of age.[95] Approximately two thirds of these neoplasms occur in the mandible, although they certainly may be observed in the maxilla[96] (Figure 29-42). A painless slow-growing mass is the most common presenting sign. Radiographically, the most common presentation is a mixed radiopaque/radiolucent lesion, frequently associated with an impacted tooth (Figure 29-43).

Histologically, the Pindborg tumor is quite unique. Discrete islands, strands, or sheets of polyhedral epithelial cells in a fibrous stroma are noted. Large areas of amorphous eosinophilic hyalinized (amyloid-like) material are also present. Calcifications, which are a distinctive feature of the tumor, develop within the amyloid-like material and form concentric rings, known as *Liesegang's rings* (Figure 29-44). The precise nature of the amyloid-like material is unknown. The material does stain as amyloid when stained with Congo red or thioflavine T. After Congo red staining, the amyloid exhibits apple-green birefringence when viewed with polarized light. It has been illustrated that the amyloid-like material may actually represent amelogenins or other enamel proteins secreted by the tumor cells.[97]

Treatment and Prognosis

Although slow growing, the Pindborg tumor is highly infiltrative and destructive and is capable of aggressive behavior.[96,97]

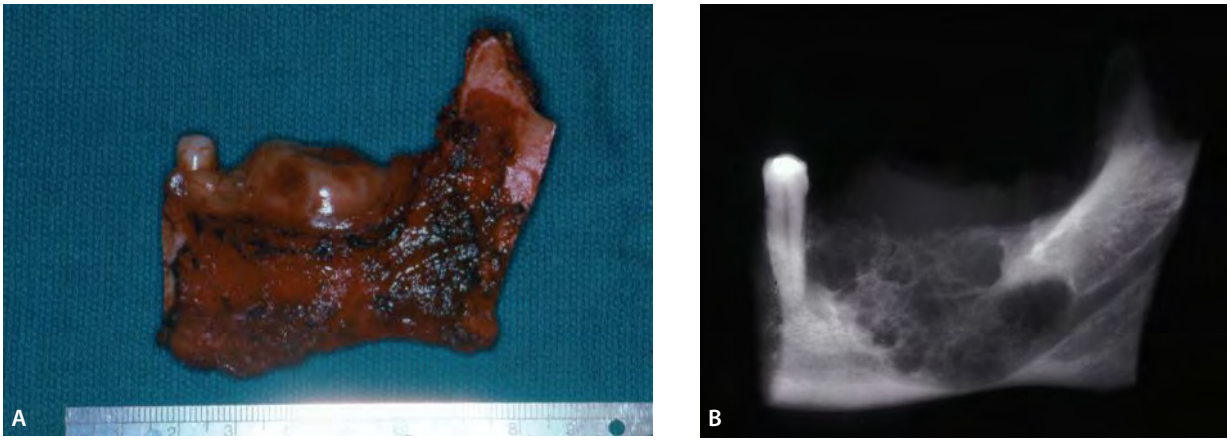


FIGURE 29-41. **A,** The patient in Figure 29-39 underwent a segmental resection of his odontogenic myxoma. **B,** As with the ameloblastoma, specimen radiographs should be obtained when resecting an odontogenic myxoma to verify the bony linear margin. A better depiction of the "stepladder" pattern of the odontogenic myxoma is noted on this specimen radiograph.



FIGURE 29-43. The panoramic radiograph **(A)** and the coronal CT scan **(B)** show a mixed radiolucent/radiopaque lesion of the left posterior maxilla.

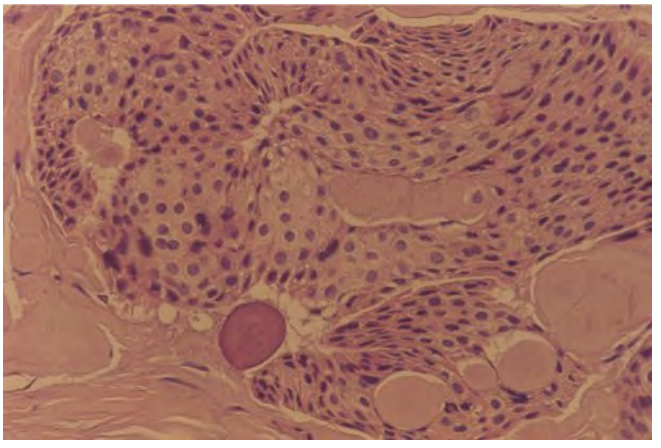


FIGURE 29-44. Incisional biopsy of the patient in Figure 29-42 shows signs indicative of the Pindborg tumor including discrete islands of odontogenic epithelium, calcification (Liesegang's rings), and hyalinized material suggestive of amyloid (H&E, original magnification ×40).



FIGURE 29-45. The patient with the Pindborg tumor in Figure 29-42 is treated with hemimaxillectomy.

Owing to the small number of reported cases and lack of consistent follow-up, evidence-based recommendations for treatment are not available. Nonetheless, the tumor is generally recommended to be treated identically to the ameloblastoma and odontogenic myxoma, with 1.0 cm bony linear margins and the appropriate attention to soft tissue anatomic barriers (Figure 29-45). When this treatment was undertaken for Franklin and Pindborg's series of tumors,[95] only one patient undergoing resection experienced recurrence.

Adenomatoid Odontogenic Tumor

The adenomatoid odontogenic tumor, regarded by many as a hamartoma, is an uncommon odontogenic lesion, accounting for 3% to 7% of all odontogenic tumors. This lesion was once believed to be a variant of ameloblastoma and was previously designated *adenoameloblastoma*.[98,99] Its clinical features and biologic behavior permit distinction from the ameloblastoma (Figure 29-46). These lesions are limited to young patients, and two thirds of all cases are diagnosed in the second decade.[100–103] The tumor is extremely uncommon in patients older than 30 years. It has a predilection for the anterior region of the jaws and is found twice as often in the maxilla as in the mandible. Females are affected about twice as often as males. Most adenomatoid odontogenic tumors are small, rarely exceeding 3 cm in diameter. In about 75% of cases, the lesion appears as a well-circumscribed unilocular radiolucency that involves the crown of an erupted tooth, frequently a canine.

Histologically, the adenomatoid odontogenic tumor is a well-defined lesion that is usually surrounded by a thick fibrous capsule (Figure 29-47). When the lesion is bisected, the central portion of the tumor may be essentially solid or may show varying degrees of cystic change with intraluminal proliferation of tissue. The lesion is composed of spindle-shaped epithelial cells that form sheets, strands, or whorled masses of cells in a scant fibrous stroma. The epithelial cells may form rosette-like structures about a central space that may be empty or contain small amounts of eosinophilic



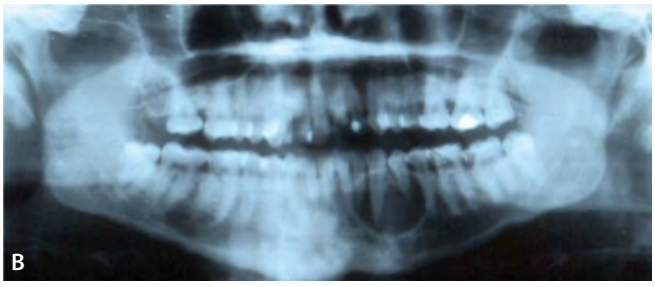


FIGURE 29-46. An expansile lesion of the lingual aspect of the left mandible **(A)** associated with a unilocular radiolucency of the left mandible **(B).**

material that may stain for amyloid.[104–106] Tubular or ductlike structures are characteristic for the adenomatoid odontogenic tumor (see Figure 29-48). These consist of a central space surrounded by a layer of columnar or cuboidal epithelial cells whose nuclei exhibit reverse polarization.

Treatment and Prognosis

Owing to this lesion being encapsulated, it separates easily from the surrounding bone. As such, an enucleation and curettage surgery is curative (Figure 29-49). Of the 499 cases of adenomatoid odontogenic tumor reported in the literature, only 1 acceptable case of recurrence has been documented.[107]

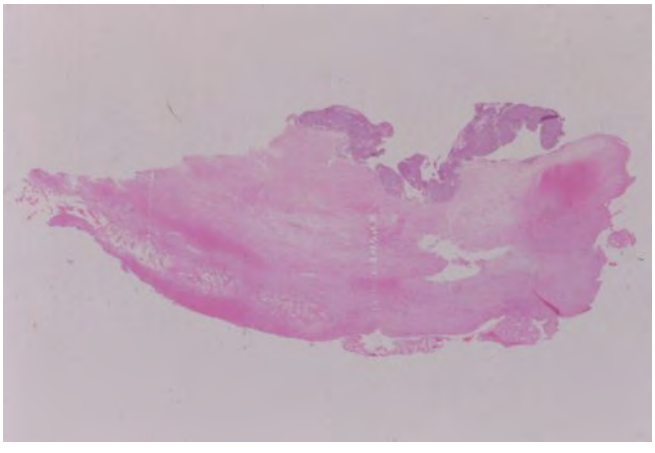


FIGURE 29-47. Incisional biopsy of the lesion in Figure 29-46 shows a well-encapsulated lesion with a thick fibrous wall (H&E, original magnification ×10).

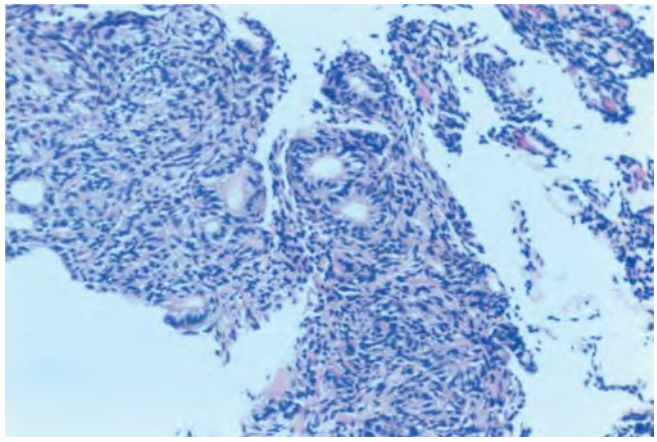


FIGURE 29-48. Ductlike structures and rosettes were also present in the biopsy of the patient in Figure 29-46 (H&E, original magnification ×40). These histologic findings are indicative of the adenomatoid odontogenic tumor.



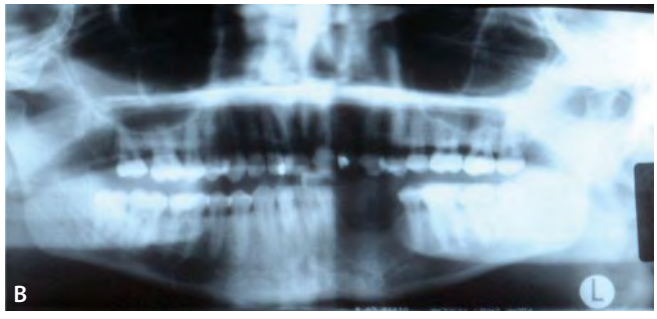


FIGURE 29-49. **A,** An enucleation and curettage surgery is performed for the patient in Figure 29-46, along with removal of the involved teeth. Erosion of the cementum of the premolar tooth is noted. **B,** The 5-year postoperative radiograph shows acceptable bony healing.

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30

CHAPTER

Benign Nonodontogenic Lesions of the Jaws

M. Anthony Pogrel, DDS, MD

Benign nonodontogenic lesions of the jaws represent a mixed group of tumors, which in many cases are difficult to classify. In addition, there are some lesions within this group that seem to occur only in the jaws, and therefore, although they do not contain any histologic or immunohistochemical evidence of odontogenic structures, the mere fact that they occur only in the jaws may mean that they are odontogenic.

The subjects discussed in this chapter are fibro-osseous disease, osteoblastoma and osteoid osteoma, aggressive mesenchymal tumors of childhood, benign tumors of bone-forming cells, synovial chondromatosis and osteochondroma, lesions containing giant cells, vascular malformations, Langerhans' cell histiocytosis, nonodontogenic cysts of the jaws, neurogenic tumors, Paget's disease, massive osteolysis (Gorham's disease), and tori.

BENIGN FIBRO-OSSEOUS DISEASE

Differences remain in the classification and diagnosis of fibro-osseous disease.[1] The general consensus is that the common entity for all of the lesions is the replacement of normal bone with a tissue composed of collagen fibers and fibroblasts that contain varying amounts of mineralized substance, which can be either bone or cementum-like material. It is difficult to differentiate conclusively between bone and cementum with light microsurgery.

For the purposes of this chapter, the term *fibro-osseous disease* is taken to include the following groups of lesions: fibrous dysplasia, cemento-osseous dysplasia, and fibro-osseous neoplasms.

Fibrous Dysplasia

Fibrous dysplasia is considered to be a developmental hamartomatous fibro-osseous disease of unknown etiology. It may represent developmental arrest in a benign fibro-osseous proliferation that lacks the ability to fully differentiate.[2] A mutation occurring in the gene coding for the alpha subunit of the stimulatory G protein GS in the guanine nucleotide binding alpha stimulating (GNAS) has been proposed to cause monostotic and polyostotic conditions and McCune-Albright syndrome.[3,4] The mutation results in defective osteoblastic differentiation.

Fibrous dysplasia is normally subdivided into four different forms:

1. Monostotic fibrous dysplasia affecting only one bone.
2. Polyostotic fibrous dysplasia affecting multiple bones.
3. McCune-Albright syndrome in which multiple lesions are associated with hyperpigmentation and endocrine disturbances, predominantly precocious puberty and/or hyperthyroidism.[5]
4. Craniofacial fibrous dysplasia confined to bones of the craniofacial complex.

The jaws are commonly associated with all forms of fibrous dysplasia. In the jaws, the onset is usually during the first and second decades, and it produces painless swelling of the involved bones (Figure 30-1). Classically, the radiographic appearance shows a ground-glass opacity without clearly defined borders (Figure 30-2). In its craniofacial form, the maxilla, zygoma, sphenoid, frontal bones, nasal bones, and base of the skull can be involved. Expansion can cause compression of nerves and blood vessels. The optic canal can be narrowed by fibrous dysplasia, although it seems unlikely that any associated vision loss can be relieved by orbital decompression.[6] The maxilla appears to be affected more often than the mandible, and females are affected more commonly than males. Typically, lesions undergo periods of activity and periods of quiescence. When they are active, they are often symptomatic



FIGURE 30-1. Swelling of the left mandible and maxilla owing to fibrous dysplasia.

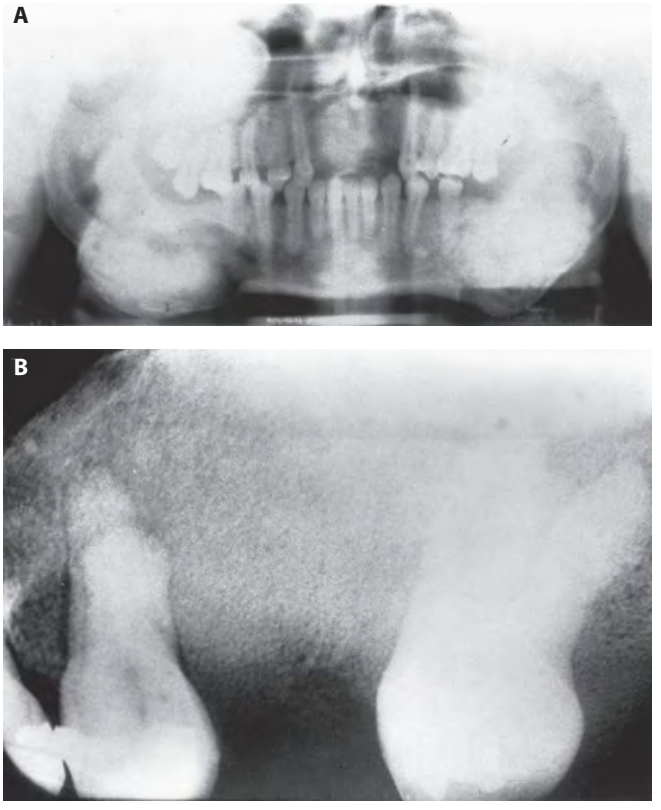


FIGURE 30-2. **A,** Radiographic appearance of patient in Figure 30-1 shows ground-glass appearance of lesions. **B,** Periapical view of typical ground-glass appearance of fibrous dysplasia.

in that the patient may perceive a throbbing or discomfort, the swelling increases, and the lesions appear hot on a bone scan (Figure 30-3) and can, in fact, mimic osteomyelitis.[7–11] In a quiescent phase, they may be totally asymptomatic. Teeth can

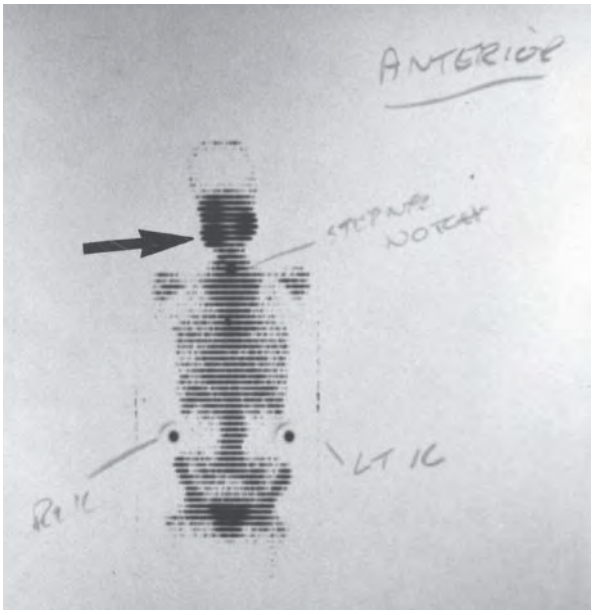


FIGURE 30-3. Bone scan of patient in Figures 30-1 and 30-2 shows area of increased uptake of isotope in both sides of the mandible and the left maxilla *(arrow)*. The isotope used was Tc-99m diphosphonate.

be displaced by the lesion (Figure 30-4). Familial cases of fibrous dysplasia have been noted.[12]

The lesions of fibrous dysplasia may be under hormonal control, particularly in McCune-Albright syndrome, and cases of increased activity and reactivation during pregnancy have been noted.[13,14] Although not normally recognized as a premalignant lesion, sarcomatous change has been noted in fibrous dysplasia.[15] Early cases appear to have been associated with the use of radiation therapy for treatment,[16,17] but cases of spontaneous sarcomatous degeneration have been noted.[18] In addition, some cases have been difficult to diagnose and may have represented a low-grade osteosarcoma from the outset.[19–21]

Classically, fibrous dysplasia appears to be a lesion that "burns itself out" when the patient is in the late teens or early



FIGURE 30-4. Teeth displaced by lesions of fibrous dysplasia.

20s, although cases of active fibrous dysplasia have been noted much later than this.

Treatment is generally symptomatic; if the lesions are asymptomatic, a biopsy diagnosis alone may be adequate without carrying out any definitive treatment. Medical treatment with bisphosphonates is often used in an attempt to slow bone turnover.[22] Surgical treatment should be limited during an active phase because the lesions are vascular and can bleed quite profusely. Treatment is best reserved for quiescent periods, at which time. cosmetic recontouring is the normal treatment of choice. Regrowth, however, can be expected after this treatment in 25% to 50% of cases, particularly if undertaken at a young age. Some investigators have suggested more aggressive surgical procedures including mandibular and maxillary resections.[23,24]

Cemento-osseous Dysplasia

The cemento-osseous dysplasias represent a pathologic process of the tooth-bearing areas and probably represent the most common manifestation of fibro-osseous disease. However, because they are frequently asymptomatic and require no treatment, they are less of a diagnostic and clinical dilemma than are the other forms of fibro-osseous disease. In this condition, there is a disordered production of bone and cementum-like tissue in the jaws. The three forms include periapical, focal, and florid osseous dysplasias, and familial gigantiform cementoma, which are probably variants of the same pathologic process but which can be differentiated by clinical and radiographic features. The etiology of these lesions remains in doubt, but local trauma may play some part, even such benign trauma as abnormal occlusal forces. A predominance of cases occur in females and also in African Americans.[24] It is suspected that the periodontal ligament may be the origin of the fibrous tissue found in the cemento-osseous dysplasias. Histologically, the three types of cemento-osseous dysplasia are indistinguishable, showing new woven bone trabeculae and/or spherules of cementum-like material, which often blend into the cortical bone. A fibrous tissue stroma is present. There is very little inflammatory component. Traumatic bone cysts have been reported in conjunction with this lesion.[25]

Periapical Cemento-osseous Dysplasia

Periapical cemento-osseous dysplasia presents as circumscribed lesions in periapical areas associated with vital teeth, with the anterior mandible being most usually involved. African American females are predominantly affected. Radiographically, the lesions can be radiolucent, of mixed density, or radiopaque, depending on their stage of development (Figure 30-5). Studies indicate that they may occur in approximately 6% of African American females.[26]

Focal Cemento-osseous Dysplasia

Lesions of focal cemento-osseous dysplasia have a predilection for middle-aged African American females and present as nonexpansile radiolucencies with associated opacities, often in edentulous areas of the mandible. They frequently occur in sites of previous dental extractions and may represent some type of abnormal healing after dental extraction. Because they are usually asymptomatic, cases are often noted on routine panoramic radiographs. They are normally well circumscribed and rarely exceed 2 cm. Differentiation from ossifying fibroma may be difficult.[27]

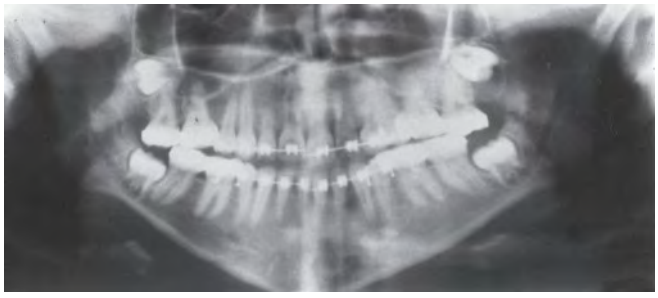


FIGURE 30-5. Periapical cemento-osseous dysplasia of the left maxilla (an atypical site). All associated teeth are vital. The patient is a 48-year-old African American female.

Florid Cemento-osseous Dysplasia

Florid cemento-osseous dysplasia has a predilection for middle-aged African American females and presents as a painless nonexpansile lesion often involving two or more jaw quadrants. Radiographically, it appears as multiple confluent lobular radiopaque masses in tooth-bearing areas (Figure 30-6). Lesions may be associated with superimposed infection and osteomyelitis and have also been associated with idiopathic bone cysts.[28] Histologically, they have an unencapsulated proliferation of cellular fibrous tissue with trabeculae or woven bone and calcification. More mature lesions may become acellular and avascular with coalescent sclerotic bone masses. Although common in African Americans, florid cemento-osseous dysplasia has been noted in all racial groups. Many



FIGURE 30-6. Florid cemento-osseous dysplasia of the mandible in a 49-year-old African American female. All associated teeth are vital.

patients are partially or totally edentulous when the condition is first discovered. Cortical expansion is usually absent or of limited degree. It has been suggested that chronic diffuse sclerosing osteomyelitis may represent a variant of this condition, but it probably represents a different condition, inflammatory in nature. The differences between the two conditions have been noted and described.[8,29,30] However, the role of bacteria in chronic diffuse sclerosing osteomyelitis has proved elusive, and in general, even authorities who strongly support an infectious origin have had difficulty isolating organisms.[31,32]

Familial Gigantiform Cementoma

Familial gigantiform cementoma represents an autosomal dominant variant of osseous dysplasia usually involving multiple quadrants with variably expansile lesions, often in the anterior mandible.[33] This particular form of osseous dysplasia has no racial predilection. The lesions often evolve during childhood and can grow rapidly. Treatment is usually surgical and symptomatic and is limited to cosmetic recontouring.

Fibro-osseous Neoplasms

Ossifying Fibroma

Ossifying fibroma (cemento-ossifying fibroma) usually presents as a well-demarcated mixed radiolucency/radiopacity with smooth and often sclerotic borders (Figure 30-7). The lesions are usually solitary and most commonly occur in the mandible. Histologically, they contain a relatively avascular cellular fibrous stroma with reticular bone trabeculae and cementum-like spherules. Most authorities now feel comfortable clearly differentiating this lesion from fibrous dysplasia. Chromosomal abnormalities have been identified in an ossifying fibroma and a cementifying fibroma.[34,35] The ossifying fibroma is believed to be a true neoplasm and occurs at a later age than does fibrous dysplasia, being most common later in the third and early in the fourth decades. Ossifying fibroma appears to be confined to the jaws and craniofacial complex, although similar lesions have been reported in the long bones.[36–38] There is a female predominance but no racial predominance, and growth rates are variable. Because it is believed to be a neoplasm, the treatment is surgical; in fact, the lesions often shell out easily at surgery, although there is a recurrence rate that has variously been reported from 1% to 63%.[39–41] For these reasons, some authorities recommend aggressive treatment for more aggressive lesions, including aggressive curettage, localized surgical resection, and segmental mandibular resection.[42,43] When present in the craniofacial complex, treatment may have to be more aggressive to protect the vital structures.[44]

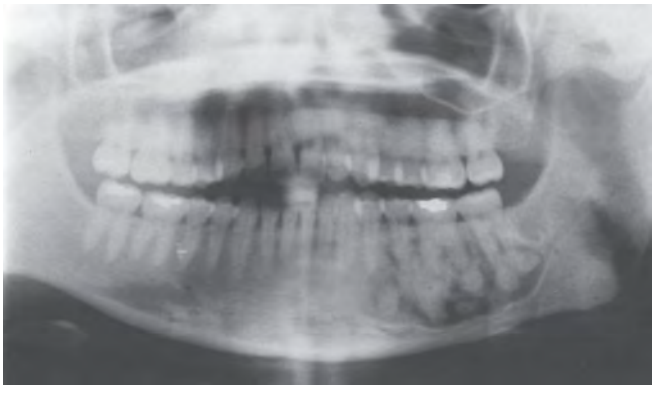


FIGURE 30-7. Ossifying fibroma of the left mandible. It appears as a well-defined mixed radiolucency/radiopacity.

Juvenile Aggressive Ossifying Fibroma

Juvenile aggressive ossifying fibroma was first described in 1952 as a variant of ossifying fibroma.[45] The lesions classically occur in younger children and adolescents and present with an aggressive behavior, but they have been noted in older patients and are not always particularly aggressive. The World Health Organization defines *juvenile aggressive ossifying fibroma* as "an actively growing lesion mainly affecting individuals below the age of 15 years, which is composed of a cell-rich fibrous tissue containing bands of cellular osteoid without osteoblastic rimming together with trabeculae of more typical woven bone. Small foci of giant cells may be present, and in some parts there may be abundant osteoclasts related to the woven bone. Usually no fibrous capsule can be demonstrated, but the lesion is well demarcated from the surrounding bone."[46] Two variants have been described: *trabecular* and *psammomatous*. The trabecular variant usually occurs in childhood, with a slight maxillary predominance, and may contain clustered multinuclear giant cells. The psammomatous variant can occur in adults as well as adolescents and often affects the orbit and paranasal tissues; frequently, it contains a whorled pattern of closely packed spherical ossicles and a myxoid component with aneurysmal bone cyst–like areas.

Although believed to be more aggressive than the more common ossifying fibroma that is found at a later age, this condition is not considered to necessitate truly aggressive surgery. Conservative excision is still the recommended treatment, although lesions involving the craniofacial structures may require more extensive surgery. Recurrence rates of between 20% and 50% have been reported, and recurrences may be more common in younger patients.[1]

OSTEOBLASTOMA AND OSTEOID OSTEOMA

Osteoblastoma and osteoid osteoma are generally believed to be variants of the same lesion and are related to fibro-osseous disease. Cementoblastoma and gigantiform cementoma are the equivalent cemental lesions and are associated with teeth. The alternative name for the osteoblastoma is *giant osteoid osteoma*, and it is generally believed to represent a larger version of the osteoid osteoma. Both are benign



FIGURE 30-8. Osteoblastoma of the left mandible in a 24-year-old female. Note mixed radiolucency/radiopacity with a radiolucent rim.

processes and are thought to represent true neoplasms. The osteoblastoma occurs primarily in the vertebrae and long bones, but it has been described in the jaws.[47–49] Clinically, it often grows rapidly and the predominant clinical feature is pain, which is generally localized to the lesion itself. Although believed to be a true neoplasm, there have been reports of regression after biopsy or incomplete removal, which could point to it being a reactive process of some kind.[50] Most cases of osteoblastoma occur in the second decade of life; they rarely occur after age 30 years. Males appear to be affected more commonly than females. In the head and neck, the mandible is the most common site.

Radiographic features are variable, usually consisting of a combination of radiolucency and radiopacity (Figure 30-8). The designation *osteoblastoma* is normally reserved for lesions greater than 2 cm in diameter. They are well circumscribed radiographically with a thin radiolucency surrounding the variably calcified contents. A sunray pattern of new bone formation similar to that described in malignant bone tumors may be evident.

The histologic appearance shows irregular trabeculae of osteoid and immature bone within a predominantly vascular stromal network. There are various degrees of calcification present. Stromal cells are generally small and slender. Differentiation must be made from the ossifying fibroma, fibrous dysplasia, and osteosarcoma.

Treatment of the osteoblastoma is generally confined to conservative surgical excision with either curettage or local excision. Recurrences are rare but have been reported and may necessitate more aggressive treatment such as en bloc resection.[51] Rare examples of malignant transformation have been reported,[52,53] but some of these may be related to an incorrect initial diagnosis.[49]

The osteoid osteoma represents a smaller version of the osteoblastoma and is thought to be a true neoplasm. It is normally less than 2 cm in diameter clinically and radiographically. It again occurs in the second and third decades of life with a male predominance. Pain is again the major clinical feature. Classically, the pain is worse at night and is relieved by acetylsalicylic acid. If the lesion is located near the cortex, it may produce a localized tender swelling. Radiographically, the lesion again shows a well-defined mixed radiolucency/radiopacity with a small radiolucent rim around the lesion, which is walled by sclerotic bone. Histologically, it resembles the osteoblastoma with a rich vascular stroma with trabeculae of osteoid and immature bone. The bone is rimmed by layers of active osteoblasts. Histologically, it is impossible to differentiate it from the osteoblastoma. Treatment is again conservative surgical excision. Spontaneous regression has also been reported clinically.

CHONDROMA

A chondroma is a benign tumor of mature cartilage. The occurrence of these lesions in the jaws is extremely rare[54]; in fact, whether they ever occur in the jaws or whether they are usually described as chondromyxomas or chondromyxoid fibromas has been questioned.[55–59] In many cases, the true diagnosis in those reported cases is actually low-grade chondrosarcoma.[60] Most reports concern the mandibular condyle, suggesting that these lesions may arise from cartilaginous remnants.[61,62] The chondroma presents as a painless slowly progressive swelling, which may result in mucosal ulceration. The gender distribution is equal, and most tumors occur in patients younger than age 50 years. Radiographically, they present as irregular radiolucent lesions, although foci of calcification may occasionally be present. Resorption of tooth roots has been reported. Histologically, the lesions contain well-defined lobules of mature hyaline cartilage. Treatment is localized, and conservative surgical excision is normally recommended. Because of the doubtful nature of these lesions and the always-present possibility of a lesion representing a low-grade chondrosarcoma, some authorities have suggested wide excision for all of these lesions as a safety precaution.[60]

OSTEOMA

Osteomas are benign tumors consisting of mature compact or cancellous bone. They may arise on the surface of bone (periosteal osteomas) or centrally within the bone (endosteal osteomas).[63] They are often discovered as asymptomatic radiopacities.

Osteomas are most commonly discovered during the second and fifth decades of life, although they have been noted in all age groups. Males appear to be affected more frequently than females.

Gardner's syndrome is an autosomal dominant condition in which patients have intestinal polyposis, multiple osteomas (usually endosteal) of the jaws, fibromas of the skin, epidermal cysts, impacted teeth, and odontomas.[64–67] The specific gene mutation associated with the condition has now been



FIGURE 30-9. Radiograph of an endosteal osteoma in the ascending ramus of the left mandible.

identified as the adenomatous polyposis coli (APC) gene on the long arm of chromosome 5 (5q21-22).[68–70] Many cases of incomplete manifestation of the syndrome have been reported. The clinical significance of this syndrome is that the intestinal polyps, which frequently occur in the colon and rectum, are premalignant and have a very high rate of malignant transformation. The associated osteomas are often found in the jaws, particularly in the angle region of the mandible, as well as the facial bones and long bones. It has been suggested that any patient with multiple mandibular osteomas should be investigated for the possibility of Gardner's syndrome. Investigation should include a detailed history of gastrointestinal disturbance and, if positive, follow-up with colonoscopy; if the diagnosis is confirmed, a prophylactic colectomy is generally recommended. Periosteal osteomas usually present as asymptomatic slow-growing bony masses. Endosteal osteomas are usually asymptomatic and are noted on routine radiographs. Radiographically, they appear as well-circumscribed sclerotic radiopaque masses (Figure 30-9). Histologically, they consist of either dense compact bone with sparse marrow spaces or lamellar trabeculae of cancellous bone with fibrofatty marrow spaces. Osteoblastic activity is often predominant.



FIGURE 30-10. Surgical specimen from the osteoma shown radiographically in Figure 30-9.

Treatment of osteomas is surgical excision (Figure 30-10). This is often necessary to establish the diagnosis. Asymptomatic cases may be followed up clinically and radiographically without treatment. After excision, recurrences are very rare.

SYNOVIAL CHONDROMATOSIS AND OSTEOCHONDROMA

Both synovial chondromatosis and osteochondroma are conditions that occur in the temporomandibular joints and may be considered variants of the chondroma and osteoma. In synovial chondromatosis, there is a proliferation of small particulate, generally unattached, chondromas within the confines of the joint capsule. Although most frequently found in the knee, they have been reported in most joints. Well-recognized cases have occurred in the temporomandibular joints (normally only the superior compartment[71]) with symptoms normally consisting of pain and swelling but most often with deviation of the mandible toward the unaffected side (Figure 30-11).[72,73] The etiology is unknown, but trauma has been suggested.[74] When these lesions become symptomatic, they should be removed via a standard preauricular approach, though an endoscopic approach has also been described.[74] Perforation into the middle cranial fossa has also been described.[75] Because it is believed that they arise from metaplasia within the synovial lining cells of the joint, it is often advocated that the lining be removed at the same time.[76] Cases have been reported in which up to 200 of these bodies were present within the temporomandibular joint (Figure 30-12).[71] After removal, recurrence has not been reported.

The osteochondroma is believed to be a benign lesion that arises predominantly in long bones from a herniation of cartilage through the epiphyseal plate. It tends to present with a predominantly osseous core with a cartilaginous cap. The lesion becomes symptomatic when function is affected, for example, a malocclusion or mandibular asymmetry develops (Figure 30-13). Cases have been reported in the mandibular condyle.[77] Cases in the temporomandibular joints appear identical in all respects to lesions in other bones of the body. However, the association with the epiphyseal plate that occurs

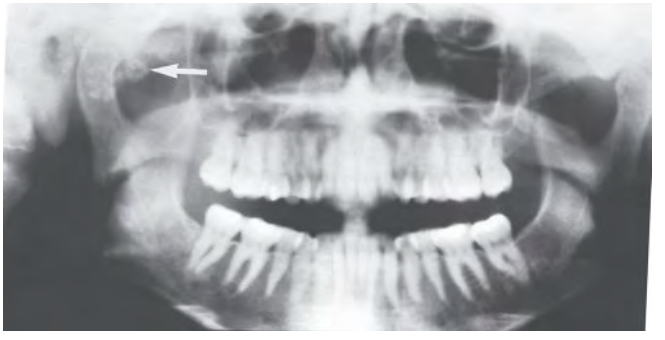


FIGURE 30-11. Panoramic radiograph shows a number of radiopaque foreign bodies in the right temporomandibular joint *(arrow)*.

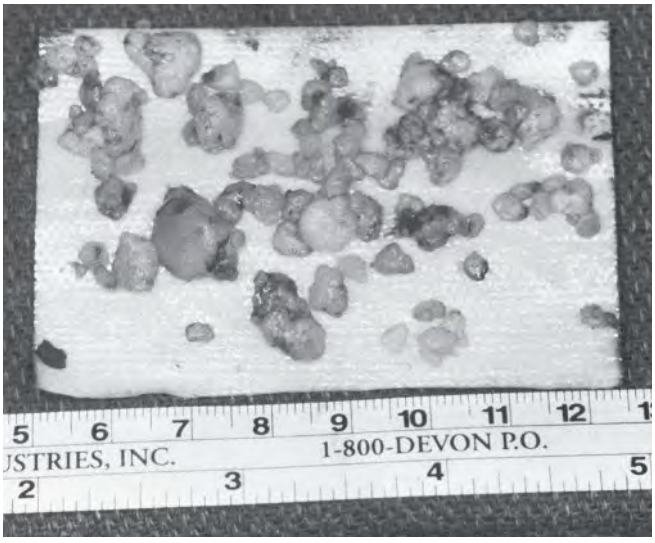


FIGURE 30-12. The synovial chondromas removed from the jaw of the patient in Figure 30-11.



FIGURE 30-13. A malocclusion caused by an osteochondroma of the left temporomandibular joint.

in the long bones is not present in the temporomandibular joint. On magnetic resonance imaging, it appears as an extraneous appendage to the temporomandibular joint and is usually more brighter than the surrounding mandible (Figure 30-14). Treatment is symptomatic; when symptoms occur, localized excision is recommended via the normal temporomandibular approach. Recurrence has been reported but is unusual.[78–80]

AGGRESSIVE MESENCHYMAL TUMORS OF CHILDHOOD

It is recognized that children and young adults can develop an aggressive and rapidly growing tumor of bone, which, although often having a benign mesenchymal appearance, nevertheless behaves very aggressively. The exact nature of these lesions remains unknown, but many have been classified

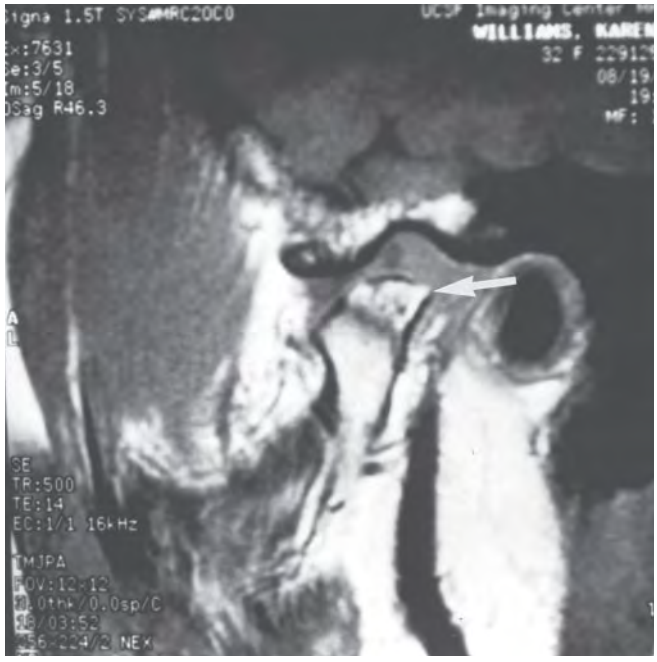


FIGURE 30-14. Magnetic resonance image of an osteochondroma of the left temporomandibular joint *(arrow)* in the patient shown in Figure 30-13.

as desmoplastic fibromas, which is the hard tissue equivalent of fibromatosis in the soft tissues. Any bone can be affected including the jaws. They are considered to be a type of intraosseous fibroma.[81]

The etiology and pathogenesis are in doubt because their aggressive behavior suggests a neoplastic process, but genetic, endocrine, and traumatic factors have also been suggested. Most occur in persons younger than age 20 years, and there is no gender predilection. The mandible is affected more frequently than the maxilla.[82] Radiographically, a unilocular or multilocular radiolucency is noted with poorly defined margins, cortical perforation, and root resorption often being present (Figures 30-15 and 30-16). Histologically, the lesion consists of interlacing bundles in a whirled



FIGURE 30-15. A desmoplastic fibroma presents as a well-defined radiolucency at the lower border of the body of the left mandible in a 3-year-old patient.



FIGURE 30-16. A desmoplastic fibroma presents as an ill-defined radiolucency of the left mandible causing displacement of teeth in a patient aged 8 years.

aggregate of collagenous tissue with elongated and spindle fibroblasts. Hypocellularity is often present. However, atypia and mitotic features are not found. Osteoid material is not produced by this lesion.

In treating this lesion, the adage "treat the biology, not the histology" is of paramount importance. Although the lesion appears benign histologically, it often behaves aggressively,[83] and the appropriate treatment is aggressive surgery, which often involves mandibular or maxillary resection (Figure 30-17). This is psychologically difficult for the surgeon to perform in a young child without a histologic diagnosis of malignancy, but the recurrence rate is very high after more conservative procedures. For lesions in inaccessible areas such as the base of the skull, radiation therapy and/or chemotherapy has been attempted with variable degrees of success.[84,85]

LESIONS CONTAINING GIANT CELLS

A number of lesions tha occur in the jaws that contain giant cells within them. Their relationship to each other, however, is ill defined. Histologically, all of the giant cell lesions appear similar, if not identical, and they usually cannot be distinguished on light microscopy alone. The clinical history, immunohistochemistry, or genetic markers have to be used to differentiate the lesions.

Central Giant Cell Granuloma

Central giant cell granuloma is a lesion occurring almost exclusively in the jaws. (A similar lesion has been described in the small bones of the fingers and toes, but its relationship to the central giant cell granuloma is unknown.[86]) Although not normally considered an odontogenic lesion, the fact that it occurs only in the jawbones probably indicates some relationship to the teeth or tooth-bearing structures. It occurs primarily in the anterior parts of the jaws in people in the second and third decades of life, but it has been recorded in all sites at all ages. Its histogenesis remains speculative. When first described it was called a "reparative giant cell granuloma,"[87–89] and it was considered a reparative lesion that was essentially self-healing. There was little evidence of this, however, and only oblique references to its self-healing properties can be found. Worth[90] showed in a study of a number of nontreated lesions that resolution often did occur as seen radiographically; even when the lesions did not resolve completely radiographically, only a fibrous scar was noted on surgical exploration. The current consensus, however, is that these are not reparative lesions and that if they are not treated, they are progressive. Most appear to follow a fairly benign course, but more aggressive lesions have been noted.[91–93] The true nature of the central giant cell granuloma remains speculative. It has been suggested that it may be an inflammatory lesion, a reactive lesion, a true tumor, or an endocrine lesion. It may behave most like a reactive lesion.

Older theories about the origin of these lesions suggested that they may be derived from the odontoclasts that were responsible for resorption of the deciduous teeth; this was said to explain why they are normally found in areas where deciduous teeth were present and are found after the deciduous teeth have resorbed.

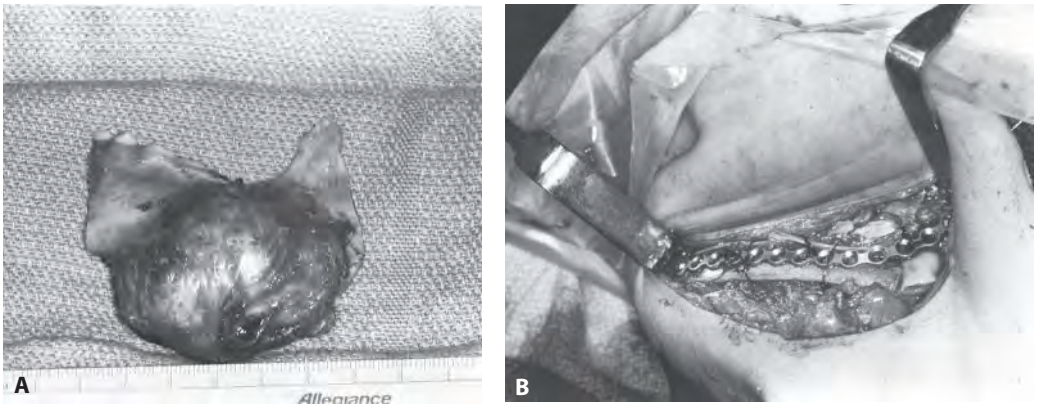


FIGURE 30-17. **A,** Resected specimen from the patient in Figure 30-15. **B,** Immediate reconstruction with ribs can often be performed in young children. Reconstruction plate and rib grafts in place.

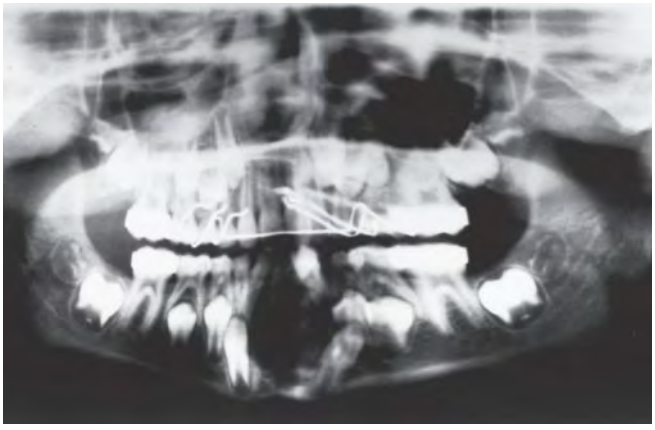


FIGURE 30-18. A central giant cell granuloma of the anterior mandible causing the displacement of teeth.

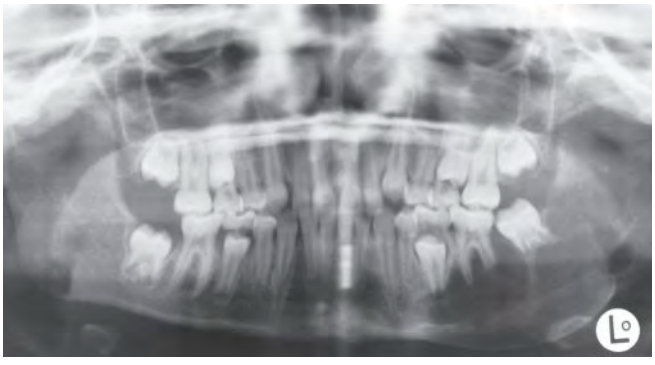


FIGURE 30-19. A central giant cell granuloma of the left angle region of the mandible, appearing as an ill-defined multilocular radiolucency, causing resorption of the distal root of the first molar (unusual).

Radiographically, the central giant cell granuloma can take a number of forms from a well-defined radiolucency, a more ill-defined radiolucency, or a multilocular radiolucency. Teeth can be displaced by the lesion, although resorption of teeth is uncommon (Figures 30-18 and 30-19).

Histologically, these granulomas contain focal arrangements of giant cells within a vascular stroma with thin-walled capillaries adjacent to the giant cells. There is a spindle cell stroma. Immunohistochemistry has shown that the giant cells are in fact osteoclasts,[94] and the spindle cells are probably the cells of origin of this lesion.[95]

Treatment is usually surgical and consists of local curettage, which is usually curative.[96] However, there is a 15% to 20% recurrence rate, and if the lesions are large, even conservative curettage may involve the loss of many teeth and possibly the inferior alveolar nerve in the mandible; it may have sinus and nasal implications in the maxilla. With the aggressive variants, more aggressive surgery has been suggested including mandibular resection and appropriate reconstruction.[97]

Because the central giant cell granuloma and the brown tumor of hyperparathyroidism cannot be separated histologically, it is advocated that hyperparathyroidism be excluded from the diagnosis by serum calcium, phosphate, and parathormone and parathormone-related protein assays in all but the single small and more benign lesions.[98]

A number of nonsurgical treatments have been suggested, all of which have their advocates. Intralesional steroids (usually triamcinolone injected into the lesion once per week for 6 wk) have been advocated and have shown some success.[99,100] Their mode of action is unknown, but they may work by suppressing the inflammatory component of the lesion. They are probably best reserved for smaller lesions that can be more easily treated by intralesional injections (Figure 30-20).

Calcitonin given by subcutaneous injection or by nasal spray has also been advocated and has met with some success (Figure 30-21).[101–105] The theory behind this treatment is that the lesion may be caused by an as-yet-undiscovered parathormone-like hormone and that the use of calcitonin antagonizes its action and allows the lesion to heal. Because some of the giant cells have been shown to have calcitonin receptors on them, this may explain calcitonin's effectiveness.[98] Treatment failures have also been reported with calcitonin.[106,107] Interferon-alfa given by subcutaneous injection has also been advocated in the treatment of the central giant cell granuloma and has again met with some success.[108] The rationale for this therapy is that the antiangiogenic action of the interferon-alfa suppresses the angiogenic component of this lesion, causing healing to occur. In most cases, surgery

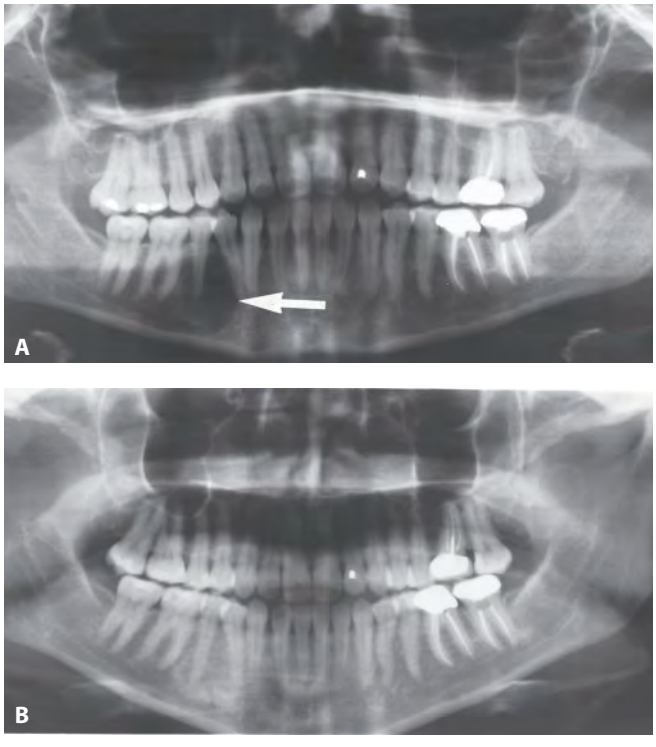


FIGURE 30-20. **A,** A central giant cell granuloma of the right mandibular bicuspid region causing displacement of the root of the first bicuspid *(arrow)*. **B,** One year after a course of six intralesional injections of triamcinolone (10 mg/mL). Note that the area is now radiopaque.

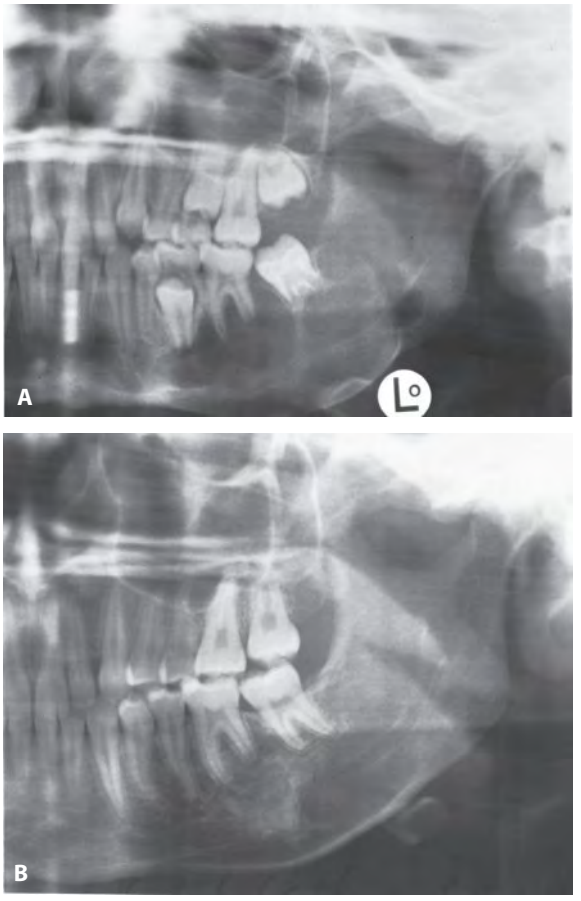


FIGURE 30-21. **A,** A central giant cell granuloma of the left posterior mandible (same case as Figure 30-19). **B,** Same case 2 years after an 18-month course of subcutaneous calcitonin injections. Note the continued development of the roots of the second molar, the cessation of resorption of the distal root of the first molar, and the radiopacity replacing radiolucency.

is still required after the interferon-alfa treatment, but it may be less radical surgery and there may be a smaller chance of recurrence. Complications and side effects of interferon can be severe, however.[109]

It has again been suggested that the central giant cell granuloma may, in fact, be a self-healing lesion, with the natural healing process stimulated by the nonsurgical therapy employed.[103]

Giant Cell Tumor

The giant cell tumor is normally found in the long bones and its presence in the jaws is not universally accepted; if it does occur, it is extremely rare. This lesion is an aggressive one and is thought by some to be a variant of a low-grade osteosarcoma. The recurrence rate after local curettage is high, and the appropriate treatment is in doubt. Some authorities advocate local curettage, whereas others have advocated resection. Histologically, it is very similar to the central giant cell granuloma, except that the giant cells are larger with more nuclei, and they are more evenly spread throughout the lesion and not as focally placed as in the central giant cell granuloma. However, in any particular case, it may be extremely difficult to make this distinction.[49]

Hyperparathyroidism

In hyperparathyroidism (primary, secondary, or tertiary), calcium is mobilized from the bones into the bloodstream to maintain homeostasis in the face of increased renal excretion. Mobilization from bone takes place focally and produces lesions in the bones (including the jaws) that are known as *brown tumors* because of their fairly distinctive coloration on surgical exploration.[110] Clinically and histologically, they are identical to the central giant cell granuloma (Figure 30-22). Therefore, whenever a lesion such as this is recurrent, aggressive, or multiple, hyperparathyroidism must be excluded by means of serum calcium, phosphate, and parathormone and parathormone-related protein assays. If these confirm a diagnosis of hyperparathyroidism, it should be treated appropriately. The lesions normally resolve without any further treatment.

Cherubism

Cherubism is a familial genetically dominant condition first described by Jones in a family in 1933.[111] Affected family mebers have multiple lesions mainly affecting the facial bones. Because of the involvement of the maxilla and orbital floor, the face has a rounded appearance and the eyes tend to look upward, giving the patient a cherubic appearance (Figure 30-23). The genetic defect in this condition has been identified on chromosome 4p16.3.[112,113]

Expression is variable, with some patients having subclinical lesions discovered only on radiographs and some having extensive and clinically obvious lesions. Spontaneous mutations also occur. Radiographically, the lesions appear honeycombed and can be very extensive. Teeth are often displaced, and in active periods, the lesions are extremely vascular (Figures 30-24 and 30-25).



FIGURE 30-22. Hyperparathyroidism shows a recurrent lesion of the left mandible with a pathologic fracture and a lesion of the right mandible.



FIGURE 30-23. A 7-year-old female with cherubism affecting the maxillofacial region.

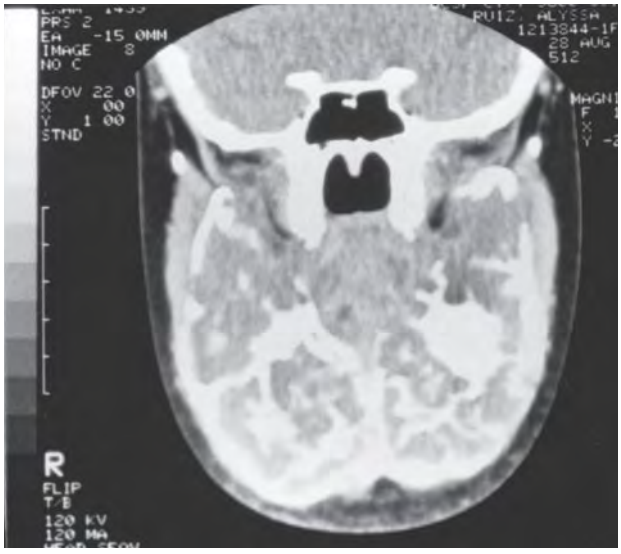


FIGURE 30-25. A coronal computed tomography (CT) scan of the patient in Figure 30-23 shows extensive involvement of the mandible and maxilla by cherubism.

Histologically, the lesions are very similar to central giant cell granuloma, with focal accumulations of giant cells in a spindle cell matrix. Perivascular cuffing is often present and, in some cases, can be used to differentiate the two lesions.

Because of its histologic similarity to central giant cell granuloma, calcitonin has been used in an attempt to cause resolution, but it has not met with variable success, suggesting that they are, in fact, different lesions.[114,115]

Treatment of cherubism is usually conservative and expectant and into the teenage years is devoted to trying to aid eruption of the teeth, which is often abnormal. Later, it is directed toward cosmetic recontouring of the affected bones. The lesions normally become less active and less vascular toward the end of the second decade and into the third decade, and it is at this time that most aesthetic recontouring is carried out.

Aneurysmal Bone Cyst

Aneurysmal bone cyst is most commonly found in the jawbones and appears to be a combination of a sinusoidal vascular lesion with a giant cell component. Radiographically, the lesion appears as a well-circumscribed soap bubble–type lesion (Figure 30-26). Histologically, the giant cell component resembles the central giant cell granuloma, whereas the vascular component is thin-walled sinusoids. Some authorities consider this to be a vascular variant of a central giant cell granuloma; others consider it a separate lesion. It responds well to aggressive curettage, although hemorrhage can be a problem. Recurrences are rare.

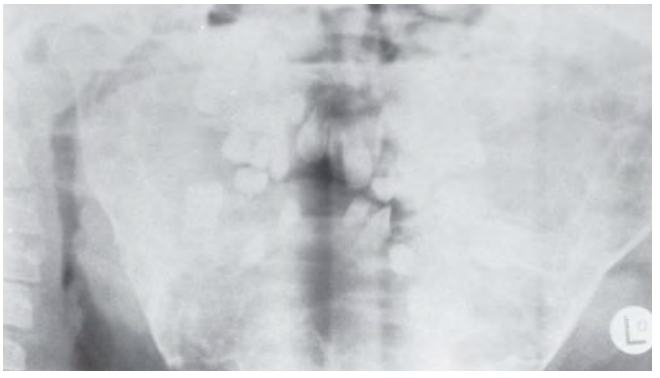


FIGURE 30-24. A panoramic radiograph of the patient shown in Figure 30-23. Note the extensive multilocular radiolucencies.

VASCULAR MALFORMATIONS

Vascular malformations can occur anywhere in the body and are thought to be developmental lesions, which can occur in soft tissue or bone. Central vascular malformations of the jaws are a rare but well-documented entity. They are in contrast to the true hemangioma, which is a neoplasm of vascular

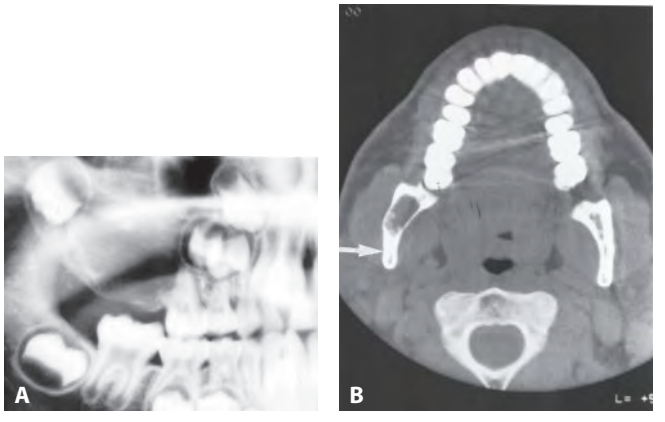


FIGURE 30-26. **A,** The "soap bubble" appearance of an aneurysmal bone cyst of the right maxilla. **B,** An axial CT scan of an aneurysmal bone cyst of the angle of the right mandible *(arrow)*.

endothelium and is normally present at birth, often enlarges, and then frequently involutes.[116] The vascular malformation generally is not present at birth, appears later, and does not involute. Vascular malformations can take a number of forms. The most practical classification is to divide them into high-flow and low-flow vascular malformations. The *high-flow vascular malformations* are either arterial lesions or arteriovenous fistulas. The *low-flow malformations* are mainly venous in nature. The clinical significance of a vascular malformation is that a central high-flow vascular malformation can cause torrential hemorrhage when surgical intervention ensues. This has been fatal on occasion.[117] Many of these lesions are asymptomatic and may even be difficult to detect preoperatively on radiographs. If there is a clinical presentation, it is often a slow-growing asymmetrical expansile lesion of the jaw, and if it is high flow, it may be associated with a bruit. Radiographically, a high-flow malformation may appear as an irregular, poorly defined soap bubble–type lesion, which may cause resorption of the roots of teeth and does not normally cause nerve involvement (Figure 30-27). Low-flow malformations are similar but are often somewhat better defined and may contain calcifications or phleboliths within them. The presence of phleboliths is diagnostic of a low-flow malformation. Diagnosis is usually confirmed by computed tomography.

To avoid the possibility of inadvertently carrying out a tooth removal or a biopsy in the presence of a high-flow malformation, a diagnostic needle aspiration should be carried out preoperatively. If bright red blood under pressure is encountered, surgery should be abandoned. Because the radiographic and clinical appearances of a vascular malformation are not diagnostic, the differential diagnosis normally includes a number of odontogenic and nonodontogenic lesions, including central giant cell granuloma, aneurysmal bone cyst, ameloblastoma, odontogenic keratocyst, and odontogenic myxoma. All of these lesions should undergo needle aspiration before biopsy or surgical treatment to rule out a high-flow vascular malformation. When a vascular malformation is suspected or

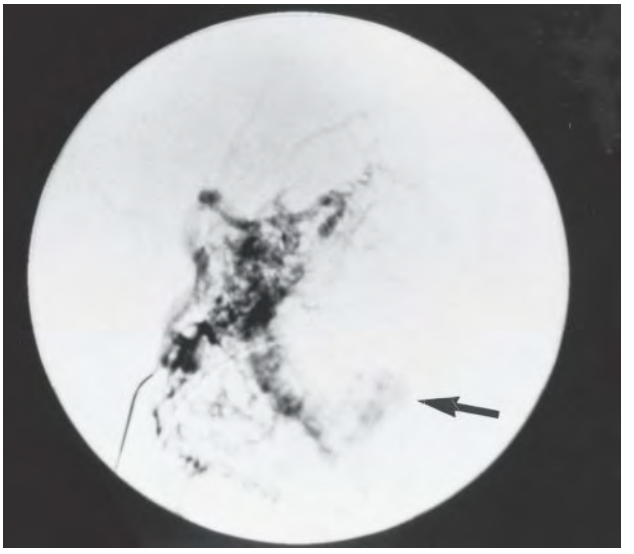


FIGURE 30-28. Subtraction angiogram of a high-flow vascular malformation of the mandible. Oblique lateral view with lower incisors *(arrow)*.

diagnosed, selective angiography is normally performed via a femoral approach (Figure 30-28). If a high-flow vascular malformation is diagnosed, treatment is normally preoperative embolization followed by wide resective surgery. The embolization can involve a number of materials, including muscle, polyvinyl, pellets, and platinum coils, which are inserted via the angiography catheter or on direct puncture. On entering the lesion, they unwind and expand (Figure 30-29).[118,119] Postembolization angiography carried out immediately after the embolization normally shows a diminution in blood flow to the lesion. However, because of the powerful angiogenic effect of these lesions (probably by production of angiogenesis growth factor), reestablishment of smaller collateral vessels usually occurs within a few days, and it is often impossible to reembolize these smaller collateral vessels. Therefore, definitive surgery should be carried out within a

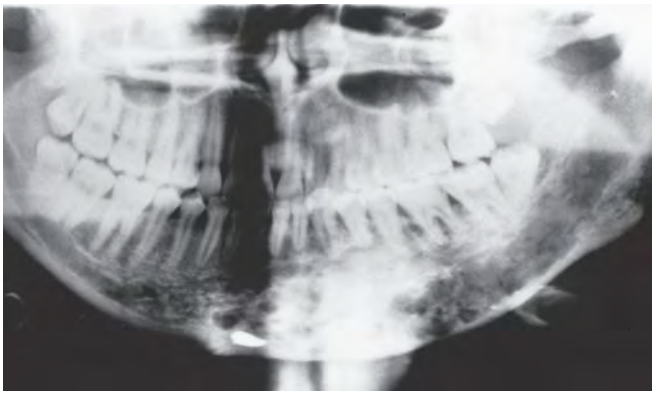


FIGURE 30-27. Radiograph of a high-flow vascular malformation of the left mandible crossing the midline. It appears as an ill-defined "soap bubble" radiolucency causing some root resorption.

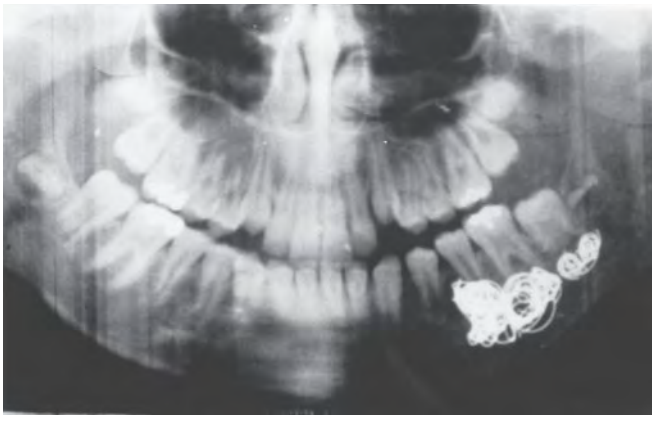


FIGURE 30-29. A high-flow vascular malformation embolized with a platinum coil.

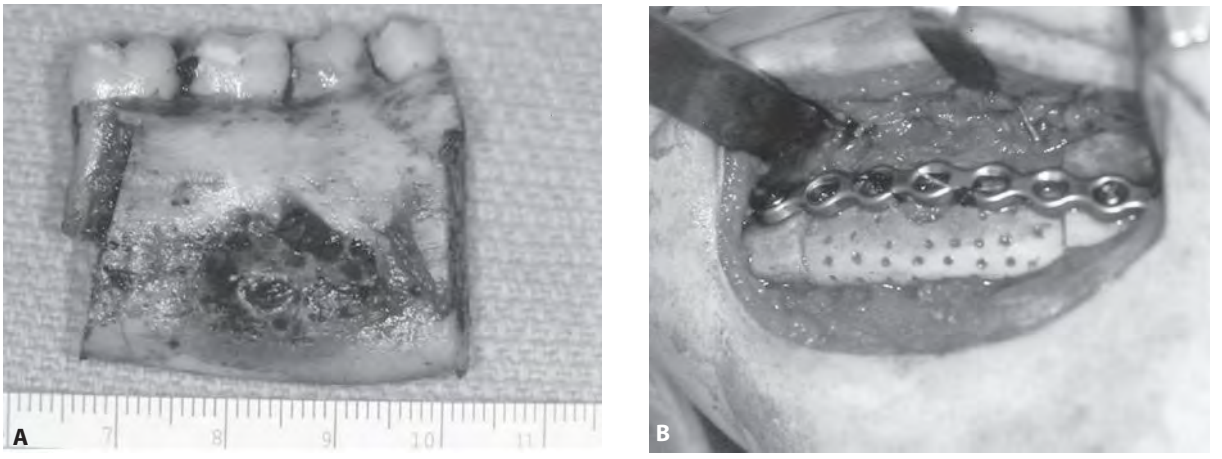


FIGURE 30-30. **A,** Resected mandible containing a high-flow vascular malformation that had been embolized previously. **B,** The same resected specimen reduced to a hollow perforated tray, filled with autogenous iliac crest cancellous bone, and replaced for an immediate reconstruction. (**A** and **B,** Photographs courtesy of J. S. Lee, DDS, MD.)

small number of days of embolization. Definitive surgery normally takes the form of resection under hypotensive anesthesia with adequate resuscitative measures available.[120–122] After resection, appropriate reconstruction can be performed. This can include the re-insertion of the resected portion of bone after curettage, thinning, perforation, and simultaneous bone grafting (Figure 30-30). Other approaches such as injection of a variety of substances into the lesion including glue, fibrin gel, and platinum coils,[119] for example, have been attempted; also, case reports exist of lesions being treated by means of local curettage after embolization, but this is not normally recommended.

Low-flow or venous malformations are not as life-threatening and are normally treated with direct puncture and an attempt to thrombose the lesion by intralesional injection of a variety of agents, including sclerosing agents, an absorbable gelatin sponge, and platinum coils. This may bring about thrombosis, allowing the necessary dental or surgical treatment to be carried out. Often, mandibular resection is not necessary but, rather, a local surgical procedure.

Langerhans' Cell Histiocytosis

Langerhans' cell histiocytosis is the term currently employed for what was previously known as histiocytosis X, and before that the three separate conditions Letterer-Siwe disease, Hand-Schüller-Christian disease, and eosinophilic granuloma. Lichtenstein[123] first suggested that the three diseases were related and that the common factor was the presence of histiocytes. The cells of origin of this disease have now been identified as the Langerhans cells, which are dendritic cells in the skin and mucosa that have a macrophage-like function. At the present time, what causes these cells to proliferate in a clonal fashion with phenotypic evidence of activation and give rise to Langerhans' cell disease is unknown.[124] The nature of this disease also eludes us. Some recent studies have suggested that it may have some of the properties of a tumor or have a viral etiology. Other studies propose that it may be a response to an overwhelming allergenic challenge, and they report cases of eosinophilic granuloma that have resolved spontaneously, further adding to the puzzle.[125] The Histiocyte Society has attempted to define all of the histiocytic diseases in a logical manner,[126] and Letterer-Siwe disease is now believed to represent the acute disseminated form of Langerhans' cell histiocytosis, whereas Hand-Schüller-Christian disease represents the chronic disseminated form, and eosinophilic granuloma represents the chronic localized form.

The acute disseminated form usually affects young children. It is multisystem in nature, affecting the skin, bones, and internal organs (especially lungs and liver), and is frequently fatal. Treatment is by chemotherapy.

The chronic disseminated form of the disease is classically associated with a triad of punched-out bone lesions (often affecting the skull and jaws), diabetes insipidus (owing to posterior pituitary involvement), and exophthalmos (owing to deposits in the posterior orbit). This normally affects an older age group, often in the second and third decades but sometimes much older. The bone lesions often affect the jaws. Although they usually appear as fairly well-defined punched-out radiolucencies (Figure 30-31), they can also be less well defined and can affect the apices of the teeth only and lead to a possible differential diagnosis of periapical infection. A frequent aspect of presentation is loose teeth; radiographically, they often appear as "floating teeth" (Figure 30-32). The treatment of the chronic disseminated form of the disease is variable and, for well-circumscribed lesions, can consist of local curettage. However, for more aggressive forms, chemotherapy is frequently employed as well. Low-dose radiation therapy has also been used on isolated lesions, and it does remain one of the very few indications for low-dose radiation therapy, often in the region of a few hundred centigray.

The chronic localized form of the disease is commonly found in the jaws and usually shows as a well-defined

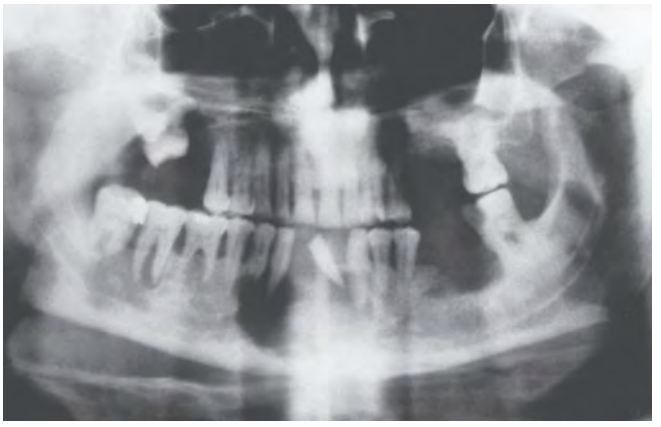


FIGURE 30-31. The multiple irregular radiolucent lesions of chronic disseminated Langerhans' cell histiocytosis in a 53-year-old male whose son died of the acute form of the disease at age 11 years.

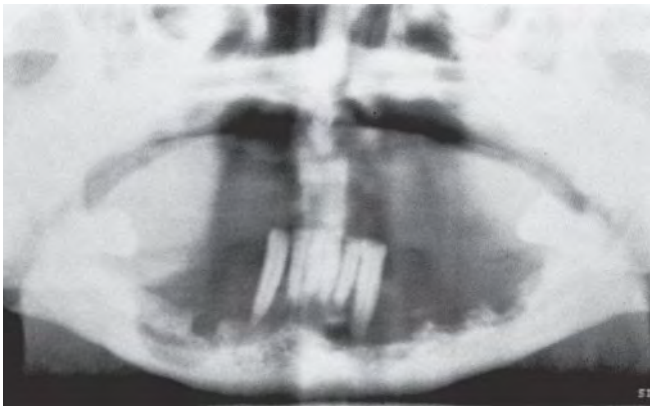


FIGURE 30-32. The "floating teeth" of Langerhans' cell histiocytosis.

radiolucency, often in the bicuspid region and more frequently in the mandible. Differential diagnosis in this case includes any fairly well-defined radiolucency. Treatment usually consists of aggressive local curettage, and the recurrence rate is low. Teeth are sacrificed as necessary. Intralesional steroids have also been employed with some success, and cases of spontaneous regression have been reported.[127]

It is generally believed that the occurrence of Langerhans' cell histiocytosis is sporadic, but clusters have been noted and there are a number of reports of a familial incidence.[124] The author has seen the disease in a father and a son. The father was diagnosed with the chronic disseminated form of the disease at age 53 years (see Figure 30-31), whereas his son died from the acute disseminated form of the disease at age 11 years.

NONODONTOGENIC CYSTS OF THE JAWS

In this section, the following are discussed: globulomaxillary lesion, nasolabial lesion, median mandibular cyst, nasopalatine duct cyst, all of which are also known as *fissural cysts, traumatic bone cyst,* and *Stafne's bone cyst.* Aneurysmal bone cyst has been discussed under "Lesions Containing Giant Cells," earlier.

Globulomaxillary Lesion

Globulomaxillary lesion was initially defined as a globulomaxillary cyst and was thought to be a fissural cyst caused by retained epithelial remnants at the fusion of the maxillary process with the globular process. It is normally found in the second or third decade. In the classic description, the lesion presents as a pear-shaped well-defined radiolucency in the maxilla between the lateral incisor and the canine. Associated teeth are classically vital, and the lesion is lined by cystic epithelium with occasional globular or ciliated epithelia.

Current thinking is that, although this lesion does exist as a radiographic and clinical entity (Figure 30-33), it is not, in fact, a fissural cyst because the proposed embryonic derivation is now known to be flawed and the supposed fusion line does not exist. It is believed that most lesions previously diagnosed as globulomaxillary cysts can now be reclassified as *keratocystic odontogenic tumor* (formerly "odontogenic keratocyst"), *radicular cysts, periapical granulomas, lateral periodontal cysts, central giant cell granulomas, calcifying odontogenic cysts,* and odontogenic myxomas.[49]

Tooth roots may be diverged by the lesion, and biopsy is usually necessary to confirm the diagnosis and enable appropriate surgical treatment to be carried out. Treatment normally consists of enucleation and curettage.

Nasolabial Cysts

The nasolabial cyst was believed to be the soft tissue counterpart of the globulomaxillary cyst. Again, it was believed to be formed at the lines of fusion of the globulomaxillary processes. Similarly, this lesion does exist, but its true origin

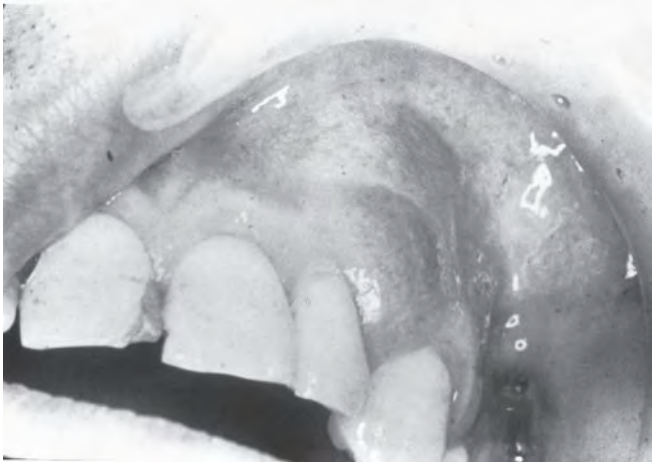


FIGURE 30-33. A globulomaxillary cyst appears as a pear-shaped swelling between the lateral incisor and the canine tooth.

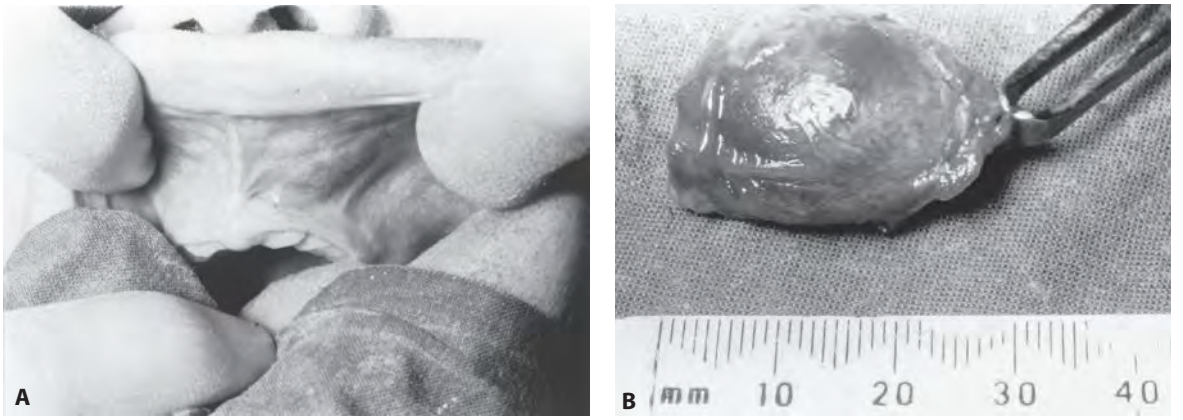


FIGURE 30-34. **A,** A nasolabial cyst causing a swelling in the buccal sulcus in the lateral incisor area. **B,** The enucleated cyst, which was confined to the soft tissues with no bony extension. Histology showed squamous epithelium with goblet cells.

remains in doubt. It could be derived from remnants that form the nasolacrimal duct. This cyst manifests itself as a soft tissue swelling in the lateral aspect of the upper lip, fairly high in the sulcus (Figure 30-34). The cyst lining is typically a pseudostratified columnar type with numerous goblet cells. Treatment is local excision.

Median Mandibular Cyst

Median mandibular cyst is a rare cyst found in the midline of the mandible. It was originally thought to form at the line of fusion of each half of the mandibular arch. Again, the embryologic theory behind this lesion is no longer thought to be plausible, and it is believed that those lesions found in the anterior mandible represent some other type of odontogenic cyst or tumor.

Nasopalatine Duct Cyst

Nasopalatine duct cyst is also known as *incisive canal cyst* and is generally located on the palatal end of the nasopalatine duct. It frequently presents as a soft swelling behind the upper anterior teeth. It is believed to be derived from the epithelial remnants of the paired embryonic nasopalatine ducts within the incisive canal and that either infection or trauma may be the stimulus for the cells to proliferate and form a cyst. These cysts appear to occur more frequently in males than in females and are most common in the fourth to sixth decades of life. Most cases are asymptomatic and either are found by chance on radiograph or present as a soft tissue swelling in the palate. Radiographically, this cyst appears as a well-defined radiolucency found in the midline of the anterior palate (Figure 30-35). In many patients, the nasopalatine duct can be identified on an occlusal radiograph; the question then arises as to when the diagnosis of nasopalatine duct cyst should be entertained. A fairly arbitrary cutoff point of 7 mm has been suggested—if the nasopalatine duct appears to be greater than 7 mm in diameter, the presence of a cyst should be suspected.[128]

Diagnosis is by biopsy, which normally shows a pseudostratified columnar epithelium lining. Treatment, if required, is surgical and consists of local curettage. This almost inevitably requires the sacrifice of the nasopalatine vessels and nerves, which results in a small area of anesthesia over the anterior palate behind the upper incisor teeth. Some patients (particularly more elderly patients) find this particularly troublesome in the articulation of some words. Recurrence rate is very low after treatment.

Traumatic Bone Cyst

Traumatic bone cyst, or cavity, has been called a number of names, including idiopathic bone cyst, simple bone cyst, and latent bone cyst or *cavity.* It is almost always asymptomatic and a chance finding on radiographs. It occurs most commonly in the mandible, particularly in the posterior mandible. It classically appears on a radiograph as a fairly well-defined radiolucency, which usually has a scalloped margin beneath the tooth roots (Figure 30-36). It is not quite as well defined as an odontogenic cyst, and the description made by Howe

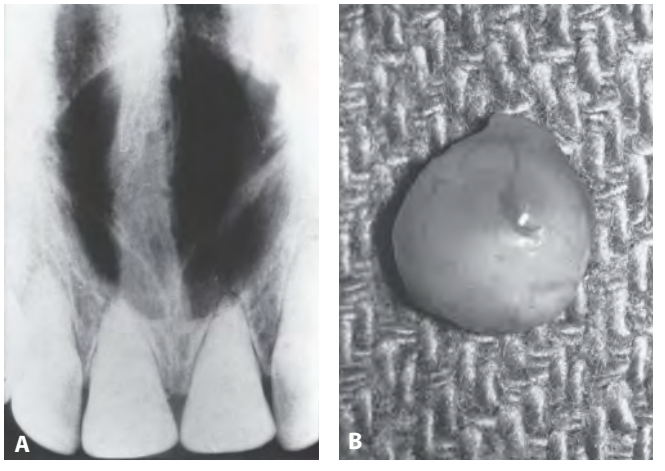


FIGURE 30-35. **A,** Palatine duct cyst appearing as a well-defined midline radiolucency. **B,** The same cyst enucleated.

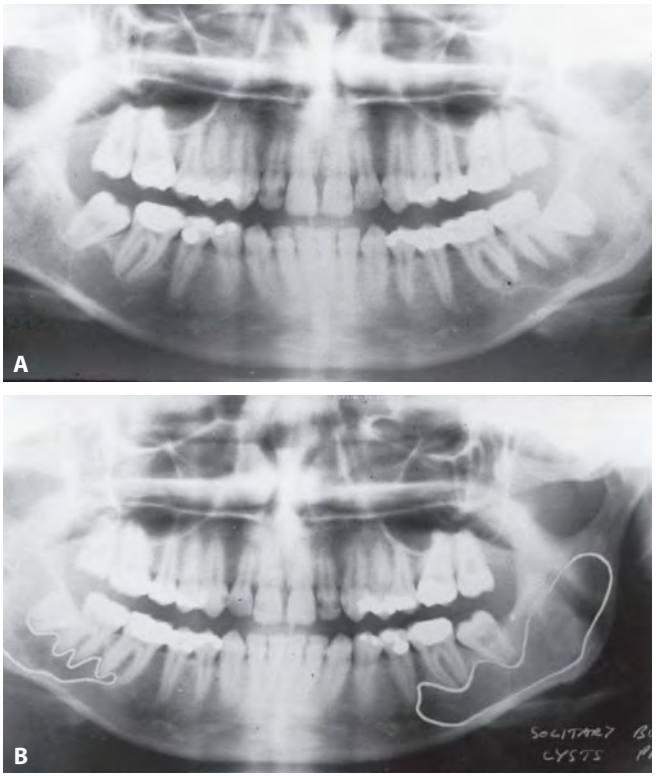


FIGURE 30-36. **A,** Bilateral poorly defined traumatic bone cysts. (Bilateral cysts are unusual.) **B,** Same radiograph shown in **A** with cysts outlined shows the size and scalloped margins around teeth.

was that it appears as a "pencil sketch for a final pen and ink drawing."[129]

The etiology of this lesion is in doubt, and suggestions have included that it may result from trauma, which can be quite mild, but results in intramedullary hemorrhage. Instead of organization and new bone formation occurring, for some reason, the blood clot liquefies and is then resorbed, leaving an empty space. On surgical exploration, these lesions are normally found to have either no lining whatsoever or just a very thin filmy lining. They are normally empty except, possibly, for a little straw-colored fluid in the base of the lesion, which could represent the last remnants of an absorbing blood clot. Studies have shown that the gaseous contents of the lesion are mainly nitrogen, and this is presumably because they contain air and the oxygen is absorbed preferentially into the bloodstream.[130]

Although these lesions have been shown to regress spontaneously, a biopsy is almost always performed to determine the diagnosis. The biopsy is normally curative because anything that causes bleeding into the lesion causes resolution. Suggested treatments have included everything from no treatment whatsoever to curettage or injection of autologous blood or packing with an absorbable gelatin sponge.[131] Recurrences are extremely rare but have been reported, sometimes in conjunction with fibro-osseous disease, as have bilateral cases.[131]

Stafne's Bone Defect

Stafne's bone defect is also known as *static bone cyst;* it is always asymptomatic and found by chance on a radiograph. It presents as a well-defined radiolucency on the lower border of the mandible, below the inferior alveolar nerve (Figure 30-37). The appearance is so diagnostic that biopsy is often not required. When this defect is explored surgically, one normally finds that it is not a totally intrabony lesion but, in fact, an indentation of the mandible on the lingual side (Figure 30-38). The indentation is normally filled with an offshoot of the submandibular salivary gland. This can be confirmed by sialography, which shows filling of the defects with the radiopaque media. Cases have also been seen that include lymphoid tissue in the cavity. It is believed that these may represent developmental lesions, although they may not present until



FIGURE 30-37. Appearance of a Stafne bone defect on panoramic radiograph below the inferior alveolar nerve on the right body of the mandible.



FIGURE 30-38. Clinical photograph of a Stafne bone defect on the lingual side of the mandible. This defect contained lymphoid tissue.

adult life. Such lesions may represent the entrapment of the salivary gland or lymphoid tissue during development of the mandible or the subsequent erosion of the lingual plate of the mandible by the tissue. Treatment is unnecessary, but enucleation is often performed as a process of diagnosis.[132]

NEUROGENIC TUMORS

Schwannoma

The schwannoma is a benign tumor of the neurilemoma or nerve sheath. Although usually found in the soft tissues, it can occur in bone, where it usually exists as a well-defined radiolucency. Following biopsy to confirm the diagnosis, treatment usually consists of surgical excision. Recurrences are rare. Histologically, lesions are well encapsulated and predominantly of spindle cells showing either an Antoni A (spindle cells arranged in palisaded whorls and waves) or an Antoni B (spindle cells with a more haphazard appearance).

Neurofibroma

Neurofibromas are believed to be derived from the fibrous elements of the neural sheath and may exist as solitary lesions or as part of generalized neurofibromatosis or von Recklinghausen's disease. This latter condition is autosomal dominant, and two distinct subsets have been defined. Neurofibromatosis 1 (NF1) has an incidence of 1 in 3500 and the gene is located on chromosome 17q11.2, whereas neurofibromatosis 2 (NF2) has an incidence of 1 in 60,000 and the gene is on chromosome 22.

Although most commonly reported in soft tissues, neurofibromas do occur in bone and have been reported on the inferior alveolar nerve, where they appear as a fusiform swelling in continuity with the inferior alveolar canal (Figure 30-39). Other bone changes associated with neurofibromatosis can include cortical erosion from adjacent soft tissue lesions or medullary resorption from interosseous lesions. In cases associated with the inferior alveolar nerve, pain or paresthesia can result.

The normally recommended treatment after biopsy is localized excision. The lesions are often vascular, and extensive blood loss has been reported from surgical management of mandibular lesions. Mandibular resection has been advocated by some authorities. The malignant transformation rate to neurogenic sarcoma of 5% to 15% in the generalized form of the disease could be a further indication for surgical removal of these lesions.

Traumatic Neuroma

Traumatic neuroma represents a misguided attempt at nerve regeneration whereby following an injury to a nerve, neurons sprout from the site of injury but, for anatomic or physiologic reasons, cannot result in a functional nerve repair. If a nerve is sectioned, an amputation neuroma can develop on the stump; if a nerve is injured along its length, either an incontinuity or a lateral neuroma can result (Figure 30-40). In the oral cavity, these latter neuromas are most often noted on the lingual and inferior alveolar nerves. On the inferior alveolar nerve, they can occur as a fusiform enlargement of the inferior alveolar canal and result most commonly after mandibular trauma, resection of pathologic lesions, and nerve involvement after dentoalveolar surgery (Figure 30-41).



FIGURE 30-39. A neurofibroma on the left inferior alveolar nerve presents as a large fairly well-defined radiolucency in the mandibular ramus *(arrow)*. The patient also had café au lait spots.

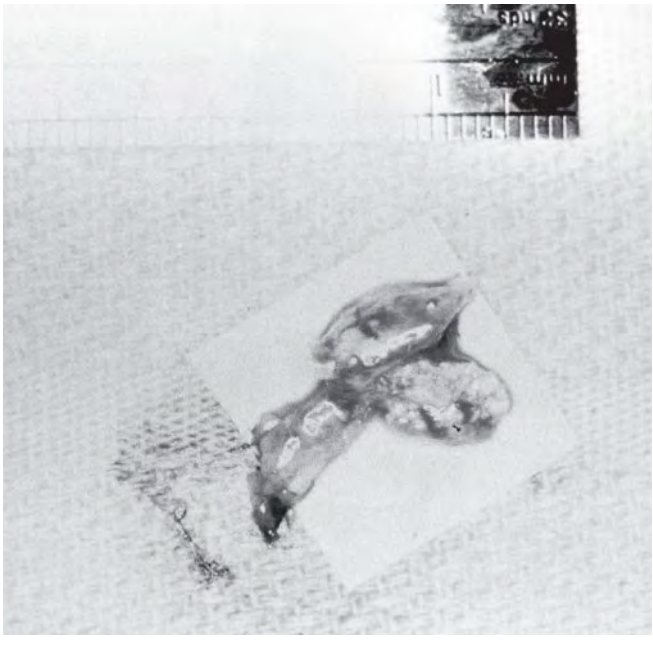


FIGURE 30-40. An excised lateral neuroma that was on the lingual nerve.



FIGURE 30-41. An incontinuity neuroma on the inferior alveolar nerve *(arrow)* as a result of the removal of a third molar. The nerve is exposed via an extraoral approach and lateral corticotomy.

If the symptoms are severe, appropriate treatment is resection of the neuroma and appropriate nerve reconstruction. Because the inferior alveolar nerve cannot be stretched significantly in the canal, repair normally involves a graft of some kind. Nerve grafts from the sural nerve or great auricular nerve have been reported, as have vein grafts, with some success.[133] The approach can be either intraoral or extraoral, but the extraoral approach generally gives better access and clinical results. However, it does have a higher morbidity, with possible risks of scarring and of damage to the mandibular branch of the facial nerve.

PAGET'S DISEASE

First described by Sir James Paget in 1876,[134] this entity still carries his name. Its alternative name is *osteitis deformans.* It is a slowly progressive bone condition of unknown etiology, predominantly affecting males over the age of 50 years. It may represent a complex interplay between genetic and environmental (possibly viral) factors, which are poorly understood. The genetic component may consist of mutations to the SQSTM1 gene that encodes the p62 protein often found in Paget's disease.

Clinically, there is hyperactive bone turnover with alternate resorption of bone, a vascular phase, and finally, a sclerosing phase. Most bones of the body are involved, and the disease can result in considerable deformity. In the facial region, the maxilla is affected more often than the mandible. Family histories have been obtained in this disease, and the genetic basis of the condition is being defined.

The classic presentation used to be a patient whose hat or gloves no longer fitted correctly or in whom false teeth, particularly the maxillary denture, did not fit owing to bone swelling. Today, these presentations are much fewer because well-fitting hats, gloves, and dentures are less commonly encountered. Initial presentation is usually related to bone deformity or pain. In the head and neck, headaches and symptoms owing to vascular and nerve compression have been noted.

The classic radiographic appearance is of a "cotton-wool" appearance in the skull and maxilla of affected patients (Figure 30-42), with hypercementosis around the roots of teeth and loss of lamina dura and obliteration of the periodontal ligament space. This does make tooth extraction extremely difficult in these patients. Root resorption has also been noted.

The histopathology shows the typical reversal lines of alternate resorption and bone deposition (Figure 30-43). Classically, patients have markedly elevated serum alkaline phosphatase levels.

Treatment is both systemic and local. Systemic treatment currently consists of the use of salmon calcitonin or non–nitrogen-containing bisphosphonates to inhibit bone

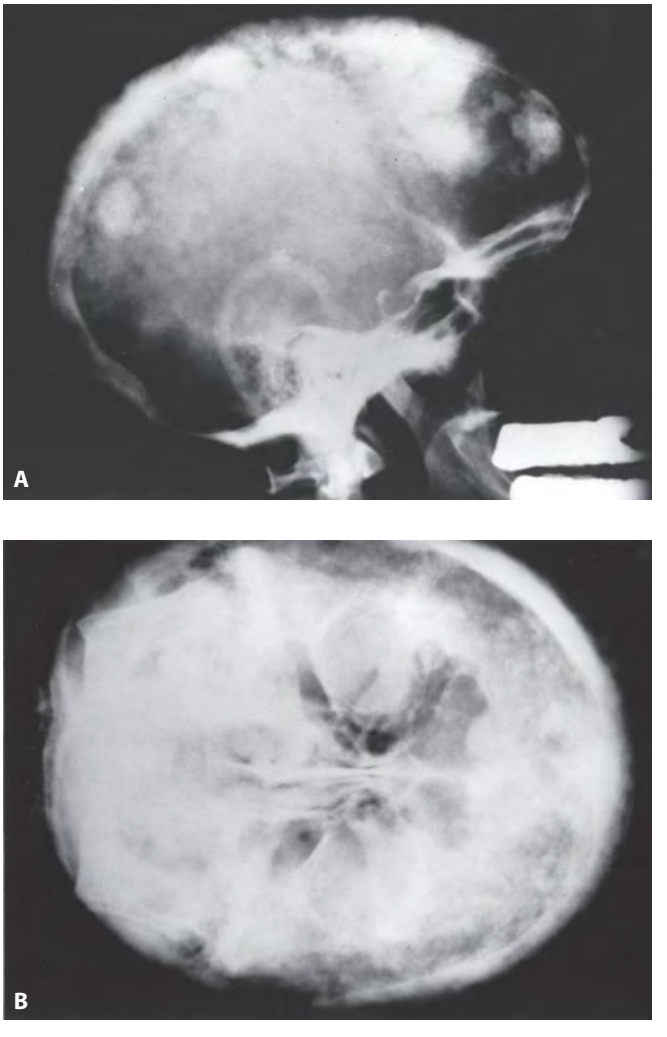


FIGURE 30-42. Lateral **(A)** and frontal **(B)** radiographs of a patient with Paget's disease show typical "cotton wool" appearance.

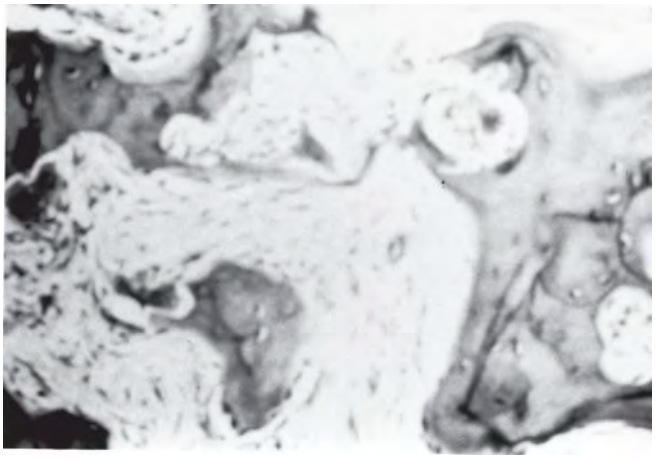


FIGURE 30-43. Histology of Paget's disease shows reversal lines of new bone deposition and resorption (hematoxylin and eosin, ×40 original magnification).

resorption. Calcitonin can be taken either subcutaneously or by nasal spray, and bisphosphonates are taken orally or by injection. Treatment causes stabilization of the bone and a lowering of the raised alkaline phosphatase levels. Localized treatment is directed to cosmetic and/or functional recontouring of bone. It should be noted that the bone of Paget's disease is often vascular, and bleeding during recontouring can be extensive. Somewhat paradoxically, however, healing is often delayed owing to the intervening sclerotic areas of bone.

The classic causes of death in patients with Paget's disease are left heart failure and osteosarcoma. Heart failure is caused by the excessive blood supply to the remodeling bone, which can cause high output or left heart failure in elderly persons. Sarcomatous change has been reported in 5% to 15% of patients with Paget's disease, which should be considered a premalignant condition.[135]

GORHAM'S DISEASE (GORHAM-STOUT SYNDROME)

Although first described in 1838, this disease was named after Gorham, who reviewed the literature and added three new cases in 1954.[136] Its alternative name is *massive osteolysis*. Gorham's disease is a rare disease of unknown etiology, usually occurring in the second to third decades of life, although it has been reported in all age groups. There is no sex or racial predilection, although an autosomal dominant inheritance pattern has been suggested. The diagnosis is usually one of exclusion. Any bone can be affected, and there is usually massive osteolysis, which is generally asymptomatic until a pathologic fracture occurs (Figures 30-44 and 30-45). The bone is usually replaced with fibrous tissue. The majority of cases are monostotic, but polyostotic cases have been reported.[136] There is no specific treatment for this disease; however, radiation therapy and surgical resection have been beneficial in selected cases. Serum biochemistry is usually

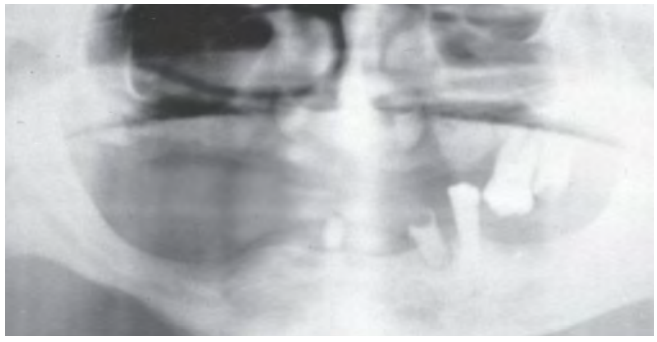


FIGURE 30-44. Early case of Gorham's syndrome with a partial loss of the right body of the mandible.



FIGURE 30-45. Axial CT scan of the patient in Figure 30-44 2 years later; note the complete loss of the right mandible.

normal, and isotope bone scans do not show excessive activity. Osteoclasts are not a prominent feature of the condition. The long-term prognosis is uncertain, but some long-term remissions have been reported.[137]

TORI

Torus Palatinus

The palatine torus appears as a bony hard swelling along the midline of the palate. It can be discrete or may be large and lobular (Figure 30-46). It usually occurs in the second or third decade of life, and has a tendency to grow throughout life. It is tempting to think that these lesions may be embryologic in their development and form at the line of fusion of the two palatal plates, but this is probably incorrect and the true nature of these lesions remains unknown. Larger versions may require surgical removal because of their interference either with speech or feeding or with prosthodontic

reconstruction. The common surgical approach is via a double Y-shaped incision (Figure 30-47) and subsequent bone removal. The bone is virtually always solid cortical bone and is actually fairly difficult to remove. The recommended technique is to make a number of vertical cuts in the bone with a fissure bur (Figure 30-48). Then the intervening ridges of bone can be snapped off and a final smoothing of the residual bone carried out, taking care not to perforate through into the nasal cavity (Figure 30-49). It may be advisable to insert a dressing plate after the procedure to prevent excessive hematoma formation and possible recurrence of the torus (Figure 30-50).

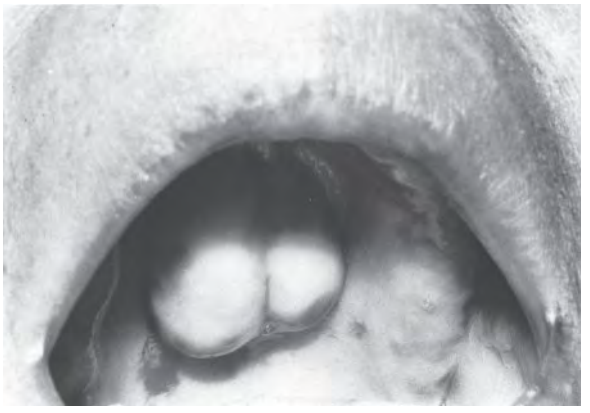


FIGURE 30-46. Large bilateral torus palatinus.

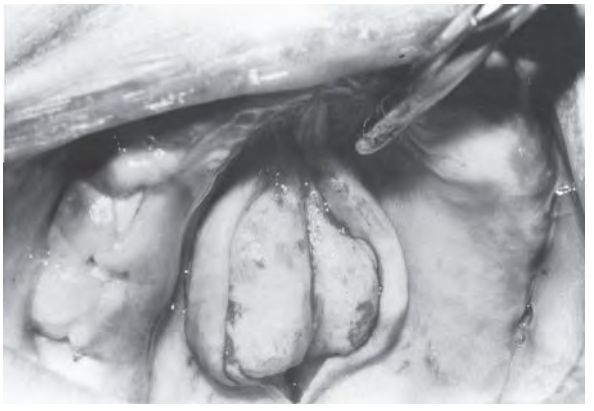


FIGURE 30-47. Torus palatinus in Figure 30-46 exposed via a double Y-shaped incision.

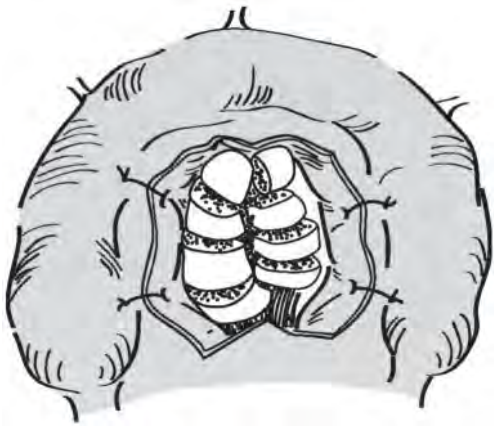


FIGURE 30-48. Diagram of the method of reducing a large torus palatinus with a number of parallel grooves.



FIGURE 30-49. Torus palatinus in Figures 30-46 and 30-48 removed with the technique described in the text.

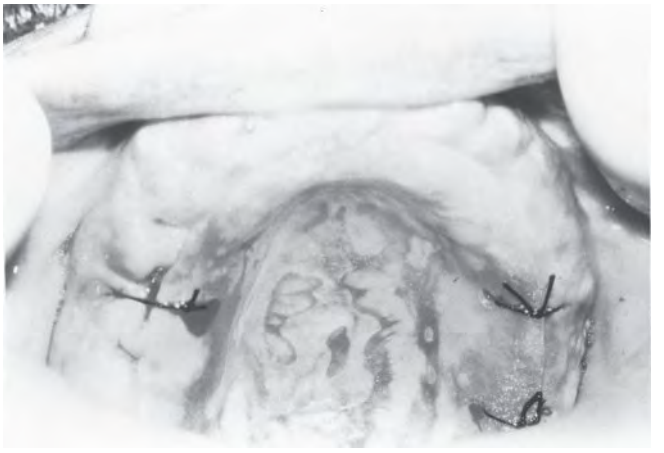


FIGURE 30-50. Dressing plate sutured in place over the wound.

Torus Mandibularis

Mandibular tori are bony exophytic growths that present on the lingual aspect of the mandible opposite the bicuspids (Figure 30-51). They are virtually always bilateral. Again, they present in early midlife and tend to grow with age. Larger versions may require removal because they interfere with tongue positioning, speech, and prosthodontic reconstruction, as well as with oral hygiene around the posterior teeth. The etiology of these lesions is in doubt; again, it is tempting to think of them as being embryologic lesions formed at the junction of the original Meckel cartilage and the neomandible, but this is almost certainly not correct.



FIGURE 30-51. Large bilateral torus mandibularis.

If surgical removal is required, it is carried out via an extensive lingual gingival margin incision with a possible releasing incision, followed by removal of the bone. This is carried out by making a number of vertical cuts with a fissure bur, as with the maxillary torus, and then snapping off the intervening ridges of bone with a periosteal elevator. The residual irregularities are then smoothed with a larger bur. Occasionally, mandibular tori are on a fairly narrow neck and can be removed in toto with a well-directioned blow from a mallet and chisel.

Recurrence of tori is rare, and it has often been noted that palatal and mandibular tori rarely occur in the same patients.

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CHAPTER 31

Oral Cancer: Etiology, Diagnosis, Classification and Staging

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ORAL CANCER

Approximately 85% to 95% of all oral cancers are squamous cell carcinomas (SCCs). However, multiple other malignant lesions can be found in the oral cavity including sarcoma, minor and major salivary gland tumors, mucosal melanoma, lymphoma, or metastatic disease. This chapter is limited to the discussion of SCC, because the other lesions are discussed elsewhere in the textbook. According to the National Cancer Institute, it is estimated that more than 35,000 new oral and oropharyngeal cancers would be diagnosed and more than 7600 people would die from this disease in 2008.[1] This number represents 3% to 4% of all cancers and is the eighth most common cancer affecting males in the United States. The most recent data on oropharyngeal cancer in the United States demonstrate a 2.6 in 100,000 mortality rate and a 10.4 in 100,000 incidence.[1] Globally, it was estimated that more than 500,000 new cases of oral and oropharyngeal cancer were diagnosed in 2008 alone. In parts of the world, such as Southeast Asia, oral and oropharyngeal cancers account for 8% to 10% of all cancer cases reported.[2,3]

The mortality rate remains high in most studies despite recent advances in locoregional control. Many studies have shown that many patients continue to present at the time of diagnosis with advanced locoregional disease (stage III or greater). This finding, despite advances in surgical and adjuvant therapies, suggests that earlier diagnosis and prevention are paramount in reduction in oral cancer prevalence and mortality.

In the United States, there are also apparent disparities in those afflicted with and dying from oral cancer. Overall, in the United States, there has been a decreasing incidence of oropharyngeal cancer over the last 30 years. The mean decrease for all demographic groups is –1.1%. Mortality rates have decreased from –0.33% to –4.48% in the same time period depending on several demographic factors. Currently, there are data to suggest that specific geographic areas within the United States are showing an increasing oropharyngeal cancer rate, especially in older white males.[1] Data have also showed a trend for African Americans to have more advanced disease compared with white Americans at the time of diagnosis. Even more alarming, when compared with equal stages at the time of diagnosis, African American men have a poorer 5-year relative survival rate compared with other races. A review of trends in 5-year relative survival rates from 2000 to 2005 shows an overall higher incidence and relative mortality rate in African Americans compared with whites[1] (Tables 31-1 and 31-2). However, there is an overall reduction in incidence and mortality rates in all races and sexes in this same period compared with earlier data.[1] These racial differences may be due to lack of health care access, public education, and socioeconomic, cultural, or biologic issues. Men, regardless of race, have been the primary group affected by oral cancer. This has been thought to

TABLE 31-1. **Incidence by Ethnicity (2002–2006)***

Ethnicity	Male (per 100,000)	Female (per 100.000)
All races	15.4	6.1
White	15.6	6.1
Black	16.7	5.8
Asian/Pacific Islander	10.8	5.4
American Indian/Alaska Native	9.2	5.1
Hispanic	9.0	3.5

*From Horner MJ, Ries LAG, Krapcho M, et al. SEER Cancer Statistics Review, 1975–2006. Bethesda, MD: National Cancer Institute. Based on November 2008 SEER data submission, posted to the SEER web site, 2009. Available at http://seer.cancer.gov/csr/1975_2006/

TABLE 31-2. **Mortality by Ethnicity (2002–2006)**

Ethnicity	Male (per 100,000)	Female (per 100,000)
All races	3.9	1.5
White	3.7	1.4
Black	6.5	1.6
Asian/Pacific Islander	3.2	1.3
American Indian/Alaska Native	3.6	1.5
Hispanic*	2.5	0.8

*From Horner MJ, Ries LAG, Krapcho M, el al. SEER Cancer Statistics Review, 1975–2006. Bethesda, MD: National Cancer Institute. Based on November 2008 SEER data submission, posted to the SEER web site, 2009. Available at http://seer.cancer.gov/csr/1975_2006/

have been related to the higher incidence of tobacco and alcohol abuse in this group. However, tobacco usage remains relatively high in adolescent and adult women.[4] How this will affect the disparity between the sexes in the future has yet to be seen and further studies of these areas are warranted.

RISK FACTORS

The etiology of SCC of the oral cavity is multifactorial and has been studied extensively. Numerous risk factors have been associated with oral cancer. No single causative agent has been identified, and many variables likely contribute along the spectrum of normal mucosa to carcinoma. The most widely studied risk factors for oral SCC are tobacco and alcohol use. New data implicating the roles of viruses are also emerging.

Tobacco use has long been associated with the development of many malignancies. The risk of oral cancer associated with tobacco use is noted to be 2 to 12 times higher than in the nonsmoking population, and up to 95% of individuals with oral cancer will have a tobacco use history.[5–9] The risk for oral cavity cancer development in nonsmokers is thought to be between 5% and 30%.[10] There are 55 known carcinogens listed by the International Agency for Research on Cancer in tobacco smoke[11] (Table 31-3). A combination of various carcinogens within tobacco, combined with the heat, may lead to a variable number of genetic mutations in the epithelium of the upper aerodigestive tract. At some point, these continued mutations, coupled with the patients' own inherent genetic susceptibility, induce a cell line capable of unregulated growth.

TABLE 31-3. **Carcinogens in Cigarette Smoke and Smokeless Tobacco**

NNN
NNK
Cadmium
Formaldehyde
Lead
Polycyclic aromatic hydrocarbons
Aromatic amines
Heterocyclic aromatic amines
Aldehydes
Miscellaneous organic compounds

NNK = 4-methyl-***N***-nitrosamine-1-(3-pyridyl)-1-butanone; NNN = ***N***-nitrosonornicotine.

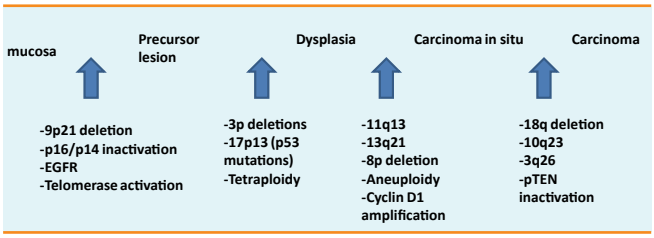
The role and significance of smokeless tobacco (SLT) products are debated in the literature.[11] Most studies attribute an increased risk in developing oral cancer; however, the relative risk is generally small. The main carcinogenic effects of SLTs, like smoked tobacco, are thought to arise from tobacco specific *N*-nitrosamines. These include *N*-nitrosonornicotine (NNN), 4-methyl-*N*-nitrosamino-1-(3-pyridyl)-1-butanone (NNK), *N*-nitrosoanatabine, and *N*-nitrosoanabasine. Only two of these compounds, NNN and NNK, have been demonstrated to be carcinogenic in humans. The raw, green tobacco leaf has very low levels of these compounds. The curing and heating processes used in manufacturing of the final tobacco product increase these to a much higher level. Cadmium, polonium-121, lead, formaldehyde, and polycyclic aromatic hydrocarbons have also been implicated as potential carcinogens in SLTs. Polycyclic aromatic hydrocarbons, in particular benzo(a)pyrene, are known to be potent carcinogens. They are important in tobacco smoking's carcinogenic activity. These compound are found in SLTs at levels near ambient environmental exposure and likely do not contribute significantly to mutagenesis. SLTs may also have some antioxidant properties as well. Carotenoids and phenolic compounds are found in SLTs; however, the protective role of these compounds in SLTs has yet to be determined.[11] In summary, SLTs do not appear to have as high a risk of mutagenesis associated with tobacco smoking. The reason for this is likely due to multiple factors. There is an increased risk of developing oral cancer with these products and it should not be viewed as a substitute for tobacco smoking.

Alcohol has long been implicated in the development of oral cancer, mainly due to its synergism when used with tobacco.[5] The concurrent use of tobacco smoking and alcohol has been shown to have a multiplicative effect, thereby significantly increasing oral and oropharyngeal cancer risks.[12,13] Alcohol has traditionally not been a recognized as a potent initiator in the development of oral SCC. However, a recent pooled analysis demonstrated that heavy alcohol use (>3 drinks/day) in nonsmokers was associated with an increased risk of developing oropharyngeal, hypopharyngeal, and laryngeal cancer when compared with nondrinkers and nonsmokers.[10] However, the role of alcohol as a promoter in the development of oral cancer when coupled with the use of smoking tobacco has been shown.[14] This may be related to the effects of contaminants in alcohol and its ability to solubilize carcinogens and enhance their penetration into oral mucosal cell lines.[5,15]

TUMOR BIOLOGY

Many genetic and cellular changes have been associated with the development of SCC (Table 31-3.1) It is believed that oral cancers arise from a premalignant progenitor cell line.

TABLE 31-3. **Progression of mutations in squamous cell carcinoma***



*Adapted from: B Perez-Ordoñez, M Beauchemin, R C K Jordan. Molecular biology of squamous cell carcinoma of the head and neck. Journal of Clinical Pathology 2006;**59:**445-453

This cell line continues to undergo clonal expansion. This clonal expansion continues with the cumulative genetic alterations and changes until frank malignancy occurs. The two major genetic alterations include inactivation of tumor suppressor genes (TSGs) and the activation of proto-oncogenes. The majority of these alterations occur in early mutagenesis, within the change from normal mucosa to premalignant mucosa. There are relatively few changes from a premalignant to a malignant state, thus demonstrating a cumulative effect. Loss of chromosome region 9p21 is found in 70% to 80% of all oral carcinoma and has been demonstrated to be an early event. *CDKN2A* gene locus on chromosome 9p21 encodes the p16 and $p14^{ARF}$ transcripts. These have been found to be responsible for p53 degradation and interfere with G1 cell cycle regulation. The loss of chromosomal 3p region, another early event, has been associated with TSG loss. Multiple other genetic abnormalities have been described.[3]

The role of viral-mediated genetic changes has also been a subject of multiple recent studies. The most common virus associated with the development of oral and oropharyngeal carcinoma is the human papillomavirus (HPV). Like cervical cancers, HPV 16 and HPV 18 are most associated with oropharyngeal SCCs. The mechanism of HPV mutagenesis appears to differ from those of tobacco and alcohol abusers. HPV viral oncoproteins may degrade and inactivate TSGs rather than mutating them.[16,17] HPV 16 and 18 encode two early gene proteins, E6 and E7, which are thought to induce the genetic and cellular changes. E6 is thought to inactivate or degrade p53, thereby arresting cellular division. E7 is believed to be involved with the degradation of Rb, which also disrupts cell cycle regulation. HPV 16– and 18–affected cell lines also show no p16 mutations, which are commonly found in oral cancers related to smoking and, in fact, may overexpress it.[3]

There is evidence supporting that HPV-mediated oral and oropharyngeal carcinomas differ from "traditional" carcinomas. Proliferative verrucous hyperplasia and verrucous carcinoma may also be associated with HPV-related cellular changes.[18–21] The risk factors associated with non–HPV-compared with HPV-related carcinomas appear to be different. Patients with HPV-positive carcinomas are likely to be younger, nonsmokers, have early lymph node metastasis, and have a social history of more frequent heterosexual and orogenital sexual contacts. Oropharyngeal, in particular tonsillar, carcinomas have the highest rates of HPV DNA detection ranging from 45% to 67% of those studied compared with oral cavity lesion whereas HPV DNA has been found in 12% to 18%.[3,16] Data also indicate that HPV-positive tumors may hold a better survival prognosis than HPV-negative tumors.[22] HPV status has not been accepted for use in standard staging or treatment regimens. The introduction of Gardisil, a recently introduced HPV vaccine for cervical cancer, may have an additional role in reducing HPV-associated oropharyngeal cancers. However, at the time of this writing, the data are still pending.

DNA ploidy is another mechanism to analyze genetic changes within malignant cells. It is a less specific means by which to analyze DNA characteristics of cells and has been studied in determining disease aggressiveness and survival characteristics. Cancer cells may be described as *diploid*, *tetraploid*, or *aneuploid* based on the number of chromosomes in the cell nucleus. Diploid cells, which are close to normal cells, have two sets of chromosomes. As the cell's DNA undergoes more mutations, the chromosomal number can become tetraploid (four times the normal chromosomes) or aneuploid (less than two or more than four chromosomes). Tumor cells that exhibit diploid chromosomal changes have been noted to have better survival rates. Those with tetraploid and aneuploid features tend to have a worse prognosis and be more resistant to therapy. More significant histologic changes have also been associated with ploidy changes.[3] Staging and clinical treatment decisions based on DNA ploidy have been investigated, but standard treatment regimens and staging criteria have yet to be changed based on DNA ploidy alone.

PREMALIGNANT DISEASE

Premalignant disease can be divided into that occurring as an isolated lesion or that associated with a condition. A *precancerous lesion* is defined as morphologically altered tissue in which the development of malignancy is more likely than with normal mucosa. A *precancerous condition* is a condition or generalized disease that does not necessarily alter the appearance of the mucosa but may be associated with a greater risk for the development of cancer. Precancerous lesions are broadly classified as leukoplakia and erythroplakia. *Leukoplakia* is defined as a white patch or plaque that cannot be characterized clinically or ascribed to any other pathologic disease.[23] Leukoplakia cannot be scraped or rubbed off and is, therefore, primarily a diagnosis of exclusion. Lesions caused by lichen planus, white sponge nevus, nicotine stomatitis, or other plaque-causing diseases do not qualify as leukoplakia. Leukoplakia is strictly a clinical diagnosis and does not imply any specific histologic diagnosis. Leukoplakia is generally asymptomatic and clinically appears as a white or off-white lesion that may be flat, slightly elevated, rugated, or smooth (Figure 31-1). It may be found as

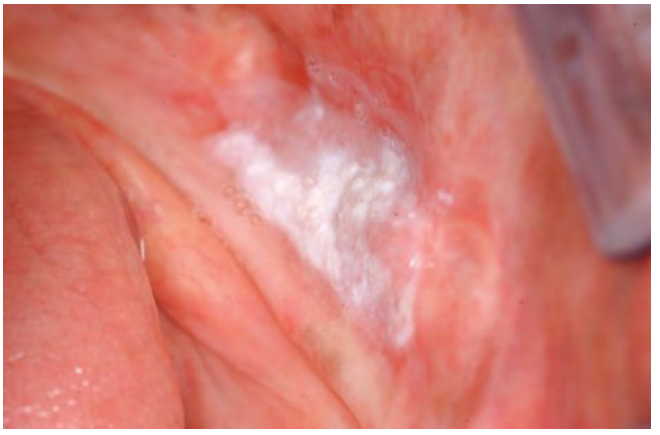


FIGURE 31-1. Typical appearance of vestibular leukopakia.

isolated or multifocal lesions and may change in morphology over time. More than 70% of the time, leukoplakia occurs on two or more surfaces and has a strong male predilection.[24,25] A more aggressive variant exists and is referred to as *proliferative verrucous leukoplakia.*

The lower lip vermilion, buccal mucosa, and gingiva account for most oral cavity leukoplakia; however, lesions found on the tongue and floor of the mouth account for most lesions exhibiting dysplasia or carcinoma. These relative frequencies change with different geographic locations and are based on local habits.

A consistent histology found in all leukoplakia is the presence of hyperkeratosis. The underlying epithelium may range from normal to invasive carcinoma. The true etiology for the development of leukoplakia is unknown; however, several causative factors have been proposed. Tobacco use, whether smoked or SLT, is most closely associated with the development of leukoplakia, and more than 70% of patients with leukoplakia are smokers.[24] Whereas several studies have shown elimination of tobacco use to be associated with resolution or decrease in the size of the lesion, others have shown poor improvement with its cessation. Ultraviolet radiation to the lower lip is frequently observed in the development of lower lip vermilion leukoplakia. Individuals with chronic unprotected exposure to sunlight are at highest risk for development. These leukoplakia lesions are frequently associated with actinic cheilitis (Figure 31-2).[26] Trauma is also associated with the development of leukoplakic lesions. Ill-fitting dentures, sharp edges on oral prostheses or teeth, or parafunctional oral habits with objects such as toothpicks can be associated with leukoplakia. Obvious traumatic lesions to the buccal mucosa such as the development of a linea alba are not considered leukoplakia. The frequency of dysplasia and carcinoma within leukoplakia is most closely associated with the lesion's location and the patient's habits. Waldron and Shafer[27] in their study of 3,256 lesions submitted to their respective oral pathology departments as "leukoplakia" found that 43% of floor-of-mouth lesions and 24% of both tongue and lip lesions contained some degree of dysplasia or carcinoma. Several studies have also looked at malignant transformation over time and found it to vary from 0.13% to 17.5%.[24–29] The results of these studies vary according to suspected causes of the leukoplakia (geographic habits) and the length of follow-up or time to biopsy of the lesion. The malignant transformation of these lesions has been studied extensively by Silverman and colleagues.[24] They note that whereas a definite rate of transformation cannot be stated, their 257 patients had a 17.5% transformation rate with an average follow-up time of 7.1 years. The second year of follow-up in their series exhibited the greatest rate of malignant transformation at 5%. If those lesions initially noted to be dysplastic on biopsy were followed, they had an even higher rate of malignant transformation, at 36.4%. Earlier studies by Silverman and coworkers[28,29] found malignant transformation rates of 0.13% and 6%. The variability in transformation rates of most studies is attributed to differences in ethnicity, drinking alcohol and tobacco usage, location of the lesions, and duration of follow-up. *Erythroplakia* is a red patch that cannot be scraped off or characterized clinically or ascribed to any other pathologic disease (Figure 31-3). Almost all true erythroplakia demonstrates dysplasia, carcinoma in situ, or invasive carcinoma. Shafer and Waldron's review[30] of biopsies submit-



FIGURE 31-2. Actinic cheilitis of the lower lip secondary to chronic unprotected sun exposure.



FIGURE 31-3. Typical appearance of erythroleukoplakia of posterior alveolus and vestibule.



FIGURE 31-4. Typical appearance of erythroleukoplakia on the lateral border of the tongue.



FIGURE 31-5. Neglected carcinoma of the lower lip.

ted under this clinical diagnosis revealed that 51% were invasive SCC, 40% were carcinoma in situ or severe dysplasia, and 9% were mild to moderate dysplasia. The most common sites of occurrence are the floor of the mouth and retromolar trigone. Lesions appear as bright red, are frequently "velvety" in appearance, and have a sharply demarcated border. The etiology of these lesions is unknown but is thought to be the same as that for leukoplakia.

Frequently, these lesions are noted to be nonhomogeneous in appearance with adjacent or intralesional leukoplakia. When observed with this morphology, they are referred to as erythroleukoplakia or "speckled erythroplakia" (Figure 31-4). These lesions also harbor an ominous potential because rates of malignant transformation have been noted of up to 23%.[24] Oral submucous fibrosis (OSF) is a precancerous condition seen predominantly in India and Southeast Asia. It is a chronic, progressive mucosal disorder most frequently associated with the habit of chewing betel quids; however, there is evidence that this lesion is multifactorial in nature with genetic, immunologic, nutritional, and autoimmune factors possibly involved.[31,32] The condition is characterized by a mucosal rigidity that leads to trismus, odynophagia with spicy foods, and difficulty with speech and swallowing. Unlike tobacco pouch keratosis, OSF does not regress with the cessation of betel quid use. Longitudinal studies have shown a malignant transformation rate of 7.6% over a 17-year period.[33]

Clinical Correlation Based on Site

The boundaries of the oral cavity extend from the vermiliocutaneous junction of the lips to the junction of the hard and soft palate posterosuperiorly and to the line created by the circumvallate papilla posteroinferiorly. Posterolaterally, the boundaries are represented by the anterior faucial pillars. The American Joint Committee on Cancer (AJCC) has divided the oral cavity into seven distinct anatomic locations from which primary lesions may develop.[34] The sites have defined boundaries; in developing these sites, the AJCC has attempted to produce a means of better studying and treating oral cancer.

Mucosal Lip

The lip begins at the junction of the vermilion border with the skin and includes only the vermilion surface or that portion of the lip that comes into contact with the opposing lip. It is well defined into an upper and lower lip joined at the commissures of the mouth.[29] It is supported by the orbicularis oris muscle and receives its blood supply from branches of the facial artery. Sensory innervation is provided by the mental nerve and motor function via branches of the facial nerve. Mucosal lip cancers represent approximately 2% to 42% of oral cavity cancers.[5,35–38] Mucosal lip cancer is seen almost exclusively in older white men as a result of chronic sun exposure (Figure 31-5). Its infrequent occurrence in dark-skinned races is further evidence of its etiology. Nodal metastasis in lip cancer is infrequent; 10% of lower lip cancers and 20% of cancers in the upper lip and commissure are found to metastasize to the nodes.[39] Metastasis from the lower lip is to the submental, submandibular, and perifacial nodes (level I more commonly than level II). Preauricular, periparotid, and submandibular nodes drain cancers of the upper lip and commissure (level II more commonly than level I). Bilateral neck metastasis may develop if the lower lip lesion is near or has crossed the midline; however, the upper lip rarely exhibits crossover between right- and left-side lymphatics.[40]

Buccal Mucosa

The buccal mucosa includes all the lining of the inner surface of the cheeks and lips from the line of contact of the opposing lips (mucovermilion junction) to the line of attachment of mucosa to the alveolar ridge (upper and lower) and pterygomandibular raphe.[34] The buccal mucosa is supported by the buccinator muscle posteriorly and the obicularis oris



FIGURE 31-6. Squamous cell carcinoma of the left buccal mucosa.



FIGURE 31-7. Biopsy-proven squamous cell carcinoma of the mandibular alveolar ridge resulting in erosion of underlying bone and loosening of dentition.

anteriorly. The vascular supply to the posterior aspect is derived from the buccal artery, a branch of the internal maxillary artery; innervation is from the buccal branches of the facial nerve along with the long buccal branch of the third division of the trigeminal nerve.

Carcinoma of the buccal mucosa represents 2% to 10% of all SCCs of the oral cavity (Figure 31-6[5,35,36,41] In Central and Southeast Asia, the use of "pan" (a combination of tobacco, betel nut, and lime) has been linked to buccal mucosa carcinoma and represents more than 40% of all oral cavity SCCs.[39] First-echelon lymphatic drainage from the buccal mucosa is level I followed by level II.[42] Cervical metastases are observed in 10% to 27% of presenting patients.[41,43,44]

Alveolar Ridge

The alveolar ridge mucosa may be divided into lower (mandibular) and upper (maxillary) components. The mucosa overlying the alveolar process of the mandible extends from the line of attachment of mucosa in the buccal gutter to the line of free mucosa of the floor of the mouth. Posteriorly, it extends to the ascending ramus of the mandible.[34] The mucosa overlying the alveolar process of the maxilla extends from the line of attachment of mucosa in the upper gingival buccal gutter to the junction of the hard palate. Its posterior margin is the upper end of the pterygopalatine arch.[34] Alveolar ridge or gingival carcinoma represents 2% to 18% of oral cancers and occurs predominantly on the mandibular alveolus (64–76%).[5,35–37,45,46] At diagnosis, approximately one third of these tumors exhibit some bony involvement.[46,47] Lymph node metastasis tends to occur more frequently in mandibular ridge tumors than in maxillary tumors. Nodal drainage is principally to levels I and II for both the maxillary and the mandibular lesions and is found in 24% to 28% of patients at diagnosis.[42,44–47] Alveolar ridge carcinomas are frequently insidious tumors masquerading as inflammatory lesions, periodontitis or gingivitis, tooth abscesses, or denture sores (Figure 31-7).

Retromolar Mucosa

The retromolar gingiva is a triangular region of attached mucosa overlying the ascending ramus of the mandible from the level of the posterior surface of the last molar tooth superiorly to the tuberosity of the maxilla. Laterally, this area merges with buccal mucosa, and medially, is in continuity with the soft palate, anterior tonsillar pillar, and floor of the mouth.[36] Tumors of the retromolar trigone frequently involve adjacent anatomic sites at the time of diagnosis (Figure 31-8). Primary symptomatic complaints with these tumors are sore throat, otalgia, and trismus. Tumors of the retromolar trigone represent 2% to 6% of all oral cavity carcinomas.[5,36,37] Lymphatic drainage from this area is predominantly to the submandibular nodes (level IB) and the upper jugulodigastric nodes (level II).[42,48] Lesions of this region tend to be more aggressive in nature with regard to developing cervical metastasis, because 27% to 56% of individuals present with metastatic disease.[49]

Floor of Mouth

The floor of the mouth is a semilunar space over the mylohyoid and hyoglossus muscles, extending from the inner surface of the lower alveolar ridge to the undersurface of



FIGURE 31-8. Ulcerative carcinoma of the left retromolar trigone with extension toward the anterior tonsillar pillar.



FIGURE 31-9. Carcinoma of the anterior floor of the mouth presents with induration, ulceration, and mild tongue fixation.



FIGURE 31-10. Carcinoma of the hard palate with extension to the alveolar mucosa.

the tongue. Its posterior boundary is the base of the anterior faucial pillar of the tonsil. It is divided by the frenulum of the tongue and contains the ostia of the submandibular and sublingual salivary glands.[35] Anatomically, it consists of the unattached mucosa overlying the mylohyoid and hyoglossus muscles. Carcinoma of the floor of the mouth represents 8% to 25% of oral cavity SCCs, and several studies have shown a fairly dramatic increase in incidence (Figure 31-9).[5,36,37] Two distinct lymphatic drainage systems have been identified in the floor of the mouth.[50] The superficial system drains bilaterally into the submandibular nodes (level I), and the deep system drains into the ipsilateral submandibular and upper and middle jugulodigastric nodes (levels I, II, and III). Studies have shown that nearly one half of all patients presenting with a floor-of-mouth carcinoma will have metastatic disease at presentation.[51–53] Shaha and associates[51] demonstrated that 60% of individuals with metastatic disease will have multiple levels involved.

Maxilla and Hard Palate

The hard palate is between the upper alveolar ridge and the mucous membrane covering the palatine process of the maxillary bones. It extends from the inner surface of the posterior edge of the palatine bone and can be divided into a hard and a soft component.[36] In the United States, only 25% of palatal SCC occurs in the hard palate with 75% occurring in the soft palate (anatomically a part of the oropharynx).[54–56] In India and Southeast Asia, where reverse smoking is popular, the proportion of hard palate lesions is greater. The hard palate represents 3% to 6% of all oral cavity SCCs (Figure 31-10).[5,35–37] There is a paucity of lymphatics to the hard palate. Approximately 10% to 25% of individuals present with evidence of metastasis, generally to levels I and II.[55,57] Hard palate lesions may also metastasize to retropharyngeal nodes or nodes that are not palpable on a clinical examination or readily removable with a traditional neck dissection. Nonhealing ulcers and poor-fitting dentures are common complaints among individuals who develop disease at this site. Recently, several papers have been published in regards to the management of the neck in patients with SCC of the maxilla. It has been suggested that tumors of the maxilla that include the maxillary alveolus and palate may have higher rates of occult lymph node metastasis than previously believed. The evidence is compelling; however, more information confirming these findings is needed before routinely performing neck dissections or adjuvant therapies for all but larger tumors of the maxilla or those with suspicious neck disease.[58,59]

Oral Tongue

The anterior two thirds of the tongue is the freely mobile portion that extends anteriorly from the line of circumvallate papillae to the undersurface of the tongue at the junction of the floor of the mouth. It has four areas: the tip, the lateral borders, the dorsum, and the undersurface (nonvillous ventral surface of the tongue). The undersurface of the tongue is considered a separate category by the World Health Organization.[34] The tongue is entirely a muscular structure composed of the extrinsic muscles—the genioglossus, hyoglossus, styloglossus, and palatoglossus—as well as the intrinsic muscles of the tongue. Blood supply to the tongue is from the paired lingual, sublingual, and deep lingual arteries. The tongue receives motor innervation via the hypoglossal nerve and taste and sensation from lingual branches of the trigeminal nerve. In the United States, SCC of the tongue is found mainly on the anterior two thirds (75%) versus the posterior one third (25%).[60] Tongue carcinoma represents 22% to 49% of all oral cancer diagnosed (Figure 31-11).[5,37,39] Several epidemiologic reviews have shown the unfortunate trend of an increase in tongue cancer and an alarming increase in the incidence of those patients diagnosed before 45 years of age.[38,61–63] Lymphatic drainage of the oral tongue is principally to level II, followed by levels III and IV.[48,50] Carcinoma of the lateral border generally metastasizes ipsilaterally, but SCC of the tip or body of the tongue may exhibit bilateral metastases. Approximately 40% of patients have evidence of clinical node metastasis at the time of diagnosis.[64]



FIGURE 31-11. Carcinoma encompassing the ventral surface of the tongue.

PRIMARY LESION ASSESSMENT

Proper lesion assessment is based on a thorough clinical evaluation. Accurate measurement of the primary lesion before biopsy is essential. Imaging techniques and pathologic measurements should not be the primary measurement for the clinical T stage. Often, biopsied SCCs are referred without accurate measurements, leaving the treating surgeon in a difficult situation relative to properly assigning a T group. In addition, postbiopsy inflammation could lead to over- or underestimation of the lesion's true dimensions. A complete evaluation of all anatomic locations within the oral cavity must be performed by visual examination and palpation to detect any mucosal abnormality. The goal in evaluating the patient is to detect any abnormal tissue and assess the extent of disease. Patients may present with myriad complaints such as a nonhealing sore in the mouth (>2 wk), loosening of teeth, ill-fitting dental prosthesis, trismus, otalgia, or weight loss. Examination of the oral cavity should include removal of all dental appliances and use of a dental mirror for indirect evaluation of the nasopharynx and hypopharynx. Bimanual palpation is critical to assess any involvement of structures such as the deep musculature of the tongue, floor of the mouth, buccal mucosa, salivary structures, or bony mandibular structures. Assessment of the lateral tongue and posterior pharynx is assisted by anterior and lateral traction on the tongue with cotton gauze. The AJCC describes the possible growth patterns of a tumor as *endophytic*, *exophytic*, or *ulcerated*.[34] These characteristics play no part in staging the primary tumor. Although depth of invasion, or tumor thickness, is not used to clinically stage the patient, several studies have shown that depth of invasion does play a prognostic role in the development of regional metastasis, especially in tongue and floor-of-mouth cancers.[65,66] The study performed by Spiro and coworkers[65] at Memorial Sloan-Kettering Cancer Center looked at primary tumor thickness in relation to risk of cervical node metastasis in SCC of the tongue and floor of the mouth. They found that patients with thin (<2 mm) cancer of these respective areas had a failure rate of 1.9% and lymph node metastasis present in 7.5% of patients versus patients whose primary tumor was thick (>2 mm), who had a 45.6% failure rate and metastatic node disease was present in 38%. This was particularly relevant in tongue and floor-of-mouth cancer.

Rarely, primary tumors may be located in areas that are difficult to assess or may be painful to assess, requiring an evaluation under anesthesia along with panendoscopy. Panendoscopy, or "triple endoscopy," involves the use of a rigid bronchoscope, esophagoscope, and laryngoscope to sequentially examine and take biopsies, if required, from the aerodigestive tract. The authors do not routinely perform rigid esophagoscopy or bronchoscopy unless the patient is symptomatic or imaging suggests the need. Computed tomography (CT) and positron-emission tomography with CT (PET/CT) are used in most cases for distant and regional metastatic evaluations. Esophagogastroduodenoscopy (EGD) with percutaneous endoscopic gastrostomy (PEG) tube placement is routinely performed during workup in patients with advanced disease that will need enteral nutritional support in the postoperative and adjuvant periods.

Warren and Gates[67] first described the notion of synchronous and metachronous tumors in 1932. A *synchronous tumor* is described as a second histologically confirmed malignancy. This malignancy must be a distinct and geographically separated by normal non-neoplastic mucosa, and not of metastatic origin from the index lesion. It must also be discovered at the time of initial tumor evaluation. If the second primary tumor is discovered at a later time, it is considered a *metachronous tumor.* Slaughter and colleagues[68] described the concept of "field cancerization" secondary to the panmucosal effects of smoked tobacco irritants and alcohol. This theory seeks to explain the relatively high prevalence of second primary malignancies in the upper aerodigestive tract and has been described on a molecular level.[69] Panendoscopy and imaging techniques are useful for discovering an often asymptomatic synchronous lesion. McGuirt[70] reported a synchronous primary lesion rate of 16% in his prospective study of 100 head and neck cancer patients. The discovery of the synchronous lesions frequently led to an alteration in the treatment plan of the index lesion. Other reported incidences of synchronous primary tumors range from 2% to 9%.[71–75] Endoscopy examination can be performed quickly and at a minimal price for the patient in terms of cost and added morbidity. The availability of flexible endoscopes, especially nasopharyngoscopes, has led to their use in many institutions, along with the conversion to flexible bronchoscopes and esophagoscopes. In addition, with the availability of tomographic and PET imaging, routine preoperative panendoscopy is currently undergoing reevaluation in many institutions.

Many authors believe that the low yield of bronchoscopy compared with chest imaging should preclude its use,

whereas others have called for selective endoscopy to investigate only symptom-driven complaints.[75–78] Should multiple primary tumors be discovered during patient evaluation, each lesion should be staged separately. Chest imaging can demonstrate suspicious, yet sometimes misleading, findings in patients with a history of lung disease or in areas where certain endemic diseases (i.e., histoplasmosis) are prevalent.

ASSESSMENT OF REGIONAL METASTASIS

Evaluation of the neck is perhaps the most critical and difficult aspect of staging oral or any head and neck cancer. The presence of a single lymph node with metastatic disease reduces the patient's 5-year survival by 50%. In turn, the presence of extracapsular spread decreases this survival by another 50%.[79] This importance has been recognized in the most recent edition of the AJCC staging manual, which has added extracapsular (ECS+ vs. ECS–) spread as a N stage descriptor in both the clinical and the pathologic examination. A retrospective study by Snow and associates[80] showed a surprisingly high rate of extracapsular tumor spread in even small lymph nodes. Their analysis showed that lymph nodes greater than 3 cm had a 73.7% chance of extracapsular spread; 2 to 3 cm, a 53.3% chance; 1 to 2 cm, a 44.3%; and less than 1 cm, a 28.8% chance.[80] Other studies have concurred with this high rate of extracapsular spread.[81,82] These drastic reductions in long-term survival underscore the importance of preoperative staging for an appropriate prognosis and treatment plan. It should be noted that staging depends not on specific lymph node level involvement, but rather on presence of nodes, size, number, and whether they are ipsilateral, contralateral, or bilateral in relation to the lesion. Traditionally, the gold standard in staging the neck has been through digital palpation of all levels of the neck bilaterally. The neck has a large number of palpable structures and a large area to be surveyed for the presence of lymph nodes. Although there is no correct order in which to evaluate the neck, each clinician should develop a sequence to utilize consistently to avoid missing any part of the examination. Observation of the neck is important to note any asymmetries or skin changes.

Most clinicians prefer to palpate the neck standing behind the patient, simultaneously palpating each aspect of the neck. We find it helpful to break the neck down into muscular triangles and examine them sequentially from the from the lower, suprasternal region to submandibular triangle to the posterior triangle. The postauricular area, parotids, cheeks, and upper and lower lips should also be palpated because lymphadenopathy and small masses may not be easily visible from observation of the lip. Lymph node chains should be evaluated for the presence of palpable masses, noting their size, surgical neck level, and whether the mass is fixed or movable. Bending the patient's head forward or slightly to the side will ease tension within the neck, allowing for better palpation of deeper structures. The thyroid gland should also be palpated for masses, enlargement, or tenderness. The trachea should be inspected for any deviation or fixation.

There is an unacceptably high incidence of observer error in evaluation of the neck by palpation alone.[83,84] Deficiencies have been observed in both the ability to recognize the presence of a clinically palpable node and in the ability to assess its size. A study by Alderson and colleagues[84] showed that both residents and staff involved in the treatment of head and neck malignancies consistently underestimated the size of smaller nodes and accuracy of assessment was independent of experience. With the advent of advanced imaging, CT, ultrasound imaging (US), and magnetic resonance imaging (MRI) have been used as adjuncts to the physical examination for both evaluating nodal disease and helping to delineate the nodes in relation to vital structures such as the carotid artery. Studies have shown that clinically negative, tumor-positive nodes may be detected on CT or MRI in 7.5% to 19% of cases.[85–90]

Cervical Lymph Node Levels

The neck is divided into six "surgical levels" based on anatomic structures (Figure 31-12). Each anatomic area of the oral cavity has a predictable lymphatic drainage pattern to the over 300 lymph nodes in the neck.[91] By grouping defined nodal groups into surgical levels, clinicians are afforded the ability to communicate with each other. It also allows clinicians to theoretically tailor their surgical management of the neck based on these known drainage patterns.

Level I includes the submental and submandibular nodal groups. Level IA, the submental group, is bounded by the hyoid bone inferiorly, the mandibular symphysis superiorly, and the anterior bellies of the digastrics muscles laterally.

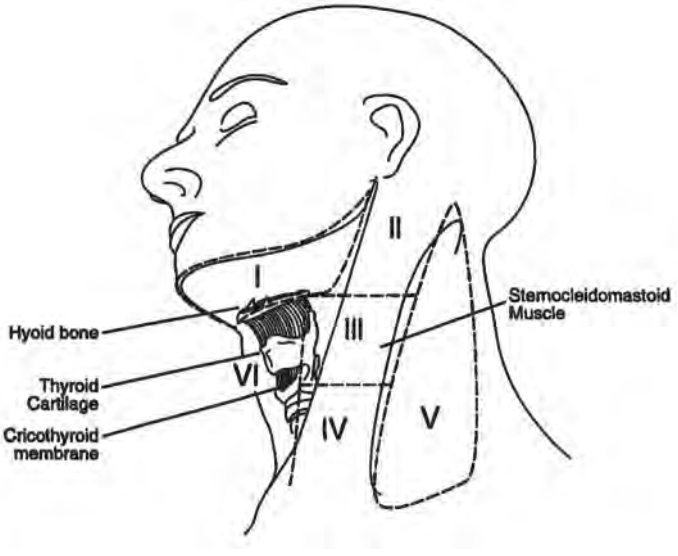


FIGURE 31-12. Lymph node levels of the neck. Levels I to VI are subdivided and described in the text.

Level IB, the submandibular group, is bounded by the posterior belly of the digastric inferiorly, the mandibular body superiorly, the anterior belly of the digastrics muscle anteriorly, and the stylohyoid muscle posteriorly.[34,92]

Level II includes upper jugular lymph nodes surrounding the internal jugular vein and adjacent spinal accessory nerve. Level IIA is bounded inferiorly by a horizontal plane made by the inferior body of the hyoid bone, superiorly by the skull base, anteriorly by the stylohyoid muscle, and posteriorly by a vertical plane defined by the spinal accessory nerve. Level IIB is bounded inferiorly by a horizontal plane made by the inferior body of the hyoid bone, superiorly by the skull base, anteriorly by a vertical plane defined by the spinal accessory nerve, and posteriorly by the lateral border of the sternocleidomastoid muscle (SCM).[34,92]

Level III includes middle jugular lymph nodes surrounding the internal jugular vein. It is bounded inferiorly by a horizontal plane defined by the inferior border of the cricoid cartilage, superiorly by the horizontal plane defined by the inferior body of the hyoid bone, anteriorly by the lateral border of the sternohyoid musculature, and posteriorly by the lateral border of the SCM or sensory branches of the cervical plexus.[34,92]

Level IV includes the lower jugular lymph nodes surrounding the internal jugular vein. It is bounded inferiorly by the clavicle, superiorly by the horizontal plane created by the inferior border of the cricoid cartilage, anteriorly by the lateral border of the sternohyoid musculature, and posteriorly by the lateral border of the SCM or sensory branches of the cervical plexus.[34,92]

Level V includes all the nodes in the posterior triangle, the spinal accessory and transverse cervical nodes, and all of the upper, middle, and lower jugular lymph nodes on the posterior aspect of the SCM.

Level VA is bounded inferiorly by the horizontal plane created by the inferior border of the cricoid cartilage, superiorly at the apex found at the convergence of the SCM and trapezius muscles, anteriorly by the posterior belly of the SCM or sensory branches of the cervical plexus, and posteriorly by the anterior belly of the trapezius muscle.

Level VB is bounded inferiorly by the clavicles, superiorly by the horizontal plane created by the lower border of the hyoid bone, anteriorly by the posterior belly of the SCM or sensory branches of the cervical plexus, and posteriorly by the anterior border of the trapezius muscle.[34,92]

Level VI includes the pretracheal, paratracheal, and prelaryngeal or so-called delphian lymph nodes. It is bounded inferiorly by the suprasternal notch, superiorly by the hyoid bone, and laterally by the common carotid arteries. This level is also known as the *anterior compartment*.[34,92]

Computed Tomography

CT is generally performed preoperatively with intravenous contrast to help delineate vascular from lymph structures. Renal function, by serum creatinine and blood urea nitrogen (BUN) levels, should be obtained before contrast administration, because they are generally nephrotoxic. The scan generally involves 3- to 5-mm slices from the skull base to the clavicles. CT scanning, including office-based cone beam scanning, is also useful to evaluate bony structures for invasion. Axial, sagittal, and coronal views are obtained routinely. Important radiographic markers for the presence of suspicious adenopathy include lymph node size, shape, and central necrosis. A lymph node is considered abnormal when it is greater than 1.5 cm in the jugulodigastric region or greater than 1 cm in other regions of the neck.[86,90] Shape has been suggested as a criterion to help distinguish pathologic nodes. The shape of a normal or hyperplastic lymph node resembles a bean, as opposed to round or spherelike metastatic nodes frequently present. Next to size, the most specific indicator of metastatic nodal disease on tomographic imaging is the presence of intranodal necrosis, independent of size and shape (Figure 31-13). Only an intranodal abscess or fatty hilar metaplasia can simulate central tumor necrosis.

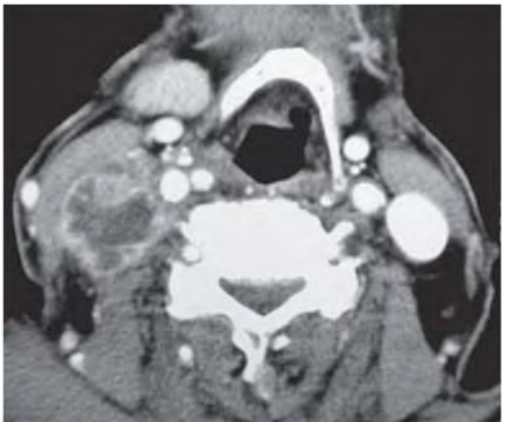


FIGURE 31-13. Axial computed tomography scan with contrast demonstrates a large right cervical node with criteria for regional metastasis.

Magnetic Resonance Imaging

MRI is another method of neck imaging that has been gaining in popularity. With superior soft tissue detail, one would expect better delineation of lymph node pathology; however, the fat that surrounds the cervical lymph nodes can interfere with imaging detection. The T_1-weighted, fat-suppressed, contrast-enhanced image is perhaps the optimal sequence to evaluate cervical metastatic disease.[86,93] MRI provides the distinct advantage of viewing the neck and primary tumor in planes not available by CT. Difficulty with the use of MRI concerns both the time and the motionlessness required for an acceptable study to be performed. Individuals with oral cancer frequently have large lesions that may compromise the airway while supine for extended periods of time. When using MRI for evaluating the neck, the same criteria concerning nodal size, shape, and central necrosis should be applied as when evaluating with CT.

Ultrasound

US evaluation of the neck has become increasingly popular in European countries. Sonography is relatively inexpensive and is tolerated well. It may be used as an initial study to help guide the clinician in deciding whether further imaging studies of the neck may be required. This is especially true in the clinically N0 neck. Sensitivity of sonography in the detection of cervical lymph node metastasis is 89% to 95% and specificity is 80% to 95%.[94–96] This specificity can be increased with the use of US-guided fine-needle aspiration (FNA).[97] Criteria for the evaluation of potentially malignant cervical nodes with sonography also involve the assessment of nodal size, shape, and presence of central necrosis. Metastatic nodes are characteristically round to spherical in shape and are frequently hypoechogenic. In the presence of extracapsular spread, loss of border definition is observed. Normal lymph nodes are frequently difficult to detect because of their high echogenicity mimicking that of the surrounding fatty tissue. US-guided FNA is also a useful tool for evaluating a neck mass suspicious for carcinoma when no other primary site can be found (i.e., "unknown primary").

Positron-Emission Tomography and PET/CT

The use of 2-18F-fluoro-2-deoxy-D-glucose (FDG) PET relies on the enhanced metabolic activity of tumoral tissue in the body, of which increased glycolysis is usually a biochemical hallmark. FDG, a radiolabeled glucose analog, is preferentially taken up within tumor cells that exhibit increased glycolysis; they can be detected from the increased signaling in that tissue (Figure 31-14). This study is unique in that it represents a functional imaging scan as opposed to a morphologic imaging scan. A prospective study by Adams and coworkers[98] showed a higher sensitivity and specificity for FDG-PET (90%, 94%) compared with CT (82%, 85%) and MRI (80%, 79%). Several other studies have produced similar results.[99–101] As with US, FDG-PET may have a unique role in the evaluation of the clinically N0 neck.[102]



FIGURE 31-14. Preoperative positron-emission tomography scan demonstrates increased activity in the right tongue and right neck at levels II and III.

FDG-PET has found a place in the evaluation of an unknown primary with success rates reported up to 60% in the identification of the index lesion.[103–105] Multiple studies have shown the advantages of PET/CT evaluations in the preoperative, post-chemoradiation, and follow-up period. Some authors, based on the high negative predictive values shown with PET/CT, suggest utilizing it for deciding on the need for planned neck dissection after definitive chemoradiation therapy and for routine surveillance for recurrent neck disease.[106] Drawbacks to the use of FDG-PET for evaluation of the neck include the inability to differentiate between cancerous and reactive inflammatory lymph nodes (i.e., after dental extraction, postbiopsy) and the poor anatomic delineation of the primary tumor and neck nodes in relation to surrounding structures, particularly those of a vascular nature. Regardless of its proposed drawbacks, PET/CT has become a standard imaging modality in the management of head and neck tumors pre- and post-treatment.

Assessment of Distant Metastasis

Final evaluation of the oral cancer patient involves a workup for possible distant metastasis. Although the percentage of individuals who present with an untreated primary tumor who already have distant metastasis is low, it is prudent to have thoroughly staged the individual for optimal treatment planning. Distant metastasis from the oral cavity most frequently involves the lung, followed by liver and bone. Therefore, routine posteroanterior and lateral chest radiographs and the evaluation of liver function tests (LFTs) are considered the minimum metastatic workup for head and neck cancer patients. Depending on abnormalities found in the chest radiograph or LFTs, locoregional extent of the disease, and degree of clinical suspicion, the surgeon may also choose to obtain a CT of the chest or abdomen and pelvis. A bronchoscopy or EGD may also be warranted if metastatic or second primary lesions are suspected based on these data. Obtaining other studies such as bone scans should be symptom-driven. An added advantage of an FDG-PET study in the evaluation of distant metastatic oral cancer is its whole body imaging of possible tumor spread. The infrequency of distant metastasis was recognized early by Crile.[107] Studies produced from the patient database at Memorial Sloan-Kettering Cancer Center have also shown relatively low rates in the eventual development of distant metastasis, ranging from 13% in individuals with floor-of-mouth cancer to 15% in patients with carcinoma of the tongue.[51,108] As new therapies lead to better locoregional control of disease, we may expect to see a greater incidence of distant metastasis or second primary lesions in long-term follow-up.

STAGING

The TNM system devised by the AJCC is designed to stratify cancer patients into different stages based on the characteristics of the primary tumor (T), regional lymph node metastasis (N), and distant metastasis (M). It is an attempt to help guide treatment and estimate patients' 5-year survivability. *T* refers to the primary lesion and is graded on greatest dimension and presence of adjacent tissue infiltration (Table 31-4). *N* refers to regional lymph node involvement and is graded on the presence of nodes, greatest dimension, and side of involvement in relation to the primary tumor (Table 31-5). *M* grades distant metastasis and is based simply on its presence (M1) or absence (M0). The AJCC staging system (Table 31-6) is designed for clinical use; however, the patient may be restaged based on final pathology after resection and designated with a *p* prefix (pTNM) or at autopsy with an *a* (aTNM). If synchronous tumors are found at presentation, the higher stage tumor should be used for stage designation, and an *m* suffix may be used to denote the multiple primary tumors (TmNM).[34]

TABLE 31-4. **Primary Tumors (T)**

Tumor	Description
TX	Primary tumor cannot be assessed
T0	No evidence of primary tumor
Tis	Carcinoma in situ
T1	Tumor ≤ 2 cm in greatest dimension
T2	Tumor > 2 cm but ≤ 4 cm in greatest dimension
T3	Tumor > 4 cm in greatest dimension
T4a*	Tumor invades adjacent structures (e.g., through cortical bone into deep [extrinsic] muscle of the tongue, maxillary sinus, skin of face) (resectable)
T4b	Tumor invades masticator space, pterygoid plates, or skull base or encases internal carotid artery (unresectable)

*Superficial erosion alone of bone or tooth socket by an alveolar primary is not sufficient to classify a tumor as T4.

Adapted from Head and neck sites. In Greene FL, Page DL, Fleming ID, et al, editors. AJCC Cancer Staging Manual. 6th ed. New York: Springer-Verlag; 2002; pp. 17–22; and Lip and oral cavity. In Greene FL, Page DL, Fleming ID, et al, editors. AJCC Cancer Staging Manual. 6th ed. New York: Springer-Verlag; 2002; pp. 23–32.

TABLE 31-5. **Regional Lymph Nodes (N)**

Node	Description
NX	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis in a single ipsilateral lymph node, ≤3 cm in greatest dimension
N2	Metastasis in a single ipsilateral lymph node, >3 cm but ≤ 6 cm in greatest dimension; or in multiple ipsilateral lymph nodes, none ≥ 6 cm in greatest dimension; or in bilateral or contralateral lymph nodes, none ≥ 6 cm in greatest dimension
N2a	Metastasis in a single ipsilateral lymph node > 3 cm but ≤ 6 cm in greatest dimension
N2b	Metastasis in multiple ipsilateral lymph nodes, none ≥ 6 cm in greatest dimension
N2c	Metastasis in bilateral or contralateral lymph nodes, none ≥ 6 cm in greatest dimension
N3	Metastasis in a lymph node > 6 cm in greatest dimension

Adapted from Adapted from Head and neck sites. In Greene FL, Page DL, Fleming ID, et al, editors. AJCC Cancer Staging Manual. 6th ed. New York: Springer-Verlag; 2002; pp. 17–22; and Lip and oral cavity. In Greene FL, Page DL, Fleming ID, et al, editors. AJCC Cancer Staging Manual. 6th ed. New York: Springer-Verlag; 2002; pp. 23–32.

DIAGNOSIS

A thorough clinical examination is the first line of defense in the detection of oral cancer. Prognosis is directly dependent on the tumor stage at diagnosis. Nearly one half of all oral cancers are not detected until they are in advanced stages. This delay may be because symptoms may not develop until later in the disease process or the socioeconomic group most likely to develop oral cancer is unable, or unwilling, to seek treatment until it has reached an advanced stage. Studies have shown that only 14% of adults in the United States have ever had an oral cancer examination.[109] A study by Holmes and associates[110] showed that detection of oral and oropharyngeal SCC during non–symptom-driven examinations was associated with a lower stage at diagnosis. These detections occurred in the dental office, whether by a dentist, dental hygienist, or oral and maxillofacial surgeon.

Biopsy

Once a clinically suspicious lesion is identified in the oral cavity, tissue diagnosis must be obtained before rendering

TABLE 31-6. **Stage Grouping**

	CHARACTERISTICS		
Stage	Tumor	Node	Metastasis
0	Tis	N0	M0
I	T1	N0	M0
II	T2	N0	M0
III	T3	N0	M0
	T1	N1	M0
	T2	N1	M0
	T3	N1	M0
IVA	T4a	N0	M0
	T4a	N1	M0
	T1	N2	M0
	T2	N2	M0
	T3	N2	M0
	T4a	N2	M0
IVB	Any T	N3	M0
	T4b	Any N	M0
IVC	Any T	Any N	M1

Adapted from Adapted from Head and neck sites. In Greene FL, Page DL, Fleming ID, et al, editors. AJCC Cancer Staging Manual. 6th ed. New York: Springer-Verlag; 2002; pp. 17–22; and Lip and oral cavity. In Greene FL, Page DL, Fleming ID, et al, editors. AJCC Cancer Staging Manual. 6th ed. New York: Springer-Verlag; 2002; pp. 23–32.

any treatment. In our practices, we educate referring providers to defer biopsy of suspicious lesions. We strongly feel that the *treating* surgeon and oncology team should be able to evaluate the primary lesion and assess for regional disease without the site being altered. In many instances, lesions are excised by the referring providers, thereby making the evaluation and initial staging more difficult. The biopsy can usually be done in an office setting or, rarely, under general anesthesia with panendoscopy if the lesion is difficult to access and patient tolerance is low. The traditional biopsy, whether incisional or excisional (for small lesions), is the gold standard. It should be emphasized that an accurate dimension of the lesion should be acquired before biopsy in order to properly stage the lesion. For histologic and treatment reasons discussed earlier, the biopsy also needs to be of adequate depth to approximate the depth of tissue invasion. When faced with a large lesion, it is best to take several biopsies from different sites in an attempt to decrease any sampling error that might be read as dysplasia, necrosis, or inflammation.

Brush cytology initially gained acceptance in the dental community as a safe, minimally invasive technique for use in the screening of clinically suspicious lesions.[111] Brush cytology differs from exfoliate cytology in that it removes an entire transepithelial layer for cytologic evaluation as opposed to the sloughing surface layer of the mucosa. Commercially available kits exist that include a brush biopsy instrument, glass slide, and fixative. The suspicious lesion is sampled by rubbing or rotating the sampling brush against its surface until pinpoint bleeding at the biopsy site is obtained, indicating sampling to the basement membrane and an adequate specimen. This specimen is then transferred to the slide, fixed in the office, and sent to the corporation for evaluation by both a computer and an oral cytopathologist.

Brush biopsy results are classified as "negative" when no epithelial abnormality is noted, "positive" when definite cellular evidence of dysplasia or carcinoma is found, "atypical" when abnormal epithelial changes of uncertain diagnostic significance are observed, and "inadequate" when an incomplete transepithelial specimen was submitted. The largest study of brush cytology by Sciubba and colleagues[112] found a sensitivity and specificity of 100%. However, as some authors have pointed out, a lack of investigation with scalpel biopsy of atypical results in "innocuous-appearing" lesions has resulted in a possible specificity exaggeration of this technique; other studies have borne this result out with reported sensitivities of approximately 90% but a specificity of only 3%.[113] The brush biopsy's highest value may lie in the general dentist's hand where she or he may encounter epithelial abnormalities on a daily basis and is reluctant to refer the patient for biopsy. It is important to remember that brush cytology is only a screening tool, and any atypical or positive results must be confirmed by an incisional biopsy. The same should be said about highly suspicious lesions read as "negative." If clinical suspicion remains high despite a negative cytology result, a biopsy should be obtained. As a general rule, questionable lesions which do not resolve in 10 to 14 days should have a scalpel biopsy, *regardless* of brush biopsy results.

CONCLUSIONS

SCC of the oral cavity and oropharynx continues to be a problem throughout the world. Volumes of research have been performed detailing the molecular biology, genetics, and pathways of tumorgenesis. New surgical and medical treatment protocols have been developed. In spite of these advances, the overall disease mortality rates continue to be exceedingly high. It is a disease that all practitioners need to be able to recognize and diagnose. The early detection, staging, and treatment of this disease are paramount in patient survival. Because SCCs of the oral cavity and oropharynx are largely preventable, patient and public education as to the prevention of this disease should be a focus of our practices. Patient education, public health measures, and regular oral cancer screenings by providers are likely the key to minimizing the mortality and morbidity of this disease in the future.

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CHAPTER 32

Oral Cancer Treatment

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Oral cavity cancers account for 30% of head and neck cancers and represent a significant challenge to clinicians. Treatment requires multidisciplinary expertise and is complicated by the complex role that the oral cavity plays in speech, mastication, and swallowing. Oral squamous carcinomas account for approximately 90% of oral cavity malignancies and are the focus of this chapter.[1] Although discussion is limited to the treatment of squamous cell cancers, oncologic principles outlined in this chapter can be applied to other malignancies affecting the oral cavity.[2–4]

Regardless of advances in diagnosis and treatment, mortality from oral cancer has not changed significantly in the past 50 years. Approximately 50% of patients diagnosed with oral cancer will ultimately die of their disease.[5,6] Advances in treatment modalities, including improved reconstructive techniques, however, have led to improvements in quality of life. Early detection and appropriate treatment of cancers remain the most effective weapons against cancers of the oral cavity. Unfortunately, public and professional awareness and knowledge of oral cancer are low. An editorial referred to oral cancer as "The Forgotten Disease."[7] Incidence and mortality for oral cancer is nearly double that of cancer of the cervix (30,300 vs. 13,500 and 8000 vs. 4400, respectively); yet few adults can remember their last oral cancer examination, whereas most women are aware of their last gynecologic examination and Pap smear.[8] Patient knowledge of other cancers, such as skin, breast, and prostate, has increased in recent years because of public awareness campaigns. Only recently, however, has oral cancer begun to receive some of the same attention. The American Cancer Society recommends a cancer-related check-up, including examination for cancers of the oral cavity, every 3 years for asymptomatic men and women aged 20 to 39 years and yearly for men and women aged 40 years and older.[9] Although the oral cavity is readily accessible for examination, results of a study by Holmes and colleagues[10] questioned whether health care professionals were screening for asymptomatic cancers. In addition, smaller symptomatic cancers often went undetected in their study and were ultimately detected at a later stage. Interestingly, all asymptomatic cancers were referred from dental practices, and the average clinical and pathologic stage of cancers referred from physician offices were statistically higher.[10] This is unfortunate because the population at highest risk for development of an oral cancer is four to six times more likely to seek care from a physician than a dentist.[11,12] Clearly, there is a need for increasing the public's awareness of oral cancer and improving screening for early oral cancers in order to improve outcomes regardless of treatment modality employed.

HISTOLOGY

Just as the molecular events leading to the development of squamous cell carcinoma are a multistep process, the histologic progression of benign mucosa to invasive cancer typically follows an orderly progression, although the timeline for this progression may be variable. Whereas squamous cell carcinoma is the most common, variations exist that require alterations in treatment.

Verrucous carcinoma is generally considered an uncommon variant of squamous cell carcinoma, representing only 5% of oral cancers.[13] It has a predilection for the buccal mucosa, and typically appears as a thick white cauliflower-like growth (Figure 32-1). The basement membrane is typically intact and the cells are very well differentiated. It is not uncommon to find focal areas of invasive squamous cell carcinoma within the excised specimen, and patients should be prepared for this eventuality. The prognosis is excellent after adequate excision.

Basaloid squamous cell carcinoma represents another rare, and some believe more aggressive, form of squamous carcinoma. It affects males predominantly and is associated with a high rate of cervical and distant metastases.[14] Histologically, basaloid cells are arranged in nests or cords.



FIGURE 32-1. Verrucous carcinoma of buccal mucosa with extension onto the adjacent maxillary alveolus.



FIGURE 32-2. Leukoplakia of the ventral tongue.

Perineural invasion and a high mitotic index are common and coincide with its tendency to recurrence and worse prognosis, with a 38% mortality at 17-month follow-up.[15] Given the aggressive nature of basaloid squamous cell carcinoma, elective treatment of the neck and postoperative radiotherapy with or without adjunct chemotherapy are probably indicated.

With regards to biopsy, an incisional biopsy in the thickest portion of the lesion should include the full thickness if possible or practical. Although sampling the interface of lesional and normal mucosa is useful in some situations, it should be avoided when possible if malignancy is suspected because it will require extension of the final resection margins beyond where the biopsy was extended into normal tissue. Adequate depth of biopsy is important because it allows the pathologist to provide a depth-of-invasion measurement on more superficial lesions, which is predictive in regard to occult metastases, and helps determine the need for elective neck dissection (see discussion on elective neck dissection later in this chapter). Depth of invasion will not influence treatment of deep, indurated, or fixed lesions. The use of slowly resorbing sutures, which will serve as a marker if an excisional biopsy is performed, is best if closure is required.

MANAGEMENT OF PREMALIGNANT LESIONS

Leukoplakia is defined as a predominantly white lesion of the oral mucosa that cannot be characterized as any other definable lesion (Figure 32-2).[16] Worldwide estimates of its prevalence range from 1.5% to 2.6%.[17] Lower socioeconomic status seems to be associated with higher prevalence.[18] The potential for malignant transformation of oral leukoplakia to invasive squamous cell carcinoma is well recognized, and leukoplakia can be considered a precancerous lesion (i.e., "a morphologically altered tissue in which cancer is more likely to occur than in its apparent normal counterpart").[16] Estimated rates of transformation, however, vary widely. This most likely relates to the heterogeneity of the lesions included in most studies. Whereas homogeneous white leukoplakia has a relatively low risk, erythroleukoplakia has a high incidence of associated dysplasia, carcinoma in situ, and frank carcinoma. In their oft-quoted study of 257 patients followed for a mean of 8 years, Silverman and coworkers[19] found transformation rates for leukoplakia to range from 6.5% for homogeneous lesions to 23.4% in erythroplasia. Lesions containing dysplasia had a transformation rate of 36.4%.[19] Of note, the risk of malignant transformation of leukoplakia seems to be higher in nonsmokers, and clinicians should consider excision or close follow-up in these patients.[20] The annual transformation rate in one population was less than 1%, which still demonstrated a 36-fold risk increase for squamous cell carcinoma in patients with oral leukoplakia over the population in general.[21]

A number of devices have recently been promoted to aid in the detection of premalignant and malignant changes based on the absorbtion patterns of different light sources. Unfortunately, none has demonstrated improvement over a thorough examination with incandescent light.[22] Also, advances in molecular biomarkers and microarray analysis have as yet failed to produce a marker or set of markers that reliably predicts which lesions will undergo malignant transformation.[23] Studies have shown that the most important factor influencing the probability of malignant transformation is the clinical appearance of the lesion (i.e., nonhomogeneous vs. homogeneous). In one study, this variable alone outweighed all others including, presence of dysplasia, site, demarcation, smoking, and surgical intervention.[24] Given its asymptomatic nature, the primary indication for treatment of leukoplakia is an attempt to prevent subsequent malignant transformation. Treatment modalities include excision, ablation, and chemoprevention. Unfortunately, no treatment modality has been shown to prevent subsequent development of squamous cell carcinoma, leading many to question the

need for treatment beyond clinical follow-up.[25,26] These and other studies were summarized in the first Cochrane review on therapy for leukoplakia, which concluded that there was no reliable therapy to prevent the transformation of leukoplakia to oral squamous cell carcinoma.[27] Furthermore, there were no effective preventive measures to halt the development of oral leukoplakia. No surgical procedures were included in the Cochrane review because of the lack of randomized clinical trials evaluating surgical excision. Chemopreventive agents including retinoids, beta carotene, green tea, and bleomycin were evaluated. Retinoids held the most promise and were associated with resolution of lesions, but at the expense of significant side effects (see "Chemoprevention" later).[25,27,28] The ultimate goal remains prevention of subsequent malignant transformation, and unfortunately, none of the agents demonstrated this reliably.

Smoking and alcohol cessation are probably the most effective nonsurgical interventions at this point, but even these have not been shown to definitively decrease the risk of malignant transformation.[26]

Surgical excision with scalpel, cautery, or CO_2 laser techniques remains an alternative for dealing with worrisome lesions. Excision of erythroplasia or erythroleukoplakia is probably indicated given the higher probablity that such lesions can harbor severe dysplasia, carcinoma in situ, or superficially invasive carcinoma. CO_2 laser ablation has been used to treat widespread superficial lesions in an attempt to limit scarring and morbidity associated with large excisions. It does not provide a histologic specimen, however, and multiple biopsies are probably indicated before ablation.[29,30] One study reported on leukoplakia that was biopsied and revealed only mild dysplasia and was subsequently completely excised and analyzed. Histopathology identified cancer within 10% of the specimens, suggesting excision rather than ablation may be more appropriate.[31] Laser ablation can be a useful technique for dealing with large areas of leukoplakia. Removal of these areas can make subsequent follow-up and detection of worrisome signs of transformation easier. Unfortunately, recurrence of leukoplakia after excision or ablation is not uncommon, and it does not necessarily prevent malignant transformation.[24,26]

Given the high rates of multiple lesions and their propensity to recur, photodynamic therapy (PDT) is gaining popularity as a potential method for dealing with multiple diffuse lesions. PDT relies on a complex interaction of a photosensitizing agent, which is preferentially concentrated in abnormal tissue, with light of various wavelengths, depending on the photosensitizer, to create necrosis through a nonthermal reaction. The most common photosensitizing agents used in head and neck cancer are Photofrin, Foscan, and d-aminolevulinic acid (ALA). The choice of the photosentitizer depends on the depth of necrosis required based on the thickness of the tumor. Tissue necrosis is mediated through the creation of singlet oxygen, a highly reactive species that induces cellular damage through several mechanisms. Advantages of PDT include minimal damage to surrounding tissues and no cumulative damage as opposed to radiation therapy, which theoretically allows unlimited treatments. Given the propensity for these patients to develop multiple lesions, this is an important advantage over excision or ablation using traditional methods. Disadvantages include marked photosensitivity, especially with regard to sun exposure, for variable lengths of time after the administration of the agent. This can be minimized by using photosensitizers, such as Foscan, which require a longer wavelength for activation. Whereas useful for thicker lesions, thinner areas of carcinoma in situ and superficially invassive cancer require a photosensitizer that has more limited penetration. Areas treated undergo healing through mucosalization with minimal or no scarring. Although a complete review of PDT is beyond the scope of this chapter, excellent reviews are available.[32–34]

PDT has been used with some success in the endoscopic treatment of dysplastic Barrett's esophagitis to prevent its transformation to adenocarcinoma.[35] Similarly, attempts have been made to treat diffuse oral leukoplakia with PDT with some success.[36] It has been reported in the management of oral dysplasia, but patients require close follow-up for recognition and salvage of new lesions.[37] In addition to its role in the management of leukoplakia, trials of PDT applied to invasive squamous cell carcinoma of selected sites in the head and neck have been reported. Copper and associates[38] reported on 25 patients with T1 and T2 lesions of the oral cavity and oropharynx treated with PDT. Complete remission was noted in 86% of lesions. Recurrences were salvaged with conventional therapy.[38] In addition to its application to mucosal lesions, interstitial delivery of light may allow treatment of more deeply situated tumors. PDT can be applied to salvage more deeply situated lesions not amenable to surgical therapy or reirradiation therapy (RRT) protocols. Although results are promising, PDT for leukoplakia and oral cavity cancers remains investigational, and its role in the management of leukoplakia and squamous cell cancers of the head and neck awaits clarification.

ROLE OF PANENDOSCOPY IN TREATMENT PLANNING

Once a histologic diagnosis of oral cancer has been made, a patient evaluation is initiated in an attempt to define the extent of the locoregional disease as well as the existence of distant metastases. The doctor-patient dialogue that began with the biopsy is now continued with the knowledge that a malignancy is present and that further work-up is necessary to define the extent of disease. Patients frequently feel a sense of urgency once a diagnosis of cancer is rendered. They want treatment initiated quickly. It is important to convey that cancer is not a surgical emergency and that the growth rate of epithelial malignancies allows for an appropriate evaluation that must be completed before making treatment recommendations. It should also be remembered that there is a high incidence of depression among the head and neck

cancer patient population. The level of family and community support should be gauged, and appropriate referrals should be made if deemed necessary. The patient's overall medical condition should also be assessed in preparation for any planned treatment. Aside from the standard history and physical examination, including head and neck examination, nasopharyngoscopy or indirect laryngoscopy in the office should be considered. Mutagens responsible for malignant transformation in the oral cavity have also had an opportunity to affect the mucosa of the remainder of the aerodigestive tract, and the rate of synchronous primary tumors (SPTs) has been reported to be as high as 20% in some head and neck cancer populations.[39] Office nasopharyngoscopy and indirect laryngoscopy may be forgone if formal panendoscopy or "triple endoscopy" under general anesthesia is planned to search for SPTs.

Following McGuirt's 1982 study of panendoscopy,[40] examination of the esophagus, larynx, and bronchus was considered mandatory in the workup of the patient with cancer of the head and neck. A mirror examination of the nasopharynx was also frequently included. McGuirt's finding of synchronous tumors in 16% of patients led most clinicians to include formal panendoscopy in their evaluation.[40] Recently, its routine use has been called into question for a variety of reasons, including cost containment, improved imaging modalities, and lower rates of SPTs of the head and neck than was previously expected. Some clinicians still believe there is a role for an examination of the primary cancer under general anesthesia, along with panendoscopy. They argue that the ability to examine some larger primary cancers is compromised in the clinical setting because of patient discomfort and that panendoscopy affords the clinician an invaluable opportunity to examine the primary cancers without this constraint. Others argue that the low yield of bronchoscopy over chest radiographs and computed tomography (CT) scans and the ability to perform an examination of the larynx with flexible nasopharyngoscopy mitigate against the usefulness of rigid laryngoscopy and bronchoscopy. Also, many patients with cancer of the head and neck receive a flexible esophagoscopy at the time of placement of a percutaneous endoscopic gastrostomy tube. For these reasons, panendoscopy should probably be symptom driven.[41]

CHOOSING A TREATMENT

Once the initial evaluation, data collection, and staging are complete, a discussion regarding treatment is undertaken. The National Comprehensive Cancer Network (NCCN) offers online access (www.NCCN.org) to standard treatment algorithms developed by experts in their respective fields. Whereas clinicians are encouraged to consult these algorithms in making treatment recommendations, significant latitude exists in choosing treatment options, reflecting the heterogenicity of oral cancer patients. The clinician and the patient are ultimately faced with deciding which treatment modality or combination offers not only the best chance for cure but also the best preservation of quality of life. Quality of life issues are becoming increasingly important in treatment planning. Despite media hyperbole on cancer treatment "breakthroughs," cancer treatment still falls into three basic categories: surgery, radiation, or chemotherapy, or some combination thereof. Choosing the appropriate treatment depends on many factors, including the patient's medical condition as well as the modalities available to the clinician. Certain therapeutic modalities, such as neutron beam radiotherapy, may hold promise for certain tumors but are limited in their availability. Although each is discussed separately in the upcoming sections, most patients will ultimately receive more than one form of treatment.

SURGERY

Surgery remains the cornerstone of most treatment regimens for oral cavity cancer. Surgery offers several advantages, including the harvest of a specimen for histopathologic analysis and the possibility of removing the cancer utilizing one treatment modality in one session. Surgical resection with frozen-section analysis of the margins is advocated by most clinicians for most stage I and stage II cancers of the oral cavity. Although primary radiation to T1 and T2 lesions may offer similar disease control, the side effects of radiation to the oral environment outweigh those of surgery in most situations. In addition, given the rate of second primary cancers in the head and neck cancer patient population, it is often better to hold radiation if possible in case it is needed in the future. The oral cancer patient population is prone to the development of second primary cancers, and some would argue that radiation for borderline indications might be withheld for future use should the need arise. RRT, although possible in some circumstances, is associated with a high degree of morbidity.

The importance of obtaining clear histologic margins has been a foundation for surgical treatment of oral cavity cancer and has been supported by several studies that have demonstrated decreased survival associated with positive margins, even if follow-up radiation is given. Unfortunately, clear pathologic margins are not always an assurance of a good outcome. Field cancerization is a concept that was proposed by Slaughter and colleagues in 1953,[42] after they reviewed resection specimens from the oral cavity and oropharynx. They found multiple foci of cancer in 11% of specimens. Areas of dysplasia also existed distant from the primary site.[42] Additional studies have shown that margins that are clear histologically may still have cells at the margin that demonstrate premalignant changes, and this can be associated with recurrence. An altered *P53* gene was identified in 52% of patients in one study, and recurrence occurred in almost half of these patients.[43] Other markers have been shown through molecular analysis to exist at margins that are clear histologically; specifically, the proto-oncogene *eIF4E* has been shown to be associated with a decreased disease-free interval (DFI) when present at the resection margin.[43]

Loss of heterozygosity (LOH) is defined as the loss of a normal allele at a particular locus within a cell's genome already harboring a deleterious mutation on the matching allele. LOH found at a histologically clear margin has also been shown to correlate with development of recurrent tumor after excision.[44] Some authors have suggested that the concept of "marginal zones" as opposed to strict "margins" may more accurately reflect what is occurring at the tumor-host interface, where molecular changes may be present even in the face of histologically normal "clear" margins. Surgeons will likely continue to debate what constitutes an adequate margin with the understanding that the heterogenicity of squamous cancers of the oral cavity makes adequate margins in one case wholly inadequate in another.[45] This concept is reflected in an interesting study by Thomson[46] on patients with unilateral squamous cell carcinomas or premalignant lesions. Biopsies were taken on the opposite side of the mouth in the same area as the contralateral cancer or premalignant lesion. These so-called mirror image biopsies revealed frank dysplasia or carcinoma in situ in 30% of patients.[46] This concept of "condemned mucosa" has caused clinicians to question the ability of surgeons to obtain clear margins in some patients and in some cancers. Studies have clearly shown, however, a decreased local control and survival in patients with positive margins even in patients receiving adjuvant therapy. Jacobs and coworkers[47] analyzed patients who had received postoperative radiation; patients with satisfactory margins suffered an 11% relapse rate, whereas patients with unsatisfactory margins relapsed 26% of the time. This is most likely due to the inability of radiation therapy to deal with the increased tumor cell burden in some cases of final positive margins.[47] Some investigators have suggested the feasibility of real-time intraoperative margin analysis for markers such as p53 or EIF4e to improve margin control.[48] In addition, improved control may be obtained in the case of unsatisfactory margins by the addition of brachytherapy to standard external beam postoperative radiation.[49] Improved survival was demonstrated in patients with positive margins who received this intensive radiation.[49] It is agreed by most that surgeons should strive for clear margins, given the impact of positive margins on survival. Excision with 1 to 1.5 cm of normal tissue beyond the obvious tumor edge is generally sufficient. Thin specimens of the margins should then be harvested from either the specimen or the wound resection periphery depending on the surgeon's preference. These thin strips are oriented for the pathologist using a specimen map (Figure 32-3). Mucosal margins as well as deep margin specimens are submitted; however, frozen-section analysis is helpful but not infallible. The role of frozen analysis of margins in oral cavity cancer has been heavily debated and its cost effectiveness has been called into question, leading some surgeons to abandon the practice. Although frozen-section analysis is highly accurate and has a high correlation with final histologic analysis of the submitted tissue samples, its ability to predict whether the entire tumor surface of the final specimen will be clear of close or involved margins is not as reliable. For this reason, frozen sections appear to be more beneficial in smaller localized tumors.[50–52]



FIGURE 32-3. Specimen map for demonstration of the orientation of frozen section specimens to the pathologist.

On occasion, a surgeon will be faced with a situation in which the frozen margins obtained at surgery were negative but the final processed specimen shows involvement of one or more margins. This phenomenon can have several explanations. First, sampling error can occur. It is not uncommon for tumors, especially those involving the tongue, to show narrow streaks or small satellite islands well ahead of the main tumor front.[53] Second, if the margins analyzed by frozen technique were taken from the tumor resection bed, then it might lie just beyond the cancerous margin. Also, tumor shrinkage of approximately 25% to 30% occurs when the tumor is removed from the body. Finally, a multifocal tumor or an undiagnosed synchronous second primary may exist.[53] When faced with this dilemma, the surgeon has several options. Re-excision of a positive soft tissue margin is difficult and rarely productive. Wound closure or reconstruction of the defect distorts tissue, and it is frequently impossible to determine exactly where the positive margin was. For this reason, a final positive margin may represent an indication for postoperative radiation therapy (see "Radiation," later). Conversely, clearance of cancer within bone with radiation is difficult and recurrence rates are high, suggesting that patients with positive bone margins should be strongly considered for re-excision. Recent reports illustrate that pathologic margins that are positive on final analysis are more likely a reflection of the aggressiveness of the particular cancer than a reflection on the surgical procedure, and histologic risk assessment is more predictive for DFI and survival than margin status.[54,55] Sutton and associates[56] found that final positive margins had a high correlation with aggressive histologic parameters such as perineural and lymphovascular invasion. Thus, the biologic aggressiveness suggested by positive margins may in itself account for the poorer outcome of patients with positive surgical margins and be an indication for multimodality therapy instead of attempts at

re-excision. Indeed, one of the main advantages of surgery as the initial treatment modality for oral cancer is the ability to obtain a specimen to examine for known indicators of biologic aggressiveness—histologic grade, lymphovascular and perineural invassion, lack of inflammatory front at the tumor-host interface, sialatropism—and use these as a guide to the need for adjuvant therapy (see discussions on "Radiation" and "Chemotherapy," later). In addition, whereas clinicians and patients are often comforted by a report of "clear margins," it must be understood that pathologic analysis at the light microscopy level is not 100% accurate and a certain number of false negatives invariably occur. This can be expecially true with regards to tissue after multimodality therpy.[57]

Despite its central role, surgery in patients with head and neck squamous cancer presents a series of variable and unique challenges that surgeons should be prepared to face. The following discussion offers an overview of some of the perioperative issues facing patients and surgeons. Subsequent sections review surgical points pertinent to specific sites within the oral cavity.

Perioperative Issues in Oral Cavity Cancer Treatment

The decision to operate on a patient with head and neck cancer must involve consideration of potential complications. Studies have demonstrated that age itself is not associated with increased complications, but medical co-morbidities are associated with both increased complications and lengthy hospital stays.[58] This is especially true regarding complex reconstructive efforts, such as vascularized tissue transfer.[59] Several factors deserve special attention in the patient undergoing surgery for a malignancy of the upper aerodigestive tract.

Airway

If there is any doubt concerning the ability of a patient to maintain an airway in the perioperative period, a tracheotomy is advisable. A tracheotomy tube does not prevent aspiration and, paradoxically, may actually increase its likelihood because of tethering of the trachea and impaired glottic closure. Tracheotomy is not without its own risks, and the nursing staff performing tracheotomy care must be well versed in suctioning and maintenance. Decanulation can generally be performed soon after edema has decreased.

Perioperative Antibiotics

Operations on the oral cavity are considered "clean-contaminated," and therefore, perioperative antibiotics are indicated. Several well-controlled studies have demonstrated that antibiotics started before the incision and continued for no more that 24 hours serve to minimize perioperative infections and emergence of resistant strains. First-generation cephalosporins and clindamycin represent the most commonly used prophylactic antibiotics in oral cancer surgery. Topical antimicrobials such as chlorhexidine and clindamycin rinses have also been shown to successfully reduce the incidence of infections.[60,61]

Alcohol Withdrawal

Many patients with oral cavity cancer are dependent on alcohol. Alcohol withdrawal has been reported to affect around 3% of head and neck cancer patients after major head and neck surgery.[62] Alcohol withdrawal can culminate in delirium tremens leading to cardiovascular collapse and death. Appropriate prophylaxis with benzodiazepines is recommended if the patient drinks daily. Lorazepam is commonly used because of its predictable onset and lack of active metabolites. Intravenous alcohol (5–10% alcohol with 5% dextrose in water) can be used in the postoperative period and slowly tapered as the patient recovers from surgery. Lansford and colleagues[62] demonstrated improved outcomes in head and neck cancer patients with alcohol withdrawal when a standardized, symptom-driven protocol for recognition and implementation of treatment with pharmacologic therapy and nutritional support was instituted.

Deep Venous Thrombosis

Patients who will be immobilized for a significant time during or after surgery should receive prophylaxis for deep venous trhombosis (DVT). This prophylaxis most commonly takes the form of mechanical compression devices that cause endothelial cells to release antithrombogenic factors and prevent stasis. It is important that these be placed and activated before surgery. Pharmacologic agents are generally reserved for known cases of thrombosis because of their propensity to cause bleeding in the postoperative setting. Low-molecular-weight heparin may be an option in this setting. If the patient has undergone microvascular reconstruction, aspirin or low-molecular-weight dextran may be indicated.

Fluid Management

Most patients undergoing surgery for oral cavity cancers can be managed without invasive monitoring of fluid status. Colloids may be needed to prevent undue amounts of crystalloid leading to a significant increase in edema. Preoperative and daily weights can be used to track fluid status. In patients with compromised cardiovascular reserve or in those undergoing large resections and free-flap reconstruction, invasive monitoring with central venous monitoring or via a Swan-Ganz catheter may be necessary. Although uncommonly performed, patients requiring bilateral resection of the internal jugular veins will need fluid restriction.

Transfusion

Controversy exists regarding the perceived impact of transfusion in the setting of head and neck cancer with some investigators demonstrating that transfusion is an independent predictor of poorer outcome.[63] Interestingly, the use of erythropoietin to correct anemia during radiation therapy has also been demonstrated to lower locoregional tumor control rates

and survival.[64] More work is needed to elucidate the interactions between anemia, transfusion, and survival. At this time, a hematocrit less than 25 generally requires transfusion and those between 25 and 30 may need transfusion based on clinical parameters.

Nutrition

Many patients with head and neck cancer will present with decreased nutritional reserves. Even patients without weight loss are often faced with therapies that will leave them unable to maintain their nutrition. The ability to bypass the upper digestive tract during intense multimodality therapy by the endoscopic placement of a gastric feeding tube (percutaneous endoscopic gastrostomy [PEG] tube) is invaluable. This procedure offers a minimally invasive "lifeline" for patients undergoing intensive therapy to the head and neck. Placement of a PEG tube has become commonplace in head and neck cancer patients despite anecdotal reports of seeding squamous cell carcinoma to the abdominal wall if the PEG is placed before resection.[65] This rare complication has led some surgeons to recommend PEG placement in the postoperative period. Even if a PEG is placed, the patient should be encouraged to continue some oral intake, because the risk of esophageal stenosis increases if the patient completely stops oral alimentation during radiation treatment. This is especially true during combined chemoradiation protocols.

Complications of Surgery

Complications of surgical resection are many and vary directly with the patient's co-morbidities, such as ischemic cardiac disease, chronic pulmonary disease, and alcoholism. Medical manifestations of preexisting chronic disease states, such as myocardial infarction, stroke, and pneumonia, however, can be precipitated by major surgery, a long general anesthetic, and a prolonged intensive care unit stay. Time under anesthesia has been shown to be an independent predictor of complications and length of stay.[58] Significant morbidity or death can be the result. Technical surgical complications, such as failure of reconstructive flaps, development of fistulas, and the other myriad problems that may require return to surgery for management, pale in significance to the greatest complication—locoregional recurrence of the cancer.

RADIATION

A complete review of radiation physics and medicine is beyond the scope of this chapter, and excellent reviews on the topic are available.[66] Surgeons dealing with oral cancer should have an understanding of radiation therapy and its advantages and disadvantages. This entails a familiarity with radiation biology and the interaction of radiation with living tissue as well as the biology of cell death. Cell death can be divided into two types: *reproductive cell death*, which results from damage to cellular genetic material, and *apoptosis*, which is programmed cell death. Reproductive cell death can occur as a result of single DNA strand breaks, which are common and easier for the cell to repair, or double-strand breaks, which are more difficult for the cell to recover from. Apoptosis occurs when a cell enters a programmed cell death mode as a result of damage. Radiation can cause either type of cell death and also slows cellular division. Classically, radiation is discussed in terms of the four Rs: *r*epair, *r*eoxygenation, *r*edistribution, and *r*egeneration.

Radiotherapy is primarily given by external beam using electromagnetic radiation or particulate components. X-rays and gamma rays represent photons. X-rays are produced by a manmade source and gamma rays are produced by radioactive decay, most commonly of cobalt 60. Particulate radiation using electrons plays an important role in head and neck cancer. Another form of particulate radiation is neutron radiotherapy, which may have a specific role in salivary gland malignancy.[67] Regardless of the source, radiation interacts with tissue to produce several types of damage to cells. The radiation particle-cell interaction may be either direct, or more commonly indirect, whereby impact with H_2O molecules creates secondary particles that interact with cellular DNA. The absorbed dosage unit of radiation energy is reported as a gray (Gy), which is 1 joule of absorbed dose per kilogram. Previously, the dosage unit was reported as a rad, which was defined as 100 ergs absorbed per gram. One gray is equal to 100 rad and 1 centigray (cGy) equals 1 rad (1 cGy = 1 rad).

In the early to mid-20th century, radiation was given as orthovoltage (125–500 KeV). Currently, radiation is delivered as megavoltage (>1 MeV). Megavoltage results in more radiation delivered to deeper tissues with less superficial (skin) damage. By comparison, a superficial radiograph unit (x-ray machine) delivers 30 to 125 KeV. Radiation therapy is typically given in daily doses of 200 cGy, except in altered fractionation schedules.

Fractionation refers to the schedule on which the radiation dose is administered. Standard radiotherapy is administered daily, 5 days a week, with weekends off. In an effort to maximize damage to the more rapidly dividing tumor cells while sparing normal tissues as much as possible, fractionation schedules have been altered. Although used primarily in clinical trials, clinicians should be familiar with the advantages and disadvantages of other fractionation schedules because it is likely that their use will become more widespread. *Accelerated fractionation* refers to an overall reduction in treatment time accomplished by giving two or more daily-dose fractions of close to conventional size each day. *Hyperfractionation* implies that the overall treatment time is conventional or slightly reduced, but an increase in total dose is achieved by giving two or more small-dose fractions on each treatment day. Each of these regimens is associated with varying degrees of early and late toxicities. For example, some clinicians believe that long-term effects such as osteoradionecrosis are increased with hyperfractionated schedules, especially when combined with concomitant

chemotherapy. This view is not universal, however, and as more experience is gained, questions regarding toxicity will be answered.[68,69]

Aside from changes in radiation schedules, other facets of radiation delivery technique have undergone recent changes. Radiation is delivered to a specific target area that is limited by shielding (defined as radiation portals or "ports") that is placed to protect areas that are not suspected of harboring tumor or that are less tolerant of radiation (i.e., the spinal cord). The radiation treatment plan is typically standardized for each subsite in the oral cavity. *Conformal radiation treatment* refers to more localized delivery of radiation to the suspect site. By linking CT images with the ability to manipulate the radiation beams, radiation therapists are able to more accurately focus the radiation dose on the tumor bed and avoid adjacent uninvolved areas that may be more susceptible to radiation damage (Figure 32-4). There is still concern that highly conformal treatment plans may result in increased recurrence rates because of the more limited field of radiation. Intensity-modulated radiotherapy (IMRT) is an example of a conformal treatment plan combined with varying radiation doses that is frequently used for head and neck patients to limit the collateral damage to surrounding areas.[70,71]

Brachytherapy or interstitial radiotherapy is administered by placing a radioactive source, typically radium (^{226}Ra) or iridium (^{192}Ir), directly into the tumor mass using needles or loop catheters. In this manner, radiation is delivered continuously. This does not allow the tumor cells to "repopulate" between fractions as in external beam therapy. Unfortunately, cells native to the area cannot recover either, which carries an increased risk of extensive radiation-induced fibrosis and osteoradionecrosis. This technique, however, allows a higher total dose of radiation to be given to a primary site than does external beam because the radiation is placed directly in the tumor mass. Brachytherapy has developed a reputation for creating chronic wounds and for increasing the risk of osteoradionecrosis when used adjacent to the mandible. Its current use is generally limited to treatment of some tongue or tongue base primaries, and it is usually combined with external beam radiation. Brachytherapy has also been advocated for treatment of close or positive margins afte surgical excision.[49] Brachytherapy patients may require a tracheotomy for airway control because of airway compromise from edema. Wound healing is also severely compromised. Some clinicians have recommended only limited biopsies in the treated area if recurrence is suspected because chronic nonhealing wounds can develop from the biopsy alone.[72,73]

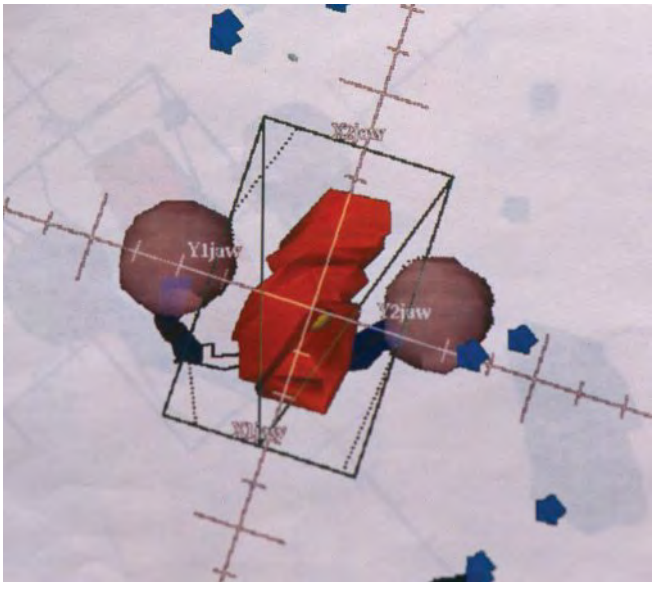


FIGURE 32-4. Three-dimensional conformal mapping of postoperative radiotherapy for esthesioneuroblastoma of the olfactory bulb. Globes and optic nerves are depicted to ensure minimal radiation damage to these structures.

Radiation can be administered with curative intent in the preoperative setting or as an attempt to shrink a tumor presurgically (neoadjunct). When the primary tumor is to be treated with radiation, the clinician must also consider elective radiation of the neck for control of occult metastases. Because of the dependence of radiation on oxygen for effectiveness, bulky neck disease with its attendant hypoxic core should probably be treated with neck dissection, either before radiation or as a planned procedure within approximately 4 weeks of the completion of radiation. Early-stage oral cavity cancer (T1 or T2) responds equally well to radiation or surgery. The morbidity of radiation and the inability to use it again in the case of a second primary cancer or recurrent disease make surgery a more attractive modality in most situations. Larger tumors (T3 and T4) generally respond poorly to radiation alone. Preoperative radiation given in an attempt to shrink larger tumors is hampered by the fact that tumors do not shrink concentrically. Viable islands of tumor cells can be left beyond the new, clinically evident margins. In theory, surgeons are committed to excising back to the original margins, something that seldom happens in clinical practice.

The primary role for radiation in oral cavity cancer is in the postoperative setting when there is potential for persistent disease. Clinical protocols vary among institutions, but there are accepted indications for postoperative radiation therapy:

- Two or more lymph nodes containing metastatic disease in a neck dissection. (Many clinicians contend that one positive node is an indication, which was supported by Shrime and coworkers[74] who demonstrated improved survival when postoperative radiation therapy was given to patients with T2 lesions and one lymph node involved.)
- Extracapsular extension (ECS) of cancer beyond the confines of a node.
- Poor histologic factors: extensive perineural or perivascular invasion, positive (close) soft tissue margins.
- Large (T3 or T4) primary cancers.

Reports have found ECS to be associated with decreased survival: disease limited to the node was associated with a 70% survival, whereas ECS was associated with a 27%

survival at 5 years.[75] Million and associates[76] found that 35% of patients with clinically negative necks converted to positive if the primary cancer was treated with surgery alone. This dropped to 5% if radiation therapy was added. Even microscopic evidence of ECS is associated with a higher rate of recurrence and death.[77] The decision to add radiation treatment must be made with a clear understanding of the morbidity of its use.

In advanced disease, clinicians are faced with a choice of preoperative or postoperative radiation treatment. Planned preoperative radiation treatment is rarely used but may lower the probability of positive margins and may allow smaller surgery (controversial). Lower doses of radiation are required because of the better oxygenation in areas not disturbed by surgery. Postoperative radiation treatment allows easier surgery and better healing in tissues not disturbed by radiation-induced fibrosis. Frozen-section analysis of margins is easier in this setting, and surgery allows improved treatment planning based on final pathology. Postoperative radiation therapy remains the mainstay in most cases of resectable cancers of the oral cavity. A study by the Radiation Therapy Oncology Group,[78] RTOG 73-03, compared 50-Gy preoperative radiotherapy with 60-Gy postoperative radiotherapy. The 10-year follow-up demonstrated no survival advantage to either regimen, but postoperative radiation treatment demonstrated superior locoregional control.[78] How much is enough? Results from an MD Anderson Cancer Center (University of Texas, Houston) study showed that 54 Gy was needed in the postoperative setting and 57.6 Gy was needed if ECS was present.[79]

Timing of the initiation of radiation therapy afte surgery is controversial. Vikram[80] demonstrated a clear survival advantage in patients whose radiation therapy was started within 6 weeks of surgery. For this reason, reconstructive options that led to reliable healing in this amount of time were advocated.[80] A more recent study failed to replicate Vikram's earlier findings, leading some to challenge the supposed impact of timing on ultimate outcome. Other studies have reported improved outcomes when postoperative radiation begins within 6 weeks and ends within 100 days of surgery for oral cavity squamous cancers.[81,82]

The future direction for radiation treatment may include the development of effective radioprotectants and radiosensitizers. Radioprotectants, such as amifostine, are given in an attempt to protect normal tissues. Amifostine was developed by the military as a possible protection from nuclear attack and has been applied to head and neck cancer patients to protect salivary gland function during radiation therapy.[83] Xerostomia is a long-term problem that has a significant effect on patients treated with radiation therapy to the head and neck, with 64% of patients reporting moderate to severe permanent xerostomia.[84] Decreased incidence of candidiasis, a frequent side effect observed in patients with radiation-induced xerostomia, has been used as an end point in amifostine therapy used for its protective effect on salivary gland function.[85] Its use is associated with side effects such as hypotension, and some patients do not tolerate it. In addition, it is costly and there remains some fear that its radioprotective effects might extend to the cancer cells as well, resulting in higher recurrence rates. Radiosensitizers are chemotherapeutic agents that enhance that effectiveness of radiation (see "Chemotherapy," later).

CHEMOTHERAPY

Until 1991, the role of chemotherapy in head and neck cancer was limited to its use in the management of recurrent and/or metastatic disease. A landmark study that changed our view of chemotherapy was from the Cooperative Studies Program of the Department of Veterans Affairs Laryngeal Cancer Study Group who reported a multi-institutional trial on patients with advanced laryngeal cancer.[86] Their study demonstrated larynx preservation and equivalent survival among patients who received induction chemotherapy followed by radiation, as opposed to traditional laryngectomy and postoperative radiation.[86] Although criticized by some for its lack of a radiation-only control group, the results fostered a renewed interest in the use of chemotherapy in the management of advanced head and neck malignancy, including squamous cell carcinoma of the oral cavity. Several reviews are available on the evolving role of chemotherapy in head and neck cancer. The following summarizes the basics of chemotherapy in oral cavity cancer and discusses several potential future applications.

Before analyzing the results of chemotherapy in oral cavity cancer, an understanding of the basic biology of chemotherapy and the associated terminology is necessary. In many ways, chemotherapy for cancer is conceptually similar to chemotherapy for infections; however, the immune system in general is not inherently competent to destroy the cancer. Chemotherapeutic agents kill a constant fraction of cancer cells, leaving behind a certain amount of resistant cells. These resistant cells subsequently divide and the tumor mass once again increases. In infectious diseases, the body's immune system aids in the destruction of the decreased burden of cells, whereas in cancer, the patient usually does not have an immune system that can deal with the rogue cell line. Similar to infections with resistant strains, multidrug protocols have been developed to counter the development of resistant cell lines in cancer. Principles of chemotherapy have been developed to overcome the development of resistant cell lines such as the use of multiple agents that have demonstrated independent activity against the cancer type, the combination of drugs with differing toxicities to allow maximum dosing of each agent, and the maintenance of short intervals between dosing agents while allowing adequate recovery of normal tissues. Solid tumor growth is governed by gompertzian kinetics, which means that growth slows as tumor bulk increases. Because chemotherapeutic agents are most effective against cells undergoing replication, smaller and faster-growing tumors are more susceptible.[87]

Assessment of the literature regarding chemotherapy is

complicated if one does not understand the definitions of complete response, partial response, stable disease, and progression. Each of these is determined by the sum of the product of the perpendicular diameters of all measurable tumors. Measurements are obtained at the beginning of treatment and at completion.

- Complete response: defined as the disappearance of all evidence of disease.
- Partial response: at least a 50% reduction in size as defined by the previous formula.
- Stable disease: less than a 50% reduction in tumor size.
- Progression: an increase of 25% or appearance of new lesions.

An important point to remember is that tumor regression must last for only 4 weeks. It is understandable, therefore, that reports of a complete response often have little impact on improved survival. The response rate represents the total percentage of patients achieving complete and partial responses. An additional problem with chemotherapy trials is patient selection bias. Increasingly, the role of co-morbidities in ultimate outcome and the impact of performance status on survival are being recognized as important contributors to survival in head and neck cancer (see later discussion). Performance status is typically reported using the Karnofsky performance status (PS), which rates patients on a scale of 0 (death) to 100 (normal, no evidence of disease), or the Eastern Cooperative Oncology Group scale, which rates patients on a scale of PS 0 (fully active) to PS 5 (death).[88,89] Most clinical trials require a certain PS to qualify, leading to enrollment of healthier patients and improved outcomes.

Timing of chemotherapy has been the subject of much investigation. Again, definitions are the key to understanding and interpretation of results of clinical trials. *Palliative chemotherapy* is given to patients with incurable disease to temporarily reduce tumor volume in the hope of improving quality of life and lengthening survival. This is typically the arena that serves as a testing ground for new therapeutic agents. *Adjuvant chemotherapy* is given to patients who have undergone treatment of their primary cancer site with surgery and/or radiation. Goals of treatment include elimination of occult disease, especially distant metastases. As the patient no longer has visible or palpable tumor with which to gauge response, agents must be selected that have proven activity against the cancer type. *Neoadjuvant chemotherapy* (also known as *induction chemotherapy*) is given to patients before definitive treatment of the primary cancer site.[87] This tactic is generally chosen in an attempt to decrease the size of the primary cancer to make definitive treatment possible. For example, a large tumor deemed unresectable may be "downstaged" by neoadjuvant chemotherapy that shrinks it to resectable proportions, although the actual stage itself does not change. As stated earlier, tumors do not shrink concentrically and islands of tumor may remain beyond the visible margin. An additional advantage to neoadjuvant therapy is the ability to evaluate response. Squamous cell carcinomas represent a heterozygous population even within the same tumor. Some will be exquisitely responsive to a particular regimen, whereas others will not. Medical oncologists can tailor their treatment more accurately if visible or palpable tumor is available to evaluate response. The biggest criticism of neoadjuvant therapy is that it delays the definitive treatment of the primary cancer. Local failure is still the biggest cause of death in oral cavity cancer, and delaying treatment of the primary site increases the difficulty of obtaining control of the primary cancer. In addition, initial chemotherapy can theoretically select more hardy cell lines that are resistant to all therapy. Critics of the Department of Veterans Affairs Laryngeal Cancer Study Group larynx trial contend that neoadjuvant chemotherapy simply selected out less aggressive cancers that would respond to radiation treatment. Currently, the role of chemotherapy that has generated the most interest is combination with radiation treatment for an "organ-sparing" approach. Chemotherapy in combination with radiation treatment can be given in a sequential or a concurrent strategy. Concurrent therapy takes advantage of the radiosensitization of certain drugs and avoids delay in treating the primary cancer site. The downside is a marked increase in side effects and toxicity that can lead to breaks in radiation treatment, which have been shown to be associated with a decrease in local control. In an attempt to control some of these toxicities, chemotherapy is usually given at the beginning of radiation treatment and frequently at the completion of radiation. Sometimes, radiation therapy is interrupted (split-course radiation) on purpose, and chemotherapy is given. Again, radiation breaks are considered to be associated with decreased control and are, therefore, not recommended.

Chemotherapeutic agents are under constant development and a complete discussion of available agents is beyond the scope of this chapter. Several principles deserve mention. In general, drugs can be divided into cell cycle–specific and non–cell cycle–specific agents, depending on whether the particular agent requires that the target cell be in a certain phase (G_0, S, G_1, or mitosis) to be effective. Agents can also be categorized based on their principle mode of action. Antimetabolites, such as methotrexate and 5-fluorouracil (5-FU), block development of certain metabolites critical for cell metabolism. *5-FU* is a fluoridated pyrimidine analog that inhibits thymidylate synthetase, blocking the generation of thymidine, which is necessary for DNA synthesis. It is frequently used in the treatment of head and neck squamous cell carcinoma. Typically it is combined with other agents, and it is a radiosensitizer. *Methotrexate*, an analog of folic acid, blocks conversion of dihydrofolate to tetrahydrofolic acid, which is a precursor of thymidylic acid and purine. This results in an interruption of DNA, RNA, and protein synthesis. Once a standard for head and neck squamous cell carcinoma, methotrexate is now typically used only for palliation. Its side effect profile and ability to be administered intramuscularly on an outpatient basis make it a good option for this purpose. *Cisplatin* and *carboplatin* are alkylating agents that

form cross-links in DNA and arrest cell division. Cisplatin is more effective in squamous cell cancer but is associated with more renal and neurologic side effects than carboplatin. *Taxanes* are plant-derived and interfere with microtubule formation, which subsequently interferes with the movement of chromosomes during mitosis. Recently, the addition of taxanes, specifically docetaxel, to traditional platinum-based chemotherapy has shown some benefit.[90] *Erbitux (cetuximab)* is a monoclonal antibody targeting the epidermal growth factor receptor (EGFR). It inhibits ligand binding to the EGFR and can stimulate antibody-dependent cell-mediated toxicity. Side effects are minimal with the exception of a variable rash reaction, the intensity of which seems to correlate with the effectiveness of the drug.[91,92] Other agents used less frequently in head and neck squamous cell cancer include *paclitaxel*, which stabilizes microtubular formation and arrests cells in G_2, and *bleomycin*, which creates DNA breaks. Agents under development include *flavopiridol*, a cyclin-dependent kinase inhibitor that has been shown to induce apoptosis (programmed cell death) in squamous cell cancer lines in vitro and for which a phase 1 trial is under way.[93,94]

Standard therapy for resectable disease remains surgery followed by radiotherapy, if indicated. To date, induction chemotherapy followed by surgery has not shown a survival benefit in oral cavity cancer. Questions remain regarding the addition of chemotherapy in the postoperative setting. The results of two major phase III trials, the European Organization for Research and Treatment of Cancer (EORTC 22931) and Radiation Therapy Oncology Group (RTOG 9501), offer some guidance.[95–97] In each study, stage III/IV patients with high-risk features, such as extranodal extension, multiple positive lymph nodes, and positive surgical margins, were randomized to receive radiation alone or concurrent radiation and platinum-based chemotherpy. Both demonstrated improved locoregional control in the combined group, but only the European trial demonstrated a survival advantage. It should also be recalled that both studies included mutliple subsites of the head and neck and only a portion of the patients represented oral cavity cancers. At this point, chemotherapy in the postoperative setting should probably be reserved for cases of of extracapsular extension and/or positive resection margins in patients whose performance status is such that it is believed they will tolerate the intensive therapy.[95–97]

In patients with unresectable disease, chemotherapy is combined with radiation treatment. Organ preservation (not to be confused with organ function) through the use of concurrent chemoradiation protocols has received much attention. Meta-analyses by El-Sayed and Nelson[98] as well as by Munro[99] have demonstrated that concurrent treatment is better than neoadjuvant therapy and that locoregional control and survival were improved in advanced head and neck cancers. Stenson and collegues[100] reported on the effectiveness of an intensive course of systemic chemotherapy and concurrent radiation for advanced-stage oral cavity cancer. They compared this group with a subset of patients that received surgery followed by chemoradiation therapy and found comparable progression-free and overall survival rates of 65.9% and 66.9%, respectively. Overall function in the chemoradiation group was judged excellent. It should be noted that the risk of osteoradionecrosis in the chemoradiation group was significant at 18%, and eight patients (7.2%) died directly as a result of treatment toxicity. The study, however, did demonstrate an alternative treatment option for advanced oral squamous cell cancers, and we will probably continue to see an increasing role for primary systemic chemotherapy in the management of patients presenting with advanced disease.[100]

In an attempt to avoid the systemic effects of chemotherapy, investigators have attempted to deliver agents topically as well as intratumorally with both intra-arterial injections and intratumoral depot forms via polymers and gels (see later discussion on recurrent tumors). A novel form of concurrent chemoradiation is the intra-arterial cisplatin and radiotherapy (RADPLAT) protocol popularized by researchers at the University of California, San Diego, and University of Tennessee at Memphis, which has shown promise for advanced cancers with bulky primary cancers and nodal disease.[101] Treatment involves supradose cisplatin delivered directly into feeder vessels of the tumor bed by microarterial catheters placed under angiography. Sodium thiosulfate, which is a neutralizing agent for cisplatin, is administered systemically, allowing doses five times larger than standard protocols. Results of patients with T4 N2–3 disease treated with the protocol revealed 4-year local control of 84%, disease-specific survival of 46%, and overall survival of 29%.[101] Unfortunately, the protocol is associated with significant toxicity, including death. Use of the RADPLAT protocol is currently limited to centers that have gained familiarity with the technique and management of the toxicities associated with it. Most of these concurrent chemoradiation protocols involved oropharyngeal and hypopharyngeal cancers and are plagued by noncompliance because of toxicity and side effects. Mucositis is intense and placement of a PEG tube is usually mandatory.[101]

Other novel techniques for minimizing the systemic side effects of chemotherapeutic regimens are under development, including the PDT discussed previously under the management of leukoplakia.

Trials of chemotherapy limited to the oral cavity are few. At this time, the clearest indication for chemotherapy in oral cancer is in metastatic and recurrent disease. Historically, the most commonly used chemotherapeutic regimen for metastatic or recurrent oral cavity squamous cell carcinoma involved combinations of cisplatin or carboplatin and 5-FU. Median survival rates of 5 to 7 months and 1-year survival of 20% were obtained. Recently, Erbitux (cetuximab) in combination with cisplatin and 5-FU has been shown to offer an advantage over platinum-based therapy alone for recurrent or metastatic squamous cell cancer of the head and neck. This is a remarkable advance considering that for the past 25 years, no other regimen has shown an advantage over plati-

num-based therapy. In addition, this was without a significant increase in toxicity and led to improved overall survival.[102] One must be cognizant of the fact that these are not curative therapies, and education of patients and families is critical to establish realistic goals and end points of therapy. Investigations continue to define a role for chemotherapy in advanced squamous cell carcinomas of the oral cavity. Unfortunately, early responses to chemotherapy have not demonstrated improvement in overall survival and only modest gains in median survival time.[103]

Current research in chemotherapeutic agents focuses on those agents that bind to specific receptors in an attempt to limit effects to target cells. Similar to the hormonal therapy used in breast and prostate cancers, investigators are experimenting with agents such as EGFR inhibitors discussed earlier.[104] Gene therapy that targets known alterations in head and neck squamous cell cancer lines, such as *TP53*, is also an area of growing research.[105] Restoration of these altered genes, possibly through viral vectors, holds promise in certain populations.[106]

CHEMOPREVENTION

An additional area of intensive research is the evaluation of *chemoprevention agents*, which are defined as agents that reverse or suppress premalignant carcinogenic progression to invasive malignancy (see "Management of Premalignant Lesions," earlier). The role of such agents would be twofold: (1) to treat premalignant lesions to prevent their evolution to invasive carcinoma, and (2) to prevent development of second primary squamous cell cancers in patients who have already undergone treatment of cancer. Given its accessibility to clinical observation, leukoplakia has been used to monitor responsiveness to certain chemoprevention agents in clinical trials. Of the agents evaluated, including retinoids, beta carotene, and vitamin E derivatives, retinoids have demonstrated the most efficacy in eliminating leukoplakia. It is important to note, however, that reversal of these lesions has not been demonstrated to reduce the risk of developing cancer, and the lesions soon return after cessation of treatment. 13-*Cis*-retinoic acid, which is more commonly used to treat acne, has been studied extensively in both the treatment of premalignant lesions and the prevention of second primary cancers. It may act through the up-regulation of a distinct retinoic acid receptor, RAR-fl, whose down-regulation is associated with development of head and neck cancer. Results of trials to date have been mixed. Although effective in eliminating leukoplakia, side effects limit its use, and lesions return after discontinuation of the drug. Second primary tumors occur in 4% to 7% of patients treated for head and neck squamous cancer and are the major cancer-related cause of death in early-stage cancer patients. The prevention of these tumors is, therefore, of critical importance. Studies of 13-*cis*-retinoic acid have shown a decreased incidence of second primary cancer but no effect on primary disease recurrence. This suggests that retinoids may prevent cancerous development in damaged cells but will not treat fully transformed cancer cells. Also, overall survival was not affected. The required doses of retinoids have side effects, including mucocutaneous toxicity (peeling and cheilitis) and elevation of liver function tests. Development of second-generation retinoids may attenuate some of these side effects. One study demonstrated a worrisome increased incidence of primary lung cancer in patients treated with beta carotene.[107,108]

Investigators continue to search for chemotherapeutic agents with more acceptable side effect profiles. One of these agents is the Bowman-Birk inhibitor, a protein derived from soybeans that has shown clinical activity against leukoplakia without the attendant side effects of retinoids.[109] Nonsteroidal anti-inflammatory drugs have been investigated since chemoprevention activity was found in some cyclooygenase-2 (COX-2) inhibitors. COX-2 influences several steps in the development of malignancies, such as apoptosis, angiogenesis, invasiveness, and immune surveillance.[110,111] COX-2 expression has been noted in high-risk premalignant lesions.[112] In addition to their potential role in chemoprevention, COX-2 inhibitors hold promise in the treatment of invasive squamous cell carcinomas.[113] Although chemoprevention offers hope for patients at high risk for development of second primary cancers and treatment of patients with high-risk lesions (see "Management of Premalignant Lesions," earlier), its use is currently restricted to clinical trials and off-label use. Further work is needed to establish a safe and effective chemopreventive regimen.

SPECIAL TREATMENT CONSIDERATIONS BY SITE

Lip

Although classified as an oral cancer, squamous cell carcinomas of the lip typically follow a different clinical course than those of oral mucosal cancers. The primary etiologic agent, sun exposure, is different from oral cancers, and the location of lip cancers usually leads to earlier discovery. The behavior of squamous cell cancers of the vermilion border is usually intermediate between squamous cell carcinoma of the skin and that of the mucosa. The vast majority arise on the lower lip where sun exposure is greatest. Most lip cancers are treated by surgical resection using 0.5- to 1.0-cm margins and frozen-section control. Although often referred to as a "wedge" resection, the actual specimen more closely resembles a shield with parallel sides and a tapering base. "Wedge" excisions may be combined with a vermilionectomy or "lip shave" procedure, removing vermilion that has suffered extensive actinic damage or contains carcinoma in situ (Figure 32-5). CO_2 laser ablation of the surface of the lip is also useful as an alternative to vermilionectomy for diffuse actinic changes. Squamous cell carcinoma of the lip shares with squamous cell carcinoma of other cutaneous sites a potential for perineural invasion. A large perineural tumor deposition

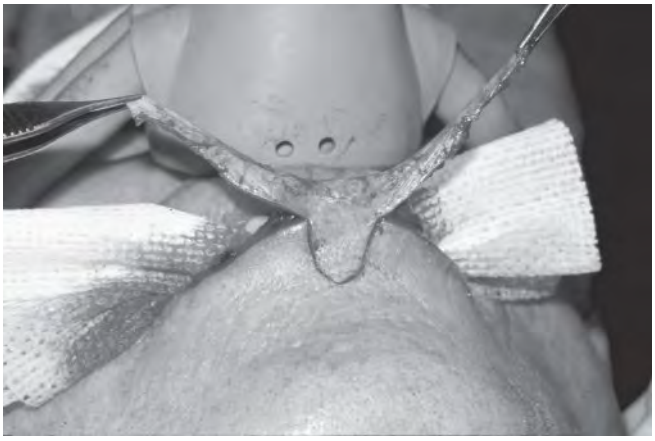


FIGURE 32-5. Vermilionectomy combined with wedge resection of lip cancer associated with diffuse actinic changes across the remainder of the lip.

along the inferior alveolar nerve, several years after a lip cancer, can be mistaken for primary intraosseous carcinoma (Figure 32-6).

Neck dissection is usually not indicated for lip cancer unless there is clinical evidence of lymph node involvement by examination or imaging. Cancers of the upper lip and commissure can metastasize to the periparotid lymph nodes, and superficial parotidectomy may be required if there are clinically enlarged nodes. Some larger lesions can be treated with radiation alone if surgical resection will result in unacceptable compromise in appearance and function.

Five-year survival for lip cancer is good for early-stage disease (90% for stages I and II).[114,115]

Buccal Mucosa

Buccal squamous cell carcinomas represent approximately 10% of oral cavity cancers in the United States compared with 41% in India. Squamous cell carcinomas of the buccal mucosa can be deceptive in their clinical course. Because of the intimacy to the buccal space and deeper structures,

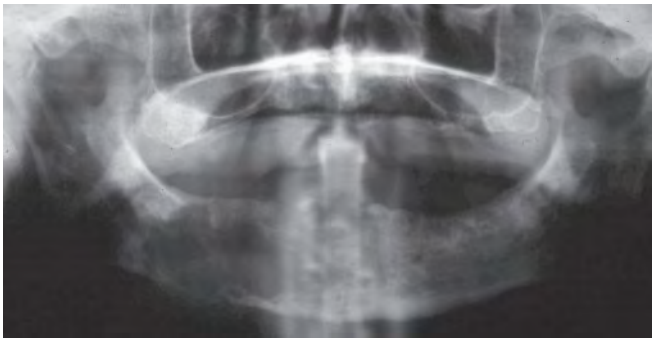


FIGURE 32-6. Apparent intraosseous squamous cell carcinoma arising from perineural spread of a previous lip carcinoma. Review of the original lip resection specimen demonstrated perineural invasion.

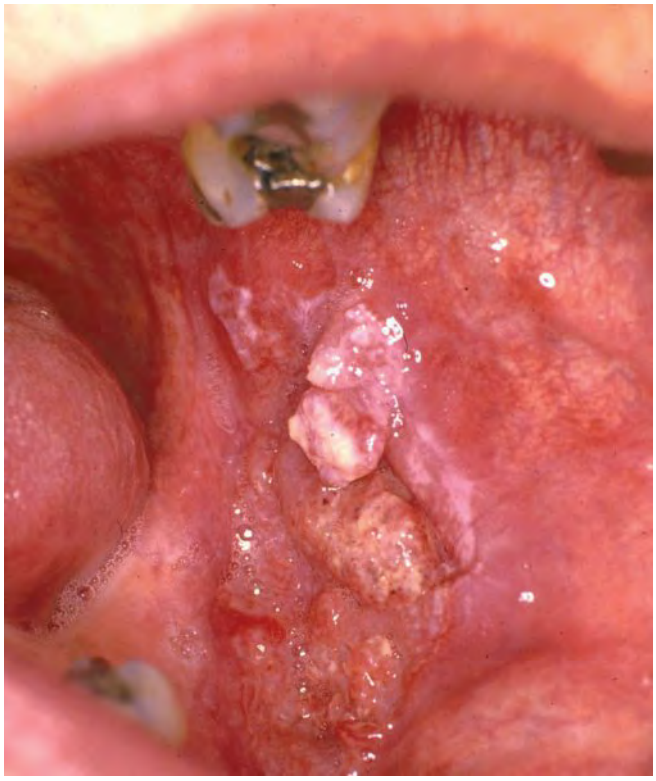


FIGURE 32-7. Squamous cell carcinoma of the buccal mucosa, deeply invading through the buccinator muscle.

cancers that penetrate the buccinator muscle can be difficult to eradicate (Figure 32-7). Patients may present with involvement of the pterygoid space posteriorly or the parotid gland laterally. Extension either superiorly or inferiorly can lead to invasion of the maxillary alveolus or mandibular alveolus respectively. These cancers often arise in wide areas of damaged mucosa, and adequate excision of these lesions often results in complex defects of the cheek that can be difficult to reconstruct. Primary radiation may be an option for smaller lesions. Although up to 50% of patients with buccal squamous cell carcinoma can present with neck metastases, the rate of occult disease in the neck is approximately 10%.[116] As with other oral cancer sites, elective treatment of the neck with radiation or surgery is indicated in T3 or T4 lesions. Consideration should also be given to elective treatment of the neck in deep T1 (>4 mm) and larger T2 lesions. Combined therapy for large lesions with surgery and radiation may offer the the best chance for cure.[117]

Two-year overall survival rates for early-stage disease treated with a variety of treatment modalities range from 83% to 100%. Stage III survival rate is 41% and stage IV is 15%. Available survival statistics, however, are often not accurate because of the inclusion of verrucous carcinoma in some of the published reports.

Diaz and colleagues at the MD Anderson Cancer Center reported on 119 consecutive patients with buccal squamous cell carcinomas, the majority of which were treated with surgery followed by radiation if indicated (positive margins,

nodal involvement).[118] Five-year survival rates for patients with stages I, II, III, and IV disease were 78%, 66%, 62%, and 50%, respectively. The significant impact of nodal involvement was noted. Five-year survival rates of 69% with nodal involvement have been reported, which decreased to 24% in cases with extracapsular extension.[118]

Retromolar Trigone

Given their proximity to the pterygomandibular space, tonsillar pillars, mandible, and tongue base, squamous cell carcinomas of the retromolar trigone (RMT) can behave in a more aggressive fashion, more like an oropharyngeal primary cancer (Figure 32-8). Smaller lesions are amenable to wide local excision with or without a marginal mandibulectomy depending on the proximity to the bone (see later discussion). Larger lesions may invade the pterygomandibular space and extend cephalad toward the skull base. Such tumors require segmental composite resections with neck dissection. Significant trismus can be an indication of pterygoid involvement and may make radiation treatment with or without concomitant chemotherapy a better option than surgery. Elective neck radiotherapy or elective neck dissection with a selective neck dissection should be considered in T2 or greater lesions. As with other sites, a depth of invasion greater than 4 mm in T1 lesions may be an indication for elective treatment of the neck.

Kowalski and colleagues[119] reported on 114 RMT cases treated with surgery with or without radiation and found 5-year survivals of 80% (T1), 57.8% (T2), 46.5% (T3), and 65.3% (T4). Overall 5-year survival was 55.3%. They recommended adjunctive radiation in stages III and IV.[119] In an excellent review of the management of RMT cancers, Genden and coworkers[120] also suggested that the addition of preoperative or postoperative radiation confers a survival advantage. Radiation therapy alone for RMT lesions is an acceptable alternative with local control and survival rates similar to those surgery, provided there is no bone involvement.[121] Surgical salvage may be indicated, however, in cases of incomplete response, and this must be explained to the patient who is unable or unwilling to undergo surgery as the primary treatment.

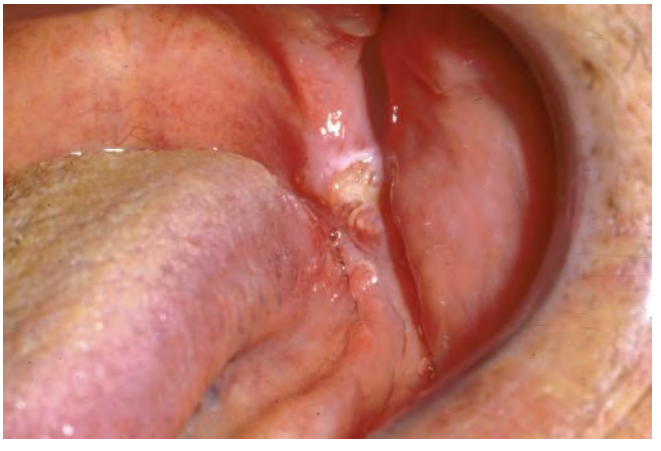


FIGURE 32-8. Squamous cell carcinoma of the retromolar trigone.

Tongue

The oral tongue, that portion anterior to the circumvallate papillae, is the most common location for intraoral squamous cell carcinoma, which typically presents as a painless indurated ulceration. If pain is present, it is usually due to secondary infection. Clinicians should attempt to determine the epicenter of the tumor and to classify it correctly because the behavior and treatment of cancers of the oral tongue is sufficiently different from those of the posterior tongue (oropharyngeal tongue or tongue base). This may be difficult in the case of larger cancers. The oral tongue poses significant challenges to clinicians. Seemingly small lesions can metastasize early and can recur after treatment. Control rates for small lesions of the oral tongue (60–80%) are lower than those of similar size in other oral cavity subsites. There are minimal barriers in the tongue to tumor invasion, and there is frequent invasion into adjacent or deeper structures at presentation. Although more commonly associated with oropharyngeal primary cancers, referred otalgia is not uncommon for cancers of the oral tongue, and limitation of tongue mobility with resultant dysarthria is associated with invasion of the deeper musculature. Magnetic resonance imaging (MRI) can be useful in evaluating the depth of invasion (Figure 32-9).

Treatment of tongue lesions should be aggressive, and strong consideration for elective treatment of the neck should be given in all cases except for the most superficial lesions (<2 mm). Although Fakih and associates[122] were not able to demonstrate a survival advantage in patients who underwent an elective neck dissection versus a watch-and-wait policy, they did demonstrate that deeper lesions were associated



FIGURE 32-9. Enhanced, T_2-weighted magnetic resonance imaging of a tongue shows right tongue tumor deeply invading tongue musculature.



FIGURE 32-10. Radial forearm flap used to reconstruct large tongue defect with nearly normal speech and swallowing.

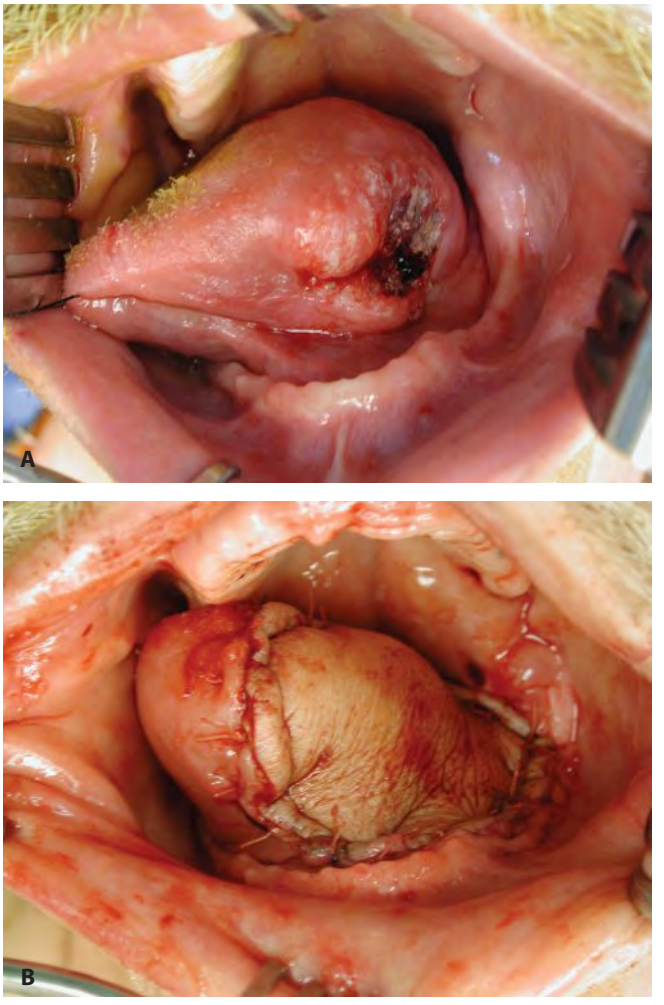


FIGURE 32-11. **A,** Large deeply infiltrative squamous cell carcinoma of the lateral tongue. **B,** Radial forearm flap tailored to the size of the resection, not the apparent size of the defect.

with a significant rate of cervical metastases. Up to 10% to 12% of tongue cancers with metastases to the neck can demonstrate "skip" metastases to level IV, and consideration should be given to extending the neck dissection to include level IV.[123] Postoperative radiation should be considered in situations in which multiple frozen-section specimens were sent before obtaining clear margins, perineural invasion, microvascular or microlymphatic invasion, or other worrisome findings that are present.

Smaller superficial tumors are amenable to wide local excision and reconstruction via primary closure, split-thickness skin grafting, or healing by secondary intention. Larger tumors are reconstructed with vascularized tissue transfer, and mandibulotomy may be needed for adequate access to large or posterior lesions. Radial forearm or lateral arm microvascular free flaps allow excellent mobility and little bulk (Figure 32-10), whereas the anterolateral thigh flap can provide bulk, if needed. Unilateral or bilateral pedicled nasolabial flaps can occasionally be used for anterior tongue lesions. Large oral tongue cancers that cross the midline present the surgeon with a difficult choice. If resection is chosen, it is often better to tailor the radial forearm flap smaller than the resected area to allow the remaining tongue musculature less bulk to move during excursions (Figure 32-11). Near-total glossectomy (resection of the oral tongue with only tongue base remaining) almost always results in high morbidity and is associated with a high incidence of aspiration that may ultimately require laryngectomy for aspiration control. Treatment with external beam radiation alone is associated with unacceptable failure rates. In this setting, consideration should be given to organ-sparing protocols with concomitant chemoradiation therapy if surgery will be associated with unacceptable morbidity. Brachytherapy combined with external beam radiation is the treatment of choice at some centers. Technical expertise is required, and most tongue brachytherapy patients require tracheotomy for airway control.

Tongue base tumors, although actually an oropharyngeal subsite and not considered an oral subsite, are discussed here for completeness. The tongue base allows tumors to grow silently, and diagnosis at an early stage is the exception rather than the rule. Most small (T1 and some T2) lesions can be treated with surgery or radiation treatment. Larger tumors are typically treated with an organ-sparing approach using chemoradiation therapy or external beam therapy, sometimes combined with brachytherapy. Results with external beam alone have been disappointing. Reconstruction of smaller posterior tongue defects is best accomplished by radial forearm or lateral arm flaps, if primary closure or healing by secondary intention is inappropriate. Larger excisions (75% to total) typically are reconstructed with a free rectus flap, although the anterior lateral thigh flap offers an alternative.[124] A new surgical modality has been developed and has recently been approved by the U.S. Food and Drug Adminstration (FDA) that entails the use of a surgical robot to access and effectively resect cancers from the base of tongue and other oro- and hypoharyngeal sites.[125] Transoral robotic surgery (TORS) may allow tumor resection with clean margins resulting in a defect that can be allowed to heal by secondary

intention. This obviates open mandibulotomy or pharyngotomy and can usually be done without a tracheotomy.

Most treatment failures of the oral tongue involve locoregional recurrence. Second primary cancer rates are high (30%), and this also contributes to treatment failure and death. Three-year survival for T1 and T2 lesions is 70% to 80%, but this decreases to 15% to 30% in patients with lymph node metastases.[126,127] Some clinicians have opined that tongue squamous cell carcinoma arising in young patients may represent a more aggressive subset and warrant more aggressive therapy. Overall survival for younger patients is actually similar to those of older patients with the same stage because of their lack of intercurrent illnesses. It was found, however, that oral tongue cancers in younger women did behave more aggressively and were associated with higher recurrence rates. This subset may warrant more aggressive initial therapy.[128,129]

Floor of Mouth

The floor of the mouth (FOM) is the second most common location for oral cavity squamous cell cancers (Figure 32-12). FOM cancers can extend along the ventral tongue and cause fixation. In addition, FOM tumors can become fixed to the mandible or extend directly into level I of the neck. McGuirt and colleagues[130] have demonstrated improved outcome with elective treatment of the neck, and elective treatment of the neck should be considered in all but the smallest thin lesions (<3 mm). Sagittal, or "inner-table" mandibulectomy may be considered in smaller tumors that abut the mandible without evidence of invasion (see later discussion), but large inner-table resection is technically difficult and can significantly weaken the mandible. Small primary cancers can be equally treated with surgery or radiation, although surgery is the choice of most clinicians. Anterior lesions may require sialodochoplasty to reroute the submandibular ducts if the submandibular gland is not removed by ipsilateral neck dissection.

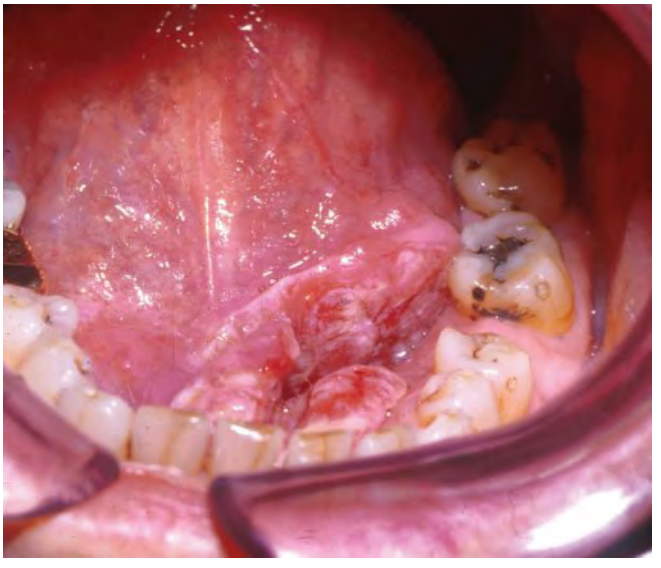


FIGURE 32-12. Advanced squamous cell carcinoma of the anterior floor of the mouth.

Smaller FOM defects can be closed primarily or left to heal by secondary intention. A partial closure of the defect will often suffice. Larger FOM defects, particularly those that include mylohyoid resection, benefit from a bulkier reconstruction, such as a vascularized radial forearm flap or bilateral nasolabial flaps placed in a one- or two-stage operation.

Local recurrence remains a problem with FOM squamous cancers and results in high rates of locoregional failure. Five-year survival rates are 64% to 80% (stage I), 61% to 84% (stage II), 28% to 68% (stage III), and 6% to 36% (stage IV).[131]

Alveolus and Gingiva

Gingival squamous cell cancers represent a unique subset in oral cavity cancers that arise on the attached gingiva and that should be differentiated from those that arise on the unattached mucosa of the alveolus. Occult neck metastasis is rare in the absence of bone invasion, and elective treatment of the neck is not necessary in smaller lesions. Larger lesions may require partial maxillectomy, or marginal or segmental mandibulectomy, if bone invasion is suspected. Indications and variations on mandibulectomy are discussed later. In general, control rates are excellent for gingival primary cancers if treated with adequate margins.

Alveolar cancer arises from the unattached mucosa of the alveolar ridge and has a different clinical behavior than gingival carcinomas. It requires more aggressive therapy and more extensive resection of bone.

Most anterior and some posterior maxillectomies for alveolar and gingival squamous cells can be accomplished via a transoral approach using techniques of orthognathic surgery (Figure 32-13).[132] The pterygoid plates require removal only if there is evidence of invasion through the posterior maxilla. Larger tumors may indicate the need for a transfacial (Weber-Fergusson) approach for adequate exposure. Posteriorly situated tumors and especially those with extension into the pterygoid or infratemporal areas may be more easily accessed via a midline mandibulotomy and mandibular swing approach. Reconstruction of maxillary defects can be accomplished with local flaps, such as the temporoparietal galeal or temporalis muscle flap, or free-flap reconstruction. The complex nature of maxillary defects and the bulk of some of the flaps, however, often leave a less than satisfactory result. Whereas most maxillectomy defects are best reconstructed primarily, prosthetic obturation of the defect may offer advantages in some situations, including a simpler operation, easier early detection of recurrence, and replacement of teeth. Dental implants in general, and the zygomatic implant in particular, can offer a vast improvement in prosthetic function.[133]

Most maxillary alveolus and gingival squamous cells that invade the sinus involve the infrastructure lying below

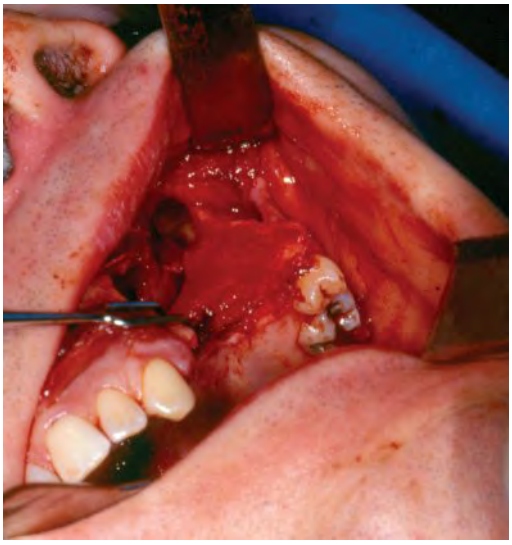


FIGURE 32-13. Le Fort I island approach to a posterior maxillary tumor, without Weber-Fergusson incision.

Ohngren's line, an artificial line that runs from the medial canthus to the angle of the mandible and separates the maxillary sinus into an infra- and a suprastructure. If superior extension results in exposure of dura mater as part of a skull base resection, flap coverage is advisable.

Overall 5-year survival for alveolar ridge carcinoma is 50% to 65% for both the upper and the lower alveolar ridge. Poor outcome is associated with advanced stage, perineural spread, and positive margins. In these cases, as well as nodal involvement, adjunctive radiation is recommended.[134]

Palate

Squamous cell cancers of the palate, hard and soft, are rare in the United States, but are more common in India owing to the phenomenon of "reverse smoking," which assaults the palate with both carcinogens and heat. The soft palate is considered an oropharyngeal subsite (Figure 32-14). Cancers arising from the hard palate, however, may extend onto the soft palate and vice versa. The periosteum of the palate acts as a significant barrier, and smaller lesions can be treated with wide local excision. Healing by secondary intention under a protective stent secured to the palate is a viable reconstructive option if the palatal bony structure is not removed. The patient should be informed that despite preservation of palatal bone, a small fistula may still develop during the healing process. Oral-nasal communications in the hard palate can be treated with an obturator or a local flap, such as an anteriorly based midline tongue flap. Oral-nasal fistulas in the soft palate are best treated with temporary obturation, because the majority will close spontaneously. Previously, it was thought that occult cervical metastases are rare among maxillary and hard palate cancers (10–25%), and elective treatment of the neck was generally not indicated except in T3 or T4 lesions.[135,136] A recent report by Schmidt and coworkers[133] found a higher incidence of occult metastasis (21.4%) with cancers involving the palate and maxilla and a high incidence of regional or distant metastasis (50%). This and other reports call into question the wisdom of observation alone for the clinically negative neck in maxillary and hard palate cancers.[137,138] In addition, metastases may occur to the retropharyngeal nodes, and consideration should be given to irradiation of the neck to include this area if suspected.



FIGURE 32-14. Squamous cell carcinoma of the right soft palate invading posteriorly to involve the anterior pole of the tonsil.

MANAGEMENT OF THE MANDIBLE IN ORAL CAVITY CANCER

Management of the mandible in oral cavity cancer continues to be the subject of much controversy. In the past, the mandible was routinely sacrificed in the treatment of FOM and tongue cancers, as it was believed that the regional lymphatics coursed through the mandibular periosteum, necessitating an in-continuity resection of the tongue, FOM, mandible, and neck dissection (commando operation). The morbidity of this approach was thought necessary to eradicate in-transit metastases, a belief that was likely based on mistranslation by McGregor[139] of an article published by Polya and von Navratil in 1902, in which they actually recommended removal of the periosteum or rim of the mandible and not a segment.

Marchetta and associates[140] subsequently demonstrated that lymphatics did not flow through the mandible and that the periosteum of the mandible actually served as a barrier to invasion. It was found that squamous cell carcinoma invasion occurs most commonly through the periodontal ligament in the dentate mandible and through the porous occlusal surface of the edentulous mandible.[140] O'Brien and colleagues[141] also demonstrated that an inflammatory front preceded cancer that stimulated subperiosteal resorption and the creation of bony clefts that allowed cancer to invade the cortex. Once the cortex was invaded, the inferior alveolar canal was usually involved, especially in edentulous mandibles.[141,142] These findings have prompted surgeons to advocate preservation of mandibular continuity through the use of a marginal mandibulectomy in cases without obvious bony involvement. It is

important to note that these principles apply only to the nonirradiated mandible because cancer invasion of a previously irradiated mandible may occur through multiple sites.

The clinical and radiographic evaluations of mandibular involvement are frequently inaccurate. Clinical findings such as impairment of inferior alveolar nerve function or fixation of the tumor to the mandible raise the index of suspicion. The history of an extraction of a tooth in an area of a cancer may suggest local mandibular invasion. Although used by some, bone scans are cumbersome to obtain and difficult to interpret accurately. A high-quality panoramic radiograph is probably the most commonly used tool to decide on mandibular marginal resection versus segmental resection. CT scanning using DentaScan software has also been used, although recent studies have shown that an MRI demonstrating enhancement of the marrow signal was a better predictor than CT scanning of mandibular involvement. Also, newer techniques that modulate the magnetic field in an attempt to examine changes in the bone marrow hold promise for evaluating mandibular involvement. Tumor invasion into the marrow space is accompanied by a lower intermediate signal. A strong bright marrow signal associated with normal marrow fat underlying the dark cortex typically excludes mandibular involvement.[143] The specific role of cone beam CT scanning in mandibular invasion assesment awaits clarification, although this modality probably offers the best-quality imaging of the mandible available in the office setting.[144]

At this time, the most accurate assessment of mandibular involvement occurs at the time of surgery when the surgeon can inspect the bone. Some surgeons send periosteum for frozen-section analysis, whereas others submit cancellous scrapings for frozen analysis.[145] Once a decision has been made to perform a marginal mandibulectomy, the surgeon has several choices for osteotomy design. Some surgeons advocate rim mandibulectomy, preserving at least a 1-cm inferior border, whereas others advocate a sagittal marginal mandibulectomy or a variation thereof.[146] Reinforcement of an aggressive marginal mandibulectomy with a reconstruction plate may be helpful to avoid later pathologic fracture (Figure 32-15). Marginal mandibulectomy can be effectively performed via a transbuccal approach.[147]

An important point is the avoidance of right angles in the osteotomy design that serve as stress risers and may lead to fracture. We prefer an osteotomy that begins in the sigmoid notch and sweeps inferiorly and anteriorly for lesions located in the posterior mandible (Figure 32-16). It is important to note that the thick cortical bone along the posterior ramus is rarely involved, even when the mandible is invaded, and it can usually be preserved and serves as an area for plating. Extending the osteotomy through the sigmoid notch also removes the coronoid process, which also decreases stress on the residual mandible. Atrophic edentulous mandibles are generally not candidates for marginal resection, although this is not an absolute rule. Wax and coworkers[148] and Shah[149] have published excellent reviews of the topic of segmental and marginal resection, and the reader is directed to their reviews for a more in-depth discussion. If the surgeon is anticipating a marginal resection, and a segmental resection becomes indicated based on operative findings, he or she is faced with a surgery for which both surgeon and patient might not be prepared. Frank discussions before surgery help prepare the patient and their family for this eventuality. Mention should be made of the possibility of return to the operating room if the final pathologic analysis reveals an unexpected amount of bone involvement, necessitating a segmental resection and more elaborate reconstruction.



FIGURE 32-15. Panorex demonstrates plate reinforcement of residual mandible after marginal mandibulectomy.

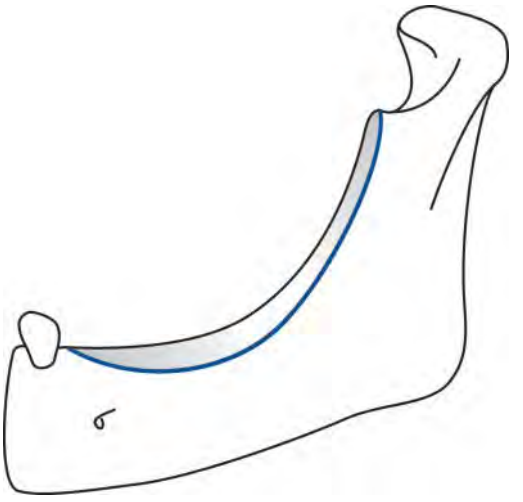


FIGURE 32-16. Outline of marginal mandibulectomy suitable for resection of tumors of the retromolar trigone and posterior maxillary alveolus.

Splitting the mandible, or mandibulotomy, is often necessary for access to large cancers, especially of the posterior tongue (Figure 32-17). Technical refinement of mandibulotomy help avoid complications. Mucosal and gingival incisions should be staggered and not placed directly overlying the proposed osteotomy site. Division of the mandible at the parasymphysis or symphysis is preferred over an osteotomy in the body region. If the mandibular osteotomy is being performed for access to the tongue, the cut should be made anterior to the mental foramen to preserve sensation. Precise preadaptation of plates will allow reestablishment of the preoperative occlusion and contour without maxillomandibular fixation.



FIGURE 32-17. **A,** Outline of incision for lip-splitting approach to the posterior oral cavity and oropharynx. **B,** Midline division of the tongue after midline mandibulotomy.

MANAGEMENT OF THE CERVICAL LYMPH NODES IN ORAL CAVITY SQUAMOUS CANCER

The ability of a cancer to metastasize most commonly manifests itself by tumor deposition and growth in regional lymph nodes. Therefore, management of the regional lymphatics is a fundamental consideration in any cancer. Lymph node status is the single most important prognostic factor in head and neck cancer with nodal involvement decreasing overall survival by approximately 50%. Unfortunately, approximately 40% of patients with oral cancer will harbor cervical lymph node metastasis at presentation. In addition to the presence or absence of lymph node involvement, prognostic features related to the regional lymph nodes include: laterality (whether the metastasis is ipsilateral or contralateral to the primary), number of involved nodes, size of nodes, anatomic level (involvement of lower neck nodes fairing much worse), ECS, permeation of perinodal lymphatics, and overall pathologic N stage.[151]

Surgical treatment of the neck is justified for two reasons: the removal of gross disease in patients with clinical evidence of nodal involvement *(therapeutic neck dissection)* or a high enough index of suspicion of occult cervical metastases to justify an *elective neck dissection (END).* Some surgeons would also suggest that unreliable patients should undergo END because follow-up may be irregular. Excluding the hard palate and lip, approximately 30% to 40% of patients with oral cavity cancer will present with cervical metastases.[152] The decision to treat the N0 neck is based on the probability of nodal involvement, with most authors recommending END if the risk of occult metastasis is greater than 15% to 20%. Evaluation of the neck for cervical metastases remains a critical component of the evaluation of the patient with oral cavity cancer. Manual palpation is regarded as the first step in this process and is usually accomplished before biopsy to avoid postbiopsy inflammatory nodal enlargement. In most necks, a lymph node must be at least 1 cm in diameter to be palpable. The accuracy and reliability of palpation are low, however, with an overall error of approximately 30% in several studies.[153] Imaging modalities including CT, MRI, ultrasonography, and positron-emission tomography (PET) have become increasingly important in the evaluation for cervical metastases and in guiding therapy.

A CT scan with contrast from the skull base to clavicles has become the most common imaging modality used for detection of cervical metastases (Figure 32-18). Specific

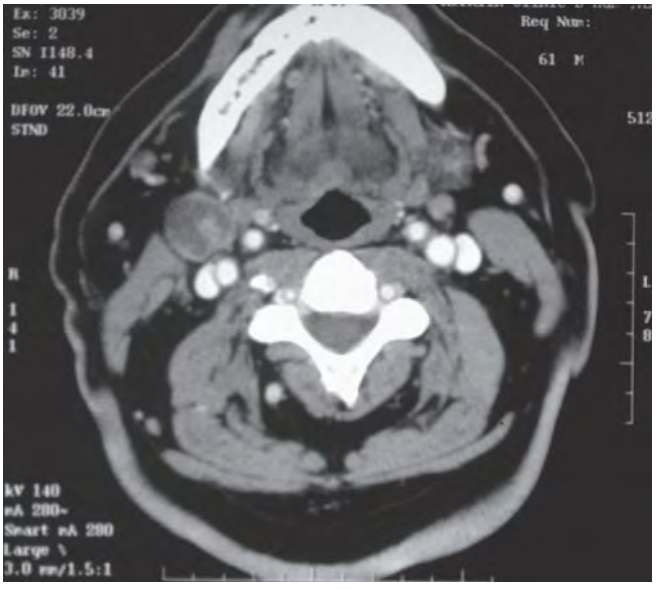


FIGURE 32-18. Computed tomography scan with contrast shows peripheral rim enhancement and central lucency of lymph node harboring metastatic squamous cell carcinoma.

criteria for nodal metastases, including node size greater than 1 cm (except the level II jugulodigastric node, which must be > 1.5 cm), central necrosis, and morphology (round instead of oval), have increased sensitivity to over 90%.[154] MRI neck evaluation has gained popularity in recent years and is typically used if the primary site is being imaged with MRI, such as for a tongue cancer. CT and MRI, in that order, are the most widely used imaging modalities for detection of occult metastases in the United States.

The characteristics of the primary tumor may also predict metastases. Spiro and associates[155] demonstrated that depth of invasion in tongue cancer was a reliable predictor of lymph node metastases in cancer of the oral tongue. They found that cancers with 2 to 8 mm depth of invasion had a significantly higher rate of lymph node metastases than those with invasion of less than 2 mm (25.7% vs. 7.5%). Depth of invasion greater than 8 mm was associated with a 41% rate of occult metastasis. Tumor thickness less than 2 mm has been associated with a 13% incidence of lymph node metastases and 3% would ultimately succumb to their disease, whereas greater than 9 mm of invasion was associated with a 65% incidence of lymph node metastases and 35% would die of their disease.[155,156] Ocharoenrat and colleagues[157] also demonstrated an increased risk of cervical metastasis in tongue cancers with a depth of invasion greater than 5 mm and correlated this with poor outcome even in early-stage (I and II) tongue cancers. Similar results were reported by Kurokawa and coworkers,[158] who found that depth greater than 4 mm was associated with an increased risk for development of late cervical metastases in patients with moderately differentiated squamous cell cancers of the tongue and diminished overall survival. This has led to recommendations that even in the absence of evidence of lymph node metastases, the neck should receive elective treatment (either END or irradiation) for thicker primary tumors, and some have suggested that depth of invassion be added to the staging of oral squamous cancers. Chen and Schmidt[159] made a persuasive argument for considering END for *any* oral primary.

Two additional imaging modalities used for evaluating nodal metastases deserve mention. Ultrasonography and positron-emission tomography with fluorodeoxyglucose (FDG-PET) with or without combined CT (PET or PET/CT) are gaining in popularity for initial staging and follow-up staging of patients with head and neck cancer. Although not commonly used in the United States, ultrasonography has been used in outpatient clinics for evaluation of oral cancer patients in Europe for some time. Ultrasonography criteria for malignant changes include nodal size and changes in echogenicity, central necrosis that will lead to an echogenic hilum, and a hypoechogenic periphery. Its ability to improve on manual palpation of cervical lymphadenopathy has led to its increased use in the United States. It can also help to evaluate carotid or jugular invasion. When performed by an experienced clinician and combined with aspiration cytology, ultrasonography is very accurate.[160] Knappe and associates[161] reported a sensitivity of 89.2% and a specificity of 98.1% in 56 patients who underwent preoperative ultrasound-guided fine-needle aspiration followed by elective or therapeutic neck dissections.

PET and PET/CT have become increasingly popular in the staging and follow-up of patients with head and neck squamous cell carcinoma. By identifying areas of high glucose uptake, PET scans allow clinicians to identify potential metastases in the preoperative workup (Figure 32-19). Standardized uptake values (SUVs) are assigned based on the amount of glucose uptake and can offer some prognostic information.[162] The overall incidence of clinically identified distant metastasis in head and neck cancer patients is 2% to 18%, and presence of distant metastases may influence the choice of initial treatment.[163,164] The role of PET scans in the evaluation of occult cervical metastases is limited by the need for at least 5 to 10 mm^3 of tumor for detection. Carlson and colleagues[165] evaluated the use of PET/CT in the preoperative staging of patients with oral/head and neck cancer and found a sensitivity and specificity of 79% and 82%, respectively, in the N0 neck and 95% and 25% for the N+ neck. They concluded that currently PET/CT was inadequate to guide the need for neck dissection nor could it guide the surgeon as to which levels should be dissected.[165] Technologic advances and refinements in technique continue, and PET scanning will likely become increasingly accurate at the detection of occult metastasis.

PET or PET/CT is frequently employed in the workup of patients with cervical metastases and unknown primaries, but



FIGURE 32-19. Positron emission tomography scan shows recurrent cancer in the left neck with three mediastinal metastases. Note the heavy physiologic uptake in the brain, heart, kidneys, and bladder.

its specific role continues to be explored.[166] It has been shown to be of some value in locating occult primary sites in patients presenting with a neck mass but has not supplanted the role of panendoscopy with directed biopsies and tonsillectomy in these patients.[167] PET is also used to examine patients who have undergone chemoradiotherapy for recurrent disease.[168] These patients are notorious for their difficulty in examination secondary to extensive changes in the soft tissue. It is recommended that at least 3 months pass after therapy completion before obtaining a PET scan because of the persistent inflammation associated with radiation and the tumoricidal effects that persist after radiation is completed. It should also be remembered that current technology requires that a focus of squamous cell carcinoma be several millimeters to be detected.[169] Findings on follow-up scans should be interpreted within the context of the entire clinical picture because false positives in postoperative and postradiated fields of the head and neck are not uncommon. In the past, it was stated that distant metastases were a late finding in head and neck cancer patients and most patients succumbed to locoregional disease. It was once thought that less than 1% of head and neck cancer patients had distant disease at presentation. As the use of PET scanning in the initial evaluation of patients with oral cavity cancers becomes more widespread, detection of distant metastases at the initial evaluation is becoming more common. One result of these improvements in detection of distant disease is *stage migration.* In other words, as our ability to detect distant disease improves, more patients are assigned a higher stage at presentation. This does not mean that patients are being diagnosed later in the course of their disease than in the past but simply that our diagnostic abilities have improved.

NECK DISSECTION IN ORAL CAVITY SQUAMOUS CELL CANCER

Appropriate surgical management of the regional lymphatics plays a central role in the treatment of oral cancer patients. Removal of the at-risk lymphatic basins serves two important purposes: (1) to remove and identify occult metastasis in patients in whom cervical metastasis are at risk; the *elective neck dissection* (note should be made here that the term "prophylactic neck dissection" should be avoided and replaced with the more accurate term *elective neck dissection* when discussing removal of at-risk lymphatic basins in the absence of clinical evidence of metastasis); or (2) to remove macroscopic disease in patients in whom metastasis are highly suspected based on imaging, clinical examination, or biopsy via fine-needle aspiration: termed *therapeutic neck dissection.* A brief overview of neck dissection follows with more comprehensive reviews outlining technique available elsewhere.[170]

The importance of treating the cervical lymph nodes was stressed by Crile in his landmark 1906 paper[171] and was later popularized by Martin and coworkers.[172] Generations of surgeons were trained in the classic radical neck dissection

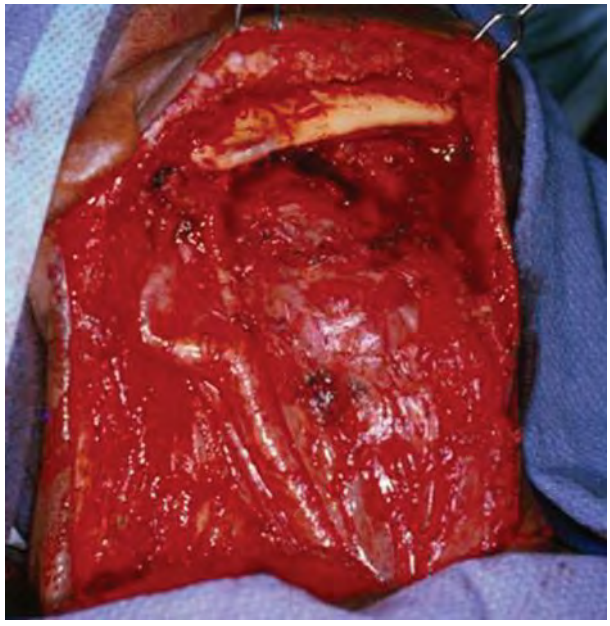


FIGURE 32-20. Standard radical neck dissection of the right neck. Note the communication with the oral cavity where an oral cancer has also been resected.

(Figure 32-20). Improved understanding of the regional lymphatic flow and nodal basins at risk for metastases from different primary locations has led to an increasing number of modifications of the standard radical neck dissection. The resultant, often misused, terminology of neck dissection was standardized by the American Academy of Otolaryngology's Committee for Head and Neck Surgery and Oncology in 1991.[173] Revisions were proposed in 2002 to improve communication among clinicians.[174] These proposed changes were primarily in regard to the selective neck dissections, and specific names such as "supraomohyoid neck dissection" were eliminated in favor of the phrase "selective neck dissection" followed in parentheses by the levels removed. The definitions pertinent to oral cavity cancer are

- Radical neck dissection: Refers to the removal of all ipsilateral cervical lymph node groups extending from the inferior border of the mandible to the clavicle, from the lateral border of the sternohyoid muscle, hyoid bone, and contralateral anterior belly of the digastric muscle medially to the anterior border of the trapezius. Included are levels I through V. This entails the removal of three important nonlymphatic structures—the internal jugular vein, the sternocleidomastoid muscle, and the spinal accessory nerve.
- Modified radical neck dissection: Refers to removal of the same lymph node levels (I–V) as the radical neck dissection, but with preservation of the spinal accessory nerve, the internal jugular vein, or the sternocleidomastoid muscle. The structures preserved should be named. Some authors propose subdividing the modified neck dissection into three types:

- Type I preserves the spinal accessory nerve.*
- Type II preserves the spinal accessory nerve and the sternocleidomastoid muscle.*
- Type III preserves the spinal accessory nerve, the sternocleidomastoid muscle, and the internal jugular vein.*

- Selective neck dissection: Refers to the preservation of one or more lymph node groups normally removed in a radical neck dissection. In the 1991 classification scheme, there were several "named" selective neck dissections. For example, the supraomohyoid neck dissection removed the lymph nodes from levels I to III (Figure 32-21). The subsequent proposed modification in 2001 sought to eliminate these "named" dissections. The committee proposed that selective neck dissections be named for the cancer that the surgeon was treating and to name the node groups removed in parenthesis. For example, a selective neck dissection for oral cavity cancer would encompass those node groups most at risk (levels I–III) and be referred to as a selective neck dissection (levels I–III).
- Extended neck dissection: Refers to the removal of one or more additional lymph node groups, nonlymphatic structures, or both, not encompassed by a radical neck dissection. For example, mediastinal nodes or nonlymphatic structures such as the carotid artery or hypoglossal nerve.

It is important to remember that classification schemes are continually changing, and as science evolves, the indications for different dissections will certainly change. For an oral cavity primary without evidence of lymph node metastases, a selective neck dissection removing lymph nodes from levels I to III is the generally accepted procedure. Shah and associates[175] demonstrated supraomohyoid neck dissection to eradicate occult metastatic disease in 95% of patients. Some surgeons, however, advocate including level IV (extended supraomohyoid neck dissection) to decrease the risk, however small, of missed occult metastases (see earlier discussion). Extension on the left side does entail an increased risk to the thoracic duct and the possibility of a chyle leak. Modifications of neck dissections have been made in an attempt to prevent the morbidity of radical neck dissection (Figure 32-22). Preservation of the spinal accessory nerve decreases the incidence of painful shoulder syndrome. Extensive skeletonization of the nerve, however, can result in significant dysfunction even if the nerve is preserved (Figure 32-23). Several studies have suggested that dissection of level IIb (above the nerve) is unnecessary in the clinically negative neck because of the low incidence of metastases in this area

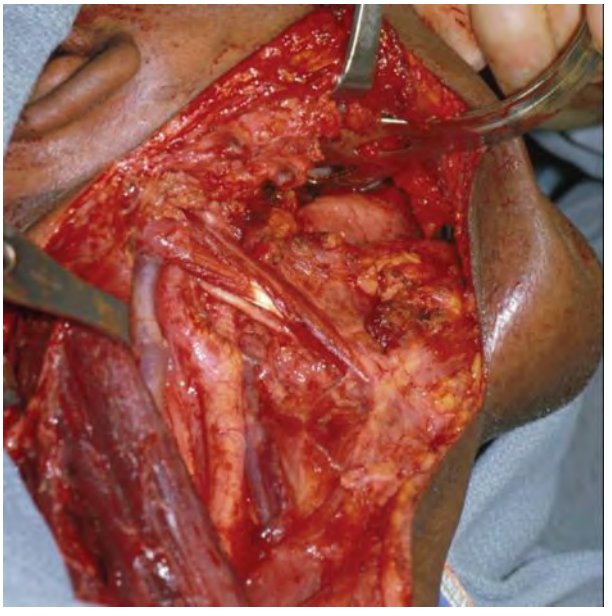


FIGURE 32-21. Supraomohyoid neck dissection, or selective neck dissection, levels I to III. The sternocleidomastoid muscle, nerve XI, and internal jugular vein are preserved.



FIGURE 32-22. Denervation of a patient's left trapezius following sacrifice of spinal accessory nerve during radical neck dissection.

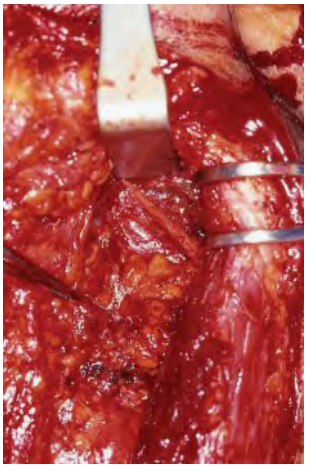
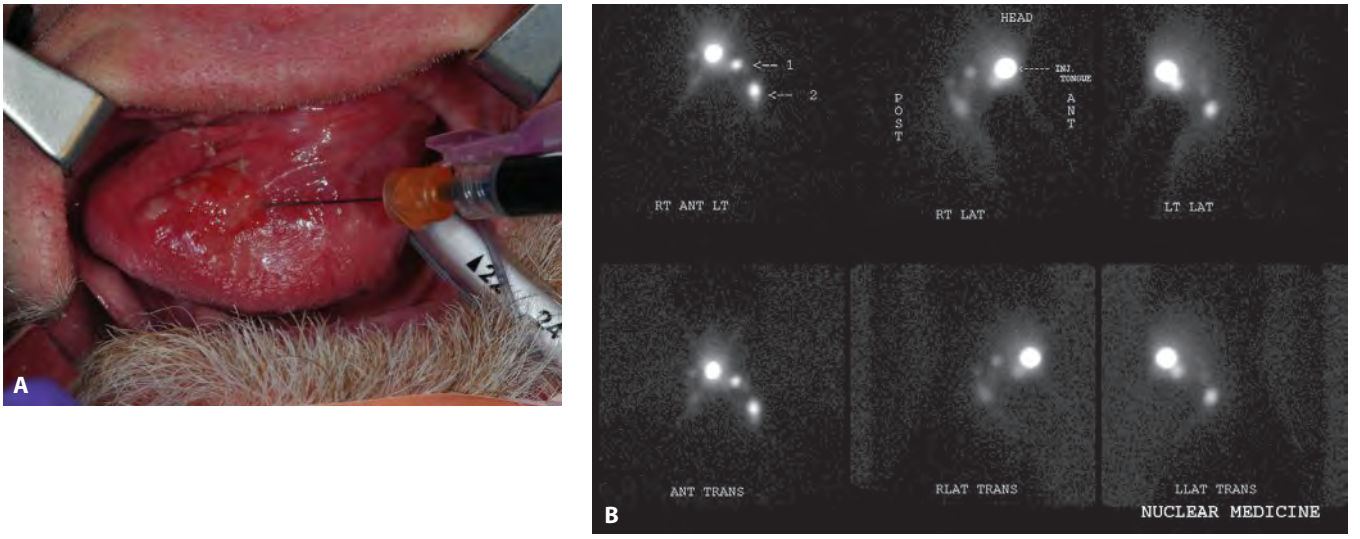
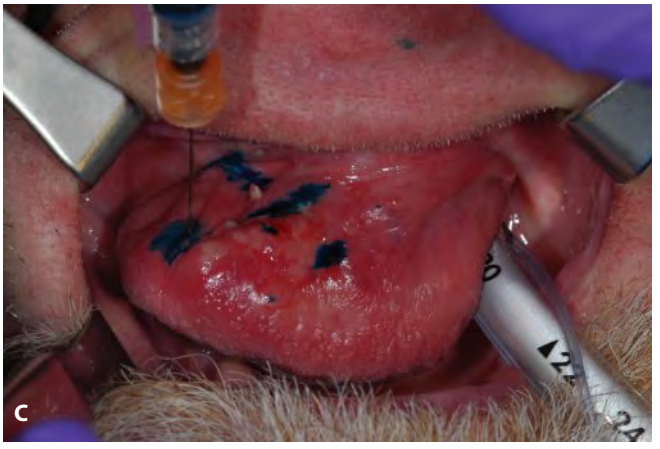


FIGURE 32-23. Dissection and skeletonization of spinal accessory nerve can produce shoulder dysfunction even if nerve is preserved.







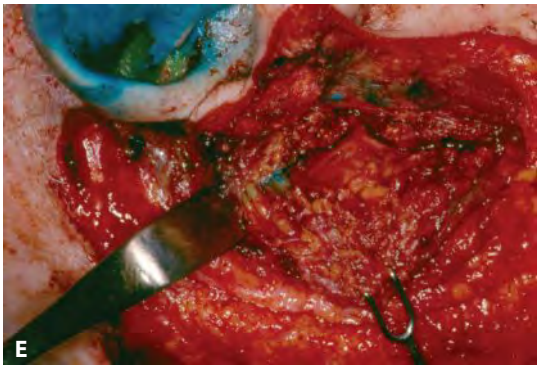


FIGURE 32-24. Sentinel node biopsy. **A,** Peritumoral injection of radiocolloid. **B,** Radiograph demonstrates radioisotope collecting in first echelon node. **C,** Peritumoral injection of isosulfan blue preoperatively. **D,** Patient marked based on radiograph and gamma probe. **E,** Note the dark staining of the sentinel node (different patient than in **A**–**D**). (Photographs courtesy of Bryan Bell DMD, MD, FACS, Portland, OR.)

(1.6%) and is recommended only if bulky disease is present in level IIa.[176]

If there is clinical evidence of lymph node metastases, controversy exists over the proper type of neck dissection (see "Therapeutic Neck Dissection," later). The application of supraomohyoid neck dissection to the N+ neck (therapeutic neck dissection) has yielded mixed results.[177] Previous studies have demonstrated that patients undergoing selective neck dissections for N0 necks have a higher rate of recurrence in the neck if positive nodes are ultimately found in the pathologic specimen. This can be improved by the addition of postoperative radiation treatment.[178] The question remains as to whether this is due to the type of neck dissection or simply the biology of the tumor.[179,180] Most surgeons advocate some form of neck dissection if there is demonstrable evidence of metastatic disease in the neck, and a diminishing number of surgeons maintain that the evidence of lymph node metastases is justification for nothing less than a standard radical neck dissection.

Another controversy regarding the evolution of neck dissection concerns the concept of in-continuity versus discontinuous neck dissections. In the past, it was considered

mandatory to remove the primary tumor in direct continuity with the neck dissection, in one specimen. Work by Spiro and Strong[181] found no adverse impact on survival when neck dissection was performed in a discontinuous manner. Bias might have occurred, however, because smaller lesions were in the discontinuity group. A study by Leemans and colleagues[182] found worse outcomes in stage II cancer of the tongue with discontinuous neck dissection, with local recurrence rates of 19.1% versus 5.3% and a 5-year survival of 63% versus 80%. Most surgeons prefer an in-continuity approach if technically feasible, without the resection of obviously uninvolved structures such as the mandible.

The controversy surrounding END versus elective neck irradiation (without neck dissection) continues. Advantages of surgery include the production of a surgical specimen that guides the need for further treatment. If no lymph nodes are identified, radiation can be held. The possibility of future second, third, or even fourth primary cancer arising in this at-risk population makes reserving radiation attractive. A comprehensive discussion of the management of cervical lymph nodes in head and neck cancer is beyond the scope of this chapter. Three excellent reviews are available and recommended.[152,183–185] Although several studies have failed to demonstrate a survival advantage in patients who undergo END versus careful follow-up and therapeutic neck dissection if a metastasis develops, most surgeons would agree that the morbidity associated with a selective neck dissection is minimal and would have a low threshold for performing it.

SENTINEL NODE BIOPSY

As the evolution toward less invasive surgical modalities proceeds, dissection of the N0 neck (staging neck dissection or END) is becoming increasingly limited. The sentinel node technique, first popularized for melanoma, has been investigated for use in head and neck cancer.[186,187] Theoretically, it allows the identification and removal of the first-echelon lymph node ("sentinel node") that would first receive metastases from a given site. The technique involves injecting the area surrounding the primary site with a radioactive-labeled material,^{99m} Tc-sulfur colloid. Various molecular weights can be chosen depending on the transit time desired. A radiograph is then taken to identify and locate the sentinel node or nodes. The patient is then taken to the operating room where the surgeon may also inject isosulfan blue dye around the primary tumor site. The dye will also drain to the sentinel node and stain it blue, assisting the surgeon in identification during surgery (Figure 32-24). The surgeon will also use a gamma detection probe counterprobe to identify the node with the highest concentration of radioactive colloid. The best results are obtained when at least two of the three localization techniques—preoperative imaging, intraoperative identification using blue dye, and/or intraoperative identification using the gamma probe—are used in combination.

The node is then removed, confirmed with the gamma probe, and if it is histologically positive, further treatment such as radiation may be indicated. In melanoma, sentinel node biopsy has a reported sensitivity of 82% to 100% and very few false negatives.[188,189]

The sentinal lymph node biopsy (SLNB) technique has been investigated in the head and neck with varying results. Problems with the application of the sentinel node technique to squamous cell cancer of the oral cavity relate to the rich lymphatic drainage with possible bilateral drainage as well as the complex anatomy in the neck, leading to difficulty in dissecting out a single node. Close proximity of the sentinel node to the primary cancer, for example, an FOM primary cancer and a closely adjacent submental node, can lead to the accumulation of colloid around the primary cancer, which obscures the sentinel node. The rich lymphovascular network can also lead to drainage to several nodes. Cevantos and coworkers[190] used the sentinel node technique in 18 oral cavity cancers with N0 necks. They compared sentinel node biopsy with CT images and PET images by obtaining a CT and PET followed by SLNB and neck dissection. They found 10 true positives, 6 positive nodes identified on frozen section, 2 positive notes on evaluation of permanent pathologic specimens, and 2 on immunoperoxidase staining for cytokeratin. In 6 specimens, the sentinel node was the only positive node. They also found 7 true negatives and 1 false negative. In 1 case, the sentinel node identified by the radioactive colloid did not contain cancer but another cervical node did. They also found that tumor in the node can lead to obstruction and redirection of lymphatic flow.[190] Pitman and associates[191] further demonstrated the use of the SLNB technique for the N0 neck. Hyde and colleagues[192] reported on 19 patients whose radiographic and clinical test results on their necks were negative and who underwent SLNB and PET scanning followed by conventional neck dissection. In 15 of the 19 patients, the sentinel node as well as the remaining nodes were negative. In 3 of the 19 patients, the sentinel node was positive along with other nodes. In 1 patient, the sentinel node was negative, but another node removed in the neck dissection was positive. The node was located close to the primary cancer, which often leads to difficulty discriminating activity due to the tumor and that of adjacent nodes. Interestingly, PET failed to reveal cancer in the 4 patients with subsequently identified cervical metastasis (see discussion on PET scanning, earlier).[192]

Shellenberger[193] offers an excellent review of the application of SLNB in oral cavity squamous cancer. An important point in the application of SLNB technique to oral cavity cancers is that multiple lymph nodes (usually 3–5) are usually removed from an area selected by the localization technique, making the procedure actually a very highly selective neck dissection.[193] Recently, Civantos and coworkers[194] released the results of a multi-institutional trial evaluating the efficacy of sentinel node technique for interrogating the N0 neck in oral cavity cancer. They reported 140 patients treated over a 3-year period among 25 institutions. The majority were oral tongue cancers, and small lesions (<6 mm diameter and "minimally invasive") were excluded. They found a negative predictive

value for SLNB of 94%, which improved to 96% with additional sectioning and immunohistochemistry. Extrapolating this to a population with an occult metastatic rate of 30% would lead to a 4% failure rate in the neck. As with previous studies, accuracy was improved with tongue lesions as compared with FOM sites. Importantly, they found acceptable results with relatively inexperienced surgeons. Their finding that additional sections and immunohistochemistry play a critical component of SLNB technique is in agreement with other studies.[194] It must be emphasized that the increased pathologic scrutiny (additional sectioning and immunohistochemistry) applied in SLNB technique has resulted in a significant number of necks being upstaged (as many as 8%), compared with traditional pathologic techniques. The clinical significance of this example of stage migration remains unclear. One wonders how many negative ENDs for the clinically N0 neck are, in fact, therapeutic and whether further treatment with comprehensive neck dissection or neck irradiation should be indicated for these micrometastasis.

THERAPEUTIC NECK DISSECTION

Patients presenting with nodal disease will usually undergo some form of therapeutic neck dissection, the nature of which varies with surgeon's preference. Some surgeons will treat all patients with suspected cervical metastases with a radical neck dissection. Most consider a modified radical neck dissection adequate, removing the internal jugular vein or sternocleidomastoid muscle if indicated by proximity to or involvement with tumor. There is some evidence that selective neck dissection may be adequate for the N+ neck in certain carefully selected patient populations (see discussion of selective neck dissections, earlier). Anderson and associates[195] reported the results of three academic centers in which patients with previously untreated clinically and pathologically N+ necks underwent selective neck dissection. They reported a regional control rate of 94.3%. Their results were comparable with those of patients undergoing more extensive neck dissections.[195] Patients presenting with massive nodal disease who are going to be treated with chemoradiotherapy or combination of brachytherapy and external beam radiotherapy can present a challenge to surgeons who are faced with the option of surgery before or after radiation. Not infrequently, surgeons are faced with complete clinical resolution of disease and neither surgeon nor patient looks forward to a neck dissection in a heavily irradiated field. There is some variation in approaches to this dilemma. Some surgeons recommend pretreatment neck dissection to remove bulky disease, whereas others plan a neck dissection 4 to 6 weeks after treatment regardless of response. Still others recommend a CT scan at 4 weeks and CT-guided biopsy of any suspicious nodes. This is followed by neck dissection if the node biopsy is positive for cancer. McHam and colleagues[196] found that clinical factors did not predict patients with residual disease after chemoradiation therapy and recommended neck dissection in all patients initially seen with N2 to N3 disease. This recommendation was made in light of a 26% to 35% complication rate in patients undergoing neck dissections after chemoradiation therapy.[196] The role of PET scanning in this situation has not been completely defined. Some investigators have demonstrated that a negative PET/CT scan may be an indication for observation alone after complete response to chemoradiotherapy, even in the face of necrotic nodes and nodes larger than 3 cm.[197] Patients with recurrent cancer after multimodality therapy typically have a poor outcome, making salvage surgery an unattractive alternative.

Surgical management of cervical lymph node metastasis, both occult and evident, continues to evolve. It is clear that metastases are an indication of aggressiveness and portend a poorer prognosis. Once the cancer has developed the necessary genetic mutations to break free and colonize independent of the primary tumor, the chance of cure with single-modality therapy diminishes. In his presidential address to the New England Surgical Society, Blake Cady referred to "… lymph node metastases as the speedometers of the oncologic vehicle, not the engine. Indicators, not governors of survival."[198] Clearly, the role of the radical neck dissection has diminished greatly over the past few decades as less invasive surgical techniques for dealing with the cervical lymphatics have gained popularity. This trend will likely continue as the role of surgery in the control of metastatic disease is better defined.[199–201]

RECURRENCE AND FOLLOW-UP SURVEILLANCE

In 1984, Vikram and coworkers[202–204] published a series of reports that discussed patterns of failure in patients treated with multimodality therapy for head and neck cancer. This classic series of articles outlined failure characteristics at the local site, neck, and distant sites as well as the development of second malignant neoplasms in patients treated at the Memorial Sloan-Kettering Cancer Center in New York.[178,202–204] Ninety percent of patients who will suffer a recurrence of oral cavity cancer will do so in the first 2 years. For this reason, patients are placed in a structured follow-up. Stage at recurrence is the most important predictor of survival, with stage I at recurrence associated with a median survival of 24.3 months and a disease-free survival at 2 years of 73%, whereas stage IV recurrence was associated with a median survival of 9.3 months and a 2-year disease-free survival of 22%.[205] Follow-up protocols vary widely and are intended to detect recurrences early. De Visscher and Manni[206] suggested the following protocols:

1. Every 2 months for 1 year.
2. Every 3 months for year 2.
3. Every 4 months for year 3.
4. Every 6 months for years 4 and 5.
5. Then yearly.

Despite this and other suggested follow-up protocols, the follow-up schedule must be tailored to the individual patient and

must take into account the patient's likelihood of having a recurrence, possible continuation of smoking or other habits, ability to travel and keep appointments, and the potential availability of local medical or dental care that might assist in follow-up surveillance. Follow-up appointments include an update of patient history and review of systems as well as clinical examination for recurrence or detection of new primaries. Questions raised by physical examination should prompt an appropriate imaging study, rebiopsy, or examination under anesthesia. Caution should be used, however, in performing biopsies in patients who have received intensive multimodality therapy, such as RADPLAT, brachytherapy, or hyperfractionated radiation schedules combined with chemotherapy. Extensive biopsy wounds are notorious for slow healing in this setting and can lead to the development of chronic wounds.

Appropriate imaging, including a baseline CT or MRI at the completion of multimodality therapy, is invaluable. PET scanning can also play a useful role in surveillance, but findings should be correlated with other clinical data, because false positives occur. Combined with CT, PET has been shown to be accurate in predicting the need for salvage surgery in some settings.[207] Failure at the primary cancer site will ultimately occur in approximately 20% of patients, and regional recurrence in the neck will occur in 10%. Death from distant metastases is rare, occurring in only approximately 1% to 4% of cases in which locoregional control is maintained. An unfortunate consequence of improved control at the primary cancer site with multimodality therapy is an increasing incidence of patients with distant metastases. In addition to recurrences, prospective studies have demonstrated that second primary cancers develop at a rate of 4% to 7% annually in patients who have had a head and neck squamous cancer. Second primary cancers are the leading cause of death among patients who have undergone treatment for early-stage oral cancer.[178,202–204]

The ability of a cancer to metastasize depends on the development of a series of genetic mutations, allowing for cells to disseminate from the primary tumor, arrest in the microcirculation, extravagate, infiltrate into stroma, and survive and proliferate as a new colony. Surveillance for distant metastases, therefore, becomes an important component of the follow-up evaluation. The lungs are the most common site for distant metastases, followed by the liver and bone. Yearly or biannual chest radiographs can detect lung metastases, the most common distant site of metastasis for oral cavity cancer, as well as primary lung cancers, which are not uncommon in the population at risk for oral cancer.[208] Given the current unavailability of an effective treatment regimen, however, some authors have questioned the use of annual or semiannual chest radiographs.[209] PET scanning may prove to be a more valuable alternative for detection of distant disease and, in the case of second primary tumors, may identify these lesions at a stage early enough that they can be sucessfully treated. Yearly laboratory work to include liver function studies is also recommended. In patients who have received radiation as part of their treatment, periodic thyroid function tests are helpful, because some will slowly become hypothyroid.

Collins[210] stated that patients with head and neck cancer are probably never cured and that it is better to consider that the host-tumor relationship has been durably altered in favor of the host. It is important to realize that approximately one third of patients with presumed localized disease will relapse and die of cancer. In advanced head and neck squamous cell carcinoma, 20% to 30% will survive, 40% to 60% of patients will suffer locoregional recurrence, and 20% to 30% will succumb to distant metastases. Hence, the majority of treatment failures remain recurrence of locoregional disease.[210] Patients with recurrent disease are reevaluated, which requires a similar evaluation as the original. Although this process is often referred to as "restaging," the original tumor-node-metastasis (TNM) stage remains unchanged. Panendoscopy and examination under anesthesia take on greater importance when a clinician is faced with examination of tissue scarred and distorted by previous surgery and radiation. Distant metastases should be ruled out to the extent possible before deciding on aggressive retreatment. If the recurrence is confined to the locoregional area, treatment decisions are limited by previous therapy. RRT protocols exist but are accompanied by significant morbidity.[211] Intensive RRT and chemotherapy protocols are being investigated and show some promise.[212] RRT protocols combining chemotherapy with additional radiation are gaining popularity in some centers. Side effects are significant, but managable in most cases, and 1- and 2-year survival rates of 40% to 50% and 15% to 27% have been reported.[213] These studies of RRT include all head and neck subsites, and RRT of local oral cavity recurrence entails much greater morbidity.

The morbidity of such treatments is significant, and their use should be restricted to clinical trials at this time. Surgical salvage remains the primary option, but the extent of salvage surgery must be considerably broader than might initially be considered. Goodwin[205] reported on the outcome of salvage surgery for recurrent head and neck cancers and found benefit in stages I and II. Success was limited in more advanced disease.[205] DFI is an important prognostic indicator, and DFIs less than 1 year are associated with poor prognosis.[214] In the future, biologic markers may play a crucial role in evaluating prognosis and guiding therapy. For example, low expression of EGFR is associated with improved outcome after salvage surgery for locoregional relapse. Clearly defined goals should be established between surgeon and patient for salvage surgery. Is the operation for cure or palliation? Palliative surgery should be undertaken very cautiously because surgical complications may greatly overshadow the palliative goals. Patients and their families must have realistic expectations as well as understand that there is no benefit from repeated surgical intervention for recalcitrant cancer.

Patients with inoperable cancer pose a unique challenge to the clinician. Because cure is no longer a realistic option, treatment modalities to prolong life and improve quality of life assume a higher priority. Chemotherapeutic options

are improving (see "Chemotherapy," earlier). Pain control becomes a significant issue in patients with recurrent head and neck cancer. Long-acting sustained-release formulations such as transdermal narcotic patches combined with short-acting narcotics for breakthrough pain are typically required. Rhizotomy is an option for intractable pain. Pain control can be a realistic goal of palliative chemotherapy or radiotherapy. Novel methods for the targeted delivery of chemotherapeutic agents into the tumor are under development. A combination of cisplatin and epinephrine gel injected into recurrent tumors demonstrated significant palliation without major side effects in most patients.[216] Wound management becomes an important issue, and dealing with large malodorous wounds can be taxing on patients and families. Patients presenting with advanced head and neck cancer will typically survive 6 to 12 months without treatment, and patients with end-stage head and neck cancer will have a median survival of 101 days.[210] The most common cause of death is aspiration pneumonia.

There is a natural tendency for clinicians to avoid the dying patient. There is a reluctance to face a disease whose biology has resisted their best efforts and whose treatment has left patients debilitated and frequently deformed. While family members and clinicians are discussing further treatment options, patients are frequently simply concerned with pain control and the effects of massive doses of narcotics on bowel function. Frank, thoughtful discussions must be held with the patients and their families regarding end-of-life issues and will help surgeons focus on these very real concerns. Hospice provides an excellent resource at multiple levels, and once the patient is enrolled, most families are greatly appreciative of the support offered by these professionals in end-of-life care.

In this era of improved treatment modalities for local and regional disease, clinicians are finding that factors unrelated to the primary cancer and beyond their control are influencing survival. It is becoming increasingly evident that factors affecting outcome in oral cancer patients are multiple and may relate more to patient characteristics than the cancer itself or the treatment they receive. Researchers are finding that genetic factors of the primary cancer have an impact on the response of the particular tumor to any treatment.[217] High expression of EGFR is associated with poor outcome and may indicate the need for more intensive multimodality therapy. Alterations in *TP53* have been associated with recurrence in squamous cell cancer of the head and neck that was refractory to radiation treatment. Future treatments may include restoration of *TP53* function.[218]

Importantly, studies are also demonstrating that co-morbidities and performance status predict survival independent of stage at diagnosis. Performance status has been shown to be a predictor of survival independent of TNM stage. Many head and neck cancer patients suffer other medical problems related to tobacco and alcohol use, and these can result in decreased overall survival despite what might be a more favorable cancer-specific survival. Ribeiro and associates[219] found that daily alcohol consumption, smoking, poor body mass index, and other co-morbidities had an independent impact on prognosis. As discussed earlier, there may indeed be a more aggressive form of squamous cell carcinoma that affects younger patients, but data from the National Cancer Data Base indicate that younger patients have a survival advantage that is most likely related to their lack of co-morbidities.[220] Frequently 5- and 10-year survival curves are affected more by these co-morbidities than the tumor characteristics recorded in the TNM system (see later discussion).[221] The TNM staging system will continue to undergo revisions to enhance its use.

FUTURE TREATMENTS

In the future, biologic markers hold out promise as the key to treatment of head and neck squamous cancers. Serving as potential targets for gene therapy, biologic markers may also determine appropriate treatment strategies and may select which patients should be treated with surgery, radiation treatment, chemotherapy, or combination treatment. Certain subpopulations of squamous cancers, those with high levels of *TP53* expression and low levels of the marker Ki-67, for example, have higher relapse rates after initial therapy. These patients may benefit from more aggressive combination treatments.[217,218]

Every few years, a new cancer therapy is heralded as the end of cancer surgery. For the present, surgery will continue to play the key role in management of oral cavity cancers, and surgeons must be knowledgeable in all diagnostic and treatment modalities as they continue their captainship of the oral cancer team. The surgeons treating oral cancer, regardless of their discipline, must learn from the contributions and mistakes of their forebears and add the benefit of their own training and experience. They must then use their knowledge base and the input of other treating colleagues to synthesize a plan of treatment tailored to the unique patient who sits before them. They must interact effectively with colleagues of other disciplines with the patient's benefit their foremost concern. They must execute the surgical components of the treatment plan with accuracy and skill. They must be supportive to their patients and their patients' families at a time of great stress in their lives and must not turn away from adversity or complication. They must accept the fact that not all patients can be cured. They should derive inspiration from those who survive and satisfaction from those who might succumb in a way made more favorable by the surgeon's input.

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CHAPTER

Lip Cancer

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Lip cancer, one of the most common cancers of the head and neck region, is one of the most easily diagnosed, with generally a good prognosis. In the United States, approximately 4300 new cases resulting in nearly 150 deaths are diagnosed annually. In some individuals, lip cancer may behave aggressively, manifested by recurrence or mortality in up to 15% of patients.[1–4] The most common malignancy of the lip is squamous cell carcinoma, whereas basal cell carcinoma accounts for only 1% of all lip carcinomas.[4–6] Other malignancies of the lip have been reported but are less common.[7,8] Generally, the behavior of lip cancer resembles skin cancer more than carcinoma of mucosal origin in the oral cavity proper. The lower lip is the most common site for lip cancer (88–98%), with only 2% to 7% arising from the upper lip and 2% to 4% at the oral commissures.[1,4–6,9–11]

EPIDEMIOLOGY AND ETIOLOGY

The incidence of lip cancer varies throughout the world, resulting in up to 30% of all malignant tumors of the oral cavity in certain regions. In the sunbelt region of the United States, the incidence of lip cancer reaches 12.7 per 100,000, making lip cancer the most common cancer of the oral cavity, and its incidence is second only to skin malignancy of the head and neck. Continental Europe and Australia have a reported annual incidence 12.0 and 13.5 cases per 100,000 population, respectively, whereas, lip cancer remains relatively uncommon in parts of Asia.[12]

In the United States, the overall incidence of lip cancer is 1.0 per 100,000 population which is a 58% improvement in overall incidence when compared with prior published National Cancer Institute SEERS (Surveillance, Epidemiology, and End Results) data.[13] However, unlike changing trends associated with other head and neck cancer sites, men continue to be affected at rates significantly higher than those in women. The incidence for males is 1.85 per 100,000 compared with 0.34 per 100,000 females.[14] Nearly 70% of newly diagnosed cases are adults 60 years of age or older. Furthermore, although the condition is most commonly associated with late middle age, lip cancer occasionally occurs in patients younger than age 30 years.[14]

The etiology of lip cancer is incompletely understood at present. Several factors have been associated with lip cancer development, but direct cause and effect has not been proved. Approximately one third of patients with lip cancer have outdoor occupations, suggesting that sun exposure may be an etiologic factor. Because of its prominence, the lower lip is at a higher risk for exposure to the sun than the upper lip. Hence, this results in the discrepancy in the distribution between upper and lower lip cancers.[15,16] Carcinoma of the lip principally affects those individuals with fair skin complexions. The prevalence of lip cancer is at least 10 times higher in whites than in those with darker skin and is extremely rare among blacks.[9,17] Although it has never been proved, darker-skinned individuals are believed to have a protective pigment in the vermilion of the lips that provides protection from solar injury.[18]

Multiple factors have been linked to lip cancer, including tobacco use, pipe smoking, thermal injury, lip trauma, poor oral hygiene, exposure to chemicals, mechanical irritants, immunosuppression, and chronic infections.[9–11,16,19–24] Immunosuppression significantly increases the risk of skin carcinoma development, especially on exposed surfaces. Immunosuppressed individuals may develop lip carcinomas at a younger age and remain at risk for the development of multiple and recurrent lip cancers.[25] Chronic infection with human papillomavirus (HPV) has been reported in 60% of cutaneous squamous carcinoma cases in patients with renal transplant history and immunosuppression; however, a matched control group in which patients did not develop squamous carcinomas demonstrated a lower incidence of HPV infection.[26] Recently, interest has developed in the association between

head and neck cancers and HPV-16 infection. Although HPV-16 is clearly associated with oropharyngeal carcinomas, its role in oral cavity and potentially lip squamous carcinomas remains controversial.[27,28] A study of nonimmunosuppressed, healthy individuals demonstrates chronic HPV presence in 30% of normal lip samples.[29] In the same cohort, patients with squamous carcinoma of the lip exhibited HPV infection at a rate of 18%, suggesting no relationship between HPV infection and carcinoma development. However, pharmacologic immunosuppression after organ transplant has been demonstrated to contribute to an increase incidence of cutaneous squamous carcinoma as patient survival times after transplant are extended.[30,31] Renal transplant patients have been reported to have a 30-fold increased risk of developing lip cancer over time.[19] Several case series have reported that a large proportion of lip cancer patients regularly use tobacco, indicating that tobacco use is etiologically associated with lip cancer development.[5,9,15,20,32–36] In 1984, Douglass and Gammon[18] reassessed the epidemiology of oral cancer and declared that there was insufficient proof for declaring tobacco as an etiologic factor in the development of lip cancer. Additional case-controlled studies concluded that no statistically significant relation exists between tobacco exposure and lip carcinoma.[37,38] The smoking of cigars and pipes is often considered an important etiologic factor; however, no convincing evidence exists that supports a causal relationship between tobacco use and developing lip carcinoma. Cigar and pipe smoking today, at best, are likely responsible for only a small fraction of lip cancers. Although alcohol and tobacco exposure remain the two factors most strongly associated with the development of oral and oropharyngeal carcinomas, these risk factors have proved to exhibit a limited influence on the developing lip carcinoma. Much of the evidence that consistantly associates prolonged and cumulative exposure to ultraviolet radiation from sunlight with the development of lip cancer has been derived from numerous case series and epidemiologic studies.[4,19–21,32,37,39]

ANATOMIC CONSIDERATIONS

Embryologically, the upper lip forms by fusing the two maxillary processes with a central median nasal process (Figure 33-1). As a result, a central midline mass with two larger lateral segments is formed. The separation of the lateral segments by this central midline mass makes metastasis from upper lip cancers to the contralateral neck exceedingly rare. Conversely, the lower lip, formed by fusion in the midline of two mandibular processes, is at an increased risk for contralateral neck metastasis, particularly with lesions near the midline. The lateral and superior borders of the upper lip are well defined at the nasolabial creases bilaterally and at the nasal base superiorly. The inferior border of the lower lip is defined along the transversely oriented labiomental crease.

The formal definition of lip cancer, established by the American Joint Committee on Cancer for the purpose of staging lip cancer, describes the lip as "beginning at the junction of the vermilion border with the skin and including only the vermilion surface or that portion of the lip that comes into contact with the opposing lip. It is well defined into an upper and lower lip joined at the commissures of the mouth."[40] This definition focuses on the unique epithelial surface of the lip vermilion and excludes cancers that arise from the adjacent skin or labial mucosa. In statistical reporting, cancers of the lip are commonly grouped with those of the oral cavity, because the lip is defined as part of the oral cavity by the American Joint Committee on Cancer.[40,41]

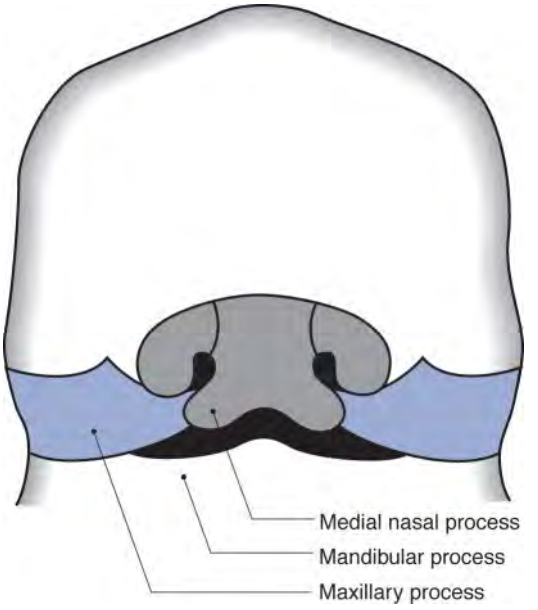


FIGURE 33-1. Developing upper lip, receiving contributions from a central medial nasal process and bilateral maxillary processes. Developing lower lip receiving contributions solely from bilateral mandibular processes.

Lymphatic drainage of the lower lip originates as an interconnecting network of lymph vessels beneath the submucosa of the vermilion.[43] It subsequently gives rise to five or six lymphatic collecting trunks that eventually terminate into regional lymph nodes. The lymphatic trunks of the central one third of the lower lip typically drain into the submental lymph nodes via perivascular and superficial facial nodes overlying the mandibular symphisis. The trunks that arise from each lateral one third of the lower lip typically drain into the ipsilateral submandibular lymph nodes. In certain individuals, the lymphatic trunks from the central one third of the lip may drain to the submandibular lymph nodes on either side.

Cervical metastasis from lip cancer occurs in fewer than 10% of patients with cancer of the lower lip and ranges from 20% to 50% in cancer of the upper lip and commissure.[5,41] In the upper lip, crossover of lymphatic drainage between the right and the left halves typically does not occur, but because of the high rate of regional metastasis, consideration of therapeutic stratagies for ipsilateral occult disease must be undertaken.[43] The upper lip also possesses five or six collecting trunks on each side of the midline that originate as

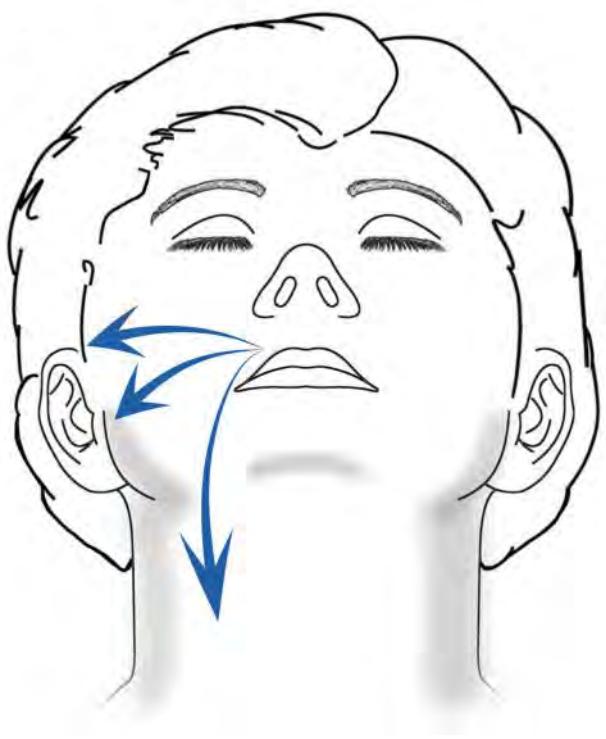


FIGURE 33-2. Patterns of lymphatic drainage from various portions of the upper lip.

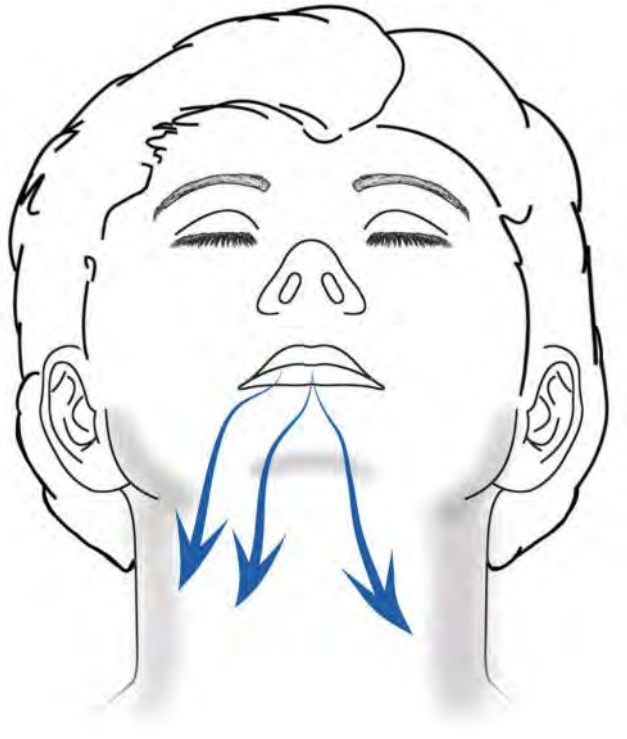


FIGURE 33-3. Patterns of lymphatic drainage from various portions of the lower lip.

delicate lymphatic vessels in the submucosa of the vermilion (Figure 33-2). The trunks ultimately terminate in the submandibular lymph nodes but occasionally also drain to the ipsilateral preauricular or infra-auricular parotid lymph nodes. Metastasis that results from cancer of the lip most commonly involves the submandibular and submental lymph nodes (level 1).[44–46] Metastasis to level II of the jugular chain rarely occurs. Cancer involving the upper lip may occasionally metastasize to the parotid lymph nodes, but contralateral metastasis is unusual for cancers of the upper lip that do not cross the midline and for lower lip cancers that do not involve the central one third of the lower lip.

Metastasis from the lower lip is primarily to the submental, submandibular, and perifacial nodes (Figure 33-3). Metastasis is found in the submandibular lymph nodes in approximately 80% to 90% of patients with metastasis from cancer of the lower lip.[5,47] Although the upper lip is responsible for fewer than 10% of lip cancer cases, its pattern of metastasis is fairly predictable, with the submandibular and parotid lymph node groups being most commonly involved.[48] Carcinoma of the commissure and upper lip spreads to the preauricular, periparotid, and submandibular nodes. Bilateral metastasis may develop if the lesion is near or has crossed the midline of the lip. Crossover between the lymphatics of the right and left sides of the upper lip rarely occurs.[4]

Cervical metastasis occurs late in the course of lip cancer in fewer than 10% of patients with cancer of the lower lip and up to 20% in cancer of the upper lip and commissure.[5] Lymph node metastasis to the upper jugular digastric chain is seen in only approximately 15% of all patients who have lymph node metastasis and is almost always seen in conjunction with ipsilateral submandibular metastasis.[5,47]

Blood supply to the upper and lower lips arises from the labial arteries, which are terminal branches from the external carotid system. The terminal blood supply forms an arterial arcade such that lesions of the lateral lip receive blood from both medial and lateral sources. Consideration of the blood supply is critical when ablative procedures require local tissue transfers to reconstuct form and function. Failure to recognize the anatomic relationship of the labial blood supply in association with the resection defect may result in the surgeon's inability to develop a well-perfused flap for rotational tissue transfer and may ultimately result in partial or complete reconstruction necrosis and failure.

MANAGEMENT

Evaluation

Because carcinomas of the lip occur on a highly visible and constantly exposed region of the body, a relatively early diagnosis is often feasible. The clinical presentation of lip carcinomas is quite characteristic, generally presenting as an exophytic or ulcerated lesion on the vermilion border, along with variable degrees of infiltration of the underlying musculature or invasion of the overlying skin or labial mucosa



FIGURE 33-4. Typical presentation of a large exophytic squamous cell carcinoma of the lower lip.

(Figure 33-4). Well-differentiated squamous cell carcinomas are often associated with hyperkeratosis and leukoplakia of the vermilion border of the lip. Primary carcinomas have a variable clinical presentation. Tumors may be ulcerative, exophytic, or endophytic on presentation. Regardless of the presenting morphologic type, any lip lesion that the clinician views as a possible malignancy should, without delay, undergo an incisional biopsy that includes both a portion of the lip lesion and a small portion of normal-appearing tissue at the margin. The factors that should be considered in planning surgical resection and reconstruction of the lips include the tumor stage, lip subsite of origin, patient preference, and histopathologic type and grade of the tumor.

Staging of lip cancers is similar to that employed for tumors of the oral cavity (Table 33-1).[40] Tumors less than 2 cm in greatest dimension are staged as T1, whereas massive tumors with invasion of deep soft tissues, adjacent bone, or overlying skin are staged as T4 (Table 33-2). Radiographic evaluation of tumors detected at an early stage that involve the lip is generally unnecessary. Conversely, advanced tumors that adhere to or invade the adjacent mandible require further radiologic evaluation.

TABLE 33-1 **Tumor Stage Grouping**

Stage 0	Tis	N0	M0
Stage I	T1	N0	M0
Stage II	T2	N0	M0
Stage III	T3	N0	M0
	T1	N1	M0
	T2	N1	M0
	T3	N1	M0
Stage IV	T4a	N0	M0
	T4a	N1	M0
	T1	N2	M0
	T2	N2	M0
	T3	N2	M0
	T4a	N2	M0
Stage IVB	Any T	N3	M0
	T4b	Any N	M0
Stage IVC	Any T	Any N	M1

TABLE 33-2. **Tumor Node Metastasis System for Lip Cancer**

T = Tumor Size	
TX	Primary tumor cannot be assessed
T0	No evidence of primary tumor
Tis	Carcinoma in situ
T1	<2 cm
T2	>2 cm and ≤ 4 cm
T3	>4 cm
T4	Tumor invades through cortical bone inferior alveolar nerve, floor of mouth, or skin of face (e.g., chin or nose)
N = Nodal Metastasis (Regional)	
NX	Regional lymph nodes cannot be assessed
N0	No regional nodes palpable
N1	Single ipsilateral node < 3 cm
N2	Metastasis in a single ipsilateral lymph node, > 3 cm but ≤6 cm; or in multiple ipsilateral lymph nodes, none > 6 cm; or in bilateral or contralateral lymph nodes, none > 6 cm
N2a	Single ipsilateral node > 3 cm but ≤6 cm
N2b	Multiple ipsilateral nodes ≤6 cm
N2c	Bilateral or contralateral nodes ≤?6 cm
N3	Node > 6 cm
M = Distant Metastasis	
MX	Distant metastasis cannot be assessed
M0	No distant metastasis
M1	Distant metastasis

Detailed studies of the mandible, including panoramic radiographs and computed tomography scans, may be necessary to delineate the extent of the bony invasion as well as any involvement of the inferior alveolar canal. Melanomas and squamous cell carcinomas are known to be neurotropic and may spread along the inferior alveolar nerve via the mental foramen.[49–51] Patients who complain of numbness or paresthesia warrant further radiologic evaluation.

Surgical Treatment

The ultimate goal of lip cancer management is long-term control of the carcinoma with preservation of the competency and aesthetics of the perioral region. Although external beam, interstitial irradiation, or surgical excision can control small primary tumors of the lip equally well, surgery is quicker, maintains blood supply and soft tissue integrity, and leaves little aesthetic or functional impairment. Locally advanced lip cancers require planned surgical resection, with reconstruction in most cases. In the past 100 years, clinicians have employed many methods to manage lip cancer. Some of the less effective methods have included direct applications of caustic agents, such as hydrochloric acid, arsenic paste, or nitric acid. In addition, laser surgery, electrocoagulation, and cryotherapy have been advocated by some. However, the two modalities that have been the most thoroughly evaluated and that have undergone the test of time are surgery and radiation therapy. These two techniques yield excellent results for very

early lip cancers, and surgery is the most common treatment selected for managing lip carcinoma of any size, particularly the larger T3 and T4 tumors. Surgeon and patient limitations dictate treatment modalities. The surgeon's experience may limited the extent of the required resection for cure based on the tumor size at presentation. Prolonged general anesthesia and advanced reconstruction modalities such as microvascular free tissue transfer or complex local tissue transfer may not be reasonable given factors such as medical co-morbidities, patient compliance, and anticipated aesthetic and functional outcomes of the reconstructive efforts.

The determination of an adequate surgical margin around a lip cancer is somewhat nebulous, and few objective data have been gathered to substantiate any recommendations for adequate excision margins. The size of the primary lesion is the most common factor that we use to determine the extent of the marginal excision. Larger cancers have typically mandated wider margins than have smaller cancers. Based on these general guidelines, a minimum of 8 to 10 mm of normal tissue around a lip cancer is recommended to facilitate its complete removal.[2,9,33,52–54] Smaller lip cancers, less than 1 cm in greatest dimension, can often be managed with slightly smaller margins of 5 mm.[54] The lip shave, or vermilionectomy procedure, is ideal for those situations wherein areas of leukoplakia, actinic cheilitis, or carcinoma in situ involve the vermilion of the lips (Figure 33-5).[55–64] These premalignant conditions require treatment but not complete full-thickness excision of the lip. This operation involves partial or entire excision of the lip vermilion. The vermilionectomy may also be used, in conjunction with a full-thickness lip excision, in individuals possessing invasive lip carcinoma and premalignant vermilion changes. Following the vermilionectomy, the residual defect is primarily closed with labial mucosal advancement flaps. Minimization of the mucosal elevation to only the required surface area required for vermilian reconstruction will preserve oral mucosal surface sensory innervation.



FIGURE 33-5. Actinic cheilitis involving the vermilion of the lower lip.



FIGURE 33-6. The proposed incisions for a shield excision of lower lip are delineated.

In situations with invasive lesions, the lip shave procedure is contraindicated, and full-thickness excision of the involved portion of the lip is the traditional procedure for management. The most commonly selected configuration of lip excision is a V, a W, or a shield (Figure 33-6). The defects resulting from the V and W excisions can easily be closed primarily with no additional mobilization of adjacent tissues (Figure 33-7). The rectangular form of excision, however, requires advancement of laterally based lip flaps to achieve a satisfactory closure. The choice of local excisional geometry and primary closure should be based on design features that maintain the aesthetic subunit morphology.

Invasion of the mandible, involvement of the mental or inferior alveolar nerve, tumor sizes of T3 or greater, or associated regional lymph node metastasis generally necessitates a more aggressive resection. Aggressive treatment requires an excision and reconstruction that is more complex than the standard full-thickness V or W excision and is discussed in detail in the following section on "Lip Reconstruction," including a marginal mandibulectomy with the resection of lip cancers that approximate the alveolar ridge or outer labial cortex of the mandible. Likewise, for rare lesions that actu-



FIGURE 33-7. Primary closure of a W excision of the lower lip.

ally demonstrate radiographic invasion of the mandible, a segmental mandibulectomy should be included in the treatment plan.

Lip Reconstruction

Lip reconstruction after surgical excision of cancer should reestablish the function and appearance of the lip. The key to functional restoration is the reconstitution of the orbicularis oris muscle. Primary surgical restoration of the orbicularis muscle after resections that exceed two thirds to three quarters of the lip length will create microstomia.

Defects of the vermilion resulting from a lip shave procedure are generally restored with labial mucosal advancement flaps.[65–67] The labial mucosal flap develops by creating a plane between the minor salivary glands and the inner surface of the orbicularis oris muscle. This flap may be mobilized into the buccal vestibule if necessary. The flap is secured to the anterior cutaneous margin of the excision to create a new vermilion cutaneous border (Figure 33-8). Other less commonly used

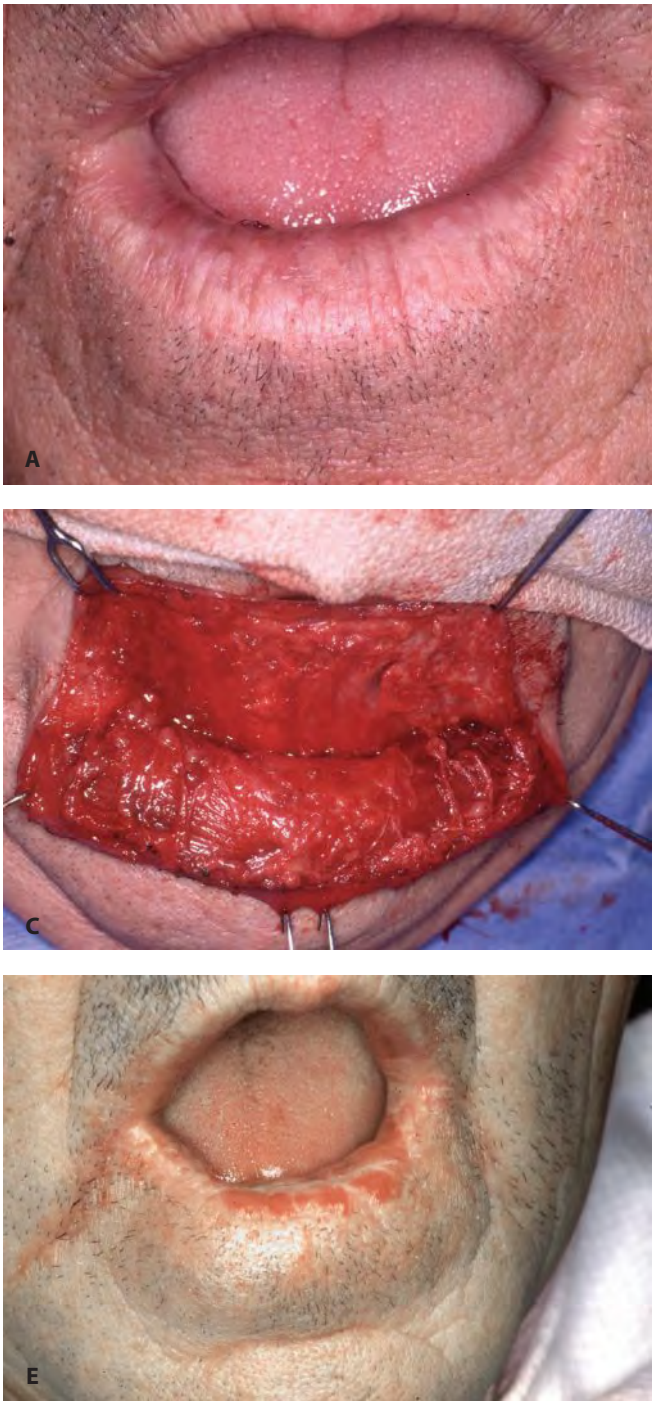


FIGURE 33-8. **A,** Actinic chelitis of the lower lip with atypia noted on biopsy. **B,** Completed excision of the lower lip vermilion. **C,** Complete undermining of the mucosa and removal of adjacent minor salivary glands help prevent rolling of the lower lip and mucocele formation, respectively. **D,** Mucosal advancement and primary closure of the surgical defect has been completed. **E,** Early postoperative results after the lip shave procedure.



FIGURE 33-9. Good aesthetic results after shield excision and primary closure of a right lower lip defect involving one third the total length of the lower lip.

flaps for vermilion reconstruction after a lip shave include cross-lip buccal mucosa flaps and tongue flaps.[67–69]

Closure may be achieved primarily when a full-thickness excision of the upper or lower lip results in a defect of up to one third of the lip length (Figure 33-9). The V-shaped excision design credited to Louis[70] is most commonly used when a primary closure is anticipated. Typically, the apex of the V is placed at or slightly above the nasolabial fold or labiomental crease and should be planned to parallel the relaxed skin tension lines.[65–68,71–73] The mucocutaneous margin can be marked before beginning the full-thickness excision to facilitate precise alignment followed by a minimum of a three-layered closure comprising mucosa, muscle, and skin as necessary to avoid unaesthetic notching of the lip as the scar matures.

Lip cancers that extend more deeply into the lip substructure but still involve a superficial length of vermilion that would otherwise produce a defect may be closed primarily via the W-shaped modification of the V configuration (Figure 33-10). This excision uses an M-plasty in place of the single apex of the V. A three-layered closure of the defect, with careful attention to detail in the reconstruction of the orbicularis oris muscle layer, is achieved.

The need to reconstruct lip defects greater than one third of the horizontal dimension mandates the use of adjacent tissue for reconstruction and has led to the development of various circumoral advancement flap techniques.[66–68,71,73–75] The most popular of these techniques includes the Karapandzic reconstruction flap (Figure 33-11). This flap consists of a transfer of the remaining lip tissue to reconstitute the lips and mouth opening, thereby maintaining a functional oral sphincter. The Karapandzic flap uses release incisions within the labiomental crease, extending around the region of the oral commissures and continuing superiorly within the nasolabial creases bilaterally. Combining sharp and blunt dissection separates the orbicularis muscles from the surrounding facial expression muscles. Neurovascular structures are preserved and transposed medially, along with the flap, and intraoral buccal mucosal release incisions are often necessary.

For this reason, the Karapandzic flap is ideally suited in situations in which two thirds to three quarters, or more, of the lower lip is resected, particularly when the resection is centrally located and leaves the lateral ends near the commissures intact. The incisions for elevation of the Karapandzic flap require mobilization of the skin and subcutaneous tissues that are superficial to the orbicularis oris muscle and mucosa and deep to the orbicularis oris muscle. At the same time, the muscle itself must be kept intact, with its nerve and blood supply preserved as tissues are rotated and sutured medially.

Among other reconstructive options for the lip, the cross-lip or opposing flaps are particularly useful in repairing moderate lip defects of one third the length of one lip.[71,76–78] These techniques transfer a full-thickness segment of lip tissue into a defect on the opposite lip. Estlander and Abbé developed the most commonly used cross-lip flap repair techniques.[77,78] Although the Estlander and the Abbé nomenclatures are used interchangeably and incorrectly at times, both techniques illustrate the important principle of tissue sharing from a normal lip to reconstuct the resected opposing





FIGURE 33-10. **A,** Typical presentation of squamous cell carcinoma of the right lower lip. **B,** W excision of the lesion sparing the right commissure.

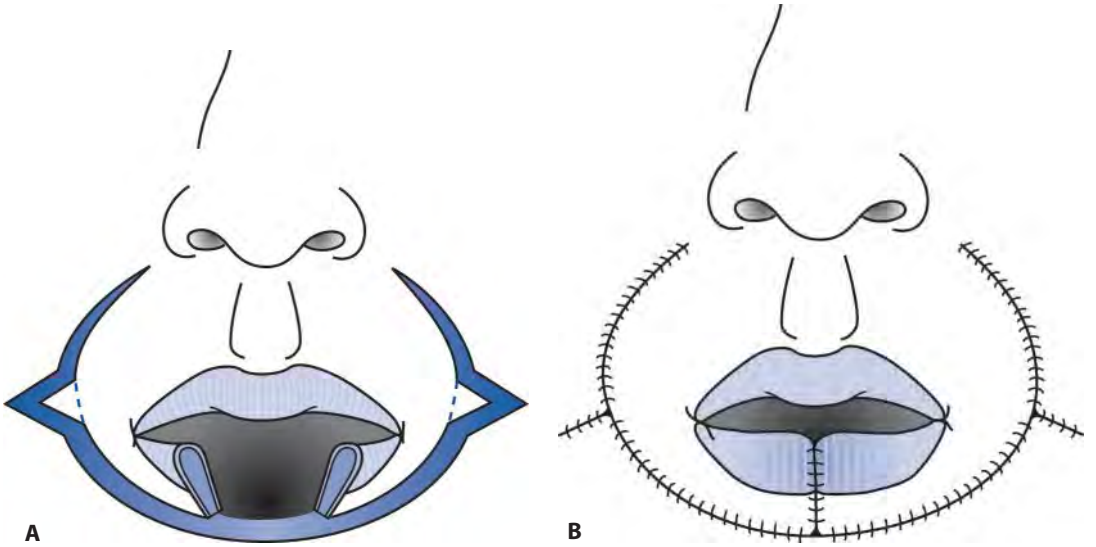




FIGURE 33-11. **A,** Defects greater than one third of the lip may require circumoral flaps, such as the Karapandzic flap depicted by this schematic. **B,** Schematic depicting the closure of the Karapandzic flap. **C,** Aesthetic postoperative results of a Karapandzic flap can be achieved. Note the recurrence of the lesion in the midline.

lip. Variations on either technique are limited only by the imagination and creativity of the reconstructive surgeon. The Abbé flap, as originally described, transfers tissue from the lower lip to a defect in the central component of the upper lip. It is, however, most often used to reconstruct lower lip defects by transferring tissue from the upper lip (Figure 33-12). The Estlander flap was used to reconstruct defects of the upper or lower lip in a single stage by transferring lip tissue around the oral commissure (Figure 33-13). All cross-lip flaps are generally referred to as *Abbé-Estlander type flaps.*

The principle of the Abbé-Estlander flap repair is that the width of the base of the triangular flap is one half that of the width of the base of the triangular surgical defect. The vertical length of the flap should match that of the defect. The cross-lip flap includes and depends on a small pedicle that carries the labial artery from the donor lip.

Generally, mark out the flap on the upper lip on the same side as the planned excision (Figure 33-14). Make a skin incision at the previously marked outline of the Abbé-Estlander flap on the lateral aspect of the upper lip. The lateral incision is deepened through both the musculature and the mucosa, extending from the vermilion border up to the



FIGURE 33-12. A drawing of the two-stage Abbé flap demonstrates the cross-lip transfer that is divided at approximately 3 weeks postoperatively.



FIGURE 33-13. A schematic drawing of the one-stage Estlander flap with the classic rounding of the commissure region.

apex of the flap. With extreme caution, perform an incision along the medial margin of the flap, beginning at the apex of the flap and working toward the vermilion border to avoid injury to the labial artery. As mobilization of the flap toward the vermilion border proceeds, separate the musculature of the upper lip bluntly with a hemostat and divide small segments of the muscle fibers a little at a time with scissors. Once the labial artery is identified, under direct vision, divide the other attachments of the musculature of the upper lip around the labial artery while keeping the mucosa of the vermilion border intact. In addition, to allow flap rotation, divide the intraoral labial mucosa on the medial aspect of the flap from the apex of the flap toward the lip. Rotate the flap 180 degrees to fill the surgical defect in the lower lip.



FIGURE 33-14. In the cross-lip flap, the transfer portion in most cases need only be 50% as large as the defect. Care should be taken to prevent injury to the superior labial artery.



FIGURE 33-15. Typical appearance of the cross-lip transfer flap at the time of suture removal, demonstrated in this lower to upper lip transfer.

Inset of the flap begins by accurate approximation of the vermilion edges of the flap and the lower lip, followed by careful multilayered closure. Bring the vascular pedicle across the open mouth and perform the second-stage release 3 weeks later (Figure 33-15). Preoperatively, instruct the patient to avoid trauma to this intervening pedicle during the immediate postoperative period.

When one commissure of the lip must be sacrificed along with the excision of the lip cancer, employ a nonbridged Estlander flap (Figure 33-16). This flap is created as a single-stage procedure without the need for secondary pedicle division. The downside of this type of flap is the development



FIGURE 33-16. At times, an extension into the labiomental crease may aid in closure of the defect, as seen marked during this cross-lip transfer flap.



FIGURE 33-17. In situations that do not require reconstruction of the vermilion surface of the lip, a cutaneous advancement flap may be appropriate, as in this case with Burow's triangles to aid in advancement.

of a somewhat unnatural, rounded commissure. The Abbé-Estlander flap can be used in reverse when a lesion of the upper lip is excised by elevating the flap from the lower lip. Alternatively, a cheek advancement flap with Burow's triangle is often useful for repairing lateral defects of the upper lip (Figure 33-17).

Several techniques designed for lip defects are too extensive for reconstruction using the Karapandzic or Abbé-Estlander techniques.[79–81] These techniques use adjacent cheek tissue in the form of laterally based advancement flaps. In the Bernard flap, the lower lip may be excised in its entirety, along with soft tissues of the mental region, and the resulting defect is closed by lateral cheek flaps to form a new lower lip (Figure 33-18). Webster and coworkers'[79] modification of Bernard's original description incorporates Schuchardt flaps to facilitate cheek advancement, allowing for setback of the commissure and to reduce the incidence of a "fish-mouth" deformity. This is accomplished by excising triangles of the skin from both sides of the upper lip. Preserve the mucous membrane to help form a new vermilion border. Excise the triangular wedges of skin from the nasolabial crease on both sides, subsequent to excision of the primary tumor. The base of this triangular excision extends from the commissure of the mouth, up to the nasolabial crease, depending on the width of the cheek flap to be mobilized (Figure 33-19A). After excising the triangular wedges, incise the mucosa from their inner aspect, except for the base, and shift the triangular flaps of the upper lip mucosa medially, along with the flaps (see Figure 33-19B). Make a counterincision in the lower mucogingival sulcus bilaterally, and mobilize both cheek flaps medially. Perform a closure of the lip musculature on both sides with interrupted sutures. The triangular wedges of the mucosa from the upper lip are everted and rolled inferiorly to provide a new vermilion surface. Mucosal closure is completed inferiorly in the mucogingival sulcus (see Figure 33-19C and D).

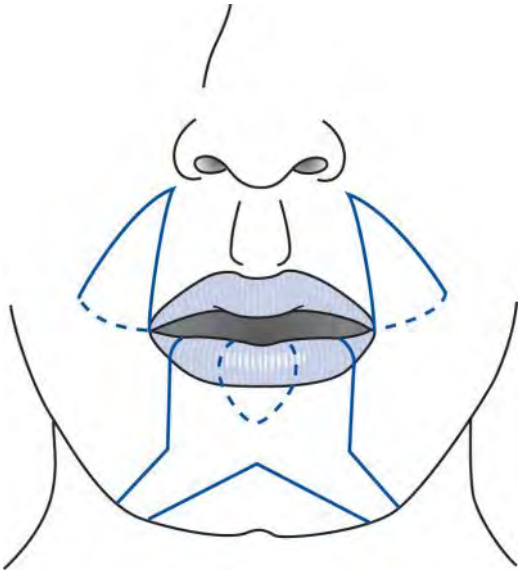


FIGURE 33-18. In situations requiring complete excision of the lower lip, the Bernard flap may provide a means for reconstruction with circumoral tissues, depicted in this schematic drawing.

The main advantage of the modified Bernard flap is its ability to reconstruct almost the whole lower lip in a single-stage procedure. The main disadvantages are potential notching at the lower lip midline closure resulting in diminished competency and saliva control as well as reduction of the size of the orifice and creating a so-called permanent smile deformity of the lips, most often produced in edentulous individuals.

The reconstruction of more massive defects that include total lip excision, as well as excising the adjacent floor of the mouth, skin, or mandible, requires the use of distant flaps, such as the deltopectoral or pectoralis major myocutaneous flap. Alternatively, use free vascularized composite flaps to reconstruct these large defects. A free flap that has been shown to be particularly useful is the composite radial forearm–palmaris longus free flap.[82–87] (Figure 33-20)

Cervical Lymphadenectomy

Patients with early cancer of the lip (stages I and II) do not generally need elective treatment of the cervical lymph nodes because the rate of occult metastasis is low. The risk for cervical metastasis increases with poorly differentiated cancer, recurrent cancer, or cancer that extends into the labial mucosa or that invades the mandible. Given the infrequency with which stage I and stage II lip cancers spread to regional lymph nodes, elective treatment of the neck is not always required. One report indicated that there was delayed cervical metastasis between 35% and 40% from lip cancer tumors 2 to 4 cm in size.[4] This report confirms a much larger rate of metastasis than that usually seen in clinical practice.

With advanced disease (stages III and IV), elective neck dissection of levels I through III is recommended (Figure 33-21). Thus, even if the patient has no palpable adenopathy



FIGURE 33-19. **A,** Proposed incisions for a classic Bernard flap reconstruction of the lower lip. **B,** The development of the Bernard flap after resection of the lower lip. **C,** Closure of the wound includes the mucosal layer, orbicularis oris layer, dermal layer, and the skin closure. **D,** Postoperative follow-up at 1 year demonstrates no recurrence and good aesthetic results.

(N0 neck), the clinician should still use elective radiation therapy or elective neck node dissection in managing patients, owing to the high rate of microscopic lymph node metastasis in these patients. Metastasis from carcinoma of the lower lip to lymphatic basins at levels IV and V is excedingly rare. In patients with lesions of the upper lip, commissure, or both, include a superficial parotidectomy. Clinically apparent lymph nodes require either radiation therapy or neck dissection for N1 nodes and combined therapy (neck dissection and radiation) for N2 and N3 nodes involvement.[88]

TREATMENT RESULTS

The cure rate for T1 and T2 lip cancers without regional metastasis is greater than 90% with surgery or radiation.[4,58] The 5-year determinate survival is approximately 80%.[1] The cure rates for cancer of the lips suggest a better prognosis than for other cancers of the oral cavity. Cancer involving the oral commissure is more aggressive, with a 5-year cure rate ranging between 34% and 50%. Cancers that include areas larger than 2 cm have cure rates of less than 80%, and those that invade deep enough to involve the mandible have a cure rate of less than 50%.[4,89] The primary cause of failure is local recurrence rather than regional node metastasis. Other adverse prognostic factors include poor histologic grade, tumor thickness greater than 6 mm, desmoplasia, stromal sclerosis, muscular invasion, and perineural invasion.[90–92] Angiogenesis has not been shown to have prognostic significance.[93] While *TP53* mutations are seen in 50% of lip cancers, the clinical significance of this observation is unknown.[94]

Generally, elective lymph node dissection in the N0 neck is reserved for advanced stage disease (stage III and stage IV).[89,95] Approximately 5% to 10% of patients with lip cancer will develop evidence of nodal involvement.[1,96,97] Without question, the presence of cervical lymph node metastasis affects survival. The average 5-year survival for patients with cervical metastasis of lip carcinoma is approximately 50%, with a range of 29% to 68%. Recurrence rates in the neck after treatment of regional metastasis are 40% for N1 disease and up to 100% for N3 disease.[1] The risk of developing a metachronous lip cancer is estimated at about 20% by 10 years follow-up.[98]







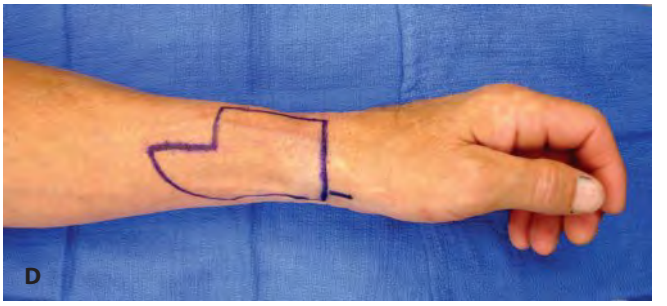




FIGURE 33-20. Free flap reconstruction of a radical resection of the lower and lateral upper lip: **A** squamous cell carcinoma involving the entire lower lip and one third of the lateral upper lip, **B** resection plan, **C** resection defect, **D** proposed radial forearm free tissue transfer design and **E** reconstructed resection defect combining a radial forearm free tissue transfer and modified Bernard advancement flap.

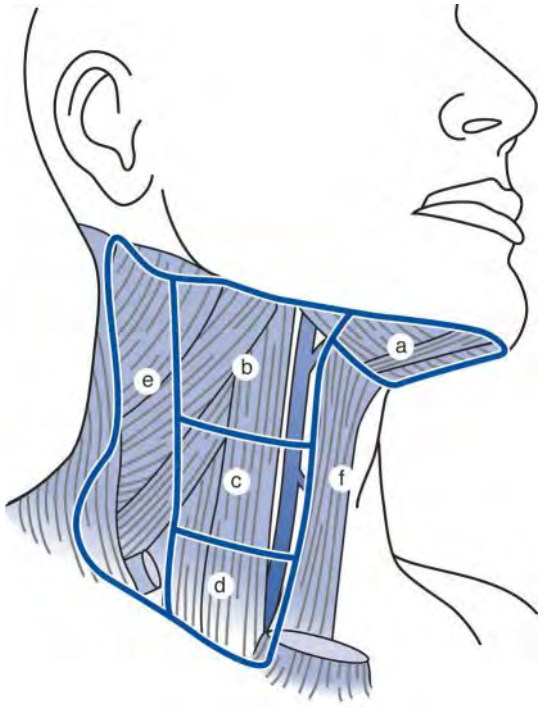


FIGURE 33-21. Diagram depicting the levels of the neck. *a* = level one; *b* = level two; *c* = level three; *d* = level four; *e* = level five; *f* = level six.

CONCLUSIONS

Lip cancer accounts for a significant percentage of all head and neck malignancies in the United States. Lip cancer arises from the lower lip in nearly 90% of cases. Etiologic factors associated with lip cancer include sun exposure, alcohol, and tobacco abuse. Commissure involvement is an adverse prognostic factor. Regional cervical lymph node metastasis is directly related to a poor prognosis. With overall cure rates of 80% to 90%, lip cancers have a more favorable prognosis than most other head and neck cancers.

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34

CHAPTER

Head and Neck Skin Cancer

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Skin cancer is the most common cancer afflicting mankind. In the United States alone, an estimated 1 million new lesions are diagnosed each year.[1] Skin cancer may be grouped into two subsets: nonmelanoma skin cancer (NMSC) and melanoma. NMSC accounts for 95% of all skin cancers. Melanoma, of which 1% to 8% occurs in the head and neck, fills the remaining 5%.[2–4]

Even with this low incidence, melanoma is responsible for about 75% of skin cancer–related deaths. The overall mortality for NMSC is relatively low, with an estimated 5-year survival rate of 95%.[5,6] Regardless, NMSC may be locally aggressive, leading to significant morbidity, disfigurement, loss of function, and high health care costs.[7] This chapter focuses on the epidemiology, etiology, clinical characteristics, and management (medical and surgical) of these cutaneous malignancies.

NONMELANOMA SKIN CANCER

Epidemiology

The NMSCs include basal cell carcinoma (BCC; 75% of NMSCs), squamous cell carcinoma (SCC; 20% of NMSCs), and a few rarer malignancies, such as Merkel cell tumor, dermatofibrosarcoma protuberans, and adnexal tumors.[5] Incidence data for the United States should be interpreted skeptically because most NMSCs are treated in outpatient clinics or private offices and are not routinely reported to cancer registries.[3] Reported yearly skin cancer rates are approximately 2 in 1000 in the continental United States, 1 in 100 on the island of Kauai, and greater than 2 in 100 in Australia.[8]

Epidemiologic studies demonstrate a positive correlation between ultraviolet (UV) radiation exposure and the incidence of NMSC. NMSC is predominantly a white disease (=98%) but does occur in blacks and Hispanics.[3] The risk of developing NMSC increases the closer one lives to the equator and the more one's outdoor activities increase the concentration of sun exposure through reflection.[8,9] Examples of the latter include work around snow, water, cement, and roofing.

For all races, 75% of NMSCs appear on body areas most chronically exposed to sunlight, such as the head, face, neck, and dorsum of the hands.[5] The incidence of BCC and SCC at early ages is comparable for men and women, but men older than 45 years have a three times greater incidence of NMSC, specifically SCC.[10,11] In men, common sites are the ears and nose, whereas in women, the nose and lower extremities are most common.

The incidence of NMSC had been increasing for decades. The mortality rate, however, has recently leveled off and is now beginning to decrease, perhaps owing to public information programs.[12] Overall, NMSC has an excellent prognosis, but approximately 2000 deaths occur annually, three fourths of which are from metastatic SCC.[13,14]

Etiology

The etiology of NMSC is multifactorial but can be broadly categorized into host-related and environmental causes. Host factors include an individual's phenotype, genetic syndromes, precursor lesions, and immunologic issues. Environmental variables include exposure to UV radiation, ionizing radiation, and chemicals.[2]

Host Factors

Tanning is the body's defense mechanism against NMSC. One's ability to tan is directly related to the amount of melanin in the skin, which is genetically determined and cannot be influenced. Skin melanin determines a person's photosensitivity. The more melanin an individual has, the less damage UV radiation inflicts. Deleterious effects of UV radiation are attenuated by the stratum corneum via refraction, reflection, and direct absorption by melanin.

Fitzpatrick[15] classified skin into six different groupings or types (Table 34-1). Each group was categorized based on the

TABLE 34-1. **Fitzpatrick Skin Types***

Type	Characterization
1.	Always burns easily, shows no immediate pigment darkening, and never tans.
2.	Always burns easily, shows trace immediate pigment darkening, tans minimally and with difficulty.
3.	Burns minimally, + immediate pigment darkening, tans gradually and uniformly (light brown).
4.	Burns minimally, ++ immediate pigment darkening, tans well (moderate brown).
5.	Rarely burns, +++ immediate pigment darkening, tans very well (dark brown).
6.	Rarely burns, +++ immediate pigment darkening, tans profusely (black).

*Fitzpatrick skin phenotypes portray the outcomes of 30 minutes of sun exposure at midday in the northern hemisphere: + indicates a relative level of pigment darkening, with +++ being the highest.

From Fitzpatrick TB. The validity and practicality of sun reactive skin types I through VI. Arch Dermatol 1988;124:869–871.

results of 30 minutes of direct sunlight to the skin in the northern hemisphere. The groups are based on the amount of melanin an individual possesses, inherent pigmentation, and sensitivity to UV light. For example, a person in type 1 is the classic freckle-faced, light-eyed redhead who burns and never tans, a Celtic type. People in type 1 are highly susceptible to skin cancer but remarkably also heal from reconstruction wounds with the least perceptible scar. Type 2 is typified by the blond-haired, blue-eyed person, a Scandinavian type. A type 3 person has olive skin and often dark eyes and occasionally burns but tans readily, a Mediterranean type. The descent of people in type 4 is Hispanic, type 5 is Arabic/Indian, and type 6 is African. Remarkably, as resistance to skin cancer increases, scarification becomes more obvious, often pigmenting or forming keloids.

Host factors contribute to a patient's ongoing risk of developing new cancers. A patient with a prior history of NMSC has a 36% to 52% 5-year risk of another cancer arising.[16,17]

Syndromes

Genetics plays a starring role in determining who gets skin cancer. Newer drugs to treat skin cancers, for example, 5% imiquimod cream applied topically three times per week, show great promise in treating skin cancers through inherent immune responses.[18] Several syndromes predispose a person to skin cancer:

- Basal cell nevus syndrome (Gorlin's syndrome) is an autosomal dominant disorder characterized by multiple BCCs, odontogenic keratocysts, bifid ribs, scoliosis, brachymetacarpalism, palmar and plantar pits, calcification of the falx cerebri, prominent supraorbital ridges, and hypertelorism. The BCCs that are produced look like small nevi (Figure 34-1) but act just like common nodular BCC. Control with a CO_2 laser or curettage and electrodesiccation (C&E) is critical before enlargement destroys anatomic structures.[19–21]
- Xeroderma pigmentosa is an autosomal recessive disorder resulting in defects in repair of DNA. UV radiation results in skin DNA damage; therefore, xeroderma pigmentosa is characterized by hypersensitivity to sun exposure and the development of multiple skin cancers. Children with this disorder must modify their lifestyles to function as night people. There are summer camps for them, at which activities begin at their wake-up time—sundown. Albinism is an autosomal recessive disorder resulting in the absence of melanin with a subsequent increase in development of skin cancer, especially SCC
- Epidermodysplasia verruciformis is an autosomal recessive disorder. It results in the development of BCC from flat warts in sun-exposed areas in homozygous individuals infected with human papillomavirus type 3 or 5

Predisposing Lesions

Several congenital and acquired lesions predispose to skin cancer:

- Nevus sebaceus of Jadassohn is a well-circumscribed. slightly raised, hairless lesion on the scalp or face present at birth that becomes verrucous and nodular during puberty. Approximately 10% of such lesions undergo malignant transformation to BCC (Figure 34-2)
- Actinic keratosis (AK), also known as solar or senile keratosis, is the most common precancerous lesion of the epidermis. AK is characterized by red, yellow, brown, or colorless macules or papules with scaly irregular surfaces, ranging in size from a few millimeters to several centimeters. Left untreated, there is a 10% to 13% risk of malignant transformation of AK to SCC (Figure 34-3); therefore, the American Academy of Dermatology recommends treatment.[22–25] All suspicious lesions should undergo biopsy. Treatment options include chemical peel, laser, cryotherapy, C&E, tangential excision, or 5-fluorouracil (5-FU)
- Cutaneous horns are hard keratotic growths that protrude from the skin. Histologically, they are advanced AK. Approximately 10% of these lesions have an underlying SCC.[26]

Immunologic Factors

Immunosuppression predisposes a person to several types of cancers including skin cancer. Immunosuppression alters the immune surveillance mechanism that typically destroys potentially malignant cells.[3] Human immunodeficiency virus (HIV) infection, lymphoproliferative disease, occult malignancy, organ transplantation, and a variety of other medical conditions result in immunosuppression. Renal transplantation patients on long-term immunosuppressive therapy not only have a higher incidence of SCC and metastasis, but also their tumors appear years earlier than in any control population.[27–30] However, most studies suggest that some other risk factor such as ionizing radiation or viral infection, along with a decreased immune system, is necessary for the development of these tumors in this subset of patients.[3,31]



FIGURE 34-1. This 30-year-old woman was first seen 10 years earlier for treatment of multiple odontogenic keratocysts. **A,** The small pigmented dots are all incipient or growing basal cell skin cancers, which are slow growing and less aggressive than most cancers resulting from sun exposure. **B,** Intraoperatively. Excision is indicated for the larger lesions; curettage and electrodesiccation or CO_2 laser is used for incipient lesions. **C,** Improvement is noted after 1 month.

Environmental Factors

Ionizing radiation, certain chemicals, and skin damage from the environment can also cause skin cancer. UV radiation has been fingered as the primary environmental culprit. There are three types of UV radiation: UVA (320–400 nm), UVB (290–320 nm), and UVC (200–280 nm). UVB rays are the most carcinogenic, triggering skin cancer via photochemical damage to DNA, injury to DNA repair mechanisms, and partial suppression of cell-mediated immunity.[5,32] UVA, originally thought to be harmless, is now known to enhance the effects of UVB as a cocarcinogen.[3] Most UVC is filtered out by the ozone layer. As the ozone layer thins, as it has over Antarctica and parts of Australia, UVC enhances the development of skin cancer. The most common historic reports for NMSC as well as melanoma are two to three childhood blistering sunburns or 3 years or more of intense sun exposure.

A comment must be made about the two methods of tanning used in tanning parlors. The method using UVA light, in our estimation, enhances new skin cancers.[32] We have seen skin cancers even in teenagers who have used tanning parlors. The "California" spray tan, a skin dye that lasts for 3 to 5 weeks, is harmless.

Chemicals such as arsenic, polycyclic aromatic hydrocarbons, and psoralens used in combination with UVA (a treatment for psoriasis) have all been implicated as originators for NMSC.[33] Patients suffering from chronic inflammatory skin conditions, such as chronic radiation keratosis, burn scars, and ulcers, have an increased risk of developing skin cancers.

Prevention

Although a doctor may be capable of treating skin cancer effectively, the informed patient is the greatest resource against the development of new cancers. Preventive measures can be classified into three types: sunscreens, clothing, and education. Sun protection is rated by sun protection factor (SPF). The SPF is a ratio of the smallest amount of radiation needed to produce erythema on protected skin compared



FIGURE 34-2. Nevus sebaceus.

with the same degree of redness produced on unprotected skin. Sunscreens function either chemically or physically. Chemical sunscreens, such as para-aminobenzoic acid, benzophenones, and cinnamates, reduce UV skin penetration by absorbing solar rays. Physical sunscreens including titanium dioxide, zinc oxide, and kaolin act as physical barriers.



FIGURE 34-3. Scaly actinic keratoses on the scalp, some of which have progressed to squamous cell carcinoma (in situ).

It is now known that regular application of SPF 30 sunblock may reduce the evolution of new actinic keratoses by 50% after 3 years. In addition, patients should be told that the application of SPF 30 does not abrogate the need for behavioral modification. For example, a farmer who applies SPF 30 at the start of the day will have the protection wear off shortly after a few hours of sweating.

Clothing may provide some protection, but a wet T-shirt has an SPF of 0 to 1.[33] High SPF clothing is available, but it is expensive and not very comfortable.

Basal Cell Carcinoma

BCCs originate from pluripotential cells in the epidermis and hair follicles. They are often slow growing and may take years to enlarge significantly.[33]

Typically, patients with BCC are categorized as Fitzpatrick types 1 to 3 with a history of sun exposure. Eighty percent to 93% of the cancers occur on sun-exposed areas of the head and neck, and 26% to 30% occur on the nose.[34] BCCs can be divided into several subtypes: superficial, noduloulcerative (or nodular), pigmented, infiltrative, micronodular, morphea-like, and basosquamous.

Superficial BCC represents approximately 10% of all BCCs (Figure 34-4).[35] They present as slightly elevated plaques or discrete macules that may be scaly. They can resemble eczema or fungal infections.

Noduloulcerative BCC is the most common type, accounting for approximately 75% of all BCCs (see Figure 34-4).[35] Clinically, they present as well-defined translucent pearly nodules that are either round or oval with rolled borders and occasional ulcerations. Telangiectasias are commonly seen coursing through the lesion.

Pigmented BCCs range from brown to blue-black and can be mistaken for melanoma. Morphea-like BCCs present as firm plaques that are yellow or white with an ill-defined border. They can be quite large and do not show more than 1 to 2 mm elevation. This tumor is likely to have positive margins after excision.

Basosquamous carcinomas have both basal and squamous cell differentiations. They have a higher growth rate as well as a higher metastatic potential than do other BCCs.

Micronodular, infiltrative, and morphea-like BCCs are the more aggressive variants of BCC and together account for 10% of BCCs.[7]

Death from BCC is rare, with a rate of metastasis of 0.0028% to 0.1%.[36] Size, depth of invasion, and histologic type are important predictors for metastasis.[10] Favored sites of metastasis include regional lymph nodes, liver, lung, bone, and skin. This rare metastasis is twice as common in males as in females.[36]

Squamous Cell Carcinoma

SCC is the second most common skin cancer and accounts for 20% of all NMSC cases. SCC is a malignant proliferation



FIGURE 34-4. **A,** Superficial and noduloulcerative basal cell carcinomas (confirmed with biopsy) are excised under local anesthesia in the office. The specimens are sent for permanent histopathology. **B,** Five days later, in the operating room, the defects are ready for reconstructive closure. **C,** Finger pressure (inherent elasticity) reveals that the largest defect is tight but amenable to advancement flap closure. **D,** The upper defect is presutured with 3-0 nylon (mechanical creep). **E,** Margins are excised (0.5- to 1-mm sharp squaring of the rounded edges with scraping of debris) and complex closures of the lower defects are accomplished. **F,** The nylon sutures are released and the tension-free upper defect is repaired with an advancement flap and M-plasty. **G,** Results at suture removal 1 week later.

of epidermal keratinocytes. Histologically, SCC is composed of nests and cords of atypical squamous cells from the epidermis infiltrating into the dermis; it often contains keratin pearls. The lifetime risk of developing SCC is 4% to 14%, and the incidence has increased by 20% in the past decade alone.[11,37,38]

Men with a fair complexion who are older tha 50 years and have had heavy sun exposure in the past several years typically get multiple actinic keratoses and SCCs. SCC presents as a painless poorly defined erythematous nodule with raised borders (Figure 34-5). Cutaneous horns or a hyperkeratotic crust with ulcerations may be present. The surrounding skin may reveal signs of chronic sun damage.

Unlike BCC, SCC may grow rapidly and metastasize. Metastasis is most common in lesions greater than 4 mm deep. The cumulative rate of metastasis is between 2% and 6%, and the 5-year survival rate for metastatic SCC is only 34%.[11,38] Metastasis can occur either through the lymphatics or by hematogenous spread, with common sites being the regional lymph nodes, the lungs, and the liver. The location of the primary lesion influences the rate of recurrence and metastasis. SCCs occurring on the lip, ear, melolabial crease,

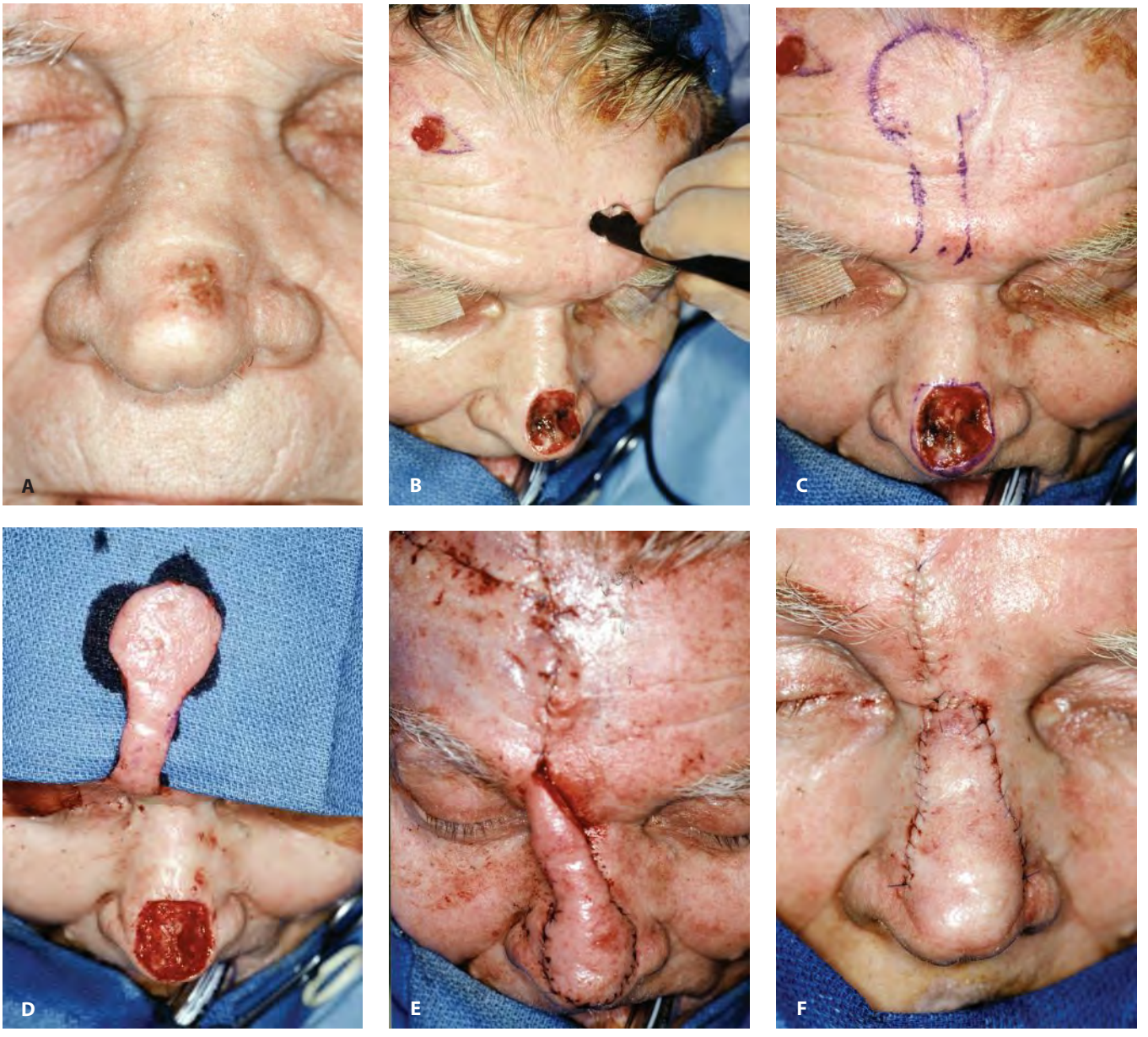


FIGURE 34-5. **A,** This 60-year-old has confirmed squamous cell cancer on his nasal tip (and basal cell cancers elsewhere). **B,** Five days after excision of the cancers, with permanent histology ensuring margin clearance, the patient undergoes Doppler ultrasonography in preparation to identify the location of the axial vessel for a paramedian forehead flap. **C,** The flap is designed on the right supratrochlear vessels. **D,** The lower recipient site has been trimmed sharply, and the pink vascular flap is ready for inset. **E,** The flap in place. **F,** The sutured flap revascularizes for 3 weeks. At this time, it may be thinned again or divided and inset according to topographic principles. *(Continued)*

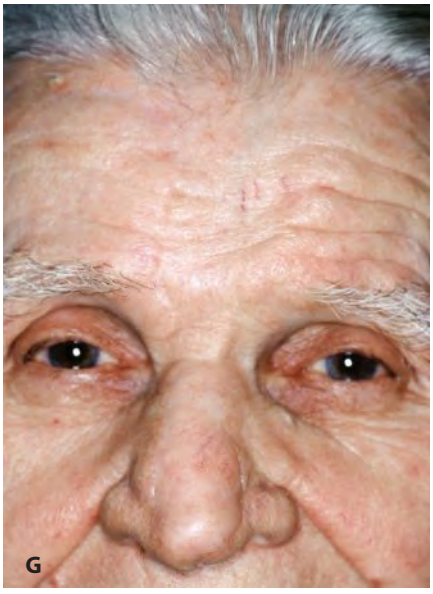


FIGURE 34-5. *(Continued)* **G,** Two months postoperative, the results of surgery are inconspicuous.

and periorbital and preauricular areas have higher rates of recurrence and metastasis (10–14%).[38,39]

The most common precursor for SCC is AK. The rate of transformation of AK to SCC is 1 in 1000 per year.[39] Approximately 40% of people older than 40 years have had at least one AK. Keratoacanthoma is a commonly confused with SCC, both clinically and histologically. Keratoacanthoma is a self-healing raised growth lesion with a central keratin-filled plug. It grows quickly but often spontaneously involutes after 2 to 6 months, leaving only a depressed white scar.

Bowen's disease is an in situ SCC presenting as a slow-growing erythematous scaling plaque with an irregular but sharp outline. These lesions rarely transform into invasive SCC.

SCC may evolve from chronically unhealed or unstable wounds, burn scars, or ulcers. These lesions, sometimes called Marjolin's ulcers, have a 20% higher rate of lymph node metastasis than does UV-induced SCC.[40,41]

Histologic features, such as the degree of differentiation, depth of invasion, and perineural involvement, as well as tumor size are prognostic indicators that may dictate selection of width of the excisional margin. More differentiated lesions have a lower invasive tendency and, hence, a better prognosis. Larger tumors and those that invade deeply along tissue planes have a greater risk of recurrence and metastasis. Tumors greater than 2 cm have a twofold increase in recurrence rate and are three times more likely to metastasize. Tumors arising in scars or wounds are usually more aggressive and have a metastatic rate between 18% and 38%.[7] With SCC, the first shot at cure is crucial because recurrent SCCs have a metastatic rate of 24% to 45%; if they metastasize, the 5-year survival rate is approximately 50%.[7]

MELANOMA

Melanoma is a potentially deadly and aggressive neoplasm resulting from the malignant transformation of melanocytes. The incidence of melanoma is increasing faster than any other cancer. It is estimated that the frequency of melanoma will double every 10 to 15 years, and that more than 40,000 new cases of melanoma will be diagnosed this year in the United States alone.[7,41,42] Melanoma occurrence increases the closer one's residence is to the equator, with residents of Queensland, Australia, having the highest rate of melanoma, approaching 1 in 14.[43] An estimated 1 in 75 people develop melanoma in their lifetime, up from 1 in 150 persons in 1985.[44] Melanoma accounts for more than three times more deaths than the combined fatalities from all other skin malignancies.[45]

Risk Factors

People in Fitzpatrick groups 1 and 2 are the most susceptible to melanoma. The role of UV is not precisely known for melanoma, but lots of freckles and a history of three or more blistering sunburns, the use of a tanning bed, and having undergone psoralen plus UVA therapy have been implicated.[43,46]

Several additional risk factors for melanoma have been identified. About 10% of patients with melanoma have a first-degree relative with the disease.[47] "Common moles," also known as acquired melanocytic nevi, can be a risk factor. Individuals with more than 100 of these moles have a 10-fold risk of developing melanoma.[4,48] When combined with a family history of melanoma, dysplastic nevi (atypical moles), which are present in approximately 10% of the population, represent a significantly increased risk of developing melanoma. Congenital (black hairy) nevi have a 4% lifetime risk of developing into melanomas.[43] Lentigo maligna, or melanotic freckle of Hutchinson, is a precursor in situ lesion that becomes malignant in approximately 5% of cases (Figure 34-6). Thirty percent of melanomas arise from preexisting lesions, whereas 70% arise de novo.

Clinical and Histologic Description

The mnemonic *ABCD* is useful in categorizing the characteristics of melanomas: *a*symmetry, *b*order irregularity, *c*olor changes or variation, *d*iameter of lesion (< or > 6 mm). The practitioner should not place the patient under casual observation (i.e., not perform a biopsy) just because these common indicators might be absent. Approximately 40% of board-certified dermatologists and more than 50% of other clinicians do not identify melanoma correctly by clinical intuition alone. Other suspicious factors include the color pink in a dark lesion and persistent itching.

Melanoma in situ is an intraepithelial lesion that can progress to an invasive lesion. When it is still in the epithelium, it is described as being in a *horizontal growth phase*, but when it invades dermis and approximates blood vessels,



FIGURE 34-6. **A,** This 65-year-old man has a lentigo maligna on his cheek and a basal cell cancer on his nose. **B** and **C,** After margin clearance by permanent histology (7 days later), the cheek defect and the nasal defect are each repaired with a rotational flap and back cut. **D,** An excellent aesthetic result is evident.

it is in a *vertical growth phase and thickens.* Hence, deeper melanomas are more deadly. Histopathologically, malignant melanoma presents as a proliferation of atypical melanocytes. The tumor originates at the epidermal-dermal junction. The cells then invade upward into the epidermis or extend downward into the dermis.

Melanomas are categorized into four main clinical and histologic subtypes: superficial spreading melanoma, nodular melanoma, lentigo maligna melanoma, and acral-lentiginous melanoma. Superficial spreading melanoma accounts for 70% of all melanomas. Clinically, superficial spreading melanoma is a flat or slightly elevated dark lesion with asymmetrical borders; it can be present for up to 5 years before invasion of the dermis.

Nodular melanoma is the second most common variant, accounting for 15% to 30% of melanomas. It appears as a raised black, brown, blue, or red nodule, perhaps with ulcerations, bleeding, or crusting. It may look just like a BCC, but contrary to BCC, the lesion grows rapidly over a few months. Approximately 5% of nodular melanomas lack pigmentation and are pinkish "amelanotic" melanomas. Nodular melanomas are thicker and metastasize rapidly.

Lentigo maligna melanoma accounts for 4% to 10% of melanomas. It arises in sun-exposed areas and occurs in the elderly.

Acral-lentiginous melanoma accounts for 2% to 8% of all melanomas in whites but is the most common type in African Americans, Asians, and Hispanics. Clinically, they present as pigmented lesions with irregular borders. Papules and nodules are frequently seen within the lesion.

Biopsy is the only fail-safe method to prove or disprove melanoma. If melanoma is suspected, incisional and excisional biopsies are much more diagnostic and prognostic than is a shave biopsy. Regardless, if a shave biopsy is performed and melanoma returns as the diagnosis, the next step is to obtain a full-thickness specimen (via punch or incisional

biopsy) to ascertain the diagnosis and confirm true depth. Neither incisional nor excisional biopsy disseminates tumor.

Incisional biopsy should be reserved for lesions greater than 2 cm or those located at anatomically restricted areas (e.g., eyelids, ears). The biopsy should be at the most raised site or the darkest area of the lesion (Figure 34-7). Full-thickness excisional biopsy with a 2-mm margin is the preferred method for lesions smaller than 2 cm.

Once the diagnosis has been established, melanoma is staged either by measuring the tumor depth from the granular cell layer of the epidermis to the farthest depth of tumor invasion (Breslow's classification) or by determining the anatomic level of invasion (Clark's classification). Melanomas measuring less than 0.76 mm have a 5-year survival rate of greater than 93%, whereas lesions larger than 4 mm thick have a 5-year survival rate of less than 50%.[49,50] Melanomas with ulceration or histologically high mitosis rates predictably worsen prognoses. Discovery of locoregional or distant metastasis lowers 5-year survival to 40% or 5%, respectively.[51] The most frequent sites of melanoma metastasis include the skin, lymph nodes, lung, liver, brain, bone, and gastrointestinal tract. Conversely, the presence of a great number of tumor-infiltrating lymphocytes and a lack of vascular invasion improve survival prognosis.[7]

Controversy exists regarding the value of elective lymph node dissection (ELND), although it is well accepted that there is no benefit to ELND performed concurrently with primary tumor resection. Our current management protocol is not to suggest ELND or sentinel node biopsy for lesions measuring less than 1 mm or greater than 4 mm. Intermediate-depth tumors (1–4 mm) are referred for sentinel node biopsy based on studies suggesting that it increases the 5-year survival rate by 10% (i.e., from 35% to 45%) (Tables 34-2 and 34-3).[52]



FIGURE 34-7. Large multicolored pigmented lesions should undergo biopsy with a full-thickness incision at the darkest area.

MANAGEMENT OF NMSC LESIONS

Regardless of the obvious appearance of some cancers, a biopsy should be performed for histologic confirmation and typing. The histologic characteristics influence clinical behavior, recurrence, and metastatic potential. Determination of margin size for tumor clearance should be based on a compilation of all available information.

TABLE 34-2. **Melanoma: Survival Rates**

Breslow Classification	Clark Classification	Tumor Thickness (mm)	5-Year Survival (%)
No evidence of primary tumor	I. Tumor in epidermis (noninvasive)	< 0.76	99
Thickness > 0.76 mm	II. Melanoma cells in papillary dermis	0.76–1.5	94
Thickness 0.76–1.5 mm	III. Melanoma cells fill papillary dermis	1.51–2.25	83
Thickness 1.51–3.0 mm	IV. Melanoma cells in reticular dermis	2.26–3.0	72
Thickness > 3.0 mm	V. Melanoma cells in subcutaneous fat	> 3.0	< 50

TABLE 34-3. **Melanoma: Biopsy Strategies**

Tumor Thickness	Margin Excision	Lymph Node Dissection?
Lentigo maligna, in situ	5 mm–1.0 cm	No
Melanoma < 1.00 mm	1 cm	No
Melanoma 1.01–4.00 mm	2 cm	Possible sentinel lymph node dissection 1.0–4.0 mm
Melanoma > 4.01 mm	2–3 cm	For diagnostic purposes

Biopsy techniques are personal. Any technique that delivers adequate histologic material for diagnosis is acceptable. Shave biopsy with a scalpel or curved razor blade is simple. Shave biopsy leaves a 5- to 6-mm saucer-shaped defect, removing epidermis and some dermis. The only drawback to shave biopsy is that histologic and prognostic features may be deeper than the shave. Thus, a shave biopsy might potentially be so superficial as to limit pathologic differentiation between an in situ and an invasive lesion. Shave biopsy is contraindicated in potential melanomas. A "pseudoshave"

biopsy might be performed with a curetted specimen before electrodesiccation.

Punch biopsy garners a full-thickness specimen. The punch has a circular cutting edge, which is pushed and turned into a suspicious lesion just like a hole saw. The punched-out defect may be sutured or may heal secondarily. Punch biopsies, although worthwhile for melanoma in which depth discernment is critical, may be too aggressive for a superficial lesion because the punch may force the tumor deeper into tissue.

Incisional and excisional biopsies are well known to surgeons. Deciding whether to use one revolves around whether the diagnosis is obvious (e.g., a dysplastic nevus in a patient with a history of them), the tumor size, and whether a small biopsy will influence the excision clearance margin. When indicated, excisional biopsies should be oriented with sutures or dye for tumor margin clearance.

Fine-needle aspiration (FNA) may also be used to obtain specimens for histologic examination of deep material. FNA is worthwhile to differentiate a dermal cyst from a parotid tumor in the periauricular region. With FNA, the pathologist aspirates tissue with a 23- or 25-gauge needle and stains and fixes the material on a glass slide.

Once the pathologic diagnosis of skin cancer is confirmed, the surgeon plans for tumor destruction by correlating tumor characteristics with patient's age, skin history, medical history, social history, and cosmetic expectations. Treatment options might include liquid nitrogen cryotherapy, standard excision, Mohs' micrographic surgery (MMS), radiation, C&E, topical chemotherapy, laser ablation, photodynamic therapy, interferon, and retinoids. We review most of these options later.

Standard Excision

Commonly, skin cancers are excised and assessed for margin clearance. Exceptions include some AKs and some superficial SCCs or BCCs, which may be treated by other modalities.[52] Excision may be done under local anesthesia or in the outpatient surgery setting. Tables 34-4 and 34-5 outline acceptable margins for clearing most lesions.

The lesion and indicated margin for clearance are outlined with a marking pen. Local anesthesia with epinephrine does not affect pathologic margin assessment but may reduce the surgeon's ability to monitor vascularity to an adjacent random flap. Clearer delineation of tumor margins may be enhanced with adjunctive procedures for melanoma, BCC, and AK. In the case of melanoma, subcutaneous extension should be viewed with a Wood's light. In the cases of BCC and AK, preexcision curettage delineates tumor margins more accurately. Some BCCs, morphea-like and infiltrative, may not be as curettable as soft tumors, but the BCCs that are curettable have a 25% higher chance of being cleared with the first excision than if excised without curettage (Figure 34-8).[52]

TABLE 34-4. **Margin Control for Squamous Cell Cancers**

Tumor Description	Margin Control
Small, well differentiated	5 mm with orientation
> 1 cm	Increase margin size
Lesion on upper lips, eyelids, nose, ears, etc.	Consider Mohs'micrographic surgery

TABLE 34-5. **Margin Control for Basal Cell Cancers**

Tumor Description	Margin Control
≤5 mm	2 mm
5 mm–1 cm	3–4 mm
1–2 cm	5–7 mm
> 2 cm, morphea-like, *or* unusual pathologic behavior	7–10 mm margin *or* Mohs'micrographic surgery or delayed reconstruction following permanent histology
Unusual pathologic behavior, recurrent tumors, tumors on lips, ear, nose, medial canthus, eyelids	Mohs'micrographic surgery *or* delayed reconstruction following permanent histology

Ideally, specimens should be examined histologically on the entire lateral and deep margin. Circumstances may reduce the likelihood of this beneficial extensive evaluation. For example, frozen histologic evaluations (while the patient is anesthetized in the operating room) are often three to four representative "loaf-of-bread" slices. You can imagine how much time would be consumed should the pathologist section and examine a large tumor in toto.[2] To abrogate the inherent limitations of frozen sections, many surgeons routinely delay reconstruction until after all margins are cleared by permanent histology or send the patient to a specialist in MMS.

Permanent histology after office excision and subsequent delayed reconstruction provides benefits to surgeon and patient alike. Office excision allows the patient to visualize the extent of the defect and to add input into personal reconstructive desires and expectations. The surgeon has the option to research effective methods of reconstruction away from the operating room and to subsequently go to the operating room with a plan; the patient will know before the surgery exactly where the scars will be located. Delayed reconstruction has been proved beneficial for patients receiving skin grafts because the delay eliminates the potential for hematoma and may allow buildup of a higher granulation base.

MMS offers the same "delayed" opportunity. The patient's entire tumor is resected before reconstruction, which may be performed on an elective basis, often up to a week later. The only surgical difference between immediate (within 24 hr) and delayed reconstruction (≥48 hr) is that defects reconstructed later are circumferentially excised for 0.5 to 1 mm to expose a new distinct margin. Debris may also need to be curetted from the base. Regardless, this step-by-step delayed technique is almost painless and does not foster infection (Figure 34-9).[51,53] Antibiotics are not necessary.



FIGURE 34-8. **A,** This 50-year-old man has what appears to be an imposing basal cell cancer. **B,** Curettage of the soft tumor allows easy visualization and control of peripheral and deep margins. **C,** Seven days after resection and margin clearance by permanent histology, a cervicofacial-type flap above the superficial musculoaponeurotic system is planned. **D,** The flap is advanced superiorly. **E,** Hairlines and sideburns are realigned. **F,** At 2 weeks, the results are very aesthetic.

Mohs' Micrographic Surgery

MMS is based on two principles: (1) most tumors spread by contiguous growth and (2) all tumor cells must be excised for cure. Dermatologist Frederic E. Mohs, MD, originated his method in the 1930s and published results in 1941. Mohs' technique evaluates the entire circumference and deep margins after frozen sections. Unlike the representative breadloaf method, in which the pathologist might suggest further removal of an entire positive superior margin of the tumor, Mohs' technique pinpoints the actual location of tumor extension. Identified tumor extensions are reexcised and rescrutinized until the tumor is totally removed. Hence, MMS is more predictable for total cure and tissue sparing as well.[54–56]

Over the years we have noted certain limitations to Mohs' technique, such as overconservative treatment for some aggressive tumors. This deficiency, not inherent in Mohs' technique, is proved by the fact that not all microscopic extensions are visible to the human eye; therefore, even tumors excised with Mohs' techniques may recur. As a result, we believe that a large SCC of the scalp is better served with an aggressive non-Mohs' excision. Controversy exists as to whether Mohs' technique is justified for melanoma and dermatofibrosarcoma protuberans, for example.

For a 1-cm noduloulcerative BCC, Mohs' technique proceeds as follows: the lesion is debulked with a curette and then excised with a 2- to 3-mm margin angled at 45 degrees toward the center of the tumor. The specimen is anatomically

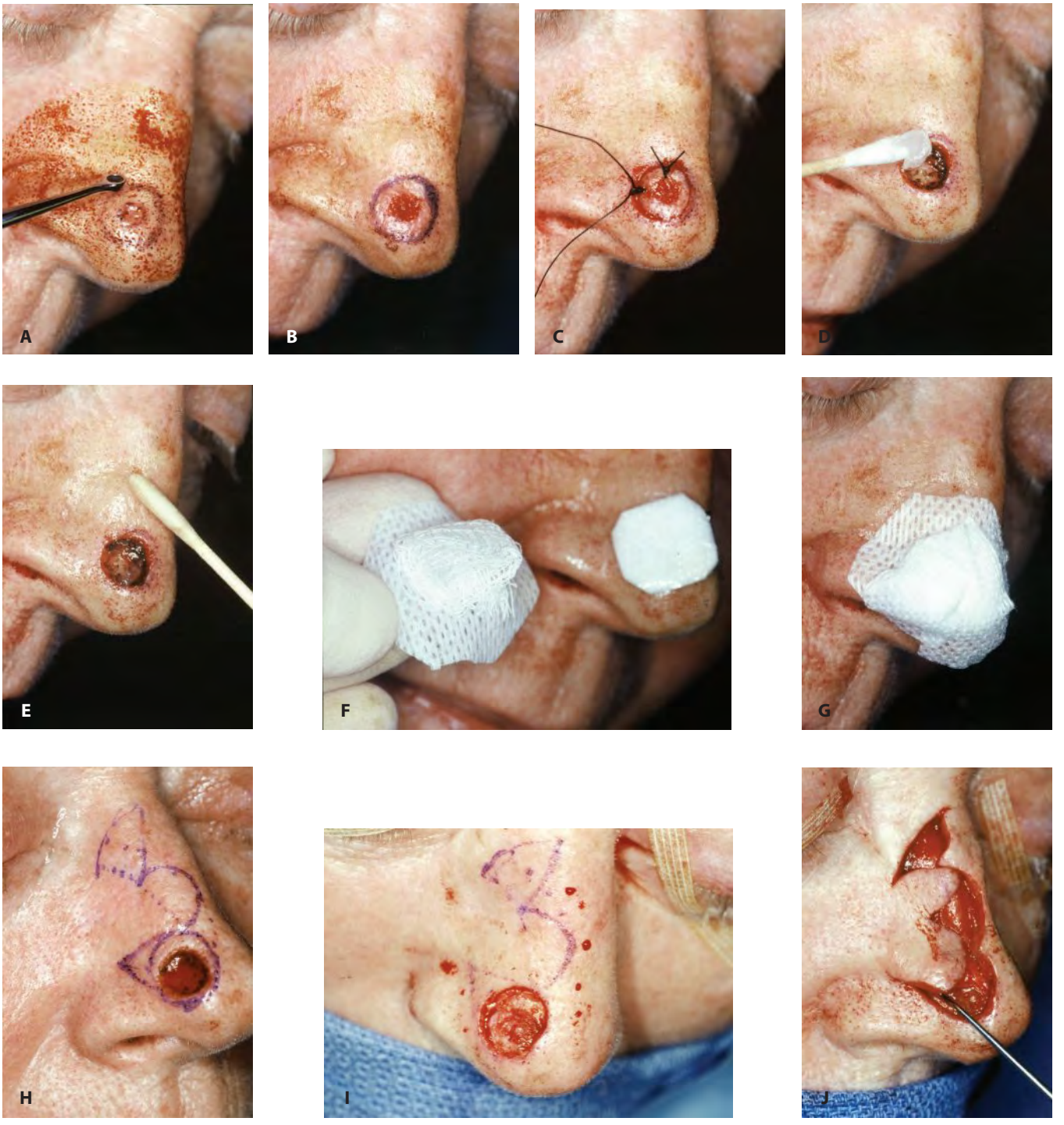


FIGURE 34-9. **A,** In the office, the surgeon, donning nonsterile gloves, curettes (with a dermatologic curette, pictured) this biopsy-proven nasal basal cell cancer. The patient is under local anesthesia. **B,** Curettage reveals that the tumor is small and superficial. **C,** After tagging the tumor for margin identification (always short 12:00 superior and long left or lateral), the wound is dressed very specifically. (If there is any potential bleeding, a piece of Surgicel may be placed at the base). **D,** Bacitracin is swabbed only within the defect. **E,** Mastisol or tincture of benzoin is wiped peripherally. **F,** A nonadherent dressing covers the wound base; the overdressing has an absorbent piece of gauze within a conforming mesh bandage, which is placed over the wound. One or 2 days later, in the office, the surgeon, donning nonsterile gloves, removes the dressing. This procedure is performed without the use of anesthesia. The area is cleaned with 50:50 peroxide and water, and the patient is instructed how to redress the wound daily after a shower (which includes washing out the defect with mild soap and water). **G,** The patient dresses the wound with bacitracin, a nonadhesive dressing, and tape only. No scabs should form. In this case, the histology analysis returned declaring that the superior tumor margin was within one high-power field. **H,** On the day of surgery, a small amount of tissue is planned for excision superiorly (and peripherally to square the margins), and a bilobed flap is planned as the defect is less than 1.5 cm in diameter. **I,** The excisions have been made. **J,** During the closure, the entire nasal dorsum is undermined submuscularly and supraperichondrally. *(Continued)*



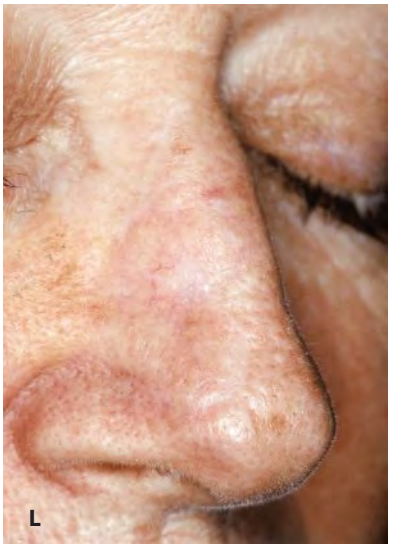


FIGURE 34-9. *(Continued)* **K,** The second lobe of the flap is oriented perpendicular to the alar rim to avoid lifting the rim. **L,** After 2 months, the result is excellent.

oriented, subdivided into numbered color-coded sections, and mapped. Mapped segments are pressed flat on their freshly cut border, frozen, and sectioned so that the entire fresh border is visualized.

The cure rates for primary BCCs less than 2 cm treated with MMS approach 99% (vs. 90–95% by routine pathologic examination).[56,57] Standard vertical breadloaf sections evaluate less than 1% of the surgical margins. Recurrent BCC cure rates range from 94% to 96% with MMS versus 85% with other modalities.[56,57] For primary SCC, MMS boasts a cure rate from 94% to 99% as opposed to 90% for non-Mohs' techniques.[39,54,58] Recurrent SCC cure rates with MMS approach 90% as opposed to 76% for other treatment modalities.[54]

Under these circumstances, MMS is indicated for the treatment of recurrent BCCs, histologically difficult BCCs (i.e., micronodular, infiltrative, and morphea-like), and BCCs in which conservation of tissue is critical (e.g., on the nose, lip, ear). For SCCs, MMS might be indicated for lower lip cancer, some poorly differentiated SCCs, and areas in which maximum tissue preservation is essential.[7]

Radiation Therapy

Radiation therapy (RT) has been mentioned for treatment of skin malignancies for almost a century, but currently, it plays a role as an adjunctive or salvage measure, rarely a curative role. The curative advantage of radiation is preservation of normal tissue next to the irradiation site. RT might, therefore, be considered for the eyelid, lip, nose, and ear. Unfortunately, RT conveys some unwanted potential side effects: cutaneous erythema, necrosis, hypopigmentation, telangiectasia, atrophy, fibrosis, hair loss, delayed healing, and risk of the development of future NMSCs when administered to younger patients.[59]

RT of tumors less than 2 mm has a cure rate of 90% and 85% to 95% for BCC and SCC, respectively.[60,61] However, larger lesions have a much lower success rate. For melanoma, local recurrence rates of up to 50% have been reported.[62] Thus, RT for melanoma is a viable option only for medically compromised patients who cannot withstand surgery or for patients who refuse surgery.

Cryosurgery

Cryosurgery destroys skin cancers and some adjacent tissue by freezing. Cryosurgery cure rates for AK, BCC, SCC, and lentigo maligna range from 94% to 99%.[63,64] Liquid nitrogen may be sprayed on the lesion directly or through a cryoprobe. Rapid freezing of the treated skin occurs as heat is transferred from the skin to the probe. Intracellular ice crystals form, and cell membranes disrupt as the temperature is lowered to –50∞C to –60∞C. When thawing occurs, electrolytes recrystallize, resulting in vascular stasis and local alterations in the microcirculation, thus producing further tissue damage.[2,65]

Most doctors freeze lesions plus a 4- to 6-mm margin to account for tumor extension. Freeze-thaw cycles may be repeated for maximal effect. Healing occurs by secondary intention, with a flat hypopigmented scar.

The side effects of cryosurgery include pain, erythema, edema, blistering, exudation, and scarring. This technique is inexpensive, and there are no costs for pathology. Hence, a lesion chosen for cryotherapy should be relatively small and well demarcated.

Curettage and Electrodesiccation

C&E is a cost-effective but technique-dependent therapy of NMSC. The lesion area is cleaned with alcohol, outlined with a provisional margin by a skin marker, and anesthetized. The lesion is curetted aggressively with the skin tensed, after which electrodesiccation (hyfrecation) for hemostasis and adjacent tissue kill occurs. This cycle may be repeated three to five times.

The major advantage to C&E is expedience, fostering treatment of multiple lesions within a single visit. Disadvantages include prolonged healing, often weeks depending on size and care, hypopigmentation, and possibly hypertrophic scar. Material from curettage may be sent for initial pathology, but margin control after C&E is not possible (unless curettage is used as a precursor to excisional pathology).

The clinician's experience and the tumor's anatomic site and size are prognostic factors limiting success after C&E. BCCs less than 5 mm have an 8.5% recurrence rate after C&E by an experienced clinician.[66] Lesions of the nose, ear, and perioral and periocular areas may recur at a rate of 16%.[66] This rate soars to 26% for lesions larger than 20 mm.[66] Therapeutic C&E is, therefore, contraindicated for larger lesions, poorly differentiated SCCs, or melanoma.

Topical Chemotherapy

Topical 5-FU or 5% imiquimod medically eliminates surface lesions. Retinoids are occasionally used concurrently. 5-FU is a thymine analog that interferes with DNA synthesis causing cell death by acting as an inhibitor of thymidylate synthase. Imiquimod induces production of interferon-alfa and messenger RNA cytokines. Application of 5-FU is recommended twice daily for 2 to 3 weeks for superficial AK and for 3 to 6 weeks for more diffuse worrisome lesions. Imiquimod is applied only three times per week but currently is much more expensive than 5-FU. Cure rates with 5-FU and imiquimod range from 92% for SCC in situ to 95% for superficial BCC and AK.[2,67]

Patients need to be warned that there is an ugly inflammatory scabby reaction during topical therapy, but the cosmetic outcome is usually very good as long as compliance is nurtured.

Lasers

The CO_2 laser focuses a beam of light with a wavelength of 10,600 nm. Laser light is absorbed by water and nonselectively vaporizes the skin. The CO_2 laser can be used as a cutting instrument (in the focused mode) to excise or ablate lesions (in a defocused mode) such as multiple AKs, superficial BCCs, and SCCs. We have found its greatest benefit in ablation of superficial AKs and superficial SCCs, both on the skin and the lower lips. Presurgical skin preparation with retinoids may foster more rapid healing. We have not prescribed preoperative antibiotics or antivirals for small localized areas but continue to do so when large areas of the face are treated.

Photodynamic Therapy

Photodynamic therapy is not widely accepted for skin cancer therapy but has been applied to lung, breast, colon, and bladder cancers. Aminolevulinic acid is wiped on a lesion; it is metabolized in cancer cells to produce porphyrins, which act as photosensitizers. Four to 6 hours later, the area is irradiated with visible light from a laser or noncoherent light source. Reactive O_2 species are generated within the cells producing cell death.[68]

Cure rates for photodynamic therapy for AKs, superficial SCCs, and BCCs are reported to be greater than 90% in some studies, but tumors thicker than 2 mm are photoresistant.[69]

Interferons

Interferons are cytokines that may affect cell growth and differentiation and accent immune responses and antiviral activity. Intralesional injection of interferon-alfa can attain cure rates of greater than 80% for superficial and noduloulcerative BCC.[70,71]

Retinoids

Retinoids are vitamin A derivatives that are crucial for control of cell growth, differentiation, and apoptosis. Topical retinoids are somewhat effective against AKs but much less so against even superficial BCCs and SCCs. Application of retinoids as a skin cancer preventive is a long-term proposition because the effects of the drug plateau at approximately 6 months and reverse shortly after discontinuation.

Retinoids do appear to act synergistically with 5-FU and may be applied in an exfoliation regimen. Noted complaints include dryness and flaking, minor side effects compared with the clinical effects of 5-FU.

Applied Skin Anatomy

The skin is composed of two layers: the superficial epidermis and, beneath it, the

dermis. The epidermis is composed of four distinct layers. From deep to superficial, they are basal cell (stratum basale), prickle cell (stratum spinosum), granular cell (stratum granulosum), and keratin (stratum corneum). Cells from the stratum basale divide and migrate upward toward the stratum corneum. The dynamic epidermis turns over and exfoliates every 30 days. This is why buried epithelium from a cyst might continue to produce sebaceous keratin.

The epidermis contains four cell types: keratinocytes, Langerhans' cells, melanocytes, and Merkel cells. Keratinocytes constitute 80% of the epidermal cell makeup.

Langerhans' cells are antigen-presenting cells, which capture and process antigens and present them to skin-specific lymphocytes. Aging and significant sun exposure both lessen the total number of Langerhans' cells. This is one partial explanation for the increase of skin neoplasms in the elderly.[72]

Melanocytes are of neural crest origin and are found in the basal layer. Melanocytes produce melanin, which in turn, protects the nucleus of the keratinocyte from UV radiation. Although numbers of melanocytes are constant for all individuals, the activity of the melanocytes differs from one race to the next. For example, melanocyte activity in darkly pig-

mented skin is higher than in light-colored skin. As with Langerhans' cells, numbers of melanocytes decrease with age, another explanation for more skin cancers developing as we get older.[73,74] Merkel cells, found in the epidermis and dermis, have an unclear function.

The dermis, situated between the epidermis and the subcutaneous fat, adheres to the epidermis at the basement membrane. The basement membrane mechanically supports the epidermis and acts as a mechanical barrier. The two dermal layers are the superficial papillary dermis and a deeper thicker reticular layer. The dermis is composed of collagen, elastic tissue, and ground substance. Collagen decreases by 1% a year throughout adulthood.[75] Topical tretinoin inhibits dermal collagenase, thus slowing the degradation rate of collagen.[76,77]

Elastic fibers in the dermis provide skin with recoil. With aging, elastic fibers decrease, causing skin laxity, bags, and jowls. Chronic sun exposure thickens elastic fibers, and clumps form in the papillary layer. Chemical peels, dermabrasion, and laser resurfacing can remove some of these clumps.[78]

The dermal ground substance is made up of glycosaminoglycans, hyaluronic acid, chondroitin 4-sulfate, fibronectin, and dermatan sulfate. These constituents hydrate the skin and maintain tensile elasticity.[78] The principal cell of the dermis is the fibroblast, whose functions include production of collagen, elastin, and ground substance. Fibroblasts enhance wound healing through contraction and production of scar.

Aging affects skin quality. Fine wrinkling, dermal atrophy, and a decrease in subdermal adipose tissue are aging phenomena. Epidermal regeneration may slow down by up to 50%, retarding secondary wound healing.[79] (Note: Isotretinoin retards epithelial regeneration chemically; hence, elective surgery should be limited on patients having used isotretinoin until the medication has been discontinued for 6–8 mo.) Natural collagen decreases in quality and quantity. Skin becomes more compact as the collagen rearranges itself into thick coarse bundles or loosely woven straight fibers. The dermal blood vessels may be collapsed, disorganized, or absent in the elderly, potentiating a greater risk for flap necrosis.[21,80]

Skin has a rich nerve supply. In the epidermis, the Merkel cell may provide touch perception. Meissner's corpuscles, located in the papillary dermis, provide fine-touch sensation. Pacinian corpuscles, located in the deeper subcutaneous tissue, mediate deep pressure and vibratory sensation. Autonomic efferent nerves innervate blood vessels and appendageal structures. Hair-bearing skin is commonly referred to as *nonglabrous* and smooth non–hair-bearing skin as *glabrous*. Skin conditions vary between individuals and from region to region with respect to mobility, color, scars, Fitzpatrick type, texture, thickness, and adnexal structures.[81]

The blood supply to the skin serves two functions: nutrition and thermal regulation. Two major routes of blood supply exist—musculocutaneous and septocutaneous arteries.[82] The musculocutaneous system traverses the muscle and enters the subcutaneous tissue in a random pattern (the basis for random skin flaps). Random-pattern blood flow to the tip of the flap is via the interconnecting subdermal plexus. The superficial vascular plexus located in the reticular dermis provides the capillary loops in the dermal papillae. The deeper vascular plexus, or subdermal plexus, lies between the dermis and the subcutaneous fat. A septocutaneous vessel travels through the septal fascia and courses parallel to the skin surface with an accompanying vein. Named septocutaneous vessels (e.g., supratrochlear) provide an axially based flap with a rich blood supply. A large interconnecting vascular arcade exists between the systems.[83] Understanding the facial vascular network is crucial to creating flaps that survive.

Flaps and Grafts and Secondary Intention Healing

Definitions and Concepts

The removal of any tumor leaves a defect. The hole created after tumor excision may be called the *primary defect*. The *secondary defect* is the wound created after tissue is transposed to close the primary defect. Every flap creates a potential secondary defect. Ideally, secondary defects should be easy to close, within relaxed skin tension lines (RSTLs), in areas of loose adjacent tissue, and within anatomic boundaries.[84]

Options for defect repair include (1) primary closure, (2) local or distant flap, (3) graft, and (4) healing by secondary intention. Elasticity and movability are two inherent skin characteristics that enable relocation and, perhaps, primary closure. Elasticity is the ability of the skin to stretch. Skin in the cheek and neck is very elastic. Movability is not related to elasticity. Temple skin is less movable than cheek skin, and the scalp is relatively immobile.

Flaps move tissue, skin, and subcutaneous from one area to another with an accompanying vascular supply. Flaps are cosmetic, use well-matched skin, and functionally protect underlying structures such as bone or cartilage, which may not have adequate blood supply to support a graft. Three types of "impure" flap movements are classically defined—advancement, rotation, and transposition—although some suggest there are only two types of movement—sliding and lifting.[84–86] *Sliding* refers to stretching or mobilizing tissue from one site to another (advancement and rotation). *Lifting* tissue across a bridge of normal tissue to close a defect is similar to transposition.[84] All flaps (except free flaps) have some pivotal restraint, whether it be adjacent skin, subcutaneous tissue, or blood vessels.

Delay increases viability to a flap by enlarging and realigning the subdermal vasculature plexus. It is now known that skin flap reliability is based on "angiosome" units; therefore, wide and thin random flaps run out of blood supply in roughly the same location. Delay may augment survivability. Methods include raising and suturing tissue without disturbing the pedicle, and tissue expansion. Subsequently (9–12 days later), the flap is mobilized.[84,87] The mechanisms that increase the

blood flow with delay include the depletion of vasoconstricting substances, formation of vascular collaterals and reorientation of vascular channels, stimulation of an inflammatory response, and release of vasodilating substances.

Aesthetic flaps are not mere hole fillers. They are designed to complement natural aesthetic units and facial borders. Defects that trespass multiple aesthetic units are designed to reproduce these independent units. For example, a cheek tumor defect that encroaches on the nose might be reconstructed with different flaps and/or grafts for the cheek and nose.

Grafts are easy to position into recipient defects and are ideal for monitoring tumors. Grafts must be placed on a well-vascularized bed. Sometimes, exposed bone should be allowed to build a granulation base before grafting. Grafts may be of full thickness or split thickness. Harvesting methods include punching, shaving with a dermatome, and excision. Graft donor sites are selected based on aesthetic and tumor considerations. Ideally, grafts to the nose are well matched with preauricular skin, but any supraclavicular facial graft (from the blush area) matches the facial color better than does any torso or thigh graft.

Healing by secondary intention is a painless but time-consuming process. It is indicated for patients who do not want more surgery, who can accept or obtain the daily care, and who can accept a scarred result. Secondary healing can be used for small defects (<1 cm) or for larger defects in areas where the resulting scar would be inconspicuous or tumor observation is critical.

Healing by secondary intention is similar to open-wound therapy. Following tumor excision and hemostasis, the wound is dressed with antibiotic ointment (e.g., bacitracin and/or polymyxin B sulfate). The outer edges of the wound are coated with an adhesive (e.g., adhesive bandage or tincture of benzoin). A nonadherent dressing is applied over the wound and a small rim of peripheral tissue. This is topped with a dry piece of gauze to absorb any blood, which is then covered with a contour mesh tape. When the defect is atop bone, the raw bone may be covered with two layers of moisture-retaining wet gauze, but any method that abrogates desiccation is acceptable (Figure 34-10).

Three days later. the dressing is removed and the wound inspected. Any oozing and crusting should be removed with a 50:50 peroxide and water solution. The wound is redressed in three layers—antibiotic ointment within the wound followed by a nonadherent dressing, which is then covered with mesh tape. The patient redresses the wound in this fashion on a daily basis to keep the area moist and free of scabs. Areas amenable to secondary epithelialization include the scalp, the retroauricular area, and some concavities away from mobile apertures. Secondary epithelialization would be a poor choice around the mouth, for example, where retraction might distort the lips.

Three caveats regarding secondary healing are useful to keep in mind. First, scabs should not form. Scabs hinder epithelialization and harbor bacteria. Second, continuous application of antibiotic ointment can lead to allergic reactions and yeast infections. This is more common with ointments that contain neomycin sulfate than with bacitracin. Alternatively, petrolatum can be substituted for the antibiotic ointment. Finally, some patients can be so incapacitated by their medical illnesses that they cannot dress their wounds. Home health care nursing can be enlisted to aid in their daily wound care.

Skin Biomechanics

Skin is a heterogeneous material with unique mechanical properties. As skin is stretched, the randomly oriented collagen and elastic fibers are stretched in the direction of the applied force. This continues until all of the available collagen and elastic fibers are recruited and no further lengthening occurs. After the maximum amount of stretch is reached, the skin may rupture. Permanent striae may scar the skin surface, as is often noted in pregnancy. Overstretching the skin collagen effaces the blood vessels under tension; thus, necrosis secondary to decreased perfusion to a distal flap may occur (Figure 34-11).[88]

Skin tension exists in all directions on the face but is greatest along the RSTLs. Ideally, elective incisions should be placed parallel to the RSTLs. Incisions made perpendicular to RSTLs (or in the lines of maximum extensibility [LMEs]) gape and heal with more obtrusive scars.[89] The rhombic flap, once considered by many as the "workhorse" facial flap, has been used less over time because some of the final legs lie within the LMEs. Today, flaps are more commonly designed with topographic units and RSTLs as primary considerations, rather than just to fill a hole.

Skin is elastic and stretches easily at low stress levels. This is related to the inherent extensibility of the skin. At higher forces, skin may become viscoelastic, that is, it can extend out a little more in spite of its thick state. This phenomenon is explicable through the two time-dependent characteristics: creep and stress relaxation.[88]

Mechanical creep refers to the change in length that is seen when skin is held under a constant stress or force. The force that is exerted to stretch skin decreases with time.[88] The surgeon routinely notes this mechanism at work after he or she *tightly* sutures an avulsive forehead wound. Two days later, the forehead is relaxed again. High stress loads, therefore, produce a degree of creep. The skin may not be totally relaxed for several months. Serial excision is a technique that harnesses the relaxation of skin over time. Wide defects may be closed sequentially over time.

Stress relaxation is the decrease in stress that occurs over time when skin is held under tension at a constant strain or is cyclically loaded.[88] It may be effected intraoperatively with the placement of a balloon under the skin or by scoring the scalp galea and pulling the skin. In addition, skin stretchers are made for this purpose (Figure 34-12).

Finally, *biologic creep* is a slow methodic stretching of skin, yielding brand new skin.[88] Skin expanders do just that (Figure 34-13).



FIGURE 34-10. **A,** This 60-year-old patient (who has diabetes and congestive heart failure) has a very rapidly growing forehead/scalp squamous cell carcinoma. **B,** In the operating room, the tumor is widely excised. **C,** The base shows tumor into the outer table of the skull, which is removed. **D,** The wound is dressed open with microfibrillar collagen peripherally to prevent bleeding, and a compression bandage over two layers of moist ointment-saturated mesh gauze. Permanent histology shows complete tumor clearance, but the patient's medical problems delay reconstruction. **E,** The patient has an excellent granulation base at 5 weeks. He elects to allow the defect site to epithelialize secondarily with daily dressing changes at home. **F,** At 8 weeks, 50% epithelialization is evident. **G,** Total epithelialization has occurred at around 3 months. He has had no tumor recurrence or metastasis after a 2-year follow-up and has deferred further reconstruction.



FIGURE 34-11. This large basal cell cancer was excised **(A)** and closed with two flaps **(B).** The patient, a smoker, had excess tension placed on the lower flap, leading ultimately to almost 1 cm of tip necrosis.

Flap Undermining

Safe flap closure of a defect is dependent on harnessing the inbred stretchable bendable nature of skin without exceeding the limits of stretch or blood supply. Some tissues can be stretched for centimeters without undermining occurring, whereas others must be separated from tethering subcutaneous tissues. Conversely, a subcutaneous island flap, totally separated from the tether of skin, depends on the mobile vascular subcutaneous pedicle.

Undermining releases the vertical attachments between the dermis and the subcutaneous planes, thereby reducing shearing forces and allowing the skin to slide and redrape in

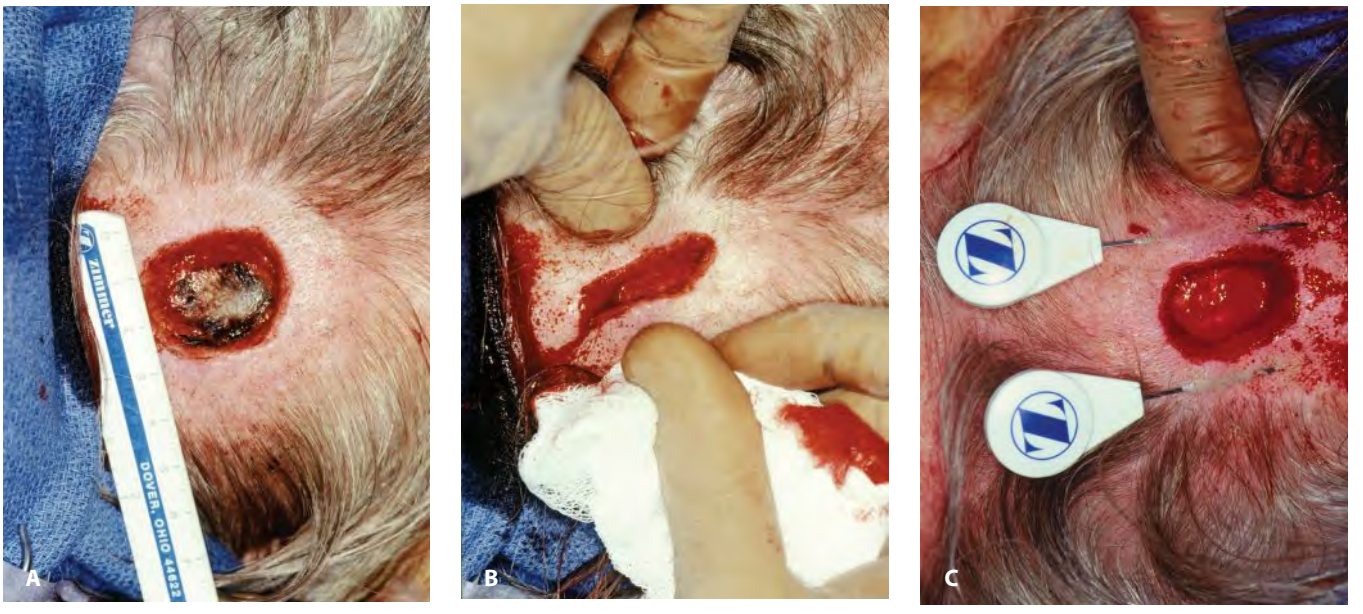


FIGURE 34-12. **A,** This 95-year-old woman has a relatively small scalp defect. **B,** Finger pressure shows that significant tension is necessary to close it elliptically. **C,** After limited subgaleal undermining, two pins are placed subcutaneously. *(Continued)*

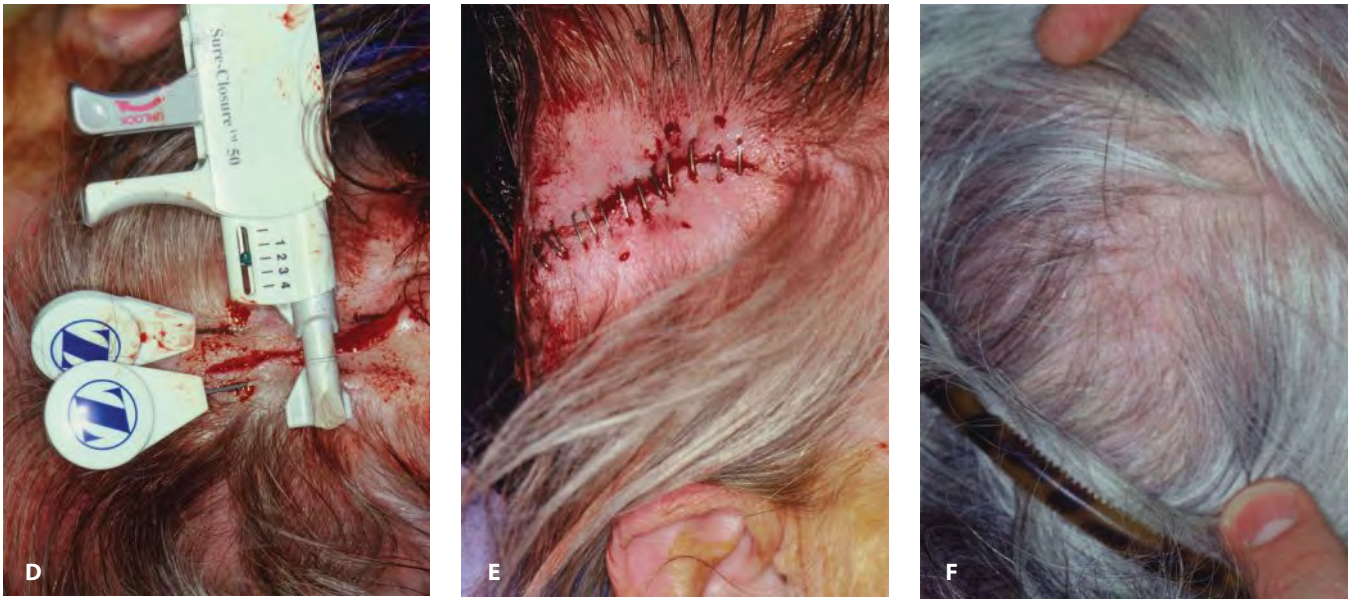


FIGURE 34-12. *(Continued)* **D,** A skin stretcher (courtesy of Life Sciences Medical) is hooked onto the pins. Slow loading is applied for 30 minutes. **E,** After the end triangles are excised, the wound is stapled closed. **F,** The site after 2 months.

another position.[78] The mobilization benefits from undermining facial skin usually occur within the first 2 cm. Animal studies reveal that undermining beyond 4 cm produces little skin edge advance and possibly a more difficult stretch of tissue.[90,91]

A correct undermining level provides the critical balance between mobility and blood supply. For example, simple random flaps, undermined in the superficial fat, are easy to raise on the cheek. Submuscular flaps maintain a robust blood supply to small, relatively immobile nasal flaps.



FIGURE 34-13. **A,** This 45-year-old heavy smoker had a squamous cell cancer removed from his midforehead and a basal cell cancer from the left temple. Direct advancement closure was impossible. **B,** After allowing some granulation tissue to form at the base, a split graft was placed on the midforehead. An 8 × 6-cm skin expander was placed laterally and expanded twice weekly for just over 2 months. **C,** The expanded rotation flap was mobilized to cover the midforehead defect. **D,** The result 1 month after closure. Note the well-healed direct closure of the right cheek defect and the depression of the left temple full-thickness skin graft.

Flap Designs

Advancement Flaps

An advancement flap is advanced linearly over a defect. It consists of a classic elliptical closure with adjacent undermining; there are no rotational or pivotal movements. Tissue elasticity provides adequate horizontal motion with a flat closure effected as Burow's triangles are removed from the ends. The length of the ellipse is three to four times the width of the defect.

Advancement flaps can be constructed with multiple modifications: simple, square, bilateral, Burow's triangle repositioning, and A- or O- to T-shaped designs.[92] The experienced surgeon realizes that the tethering forces of advancing skin also constrict the size of the leading edge. Modifications are useful in specific instances. All flaps, including simple advancement flaps, presuppose that the surgeon can disguise, adjust, transpose, or eliminate "dog-ears" or excess tissue that gathers as tissue is transposed.

There are seven ways to deal with dog-ears[93–96]:

1. Do nothing. This approach works well on the scalp because bunched-up tissue lies down with time.
2. Close opposite lines of uneven lengths by spreading out the problem—halving (Figure 34-14A).
3. Remove the excess to a hidden area—an end or middle triangle (see Figure 34-14B).
4. Lengthen the incision. This eliminates bunching (see Figure 34-14C).
5. Perform an M-plasty (sometimes called a T-plasty), which shortens the problem (see Figure 34-14D).
6. Reverse the S loop and hide the excess elsewhere (see Figure 34-14E).
7. Advance the dog-ear as a flap (subcutaneous "island") or use it as a free graft (see Figures 34-6 and Figure 34-14F).

Rotational Flaps

Curvilinear rotation flaps rotate from a tethered pivot point. These flaps fill triangular defects. The length of the arc is dependent on many variables, such as existing laxity, the size of the defect, the location, and blood supply to the flap.[85,97–101] Rotation flaps rarely fit perfected geometric schemes of success. Rather, the surgeon often finds herself or himself adjusting to the specific variables of a given situation (Figure 34-15). There are two exceptions to this complexity that have been worked out fairly precisely:

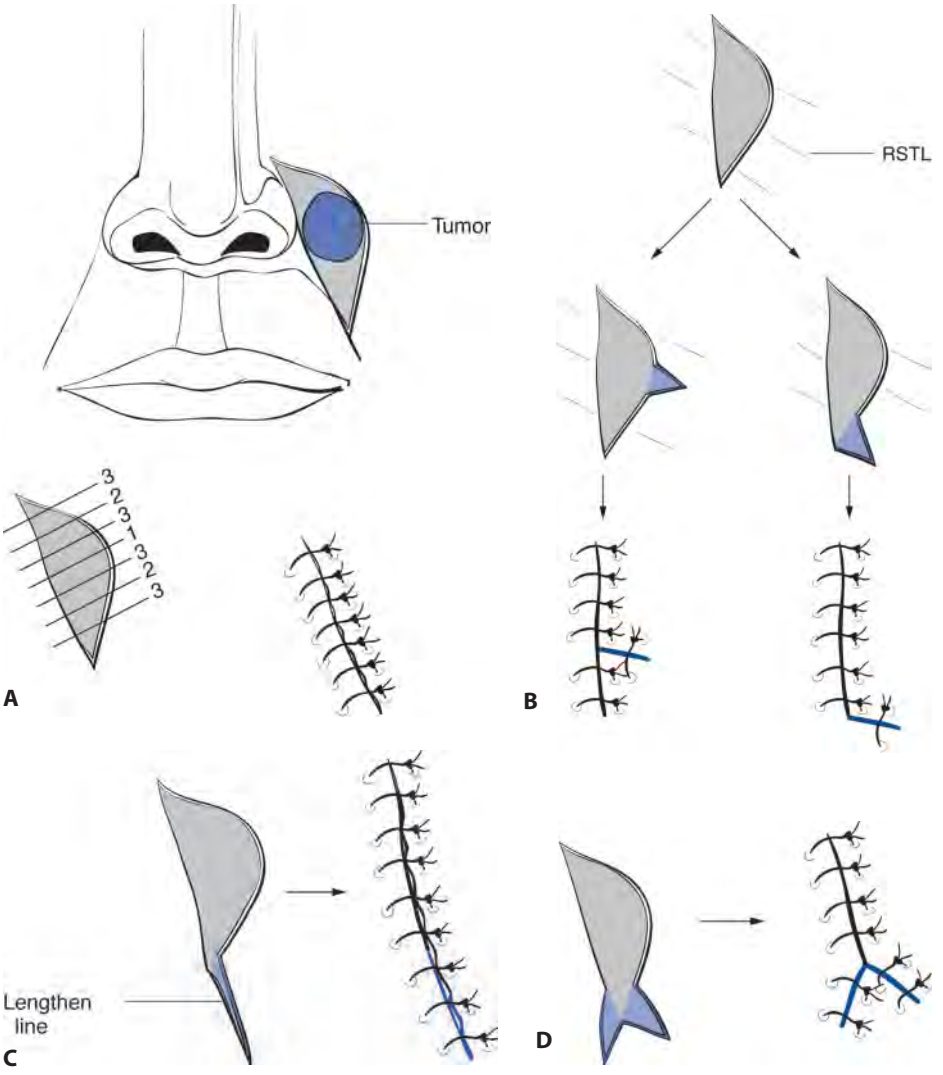


FIGURE 34-14. Six of the seven ways to deal with dog-ears (the seventh being to do nothing). **A,** Halving. Close opposite lines of uneven lengths by spreading out the problem. **B,** End or middle triangle. Remove the excess in a hidden area. **C,** Lengthen the incision. This eliminates bunching. **D,** M-plasty (sometimes called a T-plasty). This procedure shortens the problem. *(Continued)*

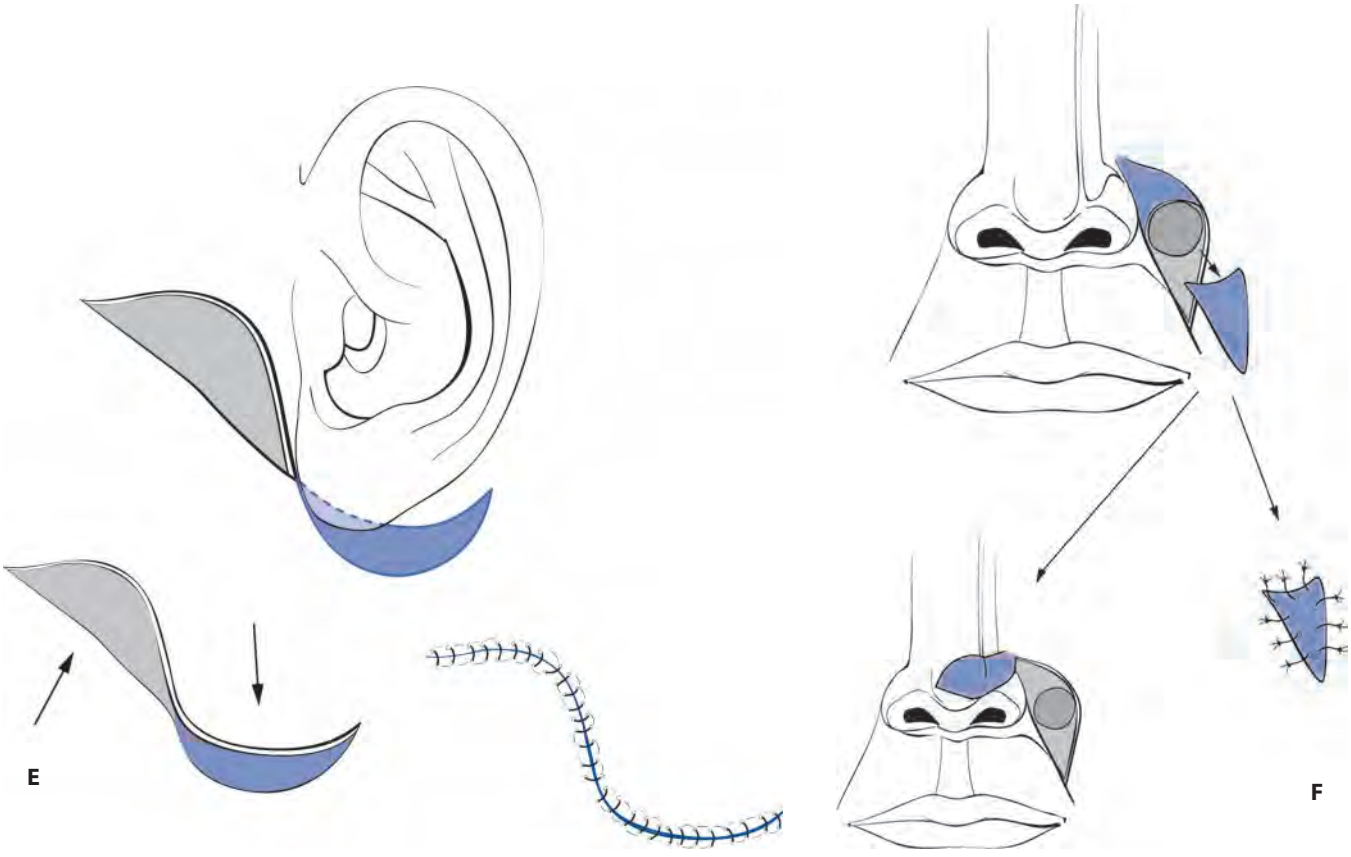


FIGURE 34-14. *(Continued)* **E,** Reverse the S. Hide the excess elsewhere. **F,** Advance the dog-ear as a flap (subcutaneous island) or use it as a free graft (see also Figure 34-6). RSTL = relaxed skin tension lines.

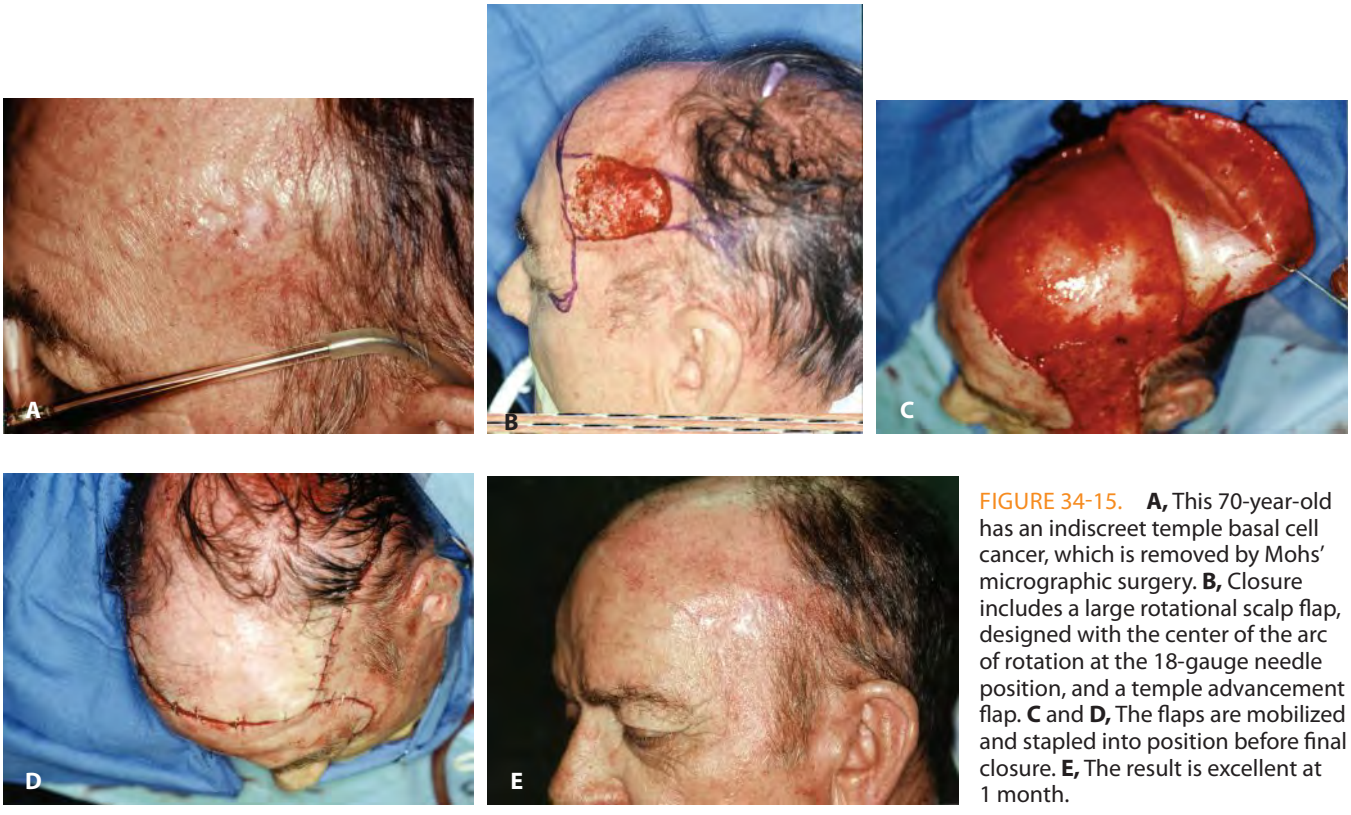


FIGURE 34-15. **A,** This 70-year-old has an indiscreet temple basal cell cancer, which is removed by Mohs' micrographic surgery. **B,** Closure includes a large rotational scalp flap, designed with the center of the arc of rotation at the 18-gauge needle position, and a temple advancement flap. **C** and **D,** The flaps are mobilized and stapled into position before final closure. **E,** The result is excellent at 1 month.

1. The nasal bilobed flap of Zitelli has excellent results when applied toward lower or middle nasal defects of 1.5 cm or less.[102] This occurs as long as the second lobe of the flap is perpendicular to the alar rim and the first lobe does not cross deep concavities such as the alar groove.
2. The scalp rotation method of Ahuja fills defects of up to 3 cm with minimal adjustments.[103]

Advantages to rotational flaps include broad-based reliable vascularity, flexibility in design, and easy placement of scars into aesthetic/cosmetic zones or RSTLs. A major advantage is that the flap can be rotated again should additional tissue need to be removed secondary to tumor concerns or should laxity be lacking.[98,104]

Disadvantages to rotational flaps include the problems associated with any pivotal flap such as standing cutaneous deformities and the need for larger flaps. Regardless, rotational flaps may be ideally designed to reconstruct medium to large defects of the cheek, neck, scalp, and forehead.

Transposition Flaps

Transposition flaps transfer defined tissue along an arc of rotation, often over normal tissue, to repair a primary defect. Actual tissue movement may be rotational, linear, or both (Figure 34-16). Transposition flaps tend to be more confined than are rotation flaps, and design is critical for success. The design/location of the pivot point is the most important factor. After tissue is transposed, flap tensions should be diffused to prevent strangulation of tissue and distortion of adjacent structures. The regional differences in tissue mobility affect the geometry of the flap design, with the classic transposition being a rhombus.

The rhombic flap is an equilateral parallelogram with oblique angles. The (Limberg) rhombic flap, first described in 1963, was an equilateral rhombus with 60- and 120-degree internal angles.[105]

According to the classic (Borges) design, eight potential rhombic flaps may close a defect. These flaps are constructed as umbrellas, drawn off the obtuse side of two potential parallelograms. These parallelograms each have two sides parallel to the LMEs. These LMEs are always perpendicular to RSTLs and run in the direction that tissue stretches most efficaciously.

The rhombic flap, whose short diagonal line parallels the LMEs and whose mobilization does not interfere with adjacent structures, is usually chosen for the rhombic transposition. The resulting tension vector in rhombic flaps lies 20 degrees from the short diagonal in a loose tissue plane.

Dufourtmental, Webster, and others modified the classic rhombic design.[106] The Dufourtmental flap was designed to close rhombic defects with acute angles approximating 90 degrees or a square defect. As with the Limberg design, the peripheral lines were equal in length, but unlike the rhombic flap, the short diagonal differed in angle size. Dufourtmental designed two isosceles triangles situated base to base. Once the sides of the triangles were drawn, the short diagonal was extended, as was one of the adjacent sides. A third line bisecting these two lines creates the first flap. The cutback line was drawn paralleling the long diagonal completing the second flap. The Webster 30-degree flap allowed for easier closure by bisecting the 60-degree angle into two 30-degree angles via an M-plasty. Here, the short diagonal had to be at least 110 degrees to prevent puckering and to maintain flap viability.

Rhombic, banner, note flaps, and others have been modified to close circular defects over all areas of the face.

Axial Pattern Flaps

Axial pattern flaps are based on named vessels in the head and neck. Classic designs include the Abbé (Figure 34-17) and Estlander flaps and the paramedian forehead flap.[97] A full description of these flaps is discussed in Chapter 40.

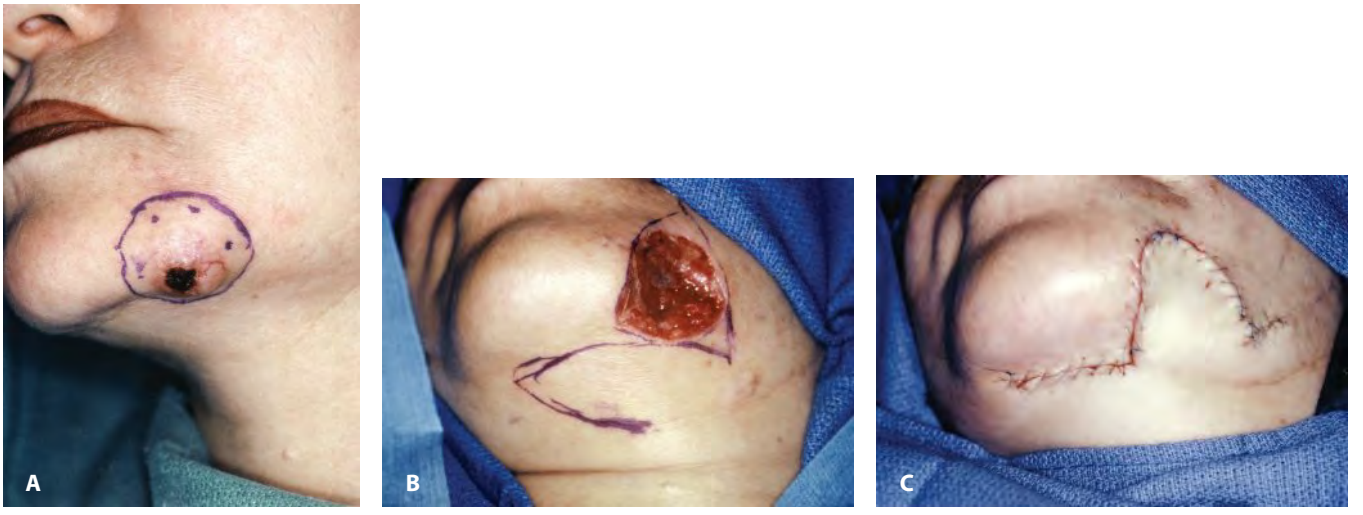


FIGURE 34-16. **A,** This 45-year-old woman has had a basal cell carcinoma for the past 3 years. **B,** It is repaired with a simple submental transposition flap. **C,** Perhaps the lateral submandibular bulkiness will need to be removed in the future, but the submental fat removal was highly esthetic.

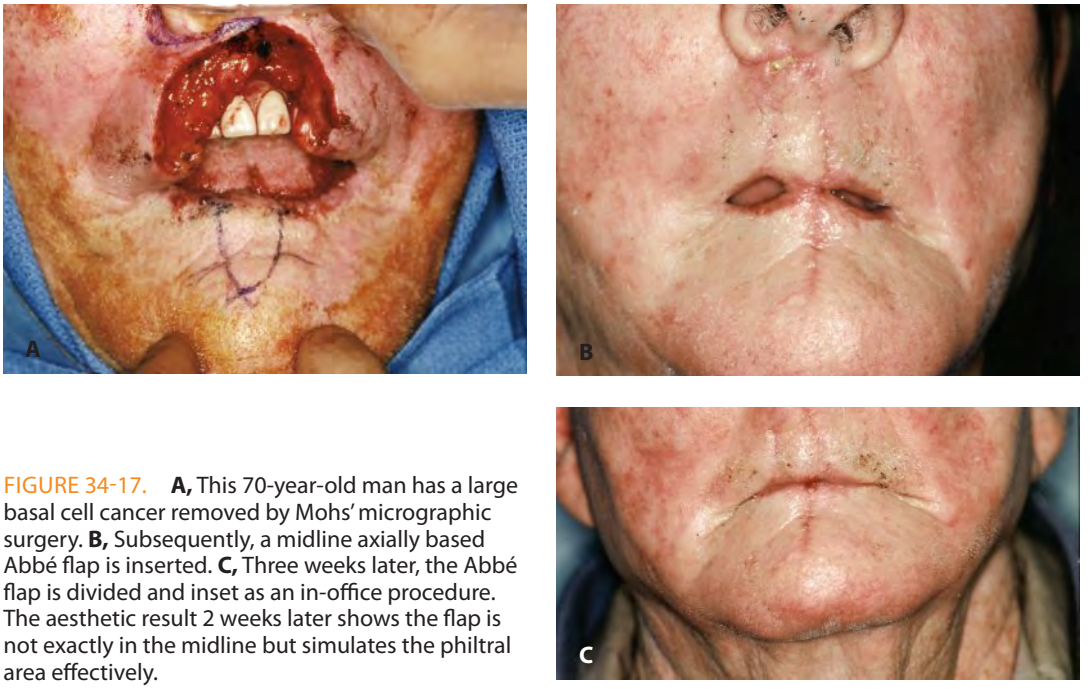


FIGURE 34-17. **A,** This 70-year-old man has a large basal cell cancer removed by Mohs' micrographic surgery. **B,** Subsequently, a midline axially based Abbé flap is inserted. **C,** Three weeks later, the Abbé flap is divided and inset as an in-office procedure. The aesthetic result 2 weeks later shows the flap is not exactly in the midline but simulates the philtral area effectively.

Skin Grafts

Skin grafting involves the removal of donor skin (epidermis and varying levels of dermis, fat, or muscle) from one area to revascularize at another. The success of skin grafts is based on factors that affect angiogenesis and capillary ingrowth into the graft.

Recipient bed vascularity and intimate graft-host contact as well as overall host health or condition affect graft success. Wounds with a poor vascular supply may not support a graft or may need to be prepared before grafting. Cartilage base, irradiated tissue, fibrosis, and foreign, crushed, or nonviable tissue can compromise success. Additional procedures, such as bringing in vascular tissue from elsewhere, may be required to optimize the recipient bed before skin grafting.[98]

Bed vascularity may be compromised by bleeding or cautery to arrest bleeding. Thus, there is an inherent benefit to delaying grafting or placing a pressure bolster bandage on top of the graft to prevent bleeding. We do not touch full-thickness graft bolsters for 6 or 7 days.

Mechanical shear forces may disrupt contact between the graft and the recipient bed, promoting graft failure. Although this may be minimized with appropriate suturing techniques as well as the placement of dressings, the force of a hard shower can dislodge a graft and should be avoided.

Wound infections rarely jeopardize skin grafts in the head and neck. It is common for surgeons to confuse the dark eschar of a failing graft in a smoker with infection. Regardless, some local measures that decrease wound bacteria include saline dressings, sulfadiazine silver, mafenide acetate cream, acetic acid solutions, sodium hypochlorite solutions, and vinegar and water.

A patient's overall medical health can influence the success of skin grafting. Autoimmune diseases such as rheumatoid arthritis, systemic lupus erythematosus, hematologic disorders, diabetes mellitus, poor nutrition, and smoking as well as medications such as corticosteroids and chemotherapeutic agents may compromise graft success.[99–101]

Full-Thickness Skin Grafts

Full-thickness skin grafts (FTSGs) are chosen when local or distant flaps are not feasible or when the FTSG would offer acceptable cosmesis and function. Examples include the multioperated face, upper nasal surface defects, nasal lining tissue, and medial canthal area. FTSGs resist contraction and may possess the texture and color of normal skin. In children, FTSGs have the potential to grow.[98]

The FTSG is preferred over the split-thickness skin graft (STSG) in areas where a wound contracture may lead to a functional deformity. An example is the lower eyelid, where wound contracture would result in ectropion. An excellent FTSG for this example would include upper eyelid skin and orbicularis oculi muscle, which has been shown to predictably revascularize.

Selection criteria for a head and neck FTSG directs the surgeon to carefully consider particulars of a variety of sites—the upper eyelid, post- or preauricular skin, and lateral neck or supraclavicular region. For example, postauricular skin is photoprotected and has few adnexal structures, which may not be suitable for nasal defects. Preauricular skin grafts in males can lead to sideburn asymmetry. Supraclavicular and neck skin is thin and may be more photodamaged than the face. In addition, a supraclavicular scar may be a nuisance for women who wear clothing with low necklines.

The harvesting of most FTSGs involves cutting out a simple template of the defect (e.g., from suture packaging) (Figure 34-18). Because an FTSG contracts by 10% to 15% after

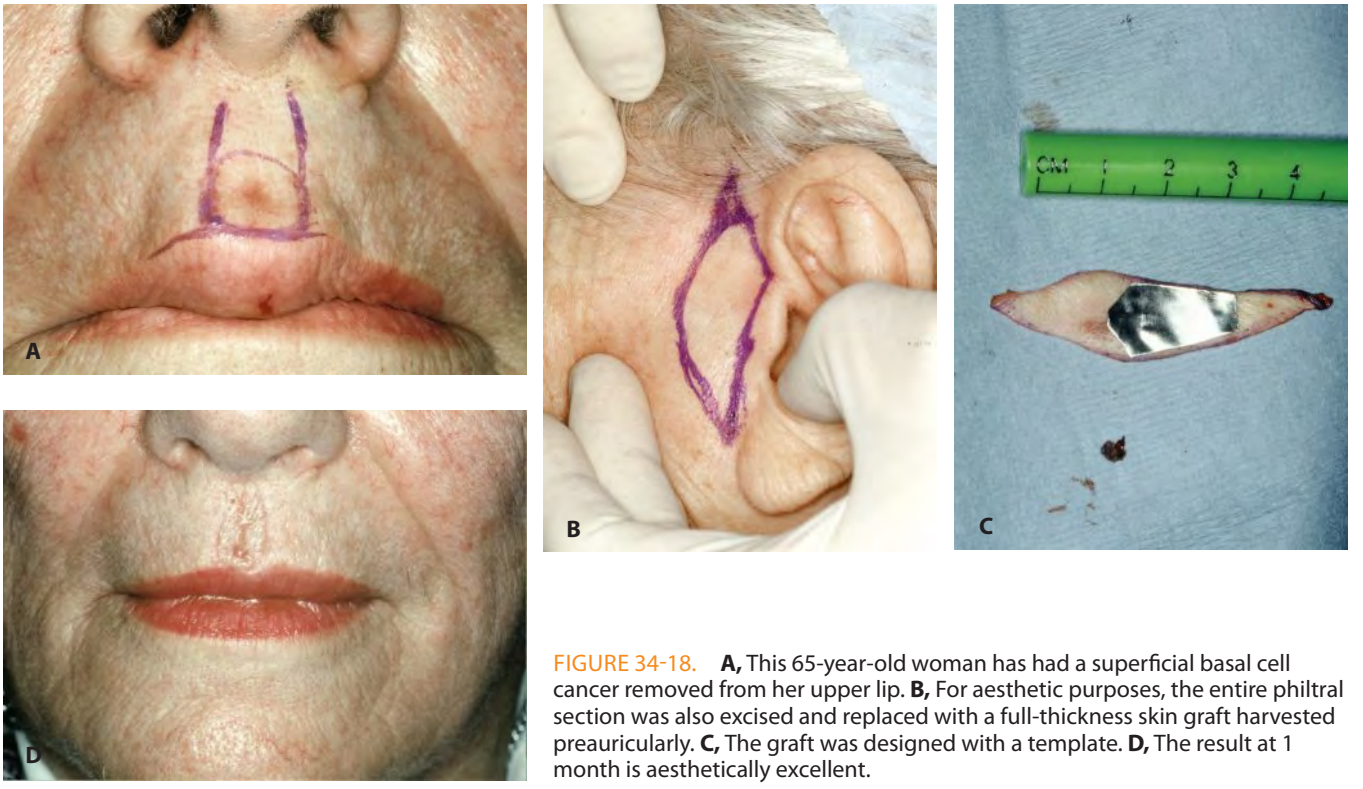


FIGURE 34-18. **A,** This 65-year-old woman has had a superficial basal cell cancer removed from her upper lip. **B,** For aesthetic purposes, the entire philtral section was also excised and replaced with a full-thickness skin graft harvested preauricularly. **C,** The graft was designed with a template. **D,** The result at 1 month is aesthetically excellent.

harvest, the donor graft pattern must be enlarged by around 20%.[102] This contracture issue is critical in areas of mobility such as in the lower eyelid. Here, grafts should be enlarged by 150% to 200% vertically to avoid ectropion/contraction occurring.[98,101]

The FTSG may be defatted with serrated scissors or by scraping with a blade. Defatting is complete when the shiny dermis is homogeneously exposed. FTSG should fit into a wound bed with maximum surface contact without any tenting.

Basting sutures may be used to affix the graft to the underlying bed to squeeze out dead space before peripheral suturing.[98,103] Peripheral sutures are easier to insert when passed from the graft through the host skin with a tapered needle.

Any nonadherent (to the graft) bolster of cotton, gauze petrolatum dressing, or plastic, for example, secured a few millimeters outside the grafted tissue is acceptable. Some surgeons prefer to remove the bolster after 48 hours to inspect the surgical site and then replace the bolster with a more conventional dressing.

FTSGs undergo an evolutionary sequence. Initially, a graft is white followed by a period of cyanosis or a bluish/violaceous hue. Subsequently, there is a period of hyperemia or a red state, which fades over time until the graft assumes its normal color. If the graft fails, the entire epidermis turns black and sloughs off, followed by reepithelialization.[98] The necrotic graft acts as a biologic dressing, allowing healing to occur by secondary intention from the wound edges as well as from adnexal structures.

Split-Thickness Skin Grafts

An STSG is defined as thin if its thickness measures 0.02 to 0.03 cm, medium from 0.03 to 0.046 cm, and thick from 0.046 to 0.076 cm. Thinner STSGs have improved survival rates compared with thicker ones because there is greater exposure of the graft to the underlying vasculature and less tissue is needed for revascularization. STSGs have a higher degree of contraction than do FTSGs and do not grow in children. Thin grafts afford less protection to the underlying tissues and do not withstand repeated trauma well. For example, an STSG may be chosen to cover a bare pericranium/skull after removal of a scalp tumor; subsequently, the patient may report breakdown sites or scabs from sleeping on the grafted sites.

STSGs are generally less pleasing cosmetically than are FTSGs and are employed for functional reconstruction (Figure 34-19).

STSGs are worthwhile for (1) wounds too large to repair with a local flap or an FTSG, (2) wounds requiring monitoring for tumor recurrence, or (3) temporary coverage of a wound before definitive reconstruction. Contraindications include areas that might compromise functional or aesthetic expectations.

STSG donor sites for facial reconstructions include the "blush zone" of the lateral neck and supraclavicular area and the scalp, owing to their similarity in color and texture. The hip, thigh, buttock, abdomen, torso, and inner aspect of the arm are also applicable at times.[98,100,104]

There are varying types of dermatomes ranging from machine to manual. The Brown dermatome allows for

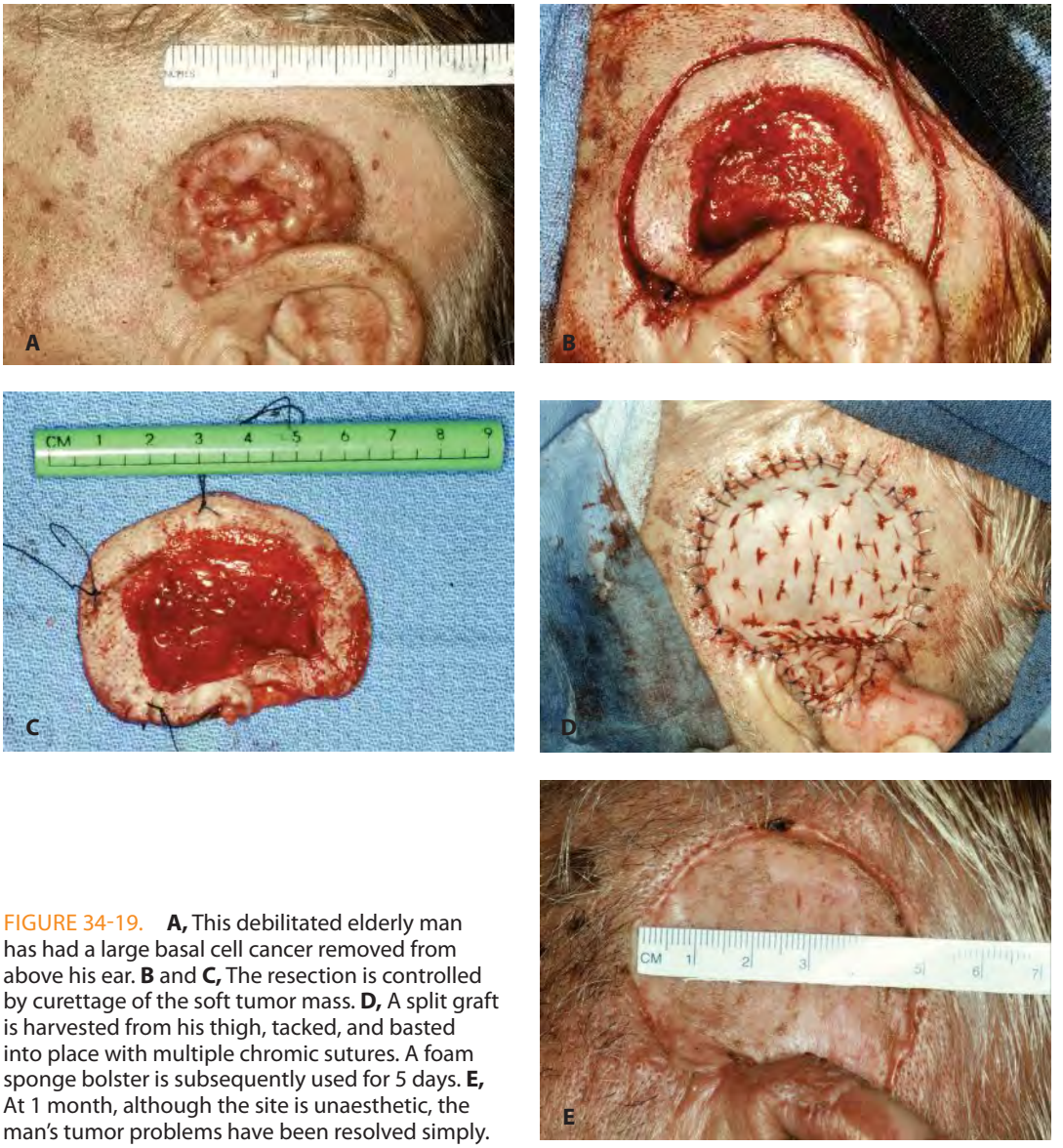


FIGURE 34-19. **A,** This debilitated elderly man has had a large basal cell cancer removed from above his ear. **B** and **C,** The resection is controlled by curettage of the soft tumor mass. **D,** A split graft is harvested from his thigh, tacked, and basted into place with multiple chromic sutures. A foam sponge bolster is subsequently used for 5 days. **E,** At 1 month, although the site is unaesthetic, the man's tumor problems have been resolved simply.

precise modification of graft thickness. The graft dimensions should be at least 25% larger than the wound defect.[98] Other dermatomes include the Padgett, Davol-Simon, Castroviejo, Reese, and Padgett-Hood dermatomes and the Weck knife.

The sterile donor site is lubricated with mineral oil. Traction and countertraction are applied, and the dermatome is engaged and advanced with a slight downward and forward pressure. The donor site bleeds if it is cut in the correct plane. After pressure or thrombin control, a semipermeable occlusive dressing covers the donor site and is left in place for 1 week to 10 days. Semipermeable membrane dressings decrease the pain of the donor site and enhance wound healing by maintaining a moist environment.[97]

Other dressing materials include Allevyn and Nobecutane spray. Allevyn is a hydrophilic polyurethane material that is highly absorbable and nonadherent. Its outer layer is waterproof and bacteria proof. The dressing is soft, absorbent, and comfortable for the patient. Nobecutane spray when applied on the wound forms a transparent plastic film. Nobecutane contains a modified acrylic resin in an organic solvent along with the bactericidal-fungicidal agent tetramethylthiuram disulfide. Brodovsky and colleagues[107] showed that this spray is an effective temporary dressing that promotes rapid painless healing. The film is shed spontaneously with epidermal regeneration.

Unlike for an FTSG, a tie-over dressing may not be necessary for an STSG. A good compression dressing and/or basting stitches may suffice to promote adherence between the graft and the underlying tissue and to prevent fluid accumulation. A variation to placing interrupted basting sutures is the spiral basting stitch. The suture is started at the edge of the graft with the "tail" left long.[108] The suture is then run along the periphery of the graft, spiraling toward the center, and then tied to the tail. The graft can then be dressed in a similar fashion to that for an FTSG.

Composite Grafts

Composite grafts contain two or more tissue layers. Composite grafts are ideal for reconstructing the nasal ala rim, auricular defects, and eyebrows. Composite grafts are able to maintain the thinness and contour of the structure with minimal contracture. The most common donor site for composite grafts is the ear, including the crus of helix, rim, antihelix, tragus, and earlobe.

A major disadvantage to composite grafts is the risk of graft failure, which is higher than for FTSGs and STSGs and is attributed to the high metabolic demands of the grafts. Harvesting (donor) adjacent dermis attached to the composite graft and inserting the deepithelialized dermis into adjacent subcutaneous tunnels (recipient) may improve vascularity substantially.[109] Cooling the composite graft with ice for 24 hours also helps.

Regardless, composite grafts are technique sensitive. Generally, composite grafts should be no larger than 1.5 to 2.0 cm. Avelar and coworkers[110] have shown composite grafts greater than 2.0 cm grafted successfully to nasal and auricular defects. Similarly, Skouge[104] has effectively grafted larger defects using a "tongue-and-groove" technique and turn-down hinged flaps. The postoperative appearance of composite grafts is distinctive. At placement, the graft is white or blanched. Within 6 hours, it becomes pink, and by 24 hours, it is cyanotic. By postoperative day 3, it resumes its pink color. Grafts that fail develop an eschar with subsequent necroses and sloughing.

Complications

There are risks to all procedures. Patients who receive skin cancer procedures should be warned of the potential for recurrence of the tumor as well as revision of any reconstructive procedure. Flap problems include necrosis, infection, hematoma, wound dehiscence, and scarring.

Smoking greatly increases the risk of necrosis. Patients who smoke one pack per day triple the risk of flap or graft necrosis compared with nonsmokers. Smoking affects the blood supply via two mechanisms. First, nicotine is a potent vasoconstrictor that may lower tissue oxygenation by greater than 50%. Nicotine effects are visible within 10 minutes and can last up to 50 minutes. Second, carbon monoxide is a competitor with oxygen for hemoglobin. It has a higher affinity for hemoglobin than does oxygen, resulting in high levels of carboxyhemoglobin. This leads to tissue hypoxia.

Infection

Infections are rare in vascularized head and neck tissues, and necrosis may be mistaken for infection. More common causes of redness include stitch abscesses, which are foreign body reactions, and allergies to antibiotic ointment. Infections, handled by drainage (when indicated), irrigation, and antibiotics, usually resolve readily.

Bleeding

Bleeding may be caused by patient factors or surgical issues. Patient factors include medical conditions such as renal failure, liver failure, collagen vascular disease, various cancers (hematopoietic malignancies), and medications. Medications that can cause bleeding include warfarin, heparin, antithrombotics, nonsteroidal anti-inflammatory drugs, acetylsalicylic acid, and cold remedies. Furthermore, commonly used herbal medications such as garlic, feverfew, and vitamin E can inhibit thrombocyte function.

The surgeon must weigh the benefits of discontinuing anticoagulants against the risks of surgery because there have been several documented cases of stroke when anticoagulants were stopped before dermatologic surgery. Consultation and coordination with the patient's internist and appropriate preoperative laboratory data are helpful. There is no need to discontinue any anticoagulant before performing a biopsy.

Surgical issues may arise intraoperatively or during the postoperative period. Decisions must be made concerning judicious cautery, the use of drains, the effect of vasoconstrictors, and postoperative pressure. Seepage may occur from any facial flap, but hematoma may necrose the flap. A hematoma, in the space created between the flap and the underlying tissue, is detrimental to flap circulation because it creates tension and acts as a physical barrier preventing cohesion to the underlying tissue base. In addition, stagnating blood may promote wound infection.

An early hematoma may often be pushed out and washed away, but a re-forming hematoma must be explored. Likewise, late collections of jellied blood should be manually extruded.

Poor Cosmetic Results

Facial flaps should restore anatomic continuity, maintain functional integrity, and provide an aesthetically pleasing result. In spite of well-executed surgical techniques, less than optimal results may occur because of unpredictable scarring and trapdoor deformity.

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35

CHAPTER

Salivary Gland Disease

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GENERAL CONSIDERATIONS

Salivary glands are divided into the paired major glands (parotid, submandibular, and sublingual), located in their specified anatomic locations in the head and neck, and the minor salivary glands found in the submucosa throughout the oral cavity, oropharynx, sinuses, and trachea. It is estimated that there are approximately 500 to 1000 lobules of minor salivary gland tissue dispersed throughout the oral/oropharyngeal mucosal and submucosal tissues of the lips, floor of mouth, hard and soft palate, tonsillar pillars, buccal mucosa, and tongue. Typically, development begins in utero around the 35th day for the major and 40th day for the minor glands; their origin is the ectoderm. Secretions by the glands in the form of saliva can be mucous, serous, or most commonly, mixed. Specifically, secretions from the parotid are mainly serous (viscosity = 1.5) and contribute about 25% of the daily saliva whereas those from the sublingual and minor salivary glands located more posteriorly in the oral cavity are more mucous in nature (viscosity = 13.4) and have only minor contribution to the daily saliva production. Seventy-one percent of the saliva is produced by the submandibular glands and is mixed in consistency with viscosity approximately 3.4. Saliva is produced at a rate of 1000 to 1500 mL/day and primarily functions as a lubricant for speech and mastication, while initiation of digestion due to enzymes and some antimicrobial properties are provided (Tables 35-1 and 35-2). The autonomic nervous system is responsible for the control of salivary secretions by the glands. Sympathetic fibers travel via arterial plexus from the superior cervical ganglion, whereas parasympathetic stimulation travels to the parotid via cranial nerve IX and to the submandibular and sublingual glands via cranial nerve VII (Figures 35-1 and 35-2).[1–4]

Anatomically, the parotids are located superficial and posterior to the masseter muscle and the mandibular ramus and are divided into superficial and deep lobes by the facial nerve (Figure 35-3). The saliva travels through a 6-cm-long, 1- to 3-mm-diameter duct, known as *Stensen's duct*, that opens into the oral cavity at the buccal mucosa adjacent to the second molar. The course of the duct involves a sharp turn at the anterior border of the masseter muscle medially through the buccinator before it enters the oral cavity (Figure 35-4). The submandibular glands are located at the submandibular triangle bilaterally formed by the two bellies of the digastric muscle, the mylohyoid, and the platysma. The gland loops behind the mylohyoid muscle and extends into the oral cavity, and it may be in intimate relationship or in continuity with the sublingual gland requiring sharp dissection during excision (Figure 35-5). Wharton's duct, 5 cm long 2 to 4mm in diameter, empties the saliva from the gland into the oral cavity at the punctum located at the midline of the anterior floor of the mouth. Along its course in the floor of the mouth, it receives saliva from the sublingual gland via the duct of Rivinus and is in close association with the lingual nerve that loops around the duct twice from lateral to medial (Figure 35-6). Finally, the sublingual gland is located between the mucosa of the floor of the mouth and the mylohyoid muscle in the sublingual space. Saliva from the gland is transferred either through Bartholin's ducts, which start within the gland and coalesce to form the ducts of Rivinus that subsequently empty into the Wharton's duct, or via short small ducts that open directly into the floor of mouth mucosa at the plica sublingualis.[2,5–16]

DIAGNOSTIC MODALITIES

The most important component in diagnosing salivary gland disorders is a detailed history and careful clinical examination. To augment the findings and aid in diagnosis, a variety of diagnostic imaging techniques are available that, for practical purposes, are employed for examination of the parotid and the submandibular glands. Plain films, either a

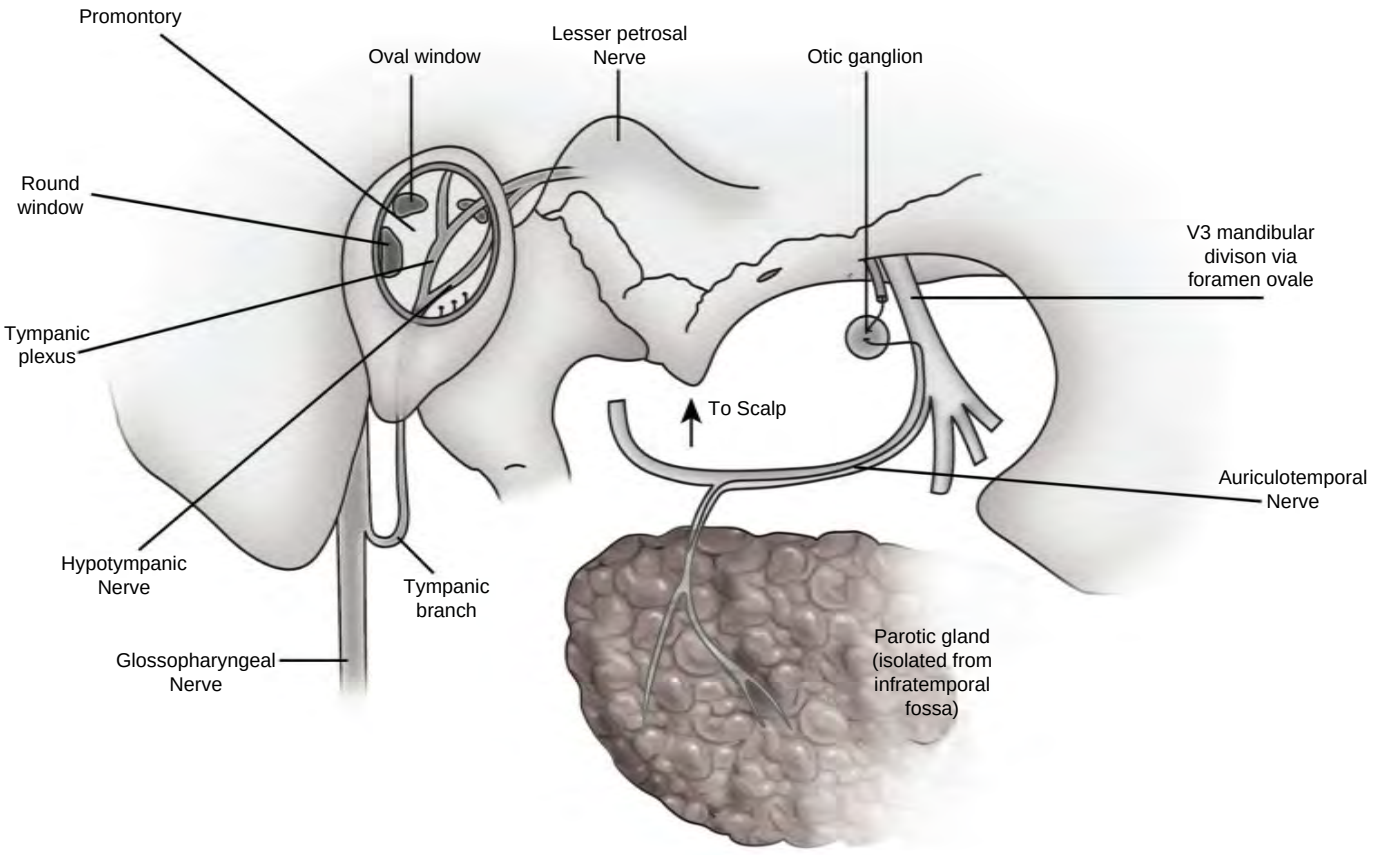


FIGURE 35-1. Innervation of the parotid gland.

mandibular occlusal and/or a panoramic radiograph, can be easily obtained in the office setting to quickly evaluate for the presence of a suspected sialolith (stone) in the submandibular gland ductal system because 80% to 85% of these stones are radiopaque (Figure 35-7). The incidence of radiopaque stones in the parotid is only 40%, limiting the value of plain films for sialolithiasis of this gland. Mucous plugs are more commonly involved in parotid obstruction and stasis. Similarly, computer tomography (CT) is very helpful for identification of radiopaque stones and, of course, provides superior information to the plain films with details about exact location of the stone and status of the duct proximally and distally (Figure 35-8).[2,7,9]

The gold standard for imaging the ductal system for salivary gland examination has traditionally been *sialography.* It is contraindicated in the presence of acute infection and iodine allergy. This modality provides important information regarding location and etiology of obstruction and the presence of inflammation and/or destruction of acini as well as allowing therapeutic dilation and irrigation of the ductal system in cases of obstruction. Specific imaging appearance of the gland parenchyma and its ductal system during the sialography are pathognomonic for normal anatomy and functional status as well as specific pathologic entities (Figure 35-9). Small stones or mucous plugs, sludge, or debris in the ductal system of the glands can be "flushed out" during the injection of the contrast. Technetium-99m scintigraphy assists in evaluation of the salivary gland parenchyma and function and also provides information for certain intraglandular tumors based on the radioisotope uptake. Approximately 80% of benign lesions will appear "cold," with the exception of Warthin's tumor and oncocytomas that will appear "hot"; lymphoid tumors will appear "warm." It has been demonstrated that, although sialography is superior in diagnosis and demonstration of obstructive conditions, scintigraphy is superior when neoplasms are suspected.

Ultrasound (US)-assisted examination of salivary gland pathology has been claimed to be superior to sialography, and with color Doppler imaging, the limitations of distinguishing between benign and malignant tumors may be improved. Distinction of fluid-filled versus solid masses can be easily and quickly made owing to the superficial position of the glands that allows for imaging with high-resolution US probes. The noninvasive nature of the study makes it an attractive alternative to other modalities especially when contraindications, such as allergy to intravenous contrast or claustrophobia, limit their use. In addition, US-guided aspiration of fluid or biopsy of deeply located masses can provide valuable information in experienced hands. At present, the main limiting factor remains the ability to acquire accurate information in cases of minor salivary gland disease, inflammation, and coexistence of small tumors with other conditions, and in the United

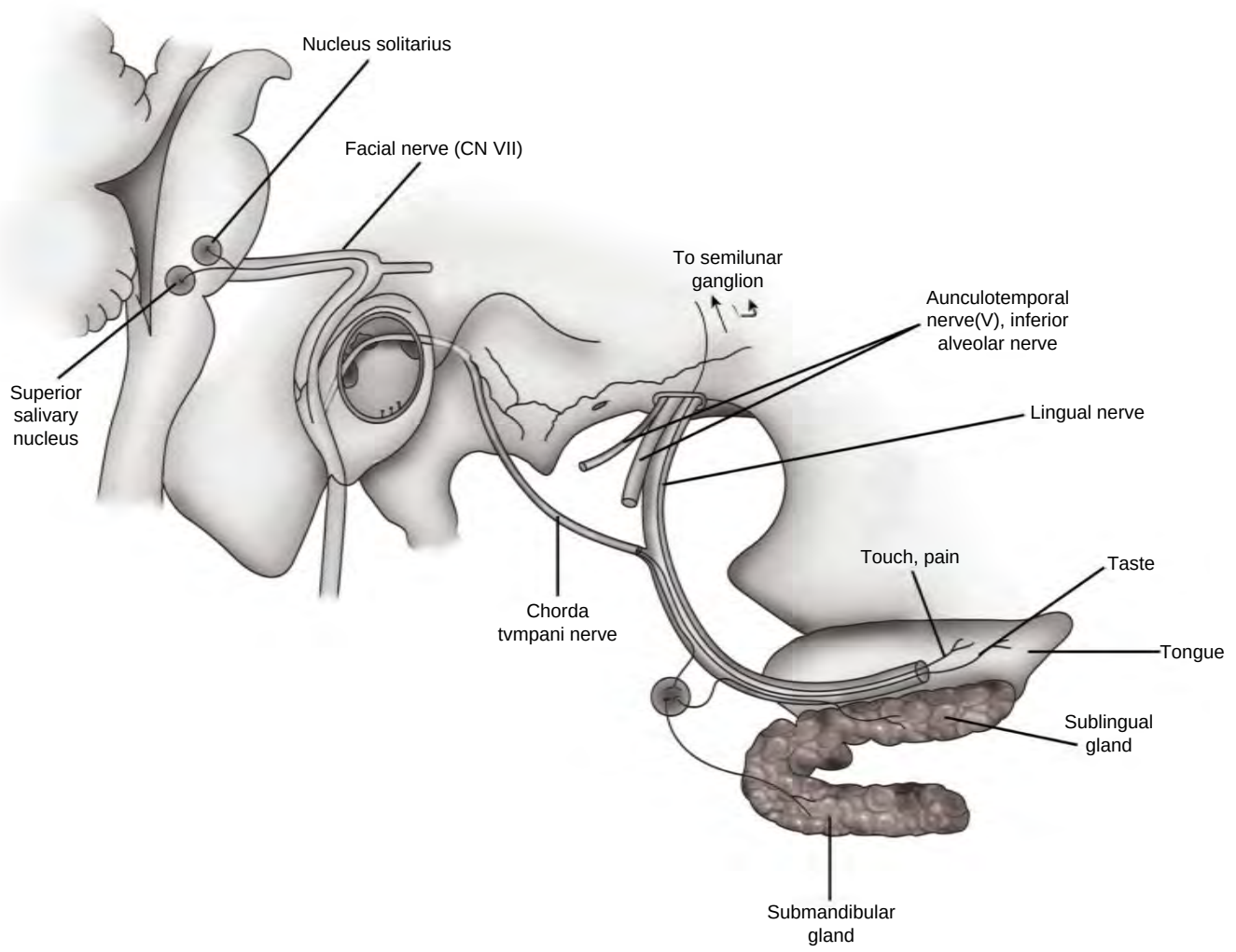


FIGURE 35-2. Innervation of the submandibular and sublingual glands.

TABLE 35-1. **Composition of Adult Stimulated Saliva**

Content	Parotid	Submandibular	Change with Flow Increase	Comments
Saliva flow rate	**0.7 mL/min**	**0.6 mL/min**		
Inorganic (mEq/L)				
Sodium	30	21	Increase.	Gland, type, and amount of stimulation dependent.
Chloride	23	20	Increase.	Equivalent to or lower than plasma.
Potassium	20	17	Increase.	Gland, type, and amount of stimulation dependent.
Bicarbonate	20	18	Increase.	Exceeds plasma concentration with stimulation. Major buffer.
Phosphate	6	4.5	Increase.	Higher than plasma concentration. Minor buffer.
Calcium	2	3.6	Increase only in high rates; otherwise no change.	Higher than plasma concentration. Free ion or bound to proteins.
Magnesium	0.2	0.3	No change.	Lower than plasma concentration (two thirds).

(Continued)

SECTION 4

TABLE 35-1. **Composition of Adult Stimulated Saliva *(Continued)***

Content	Parotid	Submandibular	Change with Flow Increase	Comments
Organic (mg/dL)				
Protein	250	150		
Urea	15	7		
Uric acid	3	2		
Total lipids	2–6	2–6		
Amino acids	1.5	—		
Fatty acids	1	—		
Ammonia	0.3	0.2		
Glucose	<1	<1		
Cholesterol	<1	—		

Modified from Mandel ID. Sialochemistry in diseases and clinical situations affecting salivary glands. Crit Rev Clin Lab Sci 1980;12:321–366.

States at least, the lack of highly trained personnel for good information acquisition and interpretation.[17–30]

Magnetic resonance imaging (MRI) remains the gold standard for evaluation of salivary tumors (benign or malignant) (Figure 35-10). MRI is superior to CT scan for evaluation of masses because it provides excellent soft tissue detail and delineation of the margins between tumor and glandular tissue. MRI is superior in detection of minor salivary gland pathology and detection of small tumors within inflammation, for example, cases of Sjögren's disease and coexistence of malignancy. In addition, MRI is the only imaging modality that can provide information regarding involvement of cranial nerves with tumor when clinical findings are not present or evident. Most recently, dynamic MRI has been used in evaluation of salivary gland function in patients with Sjögren's disease with promising results.[15,31–33]

Positron-emission tomography (PET) combined with fluorodeoxyglucose (FDG) uptake is extensively studied and accepted in cancer staging, therapy monitoring, and management of recurrent disease. Studies of the utility of FDG-PET for salivary gland neoplasms have demonstrated that there is definitely a role for FDG-PET in major salivary gland malignancies for initial staging, histologic grading, and monitoring or restaging post-treatment. In addition, FDG-PET may have a role in incidental findings of tumors or cases of benign pathology, such as Warthin's tumor, in

TABLE 35-2. **Major Salivary Molecules and Their Function**

Protein Family	Molecular Weight (kDa)	Function
Mucin 1(MG1)	>1000	Antibacterial, antiviral, digestion, lubrication, tissue coating
Secretory IgA	380	Immune response
Mucin 2 (MG2)	130	Antibacterial, antiviral, digestion, lubrication, tissue coating
Lacoferin	75–78	Tissue coating
Peroxidase	75–78	Antibacterial
Amylase	55–60	Antibacterial, Digestion, Tissue Coating
Carbonic anhydrases	42–45	Buffering
Proline-rich proteins	10–30	Mineralization
Cystatins	14	Antibacterial, antiviral, mineralization, tissue coating
Lysozyme	14	Digestion
Statherins	4–6	Mineralization, lubrication, tissue coating
Histatins	2–4	Antibacterial, antifungal, mineralization

IgA = immunoglobulin A.
Modified from Levine M. Salivary macromolecules: a structure/function synopsis. Ann N Y Acad Sci 1993;694:11; and Humphrey SP, Williamson RT. A review of saliva: normal composition, flow, and function. J Prosthet Dent 2001;85:162.

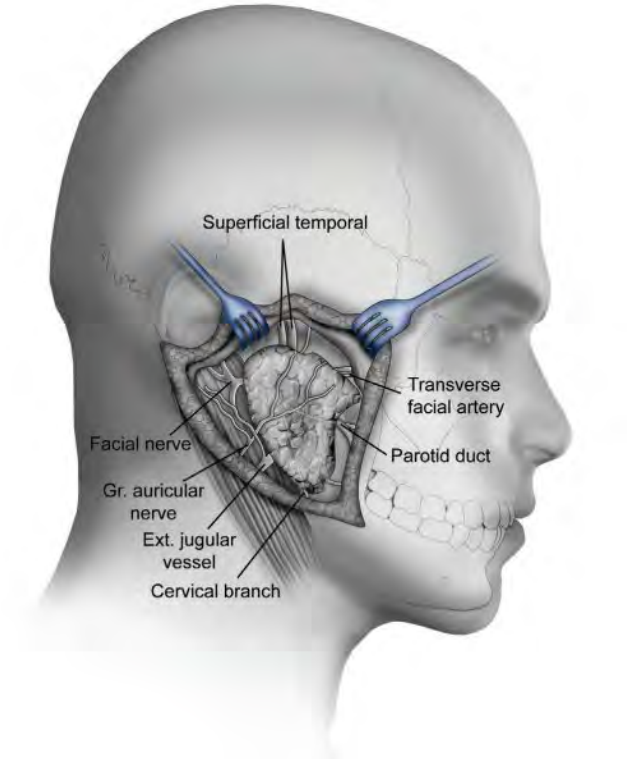


FIGURE 35-3. The parotid is divided into superficial and deep lobes by the facial nerve.

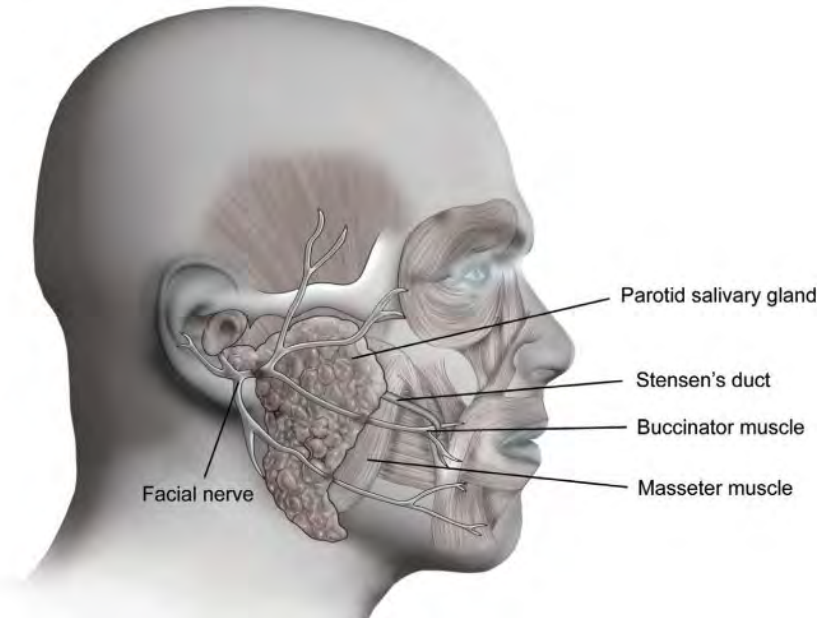


FIGURE 35-4. Stensen's duct.

which the findings of other imaging modalities, such as scintigraphy, may be misleading.[29,34–37]

The interest in using saliva as a potential diagnostic tool over the last few decades is reflected in the literature by the proliferation of information from *sialochemistry* and salivary gland immunoglobulin assay studies. The collection of saliva and analysis of its chemical composition may provide information regarding involvement of salivary glands in systemic disease processes or in cases of salivary pathology with no systemic manifestations or evaluate the functional status of the gland. Specifically, alterations of sodium and potassium relative concentrations are evident in inflammatory conditions and can be demonstrated with sialochemistry studies.[3,5,9,38–45]

Biopsy, in the form of open biopsy or *fine-needle aspiration biopsy* (FNAB), remains the gold standard for diagnosis of salivary gland tumors. Open biopsy for major salivary glands harbors the potential for facial scar, injury to the facial nerve or development of fistula or sialocele, violation of tumor capsule, and tumor spill so it is restricted to the minor gland pathology. FNAB has high specificity and sensitivity (>90%), provides accurate diagnosis, and is preferred for masses of the major glands. In addition, with the use of CT guidance, FNAB can be employed to access deeply

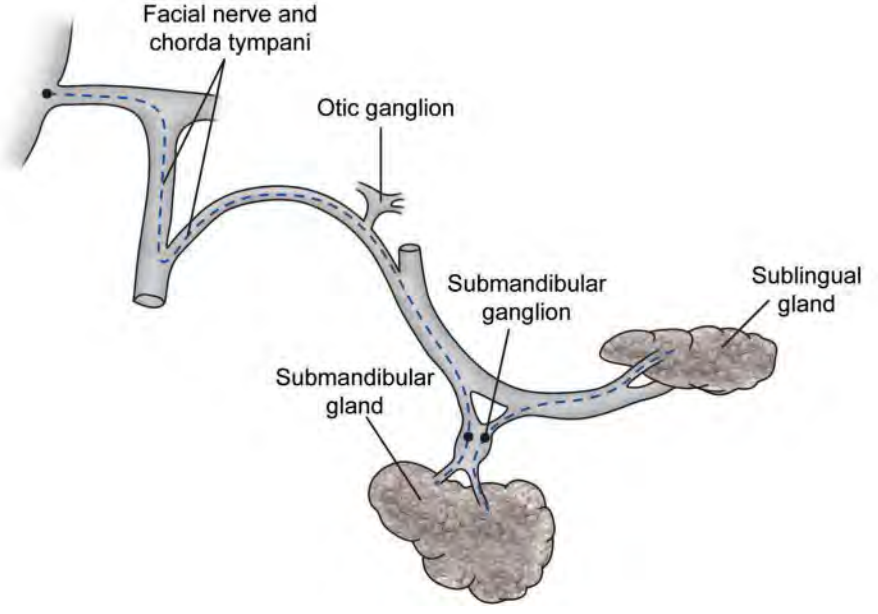


FIGURE 35-5. Relationship of the sublingual and submandibular glands.

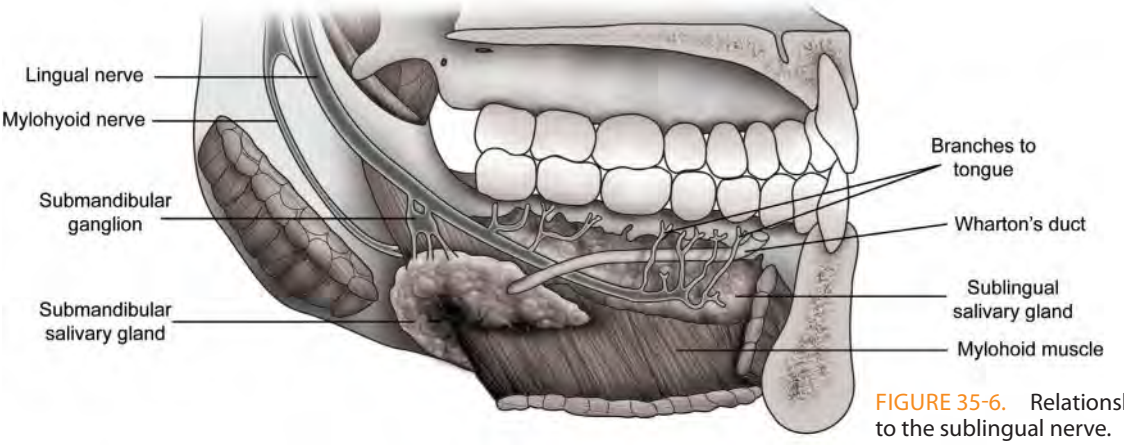


FIGURE 35-6. Relationship of Wharton's duct to the sublingual nerve.

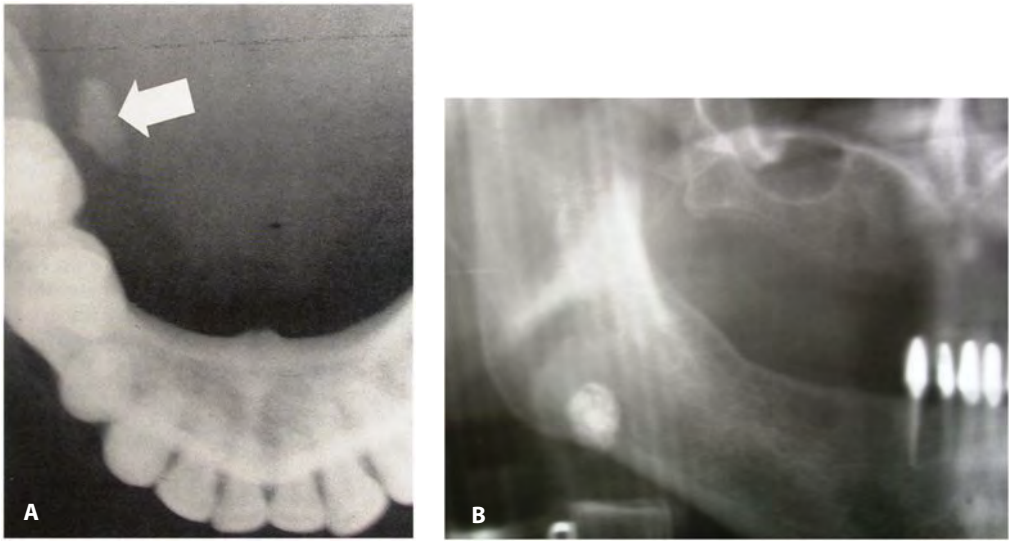


FIGURE 35-7. **A**, Occlusal radiographic film demonstrates a sialolith along Wharton's duct. **B**, Panoramic radiograph demonstrates a sialolith in close proximity to the hilum of the gland

located or small tumors that could be missed with blind approaches. Open biopsy of minor salivary glands from the lower lip remains the standard approach for diagnosis of Sjögren's syndrome (SS). A minimum of 10 minor glands are needed for histologic examination and assignment of a "focus score." Under high magnification, an aggregate of 50 or more lymphocytes in glandular tissue represents a "focus"; the presence of more than one focus in 4 mm^2 is regarded as being consistent with the salivary component of SS.[2,9,44,46]

NON-NEOPLASTIC SALIVARY GLAND DISORDERS

Obstructive Disease: Sialolithiasis

Stone or calculi formation is a common phenomenon occurring in the kidneys, gallbladder, and the salivary glands. *Sialolithiasis* occurs twice as often in men, between 30 and 50 years of age, as in women, and multiple stones may be present in 25% of the patients. An abnormality of calcium metabolism and salt precipitation appears to be the initial of a series of stages that results in formation of a nidus, which subsequently forms a calcified mass (sialolith) due to layering of organic and inorganic material. Recurrent episodes of swelling of the involved gland, occurring during meals, are the usual complaints. Careful inspection along with bimanual palpation of the involved gland and its duct will provide



FIGURE 35-8. Computer tomography demonstrates a submandibular sialolith within the ducal parenchyma

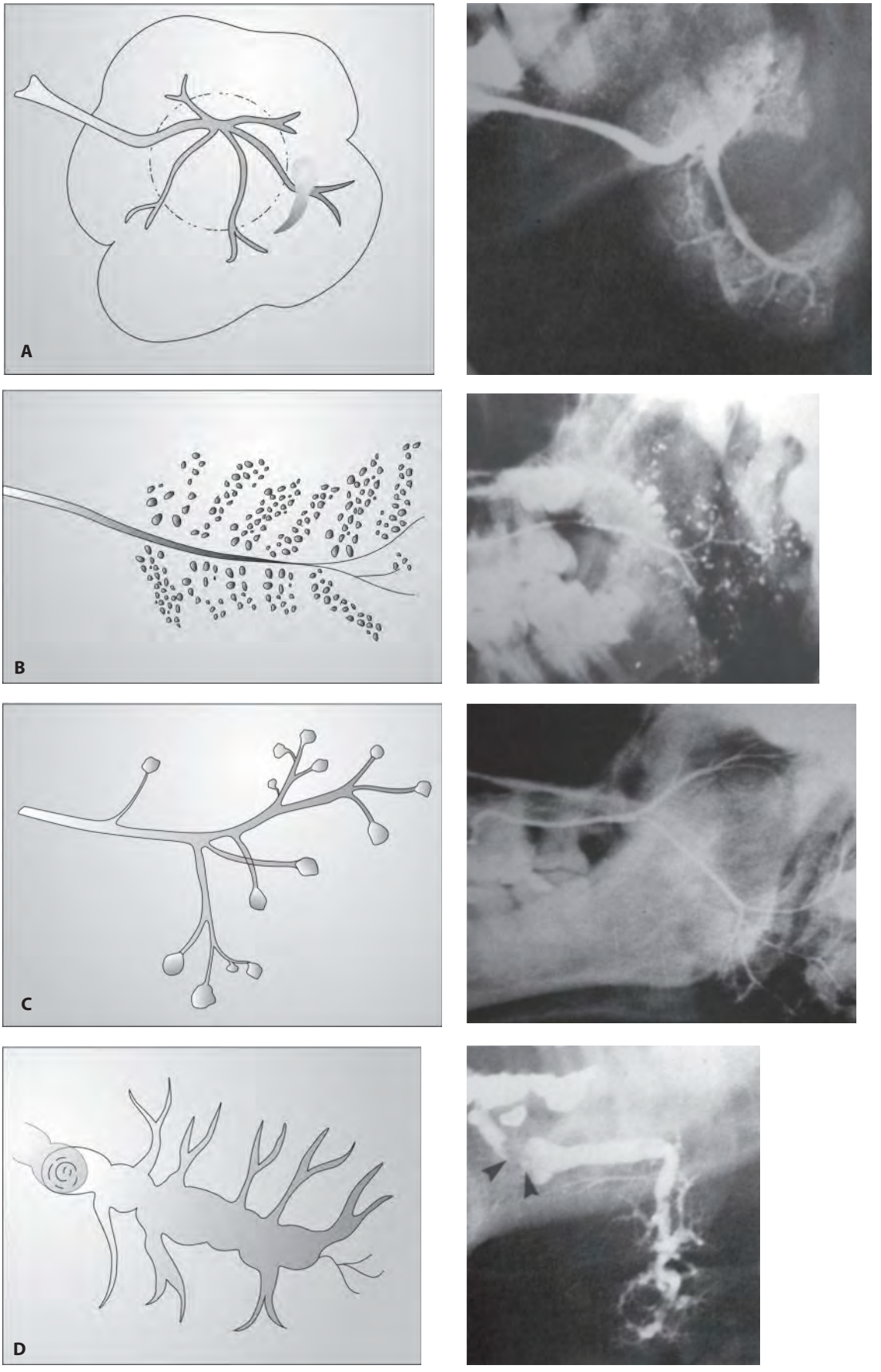


FIGURE 35-9. Schematic demonstration of sialograms. **A,** Sialolithiasis. **B,** Sjögren's disease. **C,** Sialoadenitis. **D,** Neoplasm.

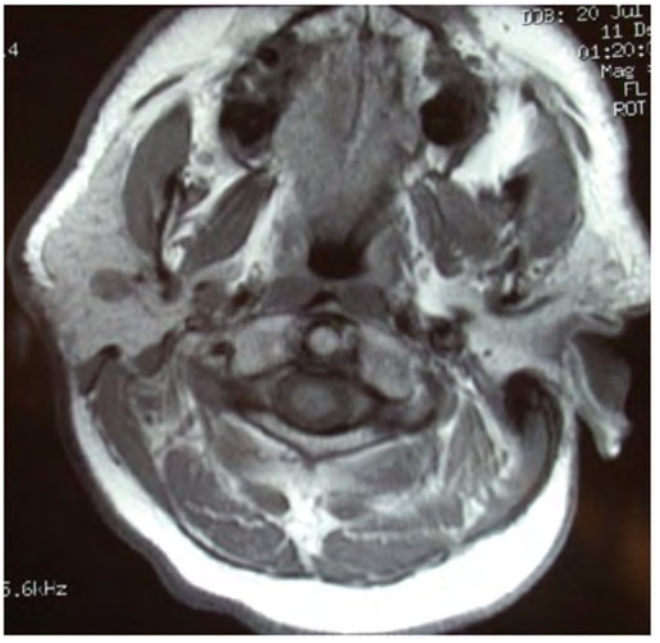


FIGURE 35-10. Magnetic resonance imaging demonstrates a mass in the right parotid.

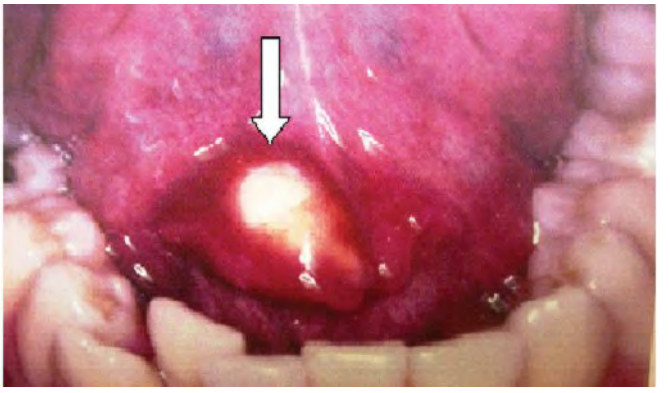


FIGURE 35-11. Sialolith at the opening of Wharton's duct.

valuable information. Occasionally, purulent discharge may be expressed from the duct when secondary infection is present, along with constitutional symptoms such as pain, fever, dehydration, halitosis, and reactive lymphadenopathy. Laboratory workup may indicate elevated white blood cell count in infected cases.

Stones occur more often in relationship to the submandibular gland, and the composition of the saliva from this gland in addition to several anatomic factors appear to be the contributing factors. Submandibular gland saliva contains twice the amount of calcium and has more alkaline pH than that produced from other glands, in part explaining the frequency of sialolith formation. The actual route of Wharton's duct from the gland to the floor of the mouth, with two sharp turns, the smaller diameter of the punctum compared with the duct, and the flow of saliva against gravity further contribute to stone formation.

Sialoliths that occur anterior to a transverse line drawn between the first mandibular molars may be visualized with occlusal films and can be approached for removal intraorally if not spontaneously passed after adequate hydration and sialagogue therapy (50% of the cases) (Figure 35-11). The duct is cannulated first, then the stone is identified and a ligature suture is place distal to it before opening the duct. After removal of the sialolith, sialodocuplasty is performed, creating a new opening in the floor of the mouth or transposing the ductal opening distally to ensure future unobstructed salivary flow. Stones located more proximally (behind the posterior border of the mylohyoid muscle) often require surgical excision of the involved gland especially when it is nonfunctional after recurrent episodes of obstruction and parenchymal destruction (Figure 35-12). Sialolithotripsy via extracorporeal electromagnetic shock waves has been shown to be successful in treatment of small stones. The number, size, and location of the stones appear to be some of the limiting factors of this technology. Carlson[47] and Yu and coworkers,[48] among others, have published useful algorithms to guide in the management of obstructive sialoadenitis and sialolithiasis.



FIGURE 35-12. Stones located proximally or within the gland parenchyma often require surgical excision of the involved gland.

Sialoliths of the parotid and Stensen's duct present the same clinical findings and complaints, but these are more commonly mucous plugs found on MRI or sialography, whereas calcified stones may be visualized with CT scan. Access depends on the location along the duct or within the glandular parenchyma. Sialoliths found in the duct after it pierces the buccinator muscle may be removed intraorally via ductal dilation or a mucosal incision over the duct. Occasionally, the proximal location of the stone, with recurrent episodes of obstruction and chronic sialoadenitis, may require superficial parotidectomy.

Sublingual gland sialolithiasis is a very rare event, with only one study available from Germany[49] reporting an incidence of 7%. The clinical findings are consistent with obstruction of Wharton's duct. Sialolithiasis of the minor salivary glands is an equally rare event, and if present, microliths are incidental histologic findings upon removal of mucoceles (mucous retention cysts) and of no clinical importance. Sialography, as stated earlier, may be employed for diagnostic and therapeutic purposes in cases of sialolithiasis caused by small stones in Wharthin's or Stensen's ducts that may be "flushed out" during the examination when secondary infection is not present.[2,7,9,10,44,50–53]

In the last decade, the use of endoscopy for examination of the salivary ductal system and retrieval of sialoliths or treatment of a variety of other conditions, such as mucous plugs, foreign bodies, ductal strictures, or kinks, has shown excellent results. Sialoendoscopy has been successfully employed for removal of sialoliths in close proximity to the hilum of the submandibular gland or the parotid without necessitating removal of the gland. The revolution in endoscopy with miniature scopes and specialized instruments that can be introduced through them into the ductal system has provided surgeons with the ability to use this approach for treatment of salivary gland obstructive disease. As a diagnostic tool, endoscopy can provide information in place of or augmenting the traditional diagnostic modalities for obstructive and inflammatory disorders of the glands. In addition, strictures or adhesions in the ductal system are excellent indications for balloon dilation via endoscopic approaches. Perhaps the major advantage of sialoendoscopy in experienced hands appears to be ability to preserve the glands and restore function with only few potential complications in a minimally invasive manner.

Intracorporeal fragmentation of larger stones may be required and can be either mechanical, laser-assisted, or performed via pneumatic methods and can be done endoscopically. With graspers or forceps introduced through the scopes, the stone can be broken in smaller pieces and removed. Alternatively, erbium:yttrium-aluminum-garnet (Er:YAG) or holmium:yttrium-aluminum-garnet (Ho:YAG) lasers may be employed to fragment the stones before removal. Damage to the laser probe from direct contact with the stone required for fragmentation is the major limitation for the Er:YAG lasers at the present time. Potential displacement of the sialolith fragments from use of Ho:YAG laser and time required are the major disadvantages of this technique. Finally, high pressure introduced via rigid probes through the endoscope has been reported for fragmentation of large sialoliths. The stones are then removed with the basket technique, but limitations of compatibility of the available probes with the endoscopes used remains a problem. Details regarding equipment, advances, case selection, risks, and potential complications of sialoendoscopy are well documented in the literature by pioneers in the field.[9,54–60]

Cystic Conditions

Cystic conditions include mucous extravasation and retention phenomena, ranulas, sialoceles, and parotid gland cysts

Perhaps the most commonly encountered "cystic condition" of the minor salivary glands, especially those of the lower lip, is the *mucocele*, which represents a mucous extravasation phenomenon. The etiology of mucocele formation is usually trauma to the labial minor salivary glands from accidental biting and is very common with younger patients (Figure 35-13). The escape of mucin into the tissues triggers an inflammatory reaction by neutrophils and macrophages. The granulation tissue that forms creates a capsule around the free mucin, while the injured gland undergoes inflammatory changes. With repeated injuries, the capsule ruptures and the mucocele disappears only to re-form after the mucosal heals. With time, scarring and fibrosis may occur and the contributing glands undergo atrophy. Approximately 20% of these cystic lesions are true retention cysts due to obstruction of the minor salivary gland opening by a microlith and occur in older individuals. In these cases, a true cystic lining is present that originates from normal but compressed ductal epithelium. Differential diagnosis includes traumatic fibromas and salivary gland or mesenchymal neoplasms. The treatment of mucoceles or mucous retention cysts is removal along with the contributing glands. "Recurrent" cases (incidence 15–30%) are usually due to incomplete initial removal or repeated trauma. Use of CO_2 laser for



FIGURE 35-13. Mucocele/mucous extravasation phenomenon of the lower lip.

treatment of the surgical bed and healing by secondary intention have been reported with some success for recurrent mucoceles.

Ranulas are mucous extravasation phenomena (mucoceles) of the floor of the mouth associated with the sublingual gland and are very common in children. Trauma and dysgenetic development of the sublingual ductal system appear to be the most accepted causative theories for this condition. Accumulation of saliva under the thin mucosa of the floor of the mouth causes the pathognomonic appearance of swelling under the tongue or "frog's belly" (*rana* = "frog") (Figure 35-14). Differential diagnosis for ranulas includes salivary gland or mesenchymal tumors and other cysts such as lymphoepithelial, dermoid, or epidermoid cysts. The mylohyoid muscle separates ranulas that are contained in the floor of the mouth from those that extend into the neck or "plunging ranulas" and may compromise the airway. CT scan or MRI will provide the necessary information for accurate diagnosis especially in cases of plunging ranulas because other neck masses or cysts need to be excluded. Differential diagnosis for neck masses include thyroid pathology, thyroglossal duct cyst, or sublingual dermoid cysts when present in the midline or lymphadenopathy, submandibular gland swelling, branchial cysts for lateral neck involvement. Marsupialization has been shown to be successful as initial treatment of ranulas contained in the oral cavity. Definitive treatment is by removal of the sublingual gland along with the ranula with care taken not to injure the lingual nerve or Wharton's duct, which requires identification and canalization of the latter.

Sialoceles are very distinct salivary retention phenomena most commonly involving the parotid and less often the submandibular glands and are the result of penetrating injury to the respective ductal system. Thinning of the skin overlying the sialocele and tenderness are common findings. Repair of the injury and prevention of fibrosis or stricture formation along with sialogogues and hydration are usually successful. In most cases, either from ductal or parenchymal injury, the sialocele can be cured with botulinium toxin injections. In cases of ductal injuries deep into the gland or when repair is not feasible, superficial parotidectomy may be considered.[2,9,44,52,53,61]

Cysts involving the submandibular gland are rare whereas those involving the parotid are fairly common, occurring as either polycystic disease or solitary entities, clinically indistinguishable from tumors. Among solitary lesions, lymphoepithelial cysts are the most commonly encountered, but they require differentiation from salivary gland neoplasms (Figure 35-15). Most often, pleomorphic adenomas, mucoepidermoid carcinomas, or Warthin's tumors may appear cystic on imaging and are discussed in detail later in this chapter. Lymphoepithelial cysts histologically consist of marked lymphoid hyperplasia and squamous lined cysts and have recently been described in patients with acquired immunodeficiency syndrome (AIDS) or AIDS risk factors. Bilateral involvement of the parotids is common, and occasional superficial parotidectomy is performed for cosmetic purposes or in cases of facial asymmetry.[62–66]

Inflammatory

Infections of major salivary glands may be due to either bacterial or viral etiology and may be acute or chronic/recurrent. Most commonly, chronic or recurrent infections are due to an



FIGURE 35-14. Clinical picture of a ranula.

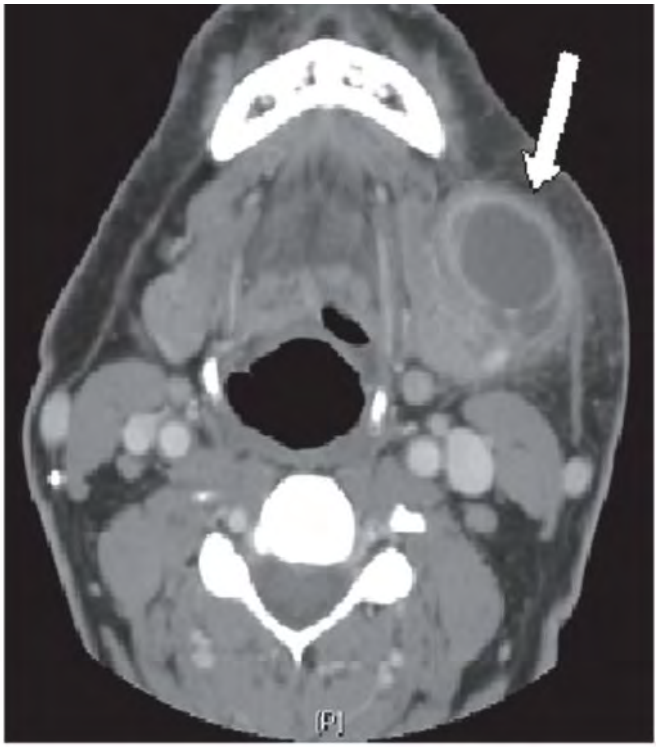


FIGURE 35-15. Computed tomography of a cyst in the right submandibular gland.

underlying etiology such as obstruction from sialoliths or ductal strictures or kinks, and treatment is correction of the primary etiology, as discussed earlier.

Acute suppurative sialoadenitis involving the parotid is the most common salivary gland infection and is usually due to ascending infection from the oral cavity in elderly, debilitated, chronically ill, or hospitalized immunosuppressed individuals. In the past, patients after surgery suffered routinely from acute suppurative sialoadenitis due to poor understanding and management of fluid balance. In general, there is a slight predilection for men over age of 60, and usually, there is underlying fluid imbalance and associated dehydration that allows superinfection with *Staphylococcus (aureus* or *viridians*, all penicillin resistant) that normally colonize the ductal openings. The decreased salivary flow allows for entrance of the bacteria into the ductal system and glandular parenchyma causing the infection. Streptococcus or other aerobic and anaerobic bacteria or fungus may be involved, and in the majority of cases, a mixed flora is isolated. As in every infection, pain, erythema, and swelling over the involved gland are present that must be differentiated from an odontogenic infection along with constitutional symptoms, fever, malaise, or tachycardia. Clinical examination will reveal a lack of saliva flow from the ductal opening or expression of thick purulent discharge when the gland is "milked." Laboratory findings include elevated erythrocyte sedimentation rate along with leukocytosis usually with a left shift consistent with the acute nature of the infection.

Treatment of acute bacterial sialoadenitis involves symptomatic and supportive care with fluid replacement via intravenous hydration and antibiotic therapy, analgesics, and antipyretics. Initial antibiotics are chosen based on the most commonly involved organisms such as a first-generation cephalosporin or semisynthetic penicillin against *Staphylococcus* species until cultures and sensitivity results are available. Occasionally, surgical drainage is required and incisions are placed in cosmetically acceptable areas avoiding injury to the facial nerve or the ducts. Hematogenous spread of bacterial infections to the parotid lymph nodes is common in infants and children and causes suppuration and nodal breakdown that require surgical drainage.

Chronic/recurrent bacterial infections are of multifactorial etiology and may involve a sequence of events that make the glands vulnerable to recurrent infections due to fibrosis and microcystic formation. These finding are well documented in the salivary gland sonography literature. Two entities are worth mentioning: chronic recurrent juvenile (parotid) sialoadenitis and chronic sclerosing submandibular sialoadenitis (Kuttner's tumor). The former is caused by a congenital abnormality of Stensen's duct (that is large with a poor seal) or the gland itself that may have poor function due to repeated viral parotitis. Long-term antibiotic use and steroids have anecdotally been reported to be successful treatments with this condition. However, sialography that shows the pathognomonic snowstorm sialectasis is frequently curative. Kuttner's tumor is the formation of a painful swollen gland commonly presenting in the elderly. It is usually associated with sialoliths or chronic nonspecific infections. Similar conditions may be found in other glands.

Mumps is the most common viral nonsuppurative condition that involves the parotid gland and is usually bilateral. Since the introduction and routine administration of attenuated vaccine for measles, mumps, and rubella (MMR) in 1967, the yearly incidence of mumps in the United States decreased from 76 to 2 cases per 100,000. The common cause is an influenza/parainfluenza-related paramyxovirus with epidemic outbreaks occurring in spring and winter months. Other potential viruses include Coxsackie A and B, Ebstein-Barr, influenza, and parainfluenza as well as human immunodeficiency virus (HIV). Fever, chills, headache, and preauricular pain occur 1 to 2 days before unilateral or bilateral swelling of the parotid glands that may last between 5 and 10 days. Supportive therapy and hydration without excessive stimulation of the glands are adequate because spontaneous resolution occurs in the uncomplicated cases. Of significance is the risk for development of mumps pancreatitis, and in 20% of adult male patients, mumps orchiitis.

Salivary glands in HIV-positive individuals are diffusely involved and the condition is referred to as *HIV associated salivary gland disease.* The usual presentation is asymptomatic swelling of one or more major glands that may fluctuate but most commonly is persistent and is due to the presence of lymphoepithelial cysts which have a pathognomonic appearance on MRI. Another common condition among HIV patients is diffuse infiltrative lymphocytosis syndrome (DILS) that causes xerostomia and sicca symptoms. Compliance with antiretroviral medications should assist with these conditions along with meticulous oral hygiene for prevention of opportunistic anterograde infections. The clinician should be careful not to miss a lymphoma of the salivary glands, which is also more common in AIDS patients and may be mistaken for one of the previously discussed entities.

Relatively rare conditions include parasitic infections or tuberculous mycobacteria or nontuberculous mycobacterial disease. The latter conditions, found in young children and immunocompromised individuals, may mimic salivary gland tumors from which they require differentiation.[9,44,45,47,52,62,64,67–75]

Noninflammatory/Autoimmune Conditions

SS is a very common chronic autoimmune disorder involving mostly the exocrine glands and affecting 0.3% to 0.6% of the population. Almost all cases involve both salivary and lacrimal glands with persistent complaints of dry mouth (xerostomia) and dry eyes (keratoconjunctivitis sicca). There is striking female predilection because 9 out of 10 patients are perimenopausal women in their 50s. SS may occur alone (primary SS) or in association with other autoimmune conditions such as systemic lupus erythematosus (SLE),

rheumatoid arthritis (RA), or scleroderma (secondary SS). The etiology is currently unknown; various environmental, viral, and hormonal causes are implicated in the development of the condition. The glandular parenchyma is infiltrated and eventually destroyed by lymphoplasmacytic infiltration and replacement. Clinical manifestations and complaints of xerostomia and dry eyes are classic findings in primary SS. Gastrointestinal, endocrine (mainly autoimmune thyroid disease), neurologic, hematologic, and connective tissue involvement are responsible for complaints and clinical findings in secondary SS. Interestingly, patients may have vague symptoms such as arthralgia, myalgia, and fatigue for years before the diagnosis of secondary SS is established. Bilateral symmetrical and recurrent parotid swelling may be present in 50% of these patients.

Sialochemistry studies, scintigraphy, contrast sialography, salivary flow rate studies, and minor salivary gland biopsy may be employed for assessment of the salivary component in SS. Lacrimal involvement is classically tested with Schirmer's test: 35-mm standardized sterile strips are placed in the fornix and the millimeters of "wetting" in 5 minutes is measured (normal > 10 mm in 5 min). Findings of sialography and scintigraphy suggestive of SS, as well as the grading system developed for in labial biopsy for diagnosis of SS, are discussed earlier in this chapter. Autoantibodies such as rheumatoid factor, antinuclear antibodies, anti-SS-A and -B (SS-A and SS-B) antibodies are found in both primary and secondary SS. Immunogenetic typing studies have demonstrated that expression of specific histocompatibility antigens (HLA) is statistically significant for each type of the disease but are only a component of the potential diagnostic criteria. In 2002, classification criteria for SS were established by the American European group and include ocular and oral symptoms, ocular and oral tests, positive labial gland biopsy, and anti-SS-A and SS-B antibodies (Table 35-3)[47]. Diagnosis of primary SS requires at least four of the six criteria whereas secondary disease requires establishment of connective tissue disorder, at least one (sicca) symptom, and two objective tests of ocular and oral involvement.

Treatment for SS is mostly supportive with artificial tears and saliva substitutes, whereas use of sialagogues may assist in stimulating flow from the remaining functional salivary gland tissue. Probably the most concerning complication associated with SS is that patients are at higher risk than the general population in developing lymphomas. The overall prevalence is estimated to be 4%.Usually, these are mucosal-associated lymphoid tissue (MALT) lymphomas, which are low-grade non-Hodgkin's type with good prognosis. If high-grade lymphomas occur, the life expectancy is affected.[9,42,43,46,51,73–78]

Necrotizing sialometaplasia is a reactive non-neoplastic process most commonly involving the minor salivary glands of the palate, although other sites may be involved (Figure 35-16). The proposed etiology, although not clear, is thought to be trauma-induced vascular infarction of the glandular lobules. Disruption of blood flow may be due to either mechanical or thermal trauma (e.g., injection of local anesthetic, smoking) or indirect causes (e.g., vascular conditions, diabetes mellitus). The lesions resolve spontaneously and the only concern is resemblance of the condition clinically to malignancy. Histologically, squamous metaplasia (pseudoepitheliomatous hyperplasia) and replacement of the ductal structures may be confused, especially by general pathologists with squamous cell or mucoepidermoid carcinoma. Good history, evidence of recent trauma, and examination of the specimen by oral pathologists would prevent misdiagnosis and unnecessary treatment.[9,52]

TABLE 35-3. **Sjögren's Syndrome Diagnostic Criteria (Symptoms and Tests)**

Symptoms (1 of 3)	OCULAR	ORAL
	Dry eyes (>3 mo) Foreign body sensation Artificial tears (3 times daily)	Dry mouth (>3 mo) Salivary gland swelling Liquids required to facilitate swallowing
Tests (1 of 2)	OCULAR	ORAL
	Schirmer's test (<5 mm in 5 min) Rose-Bengal stain Dye staining	Salivary flow <0.1 mL/min (unstimulated) Abnormal parotid sialogram Abnormal scintigraphy
	Labial salivary gland biopsy positive Positive anti SS-A and/or anti SS-B	

Benign lymphoepithelial lesion (Mikulicz's disease) was initially described in 1952 as unilateral or bilateral parotid swelling. The swelling is a result of benign infiltration of lymphocytes and histologically contains diagnostic epimyoepithelial islands. The condition is considered the manifestation of an immunologic abnormality. It may present as solitary salivary gland abnormality or as a manifestation of SS or HIV disease and usually remains benign. Malignant change may occur rarely in the lymphocyte component (lymphomas) or the epimyoepithelial component (undifferentiated carcinomas) and is associated with an increase in growth of the lesion. The majority of patients are women over 50 years of age. The cosmetic deformity caused by the parotid enlargement may require surgical excision or superficial parotidectomy.

Finally, *sarcoidosis*; a granulomatous disease of obscure etiology, often involves the salivary glands among other organs and is a common cause of bilateral parotid enlargement. Classic histologic findings of noncaseating granulomas in biopsies of involved organs and laboratory assays are used to confirm the diagnosis. Systemic treatment with steroids and immunosuppressive drugs are employed for non-spontaneously resolving cases, with good response.[9,52,79–83] Sarcoidosis may present with facial nerve palsy, uveoparotitis (Heerfordt's syndrome), and Melkerson Rosenthal syndrome with facial palsy.

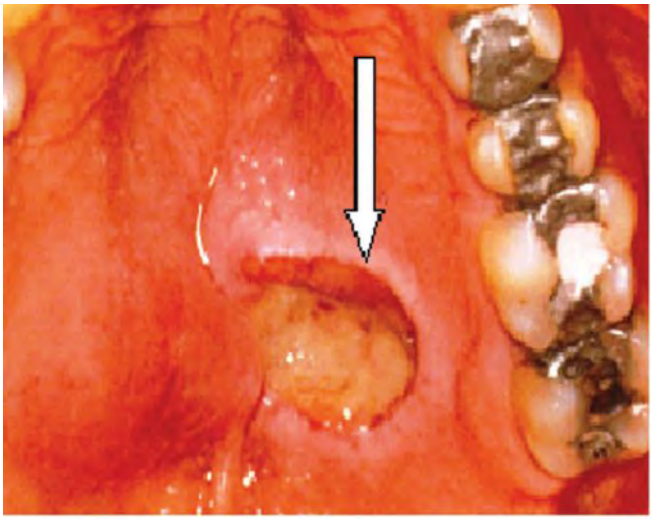


FIGURE 35-16. Necrotizing sialometaplasia of the left palate.

NEOPLASTIC SALIVARY GLAND DISORDERS

General Considerations

Salivary gland tumors account for 2% to 6.5% of all head and neck neoplasms, are more common in women with a peak incidence in their 60s and 70s, but can occur in all age groups. The majority of neoplasms occur in the parotid, and pleomorphic adenoma is the most common benign tumor and mucoepidermoid carcinoma the most common malignant tumor. The proportion of malignant tumors is greater in the minor glands (with the exception of the sublingual gland), whereas benign tumors occur more often in the major glands. Although not entirely clear, risk factors for development of salivary gland neoplasms include radiation exposure, familial or genetic predisposition, tobacco (use for specific tumors), viral etiology, and chemical exposure. FNAB, as discussed earlier, is the most commonly employed diagnostic modality for assessment of the pathology of salivary masses. Caution with malignant tumors should be exercised because the positive predictive value of FNAB is low.[83] Under light microscopy, specific features are distinctive enough for adequate diagnosis whereas immunohistochemical analysis may be employed in more complex cases.

Clinical staging, histologic type and grade, location, cranial nerve involvement (facial nerve), and patients' demographics are among the prognostic factors implicated in salivary gland malignancies. Most recent studies though have clearly demonstrated that clinical stage, particularly size, is the most critical factor of outcome rather that the histologic grade with the exception of few tumors[84,85]. The TNM staging of the American Joint Committee on Cancer (AJCC) for tumor size (T), nodal status (N), and metastasis (M) applies to malignant tumors of major salivary glands only. (The latest World Health Organization [WHO] TNM classification in 2005 appears in Table 35-4.) The major changes from the previous version is the subdivision of T4 into T4a (resectable tumors invading vital structures) and T4b (nonresectable tumors invading vital structures) and upgrade to T3 in cases of clinical or macroscopic evidence of extraparenchymal extension regardless of size. Some general behavioral differences between benign and malignant tumors are shown in Table 35-5.[85–91]

TABLE 35-4. **World Health Organization TNM Classification for Salivary Gland Neoplasms**

Stage	Tumor Size	Nodal Status	Distant Metastasis
I	0–2 cm **(T1)**	No nodal involvement **(N0)**	Not present **(M0)**
II	2–4 cm **(T2)**	No nodal involvement **(N0)**	Not present **(M0)**
III	<4cm and/or extraparenchymal tumor extension **(T3)**	No nodal involvement **(N0)**	Not present **(M0)**
	T1 or T2 or T3	Single node ipsilateral < 3 cm **(N1)**	Not present **(M0)**
IVA	T1 or T2 or T3	Single node ipsilateral > 3 cm but < 6 cm **(N2a)** or Multiple ipsilateral nodes none > 6 cm **(N2b)** or Bilateral or contralateral nodes none > 6 cm **(N2c)**	Not present **(M0)**
	Resectable tumor invading : skin, mandible, ear canal or CN VII **(T4a)**	N0 or N1 or N2 (any)	Not present **(M0)**
IVB	Unresectable tumor invading: skull base or pterygoid plates or encasing the carotid **(T4b)**	N0 or N1 or N2 (any)	Not present **(M0)**
	T1 or T2 or T3 or T4 (a or b)	Metastasis to node > 6 cm **(N3)**	Not present **(M0)**
IVC	T1 or T2 or T3 or T4 (a or b)	N1 or N2 (any) or N3	Present **(M1)**

TNM = tumor-node-metastasis.

TABLE 35-5. **Differences in Findings and Behavior between Benign and Malignant Salivary Neoplasms**

	Benign	Malignant
Findings		
Superficial ulceration	Not present	Usually present
Fixation to normal tissues (except palate)	Not present	Usually Present
CN VII involvement (nerve palsy)	Not present	Usually present
Encapsulated	Usually yes	Usually no
Pain	No	Early stages no, later yes
Behavior		
Growth	Slow	Vary, but usually rapid
Metastatic potential	No	Yes

Benign Salivary Gland Tumors

Pleomorphic adenoma (PA) is the most common benign tumor originating either from dual proliferation of cells with ductal or myopepithelial features or proliferation of a single cell with the potential to differentiate into either cell type. The myoepithelial features are responsible for determining the composition and appearance of these tumors. There is a slight male predilection and, although PAs can occur in any age, they are more common between the 40s and 50s. Eighty-five percent occur in the parotid, whereas 50% of the tumors found in the minor salivary glands are PAs, with the palate as the most common intraoral site followed by the upper lip and buccal mucosa. Classically, PAs are slow-growing, mobile (except for those found in the hard palate), painless masses that can reach gigantic sizes if left untreated (especially those involving the parotid) (Figure 35-17).

Histologically, the tumors demonstrate pleomorphic patterns of various ratios of epithelial and mesenchymal elements and are enclosed in a connective tissue pseudocapsule. The lack of true capsule, the deficiencies of the pseudocapsule, and the tumor extensions through these defects are thought to contribute to recurrences. Surgical excision is the treatment of choice for PAs. Parotidectomy (usually superficial) with facial nerve preservation, excision of the submandibular or sublingual glands, or resection to the next anatomic layer for minor glands is the treatment of choice.[91] However, currently the utility of partial parotidectomy and extracapsular dissection is debated for excision of parotid PAs. Taking care to ensure adequate removal while the tumor and its pseudocapsule are not violated is important in preventing recurrences. When recurrent, they usually appear as multiple discrete tumor foci, involve areas of scars from previous surgery, and in the majority of cases, maintain the original pathology. A rate of malignant transformation up to 25% (carcinoma ex-PA) is reported in longstanding untreated tumors or with multiple recurrences, after inadequate excision, or tumors treated with radiotherapy.[44,53–90,92–99]



FIGURE 35-17. Pleomorphic adenoma of the parotid gland.

The *metastasizing pleomorphic adenoma* (MPA) variant of this neoplasm, despite its identical benign histologic features with its primary site, is associated with a 5-year disease-specific survival rates approximately 58%. Hematogenous metastasis after surgical manipulation and incomplete removal of these tumors has been suggested to be the main etiology of their behavior. Common sites outside the head and neck are bones, lungs, and abdomen, whereas within the head and neck, an equal distribution of extralymphatic and nodal involvement is noted. Surgery is the treatment of choice for these tumors, which are considered low-grade malignancies with a lethal potential.[100–104]

Unlike PAs, *monomorphic adenomas* originate from an isomorphic epithelial cell population, and thus, they lack the mesenchymal tumor elements. Among the most common are basal cell adenoma, canalicular adenoma, myoepithelioma, oncocytic tumors, and ductal papillomas.

The vast majority, 70%, of the *basal cell adenomas* is found in the parotid and account for 1% to 2% of all salivary tumors. In the minor salivary glands,, the upper lip is the most common location, followed by the hard palate, the buccal mucosa and the lower lip. There is distinct male predilection and a mean age of 60 years. The mass is usually painless, is slow growing, and rarely recurs if adequately excised. Histologic variants include the solid, trabecular, and tubular, with the exception of the membranous variant, have no impact on prognosis. The latter histologic pattern is exclusively found in the parotid and, owing to the multifocal pattern, has demonstrated a significant recurrence rate. Local

excision, with inclusion of normal tissue in multifocal tumors, is the treatment of choice.[44,87,89,92,105,106]

In contrast to basal cell adenomas, *canalicular adenomas* occur almost exclusively in the minor salivary glands, and the most common site is the upper lip (81% of the tumors in one series). The tumor is found most commonly in female patients older than 50 years as a freely movable, painless mass. The histologic type does not alter their biologic behavior, but the lack of capsule and the fact that over 20% of these are multifocal may account for reported recurrences. Wide local excision ensuring total removal with a cuff of normal tissue is adequate treatment.[44,87,92,107–109]

Most *myoepitheliomas* are found in the parotid as well-circumscribed painless masses, presenting equally often in both genders as early as the 30s. The tumor is rare and the diagnosis occasionally requires distinction from those PAs with numerous myoepithelial cells. Three histologic forms are recognized, the spindle-cell (70%), which favors the parotid; the plasmacytoid (17%), most commonly found in the palate; and a mixture of the two (13%). Other than the potential for a diagnostic challenge, these variances have no impact on the clinical course or choice of treatment, which remains surgical excision. The tumor has no recurrence potential. Rare cases of malignant transformation have been reported in the literature.[44,87,92,110–117]

Two neoplasms make up the subcategory of oncocytic tumors: *Warthin's tumor* (or *papillary cystadenoma lymphamatosum)* and *oncocytoma*, due to presence of oncocytic-appearing epithelial cells or proliferation of oncocytic cells. *Warthin's tumor* accounts for 5% to 7% of epithelial salivary gland neoplasms, with the vast majority found in the parotid, with bilateral involvement in 4% to 6% of the cases. There is a 3:1 male-to-female predilection; patients affected are between 30 and 70 years; and this tumor is associated with cigarette smoking. The clinical presentation is that of a doughy painless mass often found in the tail of the parotid (Figure 35-18). The tumor is believed to originate from inclusion of salivary gland cells into developing lymph nodes that eventually become reactive and induce a proliferation of both tissues. The term *papillary cystadenoma lymphomatosum* is descriptive of the histologic appearance of the tumor with papillary, finger-like projections into multicystic spaces along with a lymphoid stroma. FNAB is adequate for diagnosis to rule out pleomorphic adenoma or malignancy, and the presence of a capsule allows for removal with limited potential for recurrence.[44,53–87,92,118,119]

Similarly, *oncocytomas* are small tumors found in the parotid as firm, painless, freely movable masses less than 5 cm in size. Although well encapsulated, these neoplasms may demonstrate multinodularity. Superficial parotidectomy or excision with margin of normal tissue is recommended for treatment of oncocytomas that have no recurrence potential. Caution is required in cases of oncocytosis that may occur with age and is a metaplasia of the ductal and acinal cells, seen in otherwise healthy glands and requiring no treatment. A rare malignant variant of this entity is the malignant oncocytoma that demonstrates atypical nuclear features and invasive pattern and requires more aggressive resection.[44,53,87,92,120–125]

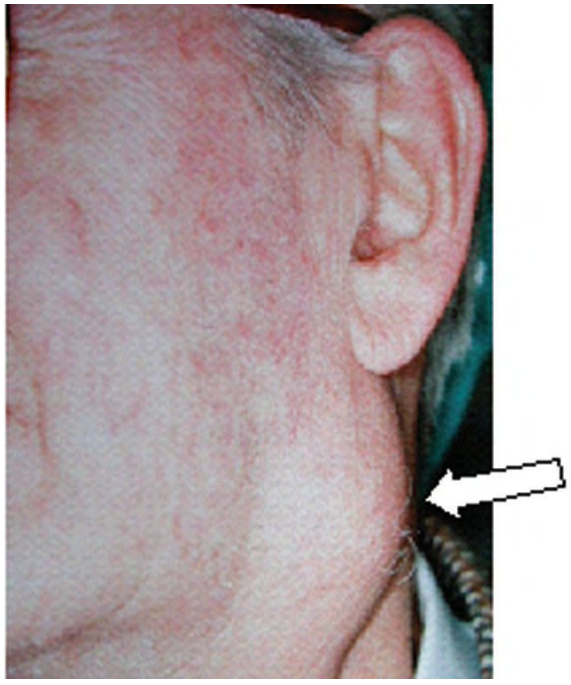


FIGURE 35-18. Warthin's tumor involving the tail of the parotid gland.

The *ductal papilloma* group includes the sialadenoma papilliferum, inverted ductal papilloma, and intraductal papilloma—all rare tumors thought to arise within the interlobular and excretory portions of the duct. All three tumors more common in middle-aged men and are usually found intraorally associated with minor salivary glands (Figure 35-19). Conservative surgery is adequate treatment for these low-recurrence potential neoplasms.[7,29,44,86,87,89,90,92,126–134]



FIGURE 35-19. Inverted ductal papilloma of the minor salivary glands.

TABLE 35-6. **Biologic Classification of Salivary Gland Malignant Tumors**

Low-Grade Tumors
Low grade MECA
PLGA
Acinic cell CA
Clear cell CA
Basal cell adenocarcinoma (rare)
Intermediate-Grade Tumors
Intermediate-grade MECA
Epimyoepithelial CA (rare)
Sebaceous CA (rare)
Adenocarcinoma NOS
High-Grade Tumors
High-grade MECA
Adenoid cystic CA
Carcinoma ex-pleomorphic carcinoma or malignant mixed tumor
Salivary ductal CA (rare)
Squamous cell CA (rare)
Oncocytic adenocarcinoma (rare)
Adenocarcinoma NOS

CA = carcinoma; MECA = mucoepidermoid carcinoma; PLGA = polymorphous low-grade adenocarcinoma; NOS: not otherwise specified.
Modified from Regezi JA, Sciubba JJ, Jordan RCK, editors. Salivary Gland Diseases. In Oral Pathology Clinicopathologic Correlations , 4th ed. Vol. 1. Missouri: Elsevier, 2003:183–217.



FIGURE 35-20. Mucoepidermoid carcinoma of the right retromolar fossa.

Malignant Salivary Gland Tumors

The biologic behavior of malignant salivary gland neoplasms can be used to classify these tumors as low-, intermediate-, or high-grade malignancies (Table 35-6). In general, the most commonly encountered tumor is the *mucoepidermoid carcinoma* (MECA) that accounts for up to 30% and 35% of the parotid and minor salivary glands malignancies (palate), respectively, and 20% of those involving the submandibular gland. MECA is the most common salivary gland malignancy in childhood, although it can present at any age, mean of 45 years, with equal gender involvement. An intraosseous variant is often encountered in the mandible at the retromolar fossa and is thought to originate from embryonically entrapped salivary gland elements (Figure 35-20). On the basis of histologic appearance and degree of differentiation, this tumor is classified as low-grade or well differentiated, composed largely of mucous-secreting cells, often forming glandular spaces, and high-grade or poor differentiated, characterized by squamous cells with rare mucous-secreting cells. An intermediate grade is described as well. The histologic findings used by pathologists to assign a grade for these tumors follow the grading criteria set by Auclair and colleagues,[87,135] Goode and associates,[136] Accetta and coworkers,[137] and Brandwein and colleagues.[138] Studies have demonstrated that tumor grade of differentiation, pattern of invasion, clinical staging, patient's age, and tumor size are all significant prognostic factors.[139,140] High-grade tumors are associated with lower 5-, 10-, and 15-year survival rates (0–22%) than low-grade tumors (90–100%).[135,141–144] Treatment for MECA varies from wide local excision for low-grade tumors to more radical resection accompanied by neck dissection and potentially radiotherapy for high-grade tumors, especially those with nodal involvement. Simple curettage of intrabony tumors increases dramatically the recurrence potential.[44,53,85,90–92]

In 1983, the *polymorphous low-grade adenocarcinoma* (PLGA) was reported under the terms "lobular carcinoma" and "terminal duct carcinoma of the salivary glands." The specific histopathologc features, clinical presentation, and behavior segregated PLGA from other tumors. Overall, PLGA is a low-grade malignancy with indolent course and low metastatic potential found almost exclusively in the minor glands, most often those in the palate. A 1- to 4-cm nonulcerated, nonpainful, firm, slow-growing, elevated submucosal swelling is the common presentation. The tumor occurs in older male or female patients in their 50s to 80s. Cervical nodal involvement is reported in 10% of the patients at the time of initial presentation. Histopathologically, the tumors lack capsule and demonstrate infiltration into surrounding tissues. Difficulties in differentiating PLGA from adenoid cystic carcinoma may be encountered on routine hematoxylin and eosin stains, especially due the presence of perineural invasion in both tumors. The perineural invasion in PLGA is of no significance, and wide local excision is adequate treatment with no need for radiotherapy.[44,90,92,145–152]

Adenoid cystic carcinoma (ACC) is a high-grade malignancy representing 25% of all salivary carcinomas with 70%

of the cases occurring in the minor glands, and the parotid is most common site among the major glands. Classically, these tumors present with tenderness or pain and neurologic findings associated with the cranial nerve invaded by the tumor. Mucosal ulceration assists in differentiation of ACC from PA in the palate (Figure 35-21). Bone invasion, presence of tumor proximally well beyond the intraoral mass, or facial nerve involvement for parotid tumors, due to perineural invasion and distant metastasis present at time of initial diagnosis, are characteristics of ACC behavior. Three histologic patterns can be identified: cribriform ("Swiss cheese"), tubular, and solid; they may coexist in the same tumor. Unlike other high-grade malignancies, ACC has good 5-year overall survival (70–90%) that drops dramatically at 10 years (30–70%) and even more at 15 years (<50%). Radical surgery with removal of underlying bone, when the palate is involved, followed by wide-field radiotherapy or superficial parotidectomy with wide-field radiotherapy is the recommended approach. Presence of metastasis (usually to the lungs and less commonly to the cervical lymph nodes) has less impact on survival than size of the tumor. Some poor prognostic factors for ACC are tumor size greater than 4 cm, tumor present at the resection margins, and presence of 30% or more of the solid variant.[44,53,90,92,153–163]

Acinic cell adenocarcinomas most commonly involve the parotid (>80%), accounting for 18% of malignant salivary tumors and 6.5% of all salivary neoplasms. In general, these are indolent neoplasms with low metastatic potential and recurrence rates, especially when they involve the minor glands. Wide local excision is usually adequate; radiation therapy is reserved for recurrences or when complete excision cannot be accomplished.

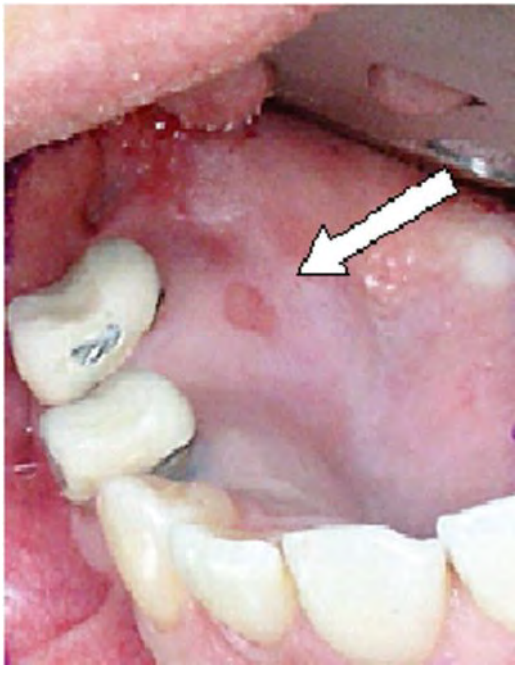


FIGURE 35-21. Adenoid cystic carcinoma of the right hard palate.

Carcinoma ex pleomorphic adenoma and *adenocarcinoma not otherwise specified* (NOS) are some additional malignant salivary tumors that may be encountered. In general, the treatment of salivary gland malignancies remains primarily surgical, although for advanced stage disease, recurrences, or not completely excised tumors radiotherapy has proved to be beneficial. Fast neuron radiotherapy has been shown to improve outcome over mixed beam radiation for stage T3 and T4 tumors, involvement of resection margins, or cases of recurrence. At present, there is no role for chemotherapy for salivary gland malignancies.[44,53,87,89–92,163–166]

SELECTIVE COMPLICATIONS OF SALIVARY GLAND SURGERY

Outcome of salivary gland surgery appears to focus on neurologic injuries, whereas gustatory sweating (Frey's syndrome) is usually discussed in parotid surgery literature. Surgical complications from approaches to the parotid (and less often the submandibular) gland specifically include injury to the facial nerve, in addition to general considerations such as scar formation and cosmetic defects. Identification of the main trunk of the facial nerve conventionally starts at its exit, before it enters the gland, or by identification of the buccal branch 4 cm anterior to the tragus in retrograde approaches. No difference in complication rates was identified between the two approaches, although the retrograde approach is considered more challenging and prolongs surgical time. Frey's syndrome, although commonly associated with parotidectomy, has been reported after removal of submandibular gland or in relationship to trauma (gunshot) to the face (Lucy Frey). Various techniques have been presented to avoid this complication and include thick flaps, use of interpositional grafts, and botulinum toxin for treatment. Injury to the lingual and hypoglossal nerves is usually due to poor surgical technique and should not be encountered if careful dissection and identification are followed.[84,167–171]

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36

CHAPTER

Mucosal and Related Dermatologic Diseases

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A myriad of conditions can cause inflammation of the oral mucosa (stomatitis). In general, these can be divided into those caused by pathogenic microorganisms (infectious stomatitis) and those not infectious in etiology (noninfectious stomatitis). In addition, this chapter addresses pigmented lesions, a common clinical manifestation of a variety of conditions.

INFECTIOUS STOMATITIS

Infections of the oral mucosa are commonly encountered in clinical practice. In general, they can be divided into bacterial, viral, and fungal.

Bacterial Infections

Tuberculosis

Tuberculosis (TB) is caused by an acid-fast bacillus, *Mycobacterium tuberculosis*. Approximately a third of the world's population is infected and the World Health Organization estimates just over 10 million new cases each year. With improved living conditions and effective antibiotics available, TB in the United States has dropped dramatically. The incidence has since increased due to immigration and human immunodeficiency virus (HIV) infection and approximately 13,000 new cases were provisionally reported in 2008.[1]

Oral involvement from TB is rare and most often occurs secondary to pulmonary disease. Most frequently, oral lesions present as enlarging ulcers or periodontal involvement. Historically, the body reacts to the organism with granulomatous inflammation and the granulomas are typically necrotizing. The organism can be identified with an acid-fast stain but false-negative stains are common and identification can be facilitated by polymerase chain reaction (PCR) amplification of the DNA of the organism. Effective multidrug protocols are available today for treatment.

Syphilis

Syphilis is a sexually transmitted disease (STD) caused by a bacteria, *Treponema palladium*. There are approximately 12 million worldwide cases of syphilis, but it is listed as a "rare disease" by the national Institutes of Health (NIH) with fewer than 200,000 cases diagnosed annually in the United States.[2]

Undiagnosed syphilis characteristically evolves through three clinical stages. Patients initially develop primary syphilis through intimate physical contact with an infected person. The primary lesion is an ulcer or chancre that occurs at the site of inoculation. Most chancres occur genitally, but the oral cavity is the most common extragenital site affected. Chancres tend to be asymptomatic, and most lesions heal without treatment within a couple months. Many patients then progress to secondary syphilis, which is disseminated and characterized by constitutional symptoms and a generalized maculopapular rash of the skin, commonly affecting the palms and soles. Symptomatic whitish plaques develop intraorally and are known as *mucous patches*. These sometimes affect the commissures and are known as *split papules*. Occasionally, verrucous or papillary lesions develop in secondary syphilis and are known as *condyloma lata*. Secondary syphilis also spontaneously resolves within months. A small number of patients evolve years later into tertiary syphilis. Tertiary syphilis significantly affects the systemic vasculature and central nervous system (CNS). Some patients develop large areas of necrotizing granulomatous inflammation known as *gummatous necrosis* or a *gumma*. The dorsal

tongue may undergo atrophy with keratosis known as *syphilitic glossitis.*

Today, syphilis is diagnosed by serology. FTA (fluorescent treponemal antibody absorption), and hemagglutination assays are sensitive and relatively specific. The incidence of syphilis has markedly declined because the infection is amenable to antibiotic regimens, particularly benzathine penicillin G.

Gonorrhea

Gonorrhea is an STD caused by the gram-negative diplococcus *Neisseria gonorrhoeae.* Worldwide, approximately 60 million persons are affected. In the United States, incidence has decreased dramatically, due in part to a national gonorrhea control program, but approximately 600,000 new cases occur annually.[3]

Like most STDs, infection occurs through direct contact and most signs and symptoms occur genitally. "Oral" involvement tends to affect the oropharynx, which is characterized by pharyngitis and sometimes with pustules or ulcers. The diagnosis can be made from smears or culture, and uncomplicated infection responds well to a single dose of ceftriaxone 125 mg intramuscularly.[4]

Viral Infections

The most important viral infections of the oral mucosa belong to the herpesvirus family of DNA viruses. Most viral infections induce neutralizing antibodies and the immune system destroys the virus. The herpesviruses are somewhat unique in that once they enter the body, they are not destroyed but are characterized by latency in which they remain dormant but have the ability to be reactivated, producing recurrent patterns of infection.

Herpes Simplex Virus

Herpes simplex virus (HSV) is ubiquitous, and most people are eventually exposed. It is important to note that patients do not need to be exposed to another person with active lesions to contract the infection, rather, 70% of the population asymptomatically shed HSV into their saliva periodically over time. At any given point in time, approximately 6% of healthy patients have HSV in their saliva by routine detection methods, and this number increases to 30% with PCR HSV DNA amplification. There are two serotypes of herpes simplex, HSV-1 and HSV-2. Type-1 causes predominantly oral disease, and type-2 predominantly genital disease. With the increasing frequency of orogenital sex, crossover is common, but the clinical manifestations of infection are not significantly altered by the type.

A small percentage, generally fewer than 10%, of initially exposed patients become clinically ill. Because these patients have no prior immunity, the primary infection is severe and the primary target is the oral cavity. This is known as *primary herpetic gingivostomatitis.* The primary infection is acute and explosive in its onset and characterized by a generalized and somewhat symmetrical stomatitis. Technically, the infection is vesiculobullous in nature but intact vesicles are rarely seen because the roof of the vesicles is very thin and invariably ruptures once formed. The resulting reaction is characterized by multiple individual superficial ulcers that are well marginated and tend to coalesce (Figure 36-1). Lesions can affect any mucosal surface and are very painful, and patients are invariably febrile with symptomatic adenopathy. The infection resolves spontaneously in 7 to 10 days, but the pain, particularly in children, makes eating and drinking a problem; dehydration can be a significant medical complicating factor.

Once exposed to HSV, whether the patient develops the primary infection or not, the virus is neurotrophic and travels up the trigeminal nerve to the ganglion where it remains latent. The virus has the ability to be reactivated and travel down the nerve to produce recurrent bouts of infection. Because the patient has been previously exposed to the virus and has some immunity, the recurrent or secondary infection tends to be milder and much more localized. In most instances, it affects the lips, most commonly at the junction of the vermilion and skin. Most patients have a prodrome

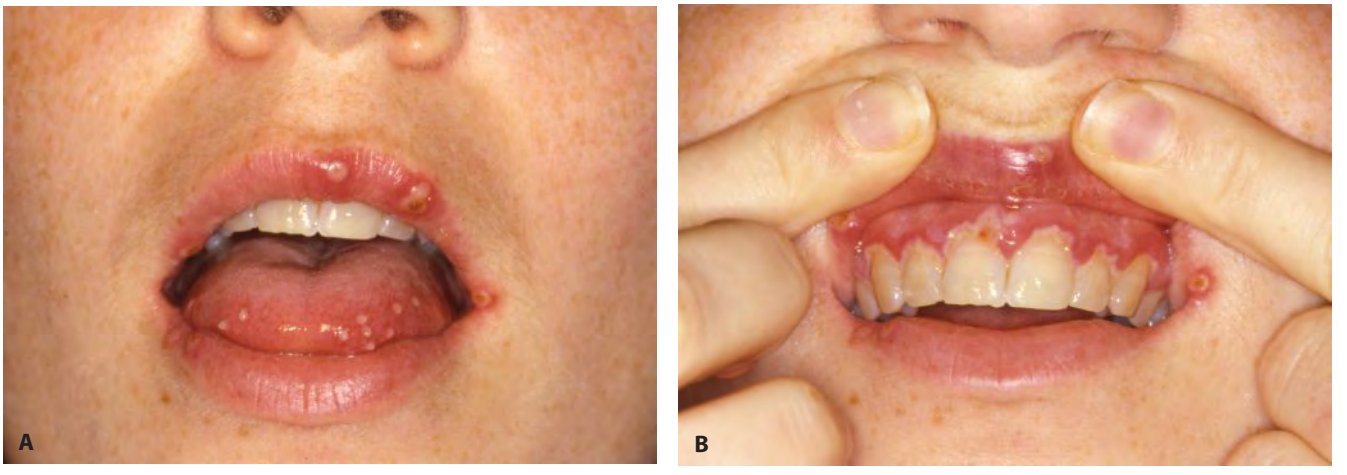


FIGURE 36-1. **A** and **B,** Multiple sharply marginated ulcers of primary herpetic gingivostomatitis.



FIGURE 36-2. Vesicular eruption of herpes labialis.

burning or tingling sensation when viral replication begins. This results in small vesicles (Figure 36-2) that quickly break, ultimately dry and crust, and heal without scarring in 7 to 10 days. This is known as *recurrent herpes labialis*, *cold sores*, or *fever blisters*.

Much less commonly, a form of recurrent intraoral herpetic infection occurs. In healthy patients, involvement is site-specific and tends to affect only masticatory mucosa or mucoperiosteum; hard palate, or attached gingiva. There may also be a prodrome with an outbreak of a cluster of multiple small well-marginated erosions or ulcers (Figure 36-3). Intraoral outbreaks tend to be more painful but resolve spontaneously. Recurrent intraoral herpetic infection is often confused with aphthous ulcers, but aphthous ulcers occur almost exclusively on the lining mucosa, not the mucoperiosteum, and tend to be larger and fewer in number.[5–9]

The infection is communicable, and historically, before the routine use of gloves and protective eyewear, occupational hazards for dentists were the development of herpetic conjunctivitis and recurrent infections of the hands (herpetic whitlow). It is important to remember that many dentists contract their infections from patients who asymptomatically excrete HSV into their saliva, not from actively infected patients.[10]

The diagnosis of herpetic infections can generally be made on the clinical features alone. If doubt exists, however, culture is reliable but inefficient and the diagnosis can be confirmed by cytologic smear. The vesicles/ulcers should be scraped with a blunt instrument like a tongue blade and spread on a glass slide. The cells must then be fixed before mailing to the laboratory. Commercially available fixatives can be purchased. Alternatively, a few drops of alcohol can be placed on the slide and allowed to air dry. Hair spray is usually a viable alternative to commercial fixatives. Virally infected keratinocytes are markedly enlarged (ballooning degeneration) and the nucleus will contain numerous eosinophilic viral inclusions, giving the nucleus the appearance of multinucleation (Figure 36-4).

Herpetic infections are self-limiting and often require no treatment, but effective antiviral therapy is available. Sun screens can reduce the frequency of herpes labialis. The literature is mixed on the efficacy for topical antiviral therapy, but for therapy to be effective, it must start at prodrome when viral replication is beginning. Topical acyclovir, penciclovir, and docosanol over the counter are available. When treating topically, it is imperative for patients to be instructed to apply the medication with a covered finger in order to prevent herpetic whitlow. There is some evidence that topical penciclovir is superior to acyclovir. Efficacy is also proved for systemic antiviral therapy. Acyclovir, valacyclovir, and famciclovir have shown therapeutic benefit. Valacyclovir is metabolized into a more bioavailable form of acyclovir and is effective at 500 mg twice daily for 5 to 7 days.[11–13]



FIGURE 36-3. Recurrent intraoral herpetic infection.

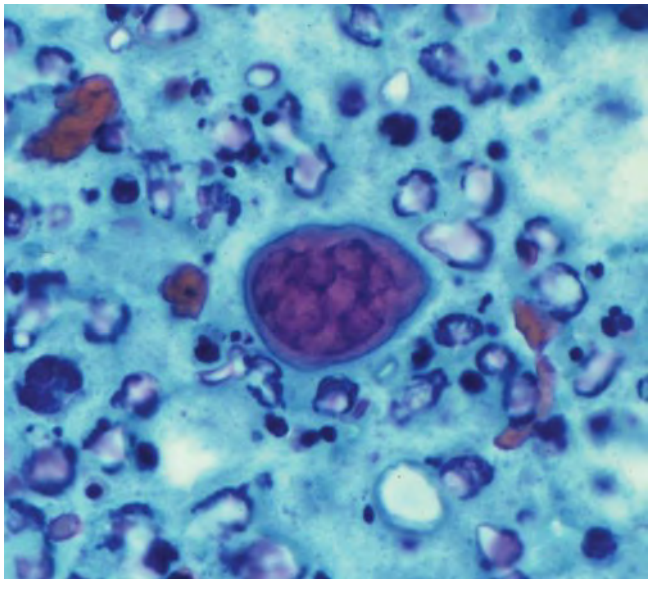


FIGURE 36-4. Cytologic smear of herpes simplex virus (HSV) infection demonstrates cytopathic effect of ballooning degeneration and multinucleation of an infected keratinocyte.

Varicella-Zoster Virus

Initial infection with the varicella-zoster virus (VZV) causes varicella or chickenpox, which produces a generalized vesiculopapular eruption on the skin and occasionally oral mucosa. Chickenpox is self-limiting, but once a person is infected, the virus is neurotrophic and travels up sensory nerves to remain latent in sensory ganglia. Reactivation causes herpes zoster or shingles, which is characterized by a vesicular eruption of the affected skin along the distribution of the nerve, characteristically stopping at the midline (Figure 36-5). The eruption is painful and most often affects the trunk, but trigeminal distribution is the second most frequent. Herpes zoster frequently affects the elderly or those immunocompromised. Herpes zoster of the external auditory canal with auditory symptoms and facial paralysis is known as *Ramsay Hunt syndrome.* VZV infections, particularly of the maxilla, can cause pain simulating a toothache, and the intrabony disease can devitalize teeth and cause bony necrosis. After an outbreak of VZV infection, about 15% of affected patients develop postherpetic neuralgia, which causes persistent pain that usually resolves within a year but can persist longer.

Epstein-Barr Virus

Epstein-Barr virus (EBV) is the cause of most cases of mononucleosis. Infection is typically through physical contact. Infection in young children tends to be asymptomatic, but in older children, teens, and young adults, it tends to produce pharyngitis, lymphadenopathy, and fever. Prodromal palatal petechiae are present in many patients. On complete blood count (CBC), patients have a leukocytosis because of significantly increased lymphocytes, many of which are cytologically atypical. The diagnosis is usually confirmed by the Paul-Bunnell test, which detects heterophil antibodies. More sophisticated immunofluorescent or enzyme-linked immunosorbent assay (ELISA) tests are also available. Mononucleosis tends to be self-limiting. The virus has an affinity for B lymphocytes, and some variants of B-cell lymphomas or leukemias are EBV-related, especially Burkitt's lymphoma. EBV has a close association with nasopharyngeal carcinoma in parts of the world. EBV is the cause of hairy leukoplakia, which is seen in immunocompromised patients, especially those with acquired immunodeficiency syndrome (AIDS). It is characterized by white plaques on the lateral tongue bilaterally.



FIGURE 36-5. Herpes zoster of V5.

Cytomegalovirus

Cytomegalovirus (CMV) tends to produce the most significant disease in neonates or those immunosuppressed. In immunocompetent patients, infection tends to be asymptomatic or produce mild flulike symptoms. A variety of cells have receptors for CMV, including salivary gland ductal epithelium where viral inclusions produce the characteristic owl's eye appearance histologically.

Coxsackievirus

Coxsackieviruses are enteroviruses that tend to produce epidemic outbreaks in children. The group A viruses produce herpangina, hand, foot, and mouth disease, and lymphonodular pharyngitis. There is significant clinical overlap among these infections, and they are not always caused by distinct strains of the virus. Herpangina is febrile and tends to produce flulike symptoms with sore throat and pharyngitis characterized by erythema with vesicles and shallow ulcers. Similar outbreaks with involvement of the hands and feet are known as *hand, foot, and mouth disease.* Other outbreaks produce hyperplasia of the small lymphoid aggregates in the palatal mucosa known as *lymphonodular pharyngitis.* Treatment tends to be symptomatic.

Fungal Infections

Most fungi are of relatively low virulence and tend to produce opportunistic infection when alterations of host or environment provide a favorable setting for fungal growth. In general, fungal infections can be divided into those that remain superficial and those that are deep.

Candidosis

Candidosis is the most common of all the intraoral fungal infections. "Candidiasis" is used synonymously, but we prefer the term *candidosis.* Most disease is caused by *Candida albicans,* although other strains can be pathogenic in humans.

Candidosis is usually not spread as a communicable disease. Candidal species can be cultured from the mouths of healthy patients approximately 50% of the time. The historical assumption was that the other 50% did not harbor the organism. With more sophisticated methods of identifying the fungus such as PCR, it has now been documented that most healthy patients harbor candidal species as a commensal organism as part of their normal oral flora.[14] *Candida* are biphasic and exist in two forms. The yeast form is considered the nonpathogenic commensal and the hyphae are pathogenic.

Because most patients normally harbor candidal species and because of its low virulence, it does not commonly produce disease in healthy patients. Rather, it is one of the prototypical opportunistic infections. Predisposing factors include age, denture wear, broad-spectrum antibiotics, any condition or medication producing immunosuppression, xerostomia, diabetes, and any generally debilitating disease. Candidosis tends to produce characteristic tissue changes that have led to clinical subclassifications of the infection, but oftentimes, there is clinical overlap among the patterns. The most characteristic of all the clinical patterns is acute pseudomembranous candidosis. Because *Candida* tends to infect the surface of the mucosa, if the infection is sufficiently severe, it will produce necrosis of the superficial keratinocytes. Normally, these necrotic cells would desquamate, but they are held together by the hyphae of the organism (Figure 36-6). This produces a buildup of necrotic cells on the surface that appear white, and because they are necrotic, they can be scraped off, hence, pseudomembrane. The base of the pseudomembrane is erythematous representing the body's inflammatory reaction to the infection. Acute pseudomembranous candidosis is usually symptomatic and often generalized with the characteristic multiple slightly raised whitish curdlike plaques that scrape off (Figure 36-7).

Acute atrophic candidosis is a clinical variant that typically follows broad-spectrum antibiotic therapy and is characterized only by erythema. It is usually symptomatic and often affects gingivae and produces tongue depapillation. Two chronic forms of atrophic candidosis exist. The most common is limited to the denture-bearing tissues of maxillary denture wearers. This is known as *chronic atrophic candidosis*, *denture stomatitis*, or *denture sore mouth*, although patients can be asymptomatic. Patients invariably wear their dentures 24 hours a day and the infection is limited to the tissues under the denture. With a good palatal seal, there is minimal salivary clearance under the denture. In addition, it is dark, warm, and moist and an ideal environment for growth of the organism. This form rarely produces pseudomembranes, but only erythema as the host reacts to the growth of the organism (Figure 36-8). Older texts discuss palatal erythema as an allergic reaction to the denture. Although hypersensitivity to plastic has technically been documented, it is exceedingly rare, and currently, palatal erythema confined to the denture-bearing areas of the maxilla should be assumed to be candidosis until proved otherwise.



FIGURE 36-7. Acute pseudomembranous candidosis.

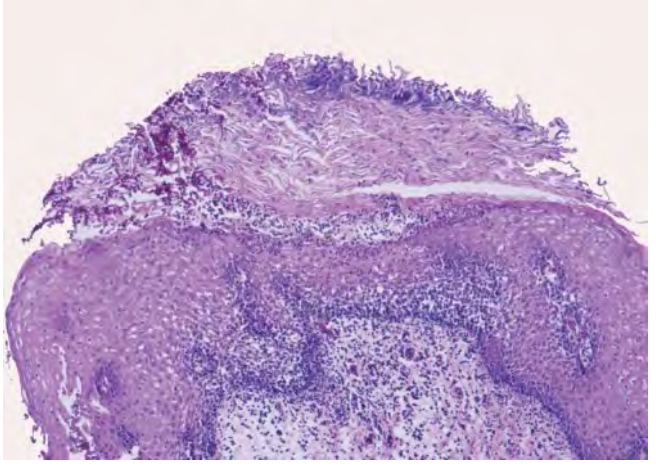


FIGURE 36-6. Pseudomembranous candidosis in which the pseudomembranes consist of necrotic surface keratinocytes.



FIGURE 36-8. Chronic atrophic candidosis.



FIGURE 36-9. Median rhomboid glossitis.

A second clinically distinct form of chronic atrophic candidosis is known as *median rhomboid glossitis* or *central papillary atrophy.* Originally speculated to be developmental, investigators were not able to find examples in large numbers of children examined.[15] It is unknown why the middorsal tongue preferentially colonizes *Candida*, but the organism can be recovered from the surface of the lesion in most patients. Lesions can be symptomatic or asymptomatic, located on the middorsal tongue, and typically atrophic with or without redness (Figure 36-9). They are sometimes firm on palpation and occasionally nodular.[14–17]

One of the rarest and most controversial forms of infection is *chronic hyperplastic candidosis*. This produces a thickened white plaquelike lesion that does not rub off (Figure 36-10). It has a predilection for tongue and commissure. Because the lesions have no clinically distinguishing features, they are usually indistinguishable from leukoplakia. Accordingly, they are also known as *candidal leukoplakia.* The diagnosis can be rendered only after biopsy because the lesions have unique histologic features characterized by epithelial hyperplasia, hyperkeratosis, and numerous yeasts and hyphae within the surface keratin. One of the controversies with this lesion is there is some evidence documenting that chronic hyperplasic candidosis should be considered a premalignant lesion because cases progressing to carcinoma are documented.[18] It is known that *Candida* can secondarily colonize any mucosa that is metabolically altered, including routine leukoplakia and lichen planus. The difficulty lies with the pathologist's ability to distinguish chronic hyperplastic candidosis from a routine leukoplakia secondarily colonized by *Candida.* The candidal organism has been shown to be capable of inducing epithelial hyperplasia experimentally. The controversy is further fueled by the fact that not all cases of chronic hyperplastic candidosis respond to antifungal medication; this is particularly true in those patients who continue to smoke.[16,19]

Angular cheilitis is characterized by cracks and fissures at one or both commissures (Figure 36-11). Many are symptomatic. In as many as 80% of cases, this is due to candidal infection, and it can be the only manifestation or it can occur with any of the intraoral forms of infection. Occasionally, it is due to bacteria, streptococcus, or staphylococcus.[17,20]

Mucocutaneous candidosis is a rare group of immunologic disorders, some with endocrinopathy, characterized by severe mucocutaneous candidosis affecting the mouth, skin, nails, and other mucosae.

The diagnosis of candidosis can generally be made from the clinical features alone, particularly if the predisposing factors can be identified. If uncertain, cytologic smear is the easiest and most efficient way to confirm the diagnosis.



FIGURE 36-10. Chronic hyperplastic candidosis.



FIGURE 36-11. Angular cheilitis.

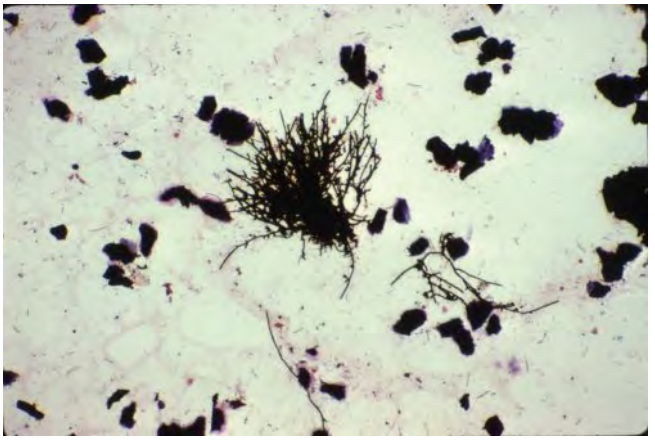


FIGURE 36-12. Cytologic smear shows pathogenic hyphae of candidosis.

Culture is usually not practical because half the population will be positive without having candidosis. Cytology has the added advantage of testing an area of clinically suspected infection and the ability to identify the organism in its pathogenic hyphal form (Figure 36-12).

A variety of effective antifungal medications are available. Identification and correction of predisposing factors will facilitate efficacy. Nystatin oral suspension or pastilles are effective and relatively inexpensive. The pastilles have an objectionable taste to many. Nystatin works topically and can be expectorated or swallowed because it is very poorly absorbed from the gastrointestinal tract and it has minimal side effects. Clotrimazole is available in a 10-mg troche that can be dissolved slowly in the mouth five times daily. Ketoconazole 100-mg tablets have been available since the 1970s, but hepatotoxicity and life-threatening side effects with terfenadine, astemizole, and cisapride have been reported.[21] Fluconazole 100-mg tabs have been shown to be more effective than ketoconazole at a dosage of 2 tablets (200 mg) initially and then 1 tablet (100 mg) daily. Rhabdomyolysis has been reported as a serious side effect when given to patients taking one of the cholesterol-lowering statins.[22] Itraconazole and amphotericin B are available as oral suspensions.[23]

Deep Fungal Infections

Deep fungal infections include histoplasmosis, blastomycosis, coccidioidomycosis, cryptococcosis, and zygomycosis. These produce ulcerative granulomatous lesions but are sufficiently rare to preclude detailed discussion. Histoplasmosis is the most common and is seen histologically as small round intracellular spores within phagocytic macrophages. Cryptococcosis is usually seen in a setting of immunosuppression, particularly AIDS. Mucormycosis, one of the zygomycoses, is a particularly invasive and destructive infection that tends to affect the sinonasal mucosa in immunosuppressed or diabetic patients.

NONINFECTIOUS STOMATITIS

Recurrent Aphthous Stomatitis (Recurrent Aphthous Ulcerations, Canker Sores)

Recurrent aphthous stomatitis (RAS) is one of the most common oral mucosal diseases and affects about 20% of the general population.[24] The word "aphtha" means ulceration. RAS is characterized by recurrent single or multiple ulcers that are covered by a yellow-white fibrinous exudate and surrounded by an erythematous halo (Figure 36-13). The onset of RAS is usually during childhood, especially in adolescence.

RAS appears to be a T-cell–mediated immunologic disorder.[25] Although the cause for RAS is still unknown, several factors have been implicated. These factors include genetic predisposition (human leukocyte antigen [HLA]-B12, B51 and Cw7), nutritional deficiencies, immunologic diseases (cyclic neutropenia, AIDS), hormonal influences, infectious agents (L form of streptococci), trauma, stress, and food allergies.

Based on clinical morphology, RAS is classified into three different types: minor, major, and herpetiform aphthous ulcers. Major/minor apthae represent a spectrum of severity. Minor aphthous ulcer is the most common form of RAS. Patients often experience prodromal symptoms of burning, itching, or stinging with the development of an erythematous macule. The macules then develop into round to oval-shaped ulcers, without vesicle formation. The ulcers are present most often on buccal and labial mucosa, followed by ventral tongue, mucobuccal fold, floor of mouth, and soft palate, and rarely affect mucoperiosteum. Major aphthae are larger, deeper, and last longer and have a strong tendency to affect the posterior part of the mouth, especially soft palate, pharyngeal wall, or tonsillar fauces. Herpetiform aphthous ulcers are the least common type and consist of numerous small ulcers that may coalesce into large irregular ulcers, resembling herpes viral infection. However, herpetiform aphthae usually occur on movable mucosa.



FIGURE 36-13. Aphthous ulcer.

Occasionally, atypical presentations of what appear to be RAS occur secondarily to systemic factors. The systemic diseases that have been reported to be associated with RAS include: Behçet's syndrome, nutritional deficiencies (folate, zinc, iron, and vitamin B deficiency), immunocompromised conditions (immunoglobulin A [IgA] deficiency, cyclic neutropenia, AIDS), inflammatory bowel diseases, celiac disease, MACIC (mouth and genital ulcers with inflamed cartilage) syndrome, PFAPA (periodic fever, aphthous stomatitis, pharyngitis, and adenitis) syndrome, Reiter's syndrome, Sweet's syndrome, and ulcus vulvae acutum.

The diagnosis of RAS is usually made by clinical history and presentation. No laboratory test provides a definitive diagnosis. The histologic features for RAS are not specific. A biopsy is not necessary unless exclusion of other similar ulcerative diseases that are in diagnostic consideration is desired.

There is no standard management for RAS and the therapeutic goal is to reduce pain and facilitate healing. Most patients with RAS can be managed by mouthwashes (sodium bicarbonate in water, chlorhexidine, or benzydamine) or topical application of corticosteroids (fluocinonide, betamethasone, or clobetasol propionate gel). For ulcers not responding to topical treatment, systemic corticosteroids and other immunosuppressants may be used. It also may be helpful to investigate possible triggering factors. For adult patients who suddenly develop persistent aphthous-appearing ulcers, it is essential to consider the possible association with underlying systemic disorders.



FIGURE 36-14. **A,** Geographic tongue. **B,** Erythema migrans.

Erythema Migrans (Geographic Tongue, Benign Migratory Glossitis, Wandering Rash of the Tongue, Erythema Areata Migrans, Stomatitis Areata Migrans)

Erythema migrans is a common inflammatory condition that primarily affects the tongue. The etiology is still unknown. It occurs in 1% to 3% of the population and appears to affect females more often than males by a 2:1 ratio.[26]

Clinically, erythema migrans is characterized by multiple well-demarcated red areas surrounded at least partially by a slightly raised, yellow-white, scalloped border (Figure 36-14). The lesions most often occur on the dorsal and lateral borders of the tongue. They typically appear quickly in one area, heal in a few days, and then develop in a different area. Most patients are asymptomatic, although some may complain of a burning sensation when eating hot or spicy food. Severe or constant soreness is rarely associated with erythema migrans. The diagnosis of erythema migrans is based on the typical clinical presentation and history. Biopsy is not necessary.

For asymptomatic lesions, no treatment is needed and patients should be assured that the condition is innocuous. For patients who experience burning sensation or soreness, topical corticosteroids may be used

Lichen Planus and Lichenoid Reactions

Lichen planus (LP) is a chronic, T-cell–mediated immunologic disorder that mostly affects the skin and mucosa. It affects mostly middle-aged adults, with a female-to-male ratio of 3:2. The skin lesions of LP are typically described as purple, pruritic, and polygonal papules that often occur on the flexor surfaces of the extremities. The surface of the papules characteristically shows a fine, lacelike network of white lines called *Wickham's striae.* Besides the skin, sites of involvement include the glans penis, the vulvar mucosa, and the nails, as well as the oral cavity.

LP is one of the most common oral mucosal diseases, and it affects as many as 2% of the population.[21] It is characterized by multifocality that presents in five different forms: reticular, erosive (ulcerative), plaquelike, bullous, and papular, but individual patients can have multiple forms, either synchronously or over time. The reticular form is the most characteristic and appears as a network of lacy white striations, occurs most often bilaterally in the buccal mucosa, and is usually asymptomatic (Figure 36-15A). The erosive form is usually symptomatic and presents as generalized erythema or ulceration and commonly affects gingiva, a condition described as desquamative gingivitis (see "Autoimmune Blistering Diseases," later; see Figure 36-15B and C). The



FIGURE 36-15. Lichen planus: reticular **(A),** erosive **(B),** erosive with ulceration **(C),** plaquelike **(D).**

plaquelike form presents as white, nonwipeable plaques that may be associated with redness (see Figure 36-15D) and commonly affects the dorsal tongue. The bullous form appears as fluid-containing vesicles but is exceedingly rare. The papular form presents as numerous small white papules that may coalesce to form the reticular form. The symptoms of LP typically wax and wane over time, and it is not uncommon for patients to present with more than one of these clinical forms.

Histologically, LP is characterized by a bandlike lymphocytic infiltrate in the superficial lamina propria, hydropic degeneration in the basal cells of the epithelium, and thickening and dissolution of the basement membrane. Apoptotic basal keratinocytes presenting as colloid bodies (hyaline or Civatte bodies) may be seen. If the patient presents with reticular areas of LP bilaterally in the buccal mucosa, the diagnosis of LP can often be made based on this clinical presentation alone. For other forms of LP, a biopsy may be necessary to rule out other erosive, ulcerative diseases or premalignant/malignant changes.

Some local and systemic conditions are known to induce lichenoid reactions that are similar to lesions seen in LP, clinically and histologically. Lichenoid reactions are known to be caused by certain medications, some dental materials, and allergic reaction to cinnamic aldehyde, a flavoring agent in food. Numerous medications have been reported to induce lichenoid lesions, producing the condition known as *lichenoid drug eruption (LDE)* (Table 36-1).[27] The most common medications that may elicit LDE are nonsteroidal anti-inflammatory drugs, angiotensin-converting enzyme inhibitors, and gold salts. Although LDE can be indistinguishable from LP, it tends to be predominantly ulcerative and may occur at sites not frequently affected by LP, such as the ventral tongue. The possibility of LDE should be considered when there is a correlation between the onset of the oral lesions and the administration of a new medication. Consultation with the patient's physician(s) is required to see whether a suitable alternative drug can be used on a trial basis to confirm the diagnosis.

Lichenoid reactions caused by dental materials probably represent a hypersensitivity to these materials. Amalgam is

TABLE 36-1. **Drugs Associated with Lichenoid Drug Eruption**

Drug Group	Examples
Antihypertensive	Angiotensin-converting enzyme inhibitors (e.g., captopril, enalapril) Beta blockers (e.g., propanolol, atenolol) Calcium channel blockers (e.g., nifedipine, cinnarizine, flunarizine) Methyldopa Thiazide diuretics Loop diuretics (e.g., furosemide)
Oral hypoglycemic (sulfonylureas)	Tolbutamide Tolazamide Chlorpropamide
Nonsteroidal anti-inflammatory drugs	Ibuprofen Naproxen Phenylbutazone Sulfasalazine and mesalazine
Second-line antiarthritics	Gold Penicillamine
Xanthine oxidase inhibitors	Allopurinol
Psychological drugs	Lorazepam Tricyclic antidepressant Lithium Carbamazepine Phenothiazines (e.g., metopromazine, levomepromazine, chlorpromazine)
Antiparasitic drugs	Antimalaials (e.g., chloroquine, quinacrine, pyrimethamine, levamisole, quinine, quinidine)
Antimicrobial drugs	Tetracycline Ketoconazole Dapsone Streptomycin Mepacrine Ethambutol, *p*-Aminosalicyclic acid Isoniazide
Chemotherapeutic agents	Hydroxyurea 5-Fluorouracil
Heavy metals	Mercurials Arsenicals Bismuth
Miscellaneous	Iodides and radiocontrast media Pyritinol Tiopronin Cyanamide Amiphenazole

the most common dental restorative material, although gold and palladium chloride have also been reported as causes. If the lichenoid lesions occur only on the mucosa adjacent to dental restorations, hypersensitivity should be considered as a possibility, because removal of the offending dental materials results in remission of the oral lesions in 50% to 90% of such cases.

Allergic reaction to cinnamon flavoring agents is also known to cause lichenoid reactions. Therefore, questions about the patient's consumption of cinnamon-containing products, such as chewing gum, soft drinks, mints, or toothpaste, should be asked of patients with oral lichenoid lesions. Patients with chronic ulcerative stomatitis, lupus erythromatosus, and graft-versus-host disease (GVHD) may present with oral lesions similar to LP.

The etiology of oral LP is essentially unknown, although stress, dental materials, medications, food allergens, hepatitis C or chronic liver diseases, and autoimmunity have been suggested as causes or contributors to the onset. The association between LP and hepatitis C is still controversial. Research studies from Italy and Japan showed that a high percentage of LP patients had hepatitis C.[28] However, this association has not been observed in the studies from France, the United States and the United Kingdom.[28–29]

Because the cause is still unclear, there is currently no cure and the goal of management is for symptomatic relief. For asymptomatic reticular LP, no treatment is needed. For patients with symptomatic lesions, topical corticosteroids such as fluocinonide, betamethasone, or clobetasol gel are usually sufficient for controlling symptoms and initiating the healing process. Oral candidiasis is a common complication of topical steroid usage, and during treatment, the patient should be monitored for this possible side effect. Other therapies such as topical retinoids, tacrolimus, pimecrolimus, cyclosporine, psoralen-UVA, and photodynamic therapy (PDT) are also occasionally used for persistent erosive lesions.

The question of whether or not LP is premalignant is a controversial and unresolved. Findings from several clinical studies have suggested that patients with LP have an increased risk for oral squamous cell carcinoma, with the malignant transformation rate varying from 0.4% to 6.25% during follow-up periods that ranged from 4 months to 22 years.[30] However, research on several surrogate end points such as telomerase activity, p53, loss of heterozygosity, and cytogenetic abnormality did not show convincing or consistent evidence of malignant potential as demonstrated in epithelial dysplasia.[31] This issue is also complicated by the fact that a lichenoid chronic inflammatory cell infiltrate can sometimes be seen in epithelial dysplasia as a host reaction to the atypical epithelial cells, and it may not be distinguishable histologically from LP, especially in mild cases of epithelial dysplasia. Many authors believe that some of the reported cases of malignant transformation were not true LP but rather misdiagnosed dysplastic leukoplakias with a secondary lichenoid inflammatory infiltrate.[26]

Considering the possible increased risk for oral cancer in LP, the current recommended management for LP patients also includes periodic follow-up at least once a year even when the lesions are asymptomatic.[32]

Lupus Erythematosus

Lupus erythematosus (LE) is a fairly common autoimmune disease that affects more than 1.5 million people in the United States.[26] Based on clinical features, LE is classified into three

forms: systemic lupus erythematosus (SLE), discoid (chronic) lupus erythematosus (DLE) and subacute cutaneous lupus erythematosus (SCLE).

SLE predominantly affects females between 15 and 40 years of age. SLE affects multiple organ systems such as skin, oral cavity, heart, joint, lung, CNS, and kidney. The patients usually experience fever, malaise, weight loss combined with arthritis, and skin rash. The characteristic butterfly-shaped rash on the malar skin and nose occurs in about half of the cases. Photosensitivity is typically described for the skin lesions. Pericarditis is the most common finding when SLE affects the heart. Kidney involvement (glomerulonephritis) occurs in about 40% to 50% of SLE patients and is the most serious complication, because it may lead to kidney failure and death. Oral lesions develop in 9% to 45% of SLE patients. They appear as erythematous, hyperkeratotic, ulcerative, or lichenoid lesions. The oral lesions are often found in the buccal mucosa, palate, and vermilion border of lower lips (lupus cheilitis).

DLE is a less aggressive form of LE, and it typically involves only the skin and oral mucosa. Patients with DLE usually have no systemic symptoms or signs. The skin lesions are often on the face and scalp and usually heal with scarring and hypo- or hyperpigmentation. Approximately 3% to 20% of DLE patients develop oral lesions. Similar to SLE, the oral lesions of DLE appear lichenoid and may be indistinguishable from erosive LP clinically. However, oral lesions of both DLE and SLE are rarely seen without the presence of the skin lesions.

SCLE presents with clinical features intermediate between SLE and DLE.

Many medications can induce a lupus-like reaction known as *drug-induced lupus erythematosus (DILE)*.[33] DILE shows clinical and serologic findings similar to those of idiopathic LE. However, it resolves after discontinuation of the offending drug. Many medications have been reported to be associated with DILE, especially the antihypertensive drugs (thiazide diuretics, calcium channel blockers, angiotensin-converting enzyme [ACE] inhibitors, beta blockers). Other medications that can induce DILE include piroxicam, nepro-xene, terbinafine, griseofulvin, bupropion, tamoxifen, lansoprazole, pantoprazole, infliximab, etanercept, efalizumab, and adalimumab.

The diagnosis of LE is based on clinical, histologic, serologic, and immunologic findings. The histologic features of the oral lesions of LE show hyperkeratosis, degeneration of the basal cells, and a lymphocytic infiltrate in the upper lamina propria. These features are also seen in oral LP and can be indistinguishable from oral LP. However, LE lesions tend to have prominent subepithelial edema, a diffuse inflammatory cell infiltrate that often shows a perivascular pattern, and patchy deposition of a periodic acid–Schiff (PAS)–positive material in the basement membrane zone. Direct immunofluorescent study (DIF) of lesional tissue demonstrates shaggy or granular deposition of immunoglobulins (most often IgG and IgM) and/or C3 at the basement membrane zone. The lupus band test is the DIF of normal-appearing skin tissue, and it shows positive staining of IgG, IgM, and/or complement deposition for most LE patients. However, a positive lupus band test is not specific, because the same finding can also be seen in patients with rheumatoid arthritis, Sjögren's syndrome, and multiple sclerosis. Serologic titers for serum antinuclear antibody (ANA) is a sensitive diagnostic test for SLE. More than 90% of SLE patients are positive with the ANA test. However, the ANA test is not specific for SLE, because the positive ANA finding can also be seen in patients with other autoimmune diseases such as rheumatoid arthritis and Sjögren's syndrome. The anti–double-stranded DNA antibody test is a more specific test for SLE, and a positive result is found in about 70% of SLE patients.

Depending on severity, the management for SLE includes nonsteroidal anti-inflammatory drugs, prednisone, and/or other immunosuppressants. Antimalarial drugs, such as hydroxychloroquine, or sulfones are also sometimes used. Patients should be advised to avoid excessive exposure to sunlight. The prognosis for SLE varies depending on the organs involved. Renal failure is the most common cause of death. The treatment for DLE is usually topical corticosteroids and the prognosis is usually good.

Graft-versus-Host Disease

GVHD is a complication that develops in allogeneic bone marrow transplant patients.[26] Allogeneic bone marrow transplant has been used increasingly in recent years for treating hematologic disorders such as leukemia, multiple myeloma, aplastic anemia, thalassemia, sickle cell anemia, and disseminated metastatic disease. Although the major histocompatibility loci are matched between the donor and the recipient, differences still may exist in the minor histocompatibility loci. These differences can trigger an immune response by the transplanted donor hematopoietic stem cells, causing them to attack the host cells and tissues and resulting in host tissue damage and organ failure.

The severity of GVHD varies and appears to be influenced by several factors. Patients who have a better match in histocompatibility loci, have received umbilical cord blood, are younger. and are female tend to have milder disease. The clinical signs of GVHD also vary, depending on the organs involved and whether it has an acute or chronic onset. Traditionally, the distinction between acute and chronic GVHD is based on whether the onset of the disease is within 100 days after the bone marrow transplant. The new guideline, recommended by the NIH and based on studies conducted in 2004 and 2005, emphasizes the clinical manifestations rather than time of onset.[34] Oral involvement is seen in about half those with acute GVHD and between 72% and 83% of those with chronic GVHD.

In the oral cavity, the graft-versus-host response can damage both oral mucosa and minor salivary glands.[35] The oral manifestations of GVHD are similar to those seen in oral LP, with diffuse symptomatic erythema with white striations,



FIGURE 36-16. Graft-versus-host disease.

plaques/papules, and ulceration (Figure 36-16). The tongue, buccal mucosa, and labial mucosa are the common sites for these lichenoid lesions. Xerostomia is also a common complaint of the patients.

The diagnosis of GVHD is often based on clinical findings. When the clinical presentation is not typical, biopsy can confirm the diagnosis. Histologically, GVHD shows features similar to those of LP.

It is known that patients with allogeneic bone marrow transplants have a higher risk for developing malignant neoplasms. Most of these malignancies are hematologic neoplasms. However, the incidence of solid tumors such as oral carcinomas is also slightly increased in this group of patients.[36] Therefore, for oral lesions that do not show lichenoid features, a biopsy is necessary to exclude the possibility of malignant or premalignant changes.

The management of oral lesions of GVHD is similar to the management of the oral lesions in autoimmune diseases. The goal is to achieve symptomatic relief, with topical corticosteroids and topical anesthetic agents being the most commonly used treatments. Topical tacrolimus has been reported to be effective for treating oral ulcerations caused by GVHD. Close follow-up for possible development of oral candidiasis is required. If significant xerostomia is present, topical fluoride should be prescribed and oral hygiene should be emphasized for preventing development of caries. Pilocarpine and cevimeline also have been used to stimulate salivary flow.

The prognosis of GVHD mainly depends on whether the condition can be controlled. The survival rate has varied from 15% to 70% at 6 years after transplantation.

Chronic Ulcerative Stomatitis

Chronic ulcerative stomatitis (CUS) is an immunologic disorder that affects mainly the oral mucosa and occasionally the skin.[26] It was first described by Jaremko and coworkers in 1990,[37] and slightly more than 40 cases have been reported in the literature.[38] However, the true incidence of this disease may be much higher than reported. Because both the clinical and the histologic features of CUS are similar to those seen in oral LP and the definitive diagnosis of CUS requires a DIF, it is very possible that CUS may be misdiagnosed as LP if DIF is not performed. However, differentiation of CUS from LP is important, because CUS typically does not respond to topical or systemic corticosteroid as well as LP does, but it can often be effectively managed by hydroxychloroquine. Although it may not be financially feasible to perform a DIF on every patient showing clinical features of LP, this test should be considered for persistent erosive LP that does not respond well to topical corticosteroid therapy.

The DIF is essential for the diagnosis of CUS and it reveals autoantibodies, mostly IgG, in the nuclei of the basal and parabasal keratinocytes. These stratified epithelial–specific antinuclear antibodies (SES-ANAs) are directed against a 70-kd nuclear protein (chronic ulcerative stomatitis protein [CUSP]). CUSP shows a DNA sequence homologous to p63 that plays an essential role in epithelial development.[39] These SES-ANAs are also present in the patient's serum and contribute to a positive indirect immunofluorescent (IIF) result.

Hydroxychloroquine has been reported to be the most effective medication for CUS. However, this medication has significant potential side effects such as retinopathy, agranulocytosis, aplastic anemia, and neuromyopathy. Therefore, close follow-up to monitor for retinopathy and periodic hematologic evaluation during therapy are necessary.

Autoimmune Blistering Diseases

Autoimmune blistering diseases (ABDs) are a group of mucocutaneous diseases characterized by bulla or vesicle formation in skin and mucosa.[26,40] These diseases share a similar pathogenetic mechanism in that the patients develop antibodies (autoantibodies) targeted to their own normal proteins (autoantigens), resulting in vesicles or bullae. The ABDs include bullous pemphigoid (BP), mucous membrane pemphigoid (MMP), pemphigus, epidermolysis bullosa acquisita (EBA), and linear immunoglobulin A bullous disease (LABD). These diseases typically have a chronic onset, progress gradually, and affect multiple sites in skin and mucosa.

The ABDs as well as other inflammatory diseases commonly present as *desquamative gingivitis*, which is not a definitive diagnosis but a clinical diagnosis for generalized gingival erythema and erosion that is associated with tissue sloughing. Desquamative gingivitis is not caused by dental plaque but is caused by many different diseases including pemphigus vulgaris (PV), MMP, LP, toothpaste allergy, EBA, LABD, LE, CUS, and paraneoplastic pemphigus (PNP), with the first four diseases being the most common causes. The definitive diagnosis for desquamative gingivitis requires histologic evaluation and a DIF. However, a simple clinical test may help to initially differentiate among these diseases. Inducing a bulla or epithelial separation by applying lateral pressure to the perilesional, normal-appearing mucosa is called a *positive Nikosky sign*. This sign is more often seen in patients with MMP, PV, LABD, or EBA.

The diagnosis of these diseases requires both histologic evaluation and DIF. Therefore, a biopsy should be taken and divided into two portions. One portion is saved in formalin for routine histologic evaluation and the other is submitted in Michel's solution (transporting medium) for DIF. Because of tissue fragility, perilesional tissue may have to be submitted for DIF.

As autoimmune diseases, the treatment goal is immunosuppression for symptomatic relief and remission. Topical and/or systemic corticosteroids are often the primary modality of treatment. For severe cases, other immunosuppressant drugs are also used in combination with corticosteroids to reduce the adverse effects of the steroids.

Pemphigus Vulgaris

The term *pemphigus* is derived from the Greek word *pemphix*, which means bubble or blister. Thus, pemphigus is a group of ABDs characterized by blister formation in skin and/or mucosa. It is further classified into six subtypes: PV, pemphigus vegetans, pemphigus foliaceus, pemphigus erythematosus, IgA pemphigus, and PNP.[41] PV is the most common. The term *vulgaris* is Latin, meaning common. Among these six subtypes, PV and PNP are especially important for oral health care providers because these two types frequently affect the oral cavity and often oral lesions are the earliest clinical manifestation of the disease.

PV is a potentially life-threatening disease with an incidence of about 0.1 to 0.5 cases per 100,000 people per year. Although all ages can be affected, PV occurs most often in middle-aged individuals. Both sexes are affected equally.

The pathogenesis of PV is known to be due to the production of autoantibodies targeted to desmoglein 1 and 3, the transmembrane proteins responsible for cell-to-cell adhesion among squamous epithelial cells. Once these proteins are damaged, the cell-to-cell adhesion is lost and the epithelial cells fall apart, which results in epithelial separation and blister formation.

A subset of PV patients develops mucosal dominant PV, in which mucosal lesions develop without skin involvement. These patients are found to have developed antoantibodies primarily targeted to desmoglein 3. Conversely, patients who develop autoantibodies against both desmoglein 3 and 1 tend to have widespread skin and mucosal lesions and show more severe disease activity.

Although the pathogenesis is known, the exact cause or triggering factor for PV is still unclear. Genetic predisposition, diet, medications, and other environmental factors have been suggested to play a role. Several medications have been reported to induce pemphigus-like drug reactions. These medications include penicillamine, rifampicin, diclofenac, phenol drugs, and ACE inhibitors such as captopril.

PV can affect essentially all mucosa—including oral, pharyngeal, laryngeal, ocular, nasal, and anal and genital mucosa. However, it affects oral mucosa most often and sometimes can be the only sign of the disease. More than 50% of PV patients have oral lesions before the development of skin lesions. Clinically, it often presents as multiple erosions and ulcerations in the oral cavity. Sloughing tissue may be seen. Because the damage to the adhesion proteins is within the epithelium, the blisters are often ruptured easily and are rarely observed clinically. Orally, the palate, labial mucosa, buccal mucosa, and gingiva are most often affected. When PV affects the gingiva, it often presents as desquamative gingivitis (Figure 36-17A). The skin lesions usually appear as vesicles that soon rupture to leave a red denuded surface (see Figure 36-17B). Severe cases can be fatal where large areas of skin slough resulting in dehydration, electrolyte imbalance, and secondary infection.

The diagnosis of PV is based on histologic and DIF findings. Histologically, PV is characterized by suprabasilar epithelial separation, in which the amount of desmoglein 3 is most abundant. The spinous cells adjacent to this separation often appear to fall apart and become round in shape, a process called *acantholysis*. The histologic findings are confirmed by DIF, which shows IgG, IgM, and complement C3 deposited in the intercellular spaces among the epithelial cells. IIF is sometimes used to measure the titer of the patient's



FIGURE 36-17. **A,** Pemphigus vulgaris presenting as desquamative gingivitis. **B,** Pemphigus vulgaris with erosion.

circulating autoantibodies, which is typically correlated with disease severity and can be used for monitoring therapeutic results.

Early diagnosis and therapeutic intervention play a pivotal role in prognosis, because control of the disease is easier to achieve. The treatment is typically systemic corticosteroids (prednisone), which may or may not be combined with other immunosuppressant agents such as cyclophosphamide and azathioprine. Recently, rituximab has also been reported to be effective for patients whose disease had not responded to prednisone or patients who could not tolerate it.[42] Topical corticosteroids are often used for treating oral lesions. PV is usually managed by physicians who are experienced in treating it, because long-term corticosteroid and immunosuppressant therapy may be needed, with required monitoring for systemic side effects. Before the development of corticosteroid therapy, the mortality rate for PV was 60% to 90%. With modern therapies, the mortality rate has been dramatically improved and is reported to be 10% or less.[26]

Paraneoplastic Pemphigus

PNP is a rare subtype of pemphigus that is associated with an underlying malignant neoplasm.[26] The associated malignancy most often is a lymphoid neoplasm or lymphoproliferative disorder, although thymoma and some solid tumors have also been reported.

Although the pathogenesis is not totally known, evidence suggests that PNP involves both the humoral and the cellular immune systems. The patient's immune reaction to the underlying neoplasm cross-reacts with adhesion molecules in the epithelium and at the basement membrane zone. The patients develop autoantibodies not only to desmoglein 1 and 3 but also to desmoplakins, desmocollins, bullous pemphigoid antigen 1 (BP230), plectin, envoplakin, and periplakin. The involvement of multiple autoantigens contributes to the various clinical, histologic, and DIF findings.

PNP typically develops in patients with a history of a previous malignancy. However, about one third of the reported cases have shown clinical symptoms and signs of PNP before the underlying malignancy was diagnosed. Clinically, PNP can present as multiple blisters in skin and mucosa. In some patients, the skin lesions appear papular and pruritic, resembling skin LP lesions. Involvement of mucous membranes is a dominant feature in all reported cases. The oral cavity is frequently involved, with the lesions appearing as widespread erosions and irregular ulcers. Lesions also may be found in the oropharynx and nasopharynx. Bloody crusting is often seen on the vermillion border of lips, a feature resembling erythema multiforme (EM). About 70% of the patients have conjunctival lesions that result in scar formation, a feature resembling MMP. Involvement of vaginal and anogenital mucosa is also seen.

The diagnosis of PNP is based on clinical, histologic, DIF, IIF, and immunoprecipitation findings. Histologically, PNP shows lichenoid mucositis, acantholysis, and both intraepithelial and subepithelial separation. DIF shows intraepithelial and/or basement membrane zone deposition of IgG and C3. The standard protocol of IIF for PNP requires using rat bladder epithelium as the substrate and it shows a unique pattern of intercellular staining. Immunoprecipitation is considered to be the gold standard for diagnosing PNP and it shows the presence of autoantibodies to the autoantigens mentioned earlier.

The treatment of PNP is similar to the treatment for PV, in addition to treatment for the underlying malignancy. However, the results have been disappointing and the prognosis is poor, with a mortality rate about 75% to 80%.

The lesions of PNP may initially respond to immunosuppressant agents that often contribute to the progression of the malignant neoplasm. The patients often die of complications of the blistering or of the immunosuppressant treatment or the progression of the malignancy. Rituximab, a monoclonal antibody to CD20, is a new therapy that has shown a promising result initially in treating patients with PNP and B-cell lymphoma. However, a recent review still showed a low survival rate, with the patients often dying of sepsis.[43]

Mucous Membrane Pemphigoid (Cicatricial Pemphigoid)

MMP is an autoimmune mucocutaneous blistering disease characterized by chronic vesiculobullous eruptions, predominantly on mucous membranes but occasionally on skin.[26] *Cicatricial* is derived from the Latin word *cicatrix*, which means "scar," because the disease is characterized by scar formation after the blisters heal, but oral scarring is rare. MMP can affect any mucous membrane and may cause serious complications, such as blindness and stenosis.

MMP typically occurs in elderly people with an average age of 60 years. It affects females more often than males. The oral cavity is the most common site for MMP, accounting for 83% to 100% of the MMP cases reported. The oral cavity is also often the site of onset and the only manifestation of the disease (oral pemphigoid). MMP usually begins with vesicles or bullae that subsequently rupture, resulting in ulceration or erosion. Although MMP is characterized by scar formation after blisters or erosive lesions are healed, scarring is less common for oral lesions. MMP can affect any oral site but the gingiva is affected most often, accounting for more than 90% of the oral cases. Desquamative gingivitis is a very common oral presentation for MMP (Figure 36-18).

Certain medications can induce a drug-related form of pemphigoid.[44] Several medications, such as lisinopril, methyldopa, D-penicillamine, and indomethacin, have been reported to induce MMP with oral lesions.[45] Other medications, including practolol, clonidine, sulfadoxine, and topical agents, such as echothiophate iodide, pilocarpine, demecarium bromide and glaucoma medication, have also been reported to induce ocular, vulval, or anal MMP.

The pathogenesis of MMP is found to be autoantibodies targeted to the various proteins in the basement membrane zone. These autoantigens include BP180, BP230, $\alpha 6\beta 4$ integrin, laminin 5, laminin 6, uncein, and 97-kd LABD



FIGURE 36-18. Mucous membrane pemphigoid as desquamative gingivitis.

antigen.[26] BP180 is the predominant autoantigen found in patients with MMP. These proteins are responsible for the adhesion of the epithelium to the underlying connective tissue. Once the proteins are damaged by the autoimmune response, the adhesion weakens, causing the epithelium to separate from the underlying lamina propria, which leads to blistering.

The diagnosis of MMP is based on histologic and DIF findings. The histologic features of MMP show chronically inflamed mucosa and subbasilar epithelial separation at the basement membrane zone. The DIF is essential for confirming the diagnosis and it shows a linear deposition of immunoglobulins (primarily IgG; less commonly IgM and IgA) and C3 in the basement membrane zone.

There is no generally accepted treatment for MMP and the goal is symptomatic relief. Topical corticosteroids are often used to control localized MMP, such as oral pemphigoid. Systemic corticosteroids and other medications such as dapsone, cyclophosphamide, and tetracycline or minocycline combined with niacinamide have been used for more severe cases. Because the most serious complication of MMP is blindness, all patients should be referred to ophthalmologists for evaluation once the diagnosis is established.

The prognosis of MMP varies. Some patients may have limited disease and no scarring. Others have significant scarring that impairs the function of the affected organs. Although MMP is a chronic incurable disease, it is rarely fatal. The prognosis for oral pemphigoid is usually good.

Bullous Pemphigoid

BP is an autoimmune blistering disease that primarily affects skin but rarely mucosa.[26] It primarily affects the elderly and oral involvement has been reported in 8% to 39% of the cases. The oral lesions are indistinguishable from MMP.

The pathogenesis of BP is similar to that of MMP except that patients with BP develop autoantibodies against only BP230 and BP180, with BP230 being the predominant autoantigen.

The diagnosis of BP is based on clinical, histologic, and DIF findings.

Linear IgA Bullous Dermatosis

LABD is a pruritic skin blistering disease that occasionally involves mucosa. It typically occurs in adults older than 60 years.[26]

Clinically, LABD is characterized by stomatitis and blisters. The definitive diagnosis is based on the DIF findings, which reveal linear deposition of IgA in the basement membrane zone. The autoantigen has been found to be the 97-kd LABD antigen, which later was identified to be a portion of BP180, the adhesion protein located in the basement membrane zone and is the predominant autoantigen found in MMP patients.

Epidermolysis Bullosa Acquisita

EBA is another rare autoimmune subepithelial blistering disease that affects skin and mucosa. Although it is predominantly a skin disease, oral lesions occur in almost 50% of the patients.[26] The autoantigen has been found to be type VII collagen in the basement membrane zone.

Clinically, the EBA patients have increased skin fragility, and the blisters of EBA often occur in trauma-prone extensor surfaces of skin, such as the knuckles, dorsal hands, elbows, and knees.[46] The blisters rupture and heal with scar formation. Oral blisters or erosive lesions typically develop in the presence of skin lesions. The oral manifestation of EBA may not be distinguishable from PV, MMP, or BP.

Erythema Multiforme and Related Disorders

EM and related disorders are a group of mucocutaneous diseases characterized by variable degrees of cutaneous and mucosal blistering and ulceration. Depending on the clinical presentation and extent of involvement, this group traditionally has been divided into three subcategories: EM minor, EM major (Stevens-Johnson syndrome), and toxic epidermal necrolysis (Lyell's disease).[26] Recent studies have suggested that Stevens-Johnson syndrome and toxic epidermal necrolysis are distinctly different from EM minor and EM major.[47] However, significant overlap in the clinical features of Stevens-Johnson syndrome and EM major is seen and a clear separation between these two entities remains controversial.[21]

EM is believed to be an immunologic disorder, although the cause is still unclear. In about half of the cases, EM is associated with a preceding infection, especially herpes simplex or *Mycoplasma pneumoniae*, or exposure to certain medications. The infectious agents and medications that have been reported to be associated with development of EM are listed in Tables 36-2 and 36-3, respectively.[47]

EM minor is typically acute onset, and an episodic or recurrent pattern has been observed clinically. It is characterized by targetoid lesions in skin, typically in the extremities and oral erythema, ulceration, or erosions. The oral lesions usually start with edema and erythema of the lips and buccal

TABLE 36-2. **Infectious Agents Associated with Erythema Multiforme**

Virus	Bacteria	Fungi	Parasites
HSV-1	*Mycoplasma pneumoniae*	*Histoplasma capsulatum*	*Trichomonas*
HSV-2	*Borrelia burgdorferi*	*Coccidiodes immitis*	*Toxoplasma gondii*
EBV	*Corynebacterium diphtheriae*		
CMV	*Streptococci*		
Varicella-zoster virus	*Legionella pneumophila*		
Adenoviruses	*Salmonella*		
Enteroviruses	*Mycobacterium leprae*		
Hepatitis virus	*Neisseria meningitides*		
Influenza virus	*Mycobacterium avium* complex		
Parvovirus B19	*Pneumococcus*		
Poliovirus	*Proteus*		
	Pseudomonas		
	Rickettsia		
	Mycobacterium tuberculosis		
	Vibrio parahaemolyticus		
	Yersinia		
	Chlamydia		

CMV = cytomegalovirus; EBV = Epstein-Barr virus; HSV = herpes simplex virus.

mucosa, followed by eruption of multiple vesicles and bullae that quickly rupture and result in ulcerations or erosions (Figure 36-19). Ulceration and hemorrhagic crusting of the vermilion border of the lips is a common and characteristic feature. Nikolsky's sign is negative. Occasionally, EM minor affects the oral mucosa exclusively.[48] The mucocutaneous lesions usually exist for 1 to 3 weeks and heal without scarring. Most patients do not have prodromal symptoms, but some may experience mild fever or chills.

EM major or Stevens-Johnson syndrome is a more severe form of EM. More than 50% of these cases are triggered by medications. In addition to skin and oral lesions, either the genital mucosa or the conjunctiva is also affected. The skin lesions are widespread and the mucocutaneous lesions last for 2 to 6 weeks. About one third of the patients have prodromal symptoms such as fever, pharyngitis, headache, and arthralgia/myalgia. The mucosal lesions sometimes heal with scar formation that may result in symblepharon of the conjunctiva or laryngeal and/or vaginal strictures.

Toxic epidermal necrolysis is considered to be the most severe form of the EM-related disorders, and 95% of the cases in adults appear to be triggered by medications. Clinically, it is characterized by diffuse sloughing of a significant portion of the skin and mucosa. The involved mucosal

TABLE 36-3. **Drugs Reported to Contribute to Development of Erythema Multiforme and Related Disorders**

Antibacterial	Sulfonamides, trimethoprim-sulfamethoxazole, aminopenicillins, cephalosporins, quinolones, tetracyclines
Antiseizure drugs	Phenobarbital, phenytoin, carbamazepine, valproic acid
Nonsteroidal anti-inflammatory drugs	Oxicam (piroxicam, tenoxicam)
Antifungal	Imidazole
Other categories	Allopurinol, chlormezanone, acetaminophen, systemic corticosteroids



FIGURE 36-19. **A,** Erythema multiforme with hemorrhagic crusting of vermilion. **B,** Erythema multiforme with panoral stomatitis.

surfaces include oropharynx, esophagus, conjunctiva, and genitalia. The lesions are often preceded by fever, sore throat, and skin tenderness.

The diagnosis of EM and related disorders is often based on the clinical findings and the exclusion of other vesiculobullous diseases. Histologically, the perilesional mucosa in EM often shows subepithelial or intraepithelial vesicles or bullae combined with epithelial necrosis and apoptotic keratinocytes. The inflammatory infiltrate is a mixture of lymphocytes, neutrophils, and often eosinophils. A perivascular inflammatory infiltrate is sometimes seen. The histologic features are not specific and DIF study is not helpful except to exclude other vesiculobullous disorders.

The management for EM and related disorders is basically symptomatic and supportive treatment. Topical or systemic corticosteroids are often used, especially in the early stage of the disease. Mouth rinse containing local anesthetic and mild antiseptic agents may help to relieve oral symptoms. In cases in which a causative drug is identified or suspected, it should be discontinued immediately. In patients with HSV-associated EM minor, antiviral therapies have been reported to be beneficial, especially in preventing recurrence. For EM major and toxic epidermal necrolysis, a multidisciplinary systemic management strategy is necessary and therapies may include systemic corticosteroids, corticosteroid-sparing immunosuppressants, and hospital-based supportive and/or intensive care.

EM is generally not a life-threatening disease except for the most severe form, toxic epidermal necrolysis, which has a poor prognosis and is associated with a 30% to 40% mortality rate. The mortality rate for Stevens-Johnson syndrome is 2% to 10%.

PIGMENTED LESIONS OF THE ORAL MUCOSA AND SKIN

Pigmented lesions involving the intraoral mucosa, the vermilion border of the lips, and the skin surfaces of the head and neck region are commonly encountered. They may appear as a single, isolated lesion or as a multifocal and sometimes diffuse process. The majority of pigmented lesions will present as a macular discoloration, without surface elevation, and palpation of the lesional area typically fails to reveal a subcutaneous or submucosal component to the lesion. Elevated, papular to nodular lesions are also encountered with some frequency, and the differential diagnosis for this group is more limited. A variety of potential mechanisms may result in pigmented lesions. Localized areas of increased melanin production are a very common cause. Intravascular blood and the breakdown products of extravasated blood account for many of these lesions. Pigmented foreign materials implanted into the connective tissue, either accidentally or purposefully, also are a cause for some of these lesions, particularly intraorally, and they are typically referred to as *tattoos*. On occasion, extrinsic pigmented materials will become adherent to the surface mucosa intraorally, simulating a pigmented lesion. For this reason, pigmented lesions should be wiped with dry gauze to rule out this possibility.

Identification of the etiology of a pigmented lesion is important because it may dictate treatment decisions. A lesion that can be confidently diagnosed clinically either as a tattoo or due to extravasated blood products normally requires only routine clinical follow-up. Lesions identified as being caused by increased melanin production or intravascular blood will more often result in a biopsy to establish a definitive diagnosis. In many instances, the clinical color of the lesion will be quite helpful in determining the etiology. Lesions produced by increased melanin production typically present as light to dark brown areas of pigmentation. These lesions will not blanch when pressure is applied. Lesions produced by extravasated blood typically present as red to reddish-purple macules. Although they also will not blanch when pressure is applied, they often are painful on palpation, representing nothing more than a bruise. Lesions produced by increased quantities of intravascular blood also will present as red to reddish-purple areas. In many cases, however, these lesions will blanch with the application of surface pressure as the intravascular blood is forced into the surrounding capillary networks. Tattoos may sometimes be identified by the unusual color of the pigmented area. They often present as slate gray to blue lesions, and they never blanch with applied pressure. The quantity of pigmented material may complicate clinical determination of the etiology of a pigmented lesion, however. As the quantity of melanin, blood and blood products, or foreign material increases, the more similar the lesions may appear, producing dark purple to indigo to black areas of pigmentation.

Localized Pigmented Lesions

Lesions Produced by Excess Melanin Production

Ephilis (Freckle)

The ephilis is an exceedingly common lesion of the skin of the face and, once recognized, requires no treatment.[49] It typically presents in childhood as a small, well-circumscribed area of uniform macular pigmentation that may vary from light to dark brown. Classically, the ephilis will darken with increased sun exposure and lighten when sun exposure decreases.[50] The number of lesions tends to increase into adulthood but then regresses with advancing age.[49] The ephilis is caused by an increased production of melanin by melanocytes in the basal cell layer of the epithelium, which is subsequently transferred to the adjacent keratinocytes. The number of melanocytes is not increased and may be diminished slightly.

Lentigo

The lentigo is a lesion of skin that clinically resembles the ephilis.[51] Two subclassifications are recognized: lentigo simplex and lentigo senilis. Lentigo simplex is of unknown etiology and usually occurs in children. Lesions are typically small, uniformly pigmented, and sharply circumscribed with

a smooth border. Unlike the ephilis, however, it tends to occur in areas not exposed to sunlight and the depth of color of the lesions does not change with increasing sun exposure.[51] They also typically are more deeply pigmented than the ephilis. Lentigo senilis is also referred to as "solar lentigo" but is best known by the lay terminology of "age spot" or "liver spot." It is an extremely common lesion in elderly white individuals, being present in more than 90% older than age 70.[49] It is only rarely encountered in persons younger than age 40. The etiology is chronic sun exposure.[50] As with lentigo simplex, the lesions show no change in pigmentation with increasing sun exposure. Unlike lentigo simplex, however, multiple lesions are typically seen and they tend to have an irregular border.[49] The skin of the face and the vermilion border of the lip are common sites of involvement. Although they have been reported intraorally, they are extremely rare in that location.[51] The histologic features of lentigo simplex and lentigo senilis are nearly identical. Both show slightly elongated rete ridges, increased melanin production, and a normal to slightly increased number of melanocytes in the basal cell layer. They often can be differentiated by the presence of solar elastosis (sun damage) in the dermis in lentigo senilis.

Melanotic Macule

The melanotic macule is the intraoral counterpart of the skin ephilis and is quite common.[52] It is sometimes referred to as *focal melanosis*. Clinically, the lesions present as small, well-demarcated, round to oval macules that are uniformly pigmented[53] (Figure 36-20). The color normally is described as light to dark brown, with occasional lesions being blue to black.[52] Unlike the skin ephilis, however, there is no association with sun exposure and there may actually be a slight increase in the number of melanocytes seen on histologic examination.[53] Whereas any oral mucosal surface may be affected, the vermilion surface of the lower lip is the most common site of occurrence. The buccal mucosa, palate, and gingiva are other commonly affected areas. Melanotic macules may be seen at any age. Women tend to be affected twice as often as men. Lesions are most often single but multiple sites of involvement can occur. They do not tend to regress and disappear.[53] Microscopic examination reveals increased melanin in the basal keratinocytes, analogous to the skin ephilis. Melanin incontinence is also often noted, however, with melanin pigment found in macrophages in the lamina propria, a feature not typically seen in the ephilis. No treatment is necessary in most instances, but removal of the lesions for aesthetic reasons, particularly when the lower lip is involved, is fairly common.

Transformation of a melanotic macule into intraoral melanoma has been documented in at least one instance, however,[54] and anecdotal reports suggest intraoral melanoma may originate as a macular pigmented lesion simulating the melanotic macule. For this reason, melanotic macules are worthy of clinical follow-up, and lesions that show any of the typical worrisome features of melanoma should be removed.

Postinflammatory Melanosis

Pigmented lesions clinically identical to the melanotic macule are occasionally encountered after traumatic injuries or other inflammatory processes. The inflammation induces overproduction of melanin by the melanocytes in the affected area, resulting in a macular area of increased pigmentation. Unlike the melanotic macule, postinflammatory melanosis resolves with loss of the inflammatory stimulation and healing.

Oral Melanoacanthoma

The oral melanoacanthoma is a relatively rare lesion. Although the etiology is not known with certainty, it is theorized to be a reactive process.[55] A source of irritation to the lesional area can be identified in many instances. It typically presents as a macular pigmented area, although occasional lesions may appear slightly elevated[56] (Figure 36-21). They are normally dark brown to black in color and may show considerable variability in the degree of pigmentation within



FIGURE 36-20. Multiple melanotic macules of the gingiva.



FIGURE 36-21. Melanoacanthoma in the buccal mucosa of a young black female.

the lesion. Although often well circumscribed, the border may be somewhat irregular.[55] Disturbingly, the melanoacanthoma often shows a very rapid growth, enlarging to several centimeters in diameter within a short period of time. Thus, melanoma is often considered high in the clinical differential diagnosis. Lesions are seen almost exclusively in blacks with a strong female predilection. Young adults are the primary population affected. The buccal mucosa is the most common site. Although usually unilateral, multifocal lesions are occasionally seen.[55] Despite its rapid growth, the process is benign and lesions have shown spontaneous regression with removal of the irritating stimulus or after incisional biopsy.[56] Histopathologic findings are distinctive. Numerous dendritic melanocytes are found throughout all levels of the epithelium. Melanocytes in the basal cell layer are also increased in number.

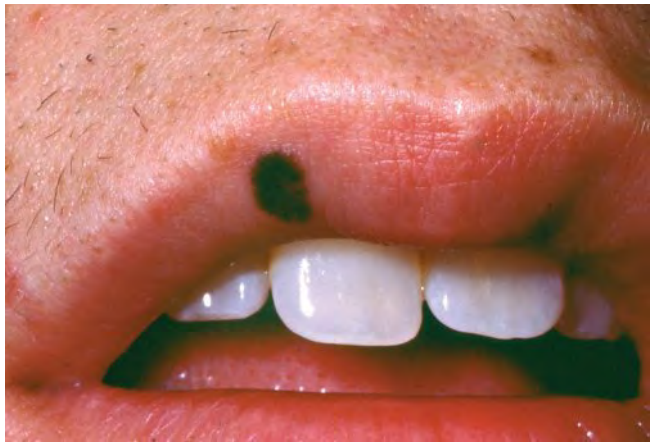


FIGURE 36-22. Junctional nevus of the upper lip.

Melanocytic Nevi

The *melanocytic nevus*, also termed *nevocellular nevus*, is generally referred to and perhaps more widely recognized as a "mole" or "birthmark." They are extraordinarily common lesions of the skin, with nearly 100% of light skinned individuals having at least one melanocytic nevus.[49] They are less common in the dark-skinned races. The skin of the face is a frequent site of occurrence. The lesions are considered developmental malformations produced by a proliferation of neural crest–derived cells (nevus cells), either melanocytes or melanocyte precursors.[49] The etiology of skin lesions is linked to sun exposure during the first two decades of life. Intermittent sunburning tends to produce more lesions than low-level chronic sun exposure, which appears to be somewhat protective.[49] Skin lesions on occasion are congenital but more commonly develop in early childhood, and nearly all lesions are present by early adulthood. Newly developing melanocytic nevi after age 35 are distinctly unusual and should be viewed with suspicion.[49] With advancing age, the lesions tend to involute, and by the eighth to ninth decades, they virtually disappear.

Nevi are subclassified histologically according to the location of the proliferating nevus cells relative to the overlying epithelium.[49] In *junctional nevi*, the nevus cells are in intimate contact with the epithelium, and the lesions are macular clinically (Figure 36-22). *Intradermal nevi* have nevus cells located separate from the epithelium in the upper dermis and produce elevated, dome-shaped nodular masses that may be sessile or polypoid. *Compound nevi*, which show nevus cell nests in both the epithelium and the upper dermis, are slightly elevated nodules. *Blue nevi* are characterized by spindled to oval nevus cells found in the deeper dermal tissues; they tend to produce elevated, flat-topped nodules with a distinctive blue color. Numerous other subtypes are also known to exist.

One important subtype is the dysplastic nevus. *Dysplastic nevi* may be a precursor to melanoma, particularly when the lesions are multiple and occur in a familial, autosomal dominant pattern. Patients with multiple lesions must, therefore, be closely monitored. The relationship between a solitary dysplastic nevus and development of melanoma is less clear. Clinically, the lesions simulate melanoma, being somewhat larger than typical nevi and show irregular and indistinct borders and variation in color. Dysplastic nevi are known to occur intraorally.[49]

Most cutaneous nevi are largely innocuous lesions that require no treatment. Chronic irritation may predispose a nevus to malignant transformation and those nevi are normally excised. Clinical changes in a preexisting nevus may herald development of melanoma and also prompt removal. Excision is also often effected per patient desires for cosmetic reasons.

In contrast to skin lesions, intraoral nevi are decidedly uncommon.[57–58] The palate is the most common site of occurrence but lesions may be seen in almost any area of the oral cavity.[58] The lips, gingiva, and mucobuccal vestibule are other relatively common sites of occurrence.[58] The dorsum of the tongue is an extraordinarily rare site of involvement, with no well-documented cases ever having been reported. Intramucosal nevi, the mucosal counterpart of intradermal nevi, are the most common subtype, accounting for more than half of all lesions[58] (Figure 36-23). Blue nevi are the second most common subtype seen intraorally. Intraoral lesions tend to be less deeply pigmented than skin lesions, with more than 20% lacking melanin pigmentation entirely, presenting instead as normal-colored nodular masses.[57–58] Because of the concern for continual irritation of the lesions when they occur intraorally, and because clinical separation from melanoma is not possible with good certainty, lesions provisionally diagnosed as intraoral nevi are typically excised.

Seborrheic Keratosis

Seborrheic keratosis is a common pigmented lesion seen primarily in the elderly. Clinically, they may resemble nevi and, thus, should be included in the differential diagnosis with nevi. They commonly do not begin to appear until age 30, a helpful contrasting feature to nevi, and they become more prevalent rather than less prevalent with increasing age.[49] They typically present as elevated pigmented lesions

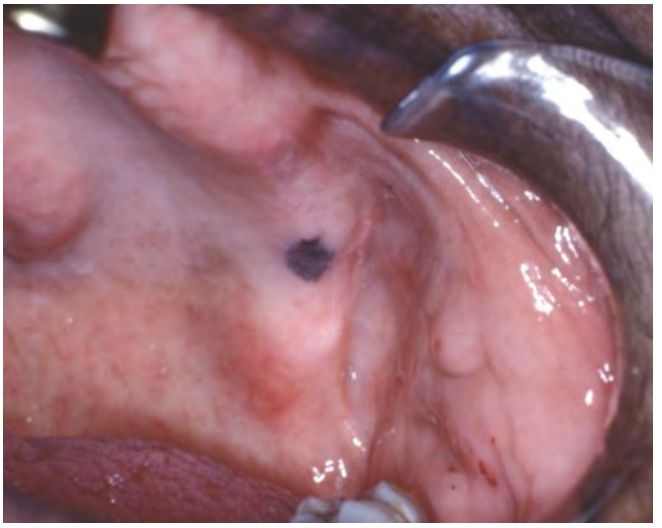


FIGURE 36-23. Intramucosal nevus of the alveolar ridge.

with a rough surface (Figure 36-24). They are often described as having a "greasy" surface and "stuck-on" appearance, also helping to separate them clinically from nevi. The cause is unknown. Chronic sun exposure may be a factor and heredity may play a role as an autosomal dominant familial pattern is seen in many affected individuals.[49] Lesions do not occur intraorally.[52] Seborrheic keratoses are innocuous lesions that seldom require removal except for cosmetic reasons.

Melanoma

Melanoma is a malignant neoplasm that arises from cells of melanocytic origin. It is the most aggressive and deadliest of all the skin cancers. Morbidity and mortality are increasing with this disease owing to its increasing incidence. The number of skin cases has been growing approximately 5% per year.[49] Whereas the majority of melanomas are believed to arise de novo from melanocytes in the basal cell layer of epithelium, origin from the cells of melanocytic nevi is relatively common. It is estimated that more than 35% of nodular and superficial spreading melanomas arise within the background of a melanocytic nevus.[49] Most cases occur in middle-aged to older adults. The etiology is likely multifactorial, with race and genetic factors having a strong influence. Sun exposure certainly plays a role, and the incidence of melanoma in susceptible individuals is known to decrease with increasing living distance from the Earth's equator. The incidence of most types of melanoma corresponds best with excessive sunburning early in life, and the lifetime risk of developing melanoma is not believed to increase significantly after the third decade of life.[49] Individuals at greatest risk for sunburning are, therefore, more likely to develop the neoplasm. This group includes individuals with fair complexion and blue eyes, often with red hair. These individuals burn easily and tan poorly and often show mottled skin pigmentation with excessive freckling.[49] Dark-skinned races have a much lower incidence of melanoma.

Early diagnosis of melanoma is critical in attempting to control the disease.[59] Melanomas tend to show two different growth patterns: a radial growth phase and a vertical growth phase. During the radial growth phase, melanoma cells grow along the epithelial/connective tissue interface but do not invade into the deeper dermis where access to blood vessels and the potential for distant metastasis are greater. Melanomas may remain in the radial growth phase for long periods of time. Once a lesion enters the vertical growth phase, however, vascular invasion and perineural spread is commonly seen and metastasis to distant sites is common. Thus, diagnosis of melanoma during the radial growth phase carries a far more favorable prognosis, and the thickness of a lesion becomes the most important prognostic factor. Cutaneous melanomas less than 1 mm in thickness and not ulcerated have a 5-year survival rate of 95% if metastasis is not already present.[49] The presence of ulceration in a lesion is often a later finding and adversely affects prognosis. Patients with documented metastatic disease at a time of diagnosis have a 5-year survival under 20%.[59]

Four histologic subtypes of melanoma are recognized. Lentigo maligna melanoma (melanotic freckle of Hutchinson) is considered an in situ disease (Figure 36-25). It presents as a macular brown lesion containing spotty areas of



FIGURE 36-24. Seborrheic keratosis of the skin of the chin.



FIGURE 36-25. Lentigo maligna melanoma of the facial skin.

darker pigmentation. It occurs on sun-exposed areas in elderly individuals, often on the face. The borders are irregular. These lesions have been shown to remain in the radial growth phase for as long as 15 years before demonstrating nodular change and progressing to invasive melanoma. Superficial spreading melanoma is the most common form, representing approximately 70% of the cutaneous lesions. It occurs in non–sun-exposed areas with some frequency, showing a propensity for the upper back in men and the lower legs in women. Clinically, the lesions are irregular in outline and show a variation in pigmentation. Areas of depigmentation within the lesion may be seen (Figure 36-26). The surface may also be irregular with nodular foci and/or ulceration developing within an otherwise macular lesion. As the name would indicate, *superficial spreading melanoma* shows radial growth primarily but progresses to the vertical growth phase more quickly than lentigo maligna and carries a poorer prognosis. Nodular melanoma shows prominent vertical growth early in its development without a significant radial growth phase and is not as strongly associated with sun exposure. It typically presents as a rapidly growing, ulcerated, darkly pigmented mass that is deeply invasive at the time of initial diagnosis. Metastasis is common and the prognosis is poor. Acral-lentiginous melanoma is the rarest subtype of melanoma but the most commonly seen in black individuals. It is an aggressive type that tends to metastasize early. It begins as a macular area of irregular dark pigmentation. The palms of the hands, soles of the feet, and underneath the nails are common locations. Intraoral melanomas often present as this subtype and are sometimes referred to as *mucosal lentiginous melanoma.*

Clinical features worrisome for melanoma that are quite helpful in recognizing lesions in the earliest stages of development utilize the *ABCD* algorithm.[59] These include (1) variation in the surface *a*rchitecture of the lesion, with areas of nodularity and ulceration within a macular area of pigmentation, (2) irregularity of the *b*order of the lesion, (3) variation in the *c*olor of the lesion with pink, tan, brown, blue, black, or even depigmented areas within a single lesion, and (4) a *d*iameter greater than 6 mm. Incisional biopsy of large lesions to establish a diagnosis is acceptable. Previously, incisional biopsy of melanoma was discouraged because it was thought to enhance the likelihood of metastatic spread. This has been shown not to be the case.



FIGURE 36-26. Malignant melanoma of skin.

Treatment of melanoma consists of assured surgical excision with a wide margin of surrounding normal tissue. Sentinel lymph node dissection now is often performed to assess metastatic status. Radiation therapy is unusual because it is found to be generally ineffective against melanoma. Chemotherapy, immune system modulation therapy, and genetically directed treatment modalities are also being used with greater frequency.[49]

Intraoral melanoma luckily is a rare occurrence. Most patients are older than age 50 at initial presentation. Clinically, intraoral lesions often begin as irregular macules that represent the radial growth phase of the lesion, the so-called mucosal lentiginous type of melanoma.[60] A nodular invasive growth pattern soon develops within the area of macular pigmentation and ulceration is frequent. The hard palate and the gingiva are the most commonly affected sites.[60] Extension of the melanoma into the underlying bone is often noted and heralds a particularly poor prognosis. The prognosis for oral melanoma is poor even in the absence of bone involvement. Average 5-year survival in oral melanoma is less than 20%.[60] Surgical treatment, therefore, is always very aggressive.

Vascular Lesions

Vascular lesions normally present as red to reddish-purple areas and are, therefore, usually easily recognizable as vascular in origin. As the quantity of blood increases, the lesions will darken in color and may be confused with lesions produced by melanin. Three mechanisms are possible in the production of vascular lesions. The normal blood vessels in an area may be markedly dilated, there may be an increased number of blood vessels present in an area, or blood may be spilled from the vessels into the connective tissue compartment. Lesions produced by dilation of existing blood vessels or proliferation of new blood vessels may blanch with applied pressure over their surface, clearly identifying the intravascular nature of the blood. Lesions produced by blood spillage are often mildly painful on palpation but do not blanch.

Telangiectasia and Varix

Telangiectasia represents a dilation of the capillary vasculature in an area. It usually presents as multiple, small, bright red lesions and can involve any soft tissue site. The lesions may be flat to slightly elevated. They are asymptomatic and require no treatment in most instances. A varix is a dilated vein. It can be found at any age but is seen with increased frequency in older individuals.[61] The lips, floor of mouth, and ventral tongue are common sites.[61] Varices often are reddish-purple to blue in color due to the quantity of blood present in the dilated vessel (Figure 36-27). They are soft on palpation and will blanch with pressure unless thrombosed. No treatment is necessary, although thrombosed varices are often biopsied because of their firm nature on palpation and the clinical concern to rule out melanoma.



FIGURE 36-27. Varix of the upper lip.



FIGURE 36-28. Multiple lesions of Kaposi's sarcoma affecting the palate.

Hemangioma

Hemangiomas represent a proliferation of blood vessels. Some debate remains as to whether they represent developmental anomalies or true neoplasms.[61] Spontaneous regression occurs routinely for skin lesions, suggesting developmental anomalies, but regression for oral lesions is less common.[62] The head and neck area is commonly involved and intraoral lesions are frequent.[63] They are seen most often in children and are sometimes congenital. The quantity of blood vessels and their location within the tissue determine the clinical presentation. Lesions may be flat or produce enlargement of the involved area. Intraorally, hemangiomas are red to blue in color, soft to palpation, and usually blanch with applied surface pressure. The color of the lesion is determined by the size of the blood vessels in the lesion. Capillary hemangiomas are normally red; cavernous hemangiomas tend to be blue to purple. Thrombosis within the lesional blood vessels is not uncommon and will produce lesions with a firm, sometimes multinodular character. Whereas any area of the oral mucosa may be involved, the lips, buccal mucosa, and tongue account for a large number of cases.[54] Hemangiomas are typically diffuse and poorly delineated clinically, making surgical removal problematic at times. Incision through a lesion may cause extensive bleeding. Other treatment options include embolization, cryotherapy, and laser surgery.[53,54] Sclerosing agents have also been utilized but tend to produce less reliable and predictable results. Treatment may not be necessary unless the lesion may be subject to trauma or it becomes a functional or cosmetic problem for the patient.

Angiosarcoma and Kaposi's Sarcoma

Angiosarcomas represent a malignant proliferation of blood vessels. They are very rare lesions overall. The skin in the scalp and forehead area is a common location.[55] They tend to be aggressive lesions with a poor prognosis and are treated with radical surgical excision and radiotherapy.[55] Angiosarcoma involving the oral mucosa is decidedly rare. Kaposi's sarcoma (KS), a subtype of angiosarcoma linked to human herpesvirus 8 and HIV infection, is more often seen intraorally. At the height of the AIDS epidemic, KS was commonly encountered, but with the advent of antiretroviral therapy, the incidence of KS has fallen dramatically.[65] KS is also encountered in immunosuppressed patients secondary to organ transplantation. When the oral mucosa is affected, the hard palate and gingiva are most often involved (Figure 36-28). Lesions may begin as macular reddish lesions that subsequently enlarge and become nodular, often ulcerating. Pigmentation increases with increasing size and can produce dark brown to black lesions.[65] The lesions may or may not blanch with applied pressure. Treatment for KS may not be necessary. In patients with AIDS, antiretroviral treatment may result in regression of the lesions.[65] Surgery, radiation therapy, and chemotherapy have also been utilized.

Lesions Produced by Extravasated Blood

Petechia, Purpura, and Ecchymosis

These lesions all represent a form of bruising with the size of the lesion being the differentiating factor. *Petechiae* are defined as pinpoint areas of hemorrhage and are typically associated with breakage of capillary blood vessels. *Purpuras* are slightly larger, measuring up to 1 cm in diameter, and are often associated with small veins. *Ecchymoses* are larger than 1 cm and are typically associated with larger blood vessels. They may be mildly tender to palpation but do not produce palpable evidence of a deep component. Trauma is the usual cause, but any condition producing vascular fragility may produce these lesions. No treatment is typically necessary.

Hematoma

A hematoma is the result of bleeding into deep tissue spaces. Trauma to the area is a consistent etiologic finding. A large quantity of blood is present, which subsequently clots, producing a deep component to the lesion on palpation. Pain on palpation may be significant. The tongue and buccal mucosa are common sites, owing to the frequency of associated trauma in these areas. Treatment is not necessary and the lesions resolve slowly over time.

Tattoos

Tattoos are frequently encountered oral lesions.[66] They are produced by implantation of foreign materials into the

subepithelial connective tissues. Amalgam is the most common culprit[57] (Figure 36-29). Other metals, graphite, and ink have also been identified as a source. Any area of the oral mucosa may be affected, but the gingiva is the most common site.[57] The lesions are typically macular and discovered on routine examination. Occasional lesions are elevated. The color will vary depending upon the nature of the implanted material. Amalgam often produces a slate gray to blue to black discoloration. If large metal fragments are implanted, radiographic studies may verify the clinical impression and negate the need for treatment. A large number of tattoos do not show up on radiographic examination, however. Treatment is sometimes necessary for cosmetic reasons but more commonly when the clinical differential diagnosis includes other, more worrisome lesions.

Generalized Pigmented Lesions

In contrast to localized pigmentations, generalized pigmented lesions normally signal a systemic etiology for the process. This may be due to hereditary factors, metabolic disorders, endocrine abnormalities, or the effects of medications. Lesions produced tend to be macular brown pigmentations involving a diffuse area of the mucosa. A symmetrical distribution to the lesions may be seen. Generalized pigmented lesions linked to genetic factors include physiologic (racial) pigmentation, Peutz-Jeghers syndrome, neurofibromatosis I, and Albright's syndrome. Endocrine and metabolic disorders may also produce generalized pigmentations. These include pulmonary disease, heavy metal intoxication, Addison's disease, pregnancy, and menopause. Perhaps the most common cause for generalized pigmented lesions is secondary to the effects of medications. Antimalarials, birth control medication, minocycline, phenothiazines, and some cancer chemotherapeutic agents are most often implicated.



FIGURE 36-29. Amalgam tattoo secondary to endodontic surgery with amalgam retrofill.

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37

CHAPTER

Pediatric Maxillofacial Pathology

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INTRODUCTION

The complex development of the facial skeleton and the presence of odontogenic tissue contribute to formation of a variety of cysts, tumors, and pathologic conditions that either are unique or occur more often in this region in the pediatric age group. Tumors of the maxillofacial region in children are generally infrequent and may represent a variety of clinical and pathologic entities. The rarity of these conditions explains the lack of numerous comprehensive case series that specifically address pediatric maxillofacial pathology. Nevertheless, the maxillofacial surgeon should be familiar with at least the most commonly encountered cysts and tumors, odontogenic or nonodontogenic in origin, their behavior, and the appropriate treatment protocols. Even more important is an understanding of the potential outcomes of treatment of these conditions in the immature, developing child. The purpose of this chapter is to provide a comprehensive review of pediatric maxillofacial pathology along with the currently available treatment recommendations.

Cysts and tumors in the maxillofacial skeleton are traditionally classified as *odontogenic* or *nonodontogenic* based on the tissue of origin**.** The contributing tissue, epithelium or mesenchyme, is used to further subclassify odontogenic pathology, whereas nonodontogenic tumors are grouped based upon the cell line of origin and their histologic appearance or behavior[1–3] (Tables 37-1 and 37-2). However, treatment should be based not only on the histologic appearance of these entities but also on their known clinical and biologic behavior. Finally, salivary gland tissue pathology is unique to the maxillofacial skeleton and certain inflammatory conditions, cysts, and tumors may occur more commonly in children than in adult patients.

ODONTOGENIC PATHOLOGY

Gingival cysts of the newborn, or dental lamina cysts of the newborn, or *Bohn's nodules* are superficial, raised nodules of edentulous alveolar ridges in infants and neonates. These are thought to originate from entrapment and proliferation of primary dental lamina after tooth formation and consist of a keratin-producing epithelial lining. The proliferation of the dental lamina remnants is limited in overall extent and potential and these cysts degenerate, involute, or rupture spontaneously in the oral cavity by 3 months of age.

TABLE 37-1. **Classification of Pediatric Odontogenic Tumors in the Maxillofacial Skeleton Based on the Tissue of Origin**

Epithelial Origin Tumors	Mesenchymal Origin Tumors	Mixed Origin Tumors
Ameloblastoma	Odontogenic myxoma	Ameloblastic fibroma
AOT	Cementoblastoma (true cementoma)	Odontoma
KCOT	Cementifying fibroma (COF)	Ameloblastic fibro-odontoma

AOT = adenomatoid odontogenic tumor; COF = cemento-ossifying fibroma; KCOT = keratocystic odontogenic tumor.

Epithelial inclusion cysts that form along the midline of the palate are known as *palatine cysts of the newborn*, or *Epstein's pearls.* Similarly, these cysts are developmental in origin. During the fusion of the palatal shelves and nasal processes, entrapment of keratin-producing epithelium at the interface may occur. Epstein's pearls fuse with the overlying epithelium and generally require no treatment. The vast majority of these cysts are clinically undetectable, and an incisional or excisional biopsy to confirm the diagnosis is typically not required or justified. In most cases, reassurance for the parents and the pediatrician may be all that is needed.[4–7]

Developmental cysts associated with impacted teeth in children are more common in the maxillary canine and mandibular second molar region, whereas in the teenage years, as in adulthood, the third molar region is the most common location. Delay in eruption of teeth necessitates radiographic

TABLE 37-2. **Classification of Pediatric Nonodontogenic Cysts and Tumors in the Maxillofacial Skeleton Based on the Cell Line of Origin**

Benign Mesenchymal Origin Tumors	Hematopoietic reticuloendothelial Tumors	Neurogenic Origin Tumors	Vascular Pathology	Epithelial Neoplasm	Mesenchymal Neoplasm
Giant cell lesions	Burkitt's lymphoma	Congenital gingival granular cell tumor (congenital epulis	Hemangioma	Squamous cell carcinoma (extremely rare in children)	Ewing's sarcoma
Fibro-osseous lesions	Langerhans' cell histiocytosis	Mucosal neuroma	Vascular malformation		
Desmoplastic fibroma	Hodgkin's disease	Neurofibroma	Aneurysmal bone cyst		
	Non-Hodgkin's lymphoma	Melanotic neuroectodermal tumor of infancy			

investigation that may reveal the absence of the tooth or the presence of a radiolucent lesion associated with the crown of an impacted permanent or a supernumery tooth. By definition, a *dentigerous cyst* is precisely this, a cyst that attaches to the tooth cervix (cementoenamel junction) and contains the crown of the impacted tooth. The tissue of origin is considered to be the reduced enamel epithelium or remnants of the enamel organ. Simple enucleation and curettage along with removal of the impacted tooth, in cases of third molar involvement, is usually adequate treatment. When the canine or second molar teeth are involved, extraction of teeth should be avoided, especially if the teeth can be orthodontically moved into the arch, thereby effectively treating the cyst. The recurrence potential of a dentigerous cyst is negligible, but these cysts may cause bone expansion, local destruction, root resorption, or displacement of teeth, which occurs more commonly with longstanding lesions (Figure 37-1). Rarely, neoplastic transformation to ameloblastoma (i.e., mural ameloblastoma) has been described, although the possibility of a misdiagnosis should be considered in these cases, and rebiopsy may be required.[4–7]



FIGURE 37-1. Dentigerous cyst in the left mandible displacing tooth no. 17 of a 16-year-old girl.

Epithelial Odontogenic Tumors

Although *ameloblastoma* is the most common epithelial-origin odontogenic tumor and should always be included in the differential diagnosis of radiolucent lesions in the jaws, it is a rare tumor in children. In general, there is an equal gender predilection, but the overwhelming majority of these tumors are reported in the posterior mandible. Among the biologic forms described, the *unicystic* variant is the one encountered with some frequency among adolescents and is further discussed here. Clinically and radiographically, this tumor resembles a cyst and is associated with an impacted tooth (Figure 37-2). Common findings associated with the unicystic ameloblastoma include symptomatic bony expansion, cortical perforation, unilocular appearance, tooth and root displacement, and root resorption. In the majority of cases, the diagnosis of ameloblastoma is made after a "cystic" lesion is removed or biopsied. Clear serous fluid is



FIGURE 37-2. Ameloblastoma involving the left posterior mandible extending into the ramus of a 14-year-old boy.

usually aspirated from this lesion, and a bony cavity is usually encountered upon surgically entering the lesion.

Classically, there are three histologic patterns of cystic ameloblastoma, based upon removal and histopathologic examination of the entire lesion. The term *luminal* (unicystic) ameloblastoma is used when examination of a cystic lesion composed of a fibrous tissue wall reveals ameloblastic epithelium. The classic ameloblastoma-like characteristics of a palisading cuboidal basal cell layer with hyperchromatic nuclei and reverse polarization are present but contained within the cystic wall itself. When these typical tumor-like elements are found to project into the lumen of the cystic lesion as nodules but are still contained within the cystic lining, the term *intraluminal* ameloblastoma is applied. Lastly, when ameloblastic epithelial nests or islands are found to infiltrate through the cystic wall, the term *mural* ameloblastoma is employed, and these tumors may demonstrate a more aggressive behavior. Enucleation and curettage of cystic ameloblastomas is recommended as sufficient treatment in children, but long-term follow-up studies to support this approach are lacking owing to the overall rarity of these tumors. The recurrence potential, however, should be carefully considered especially in cases of mural ameloblastoma, and this will dictate the need for long-term follow-up. Conventional solid, or multicystic, ameloblastomas do occur in children, although rarely, and on occasion, extension of a unicystic ameloblastoma through the fibrous capsule may be encountered. On these occasions, surgical resection to the next anatomic barrier with a 1-cm linear bony margin should be performed as adequate treatment.[8–14]

Adenomatoid odontogenic tumor (AOT) is an epithelial odontogenic tumor, or more accurately, a hamartoma, that most likely originates from the reduced enamel epithelium. AOT may occur in children as young as 5 years of age, is more common in females (two thirds), is associated with an impacted tooth (two thirds), shows predilection for the anterior jaws (two thirds), and occurs in the maxilla (two thirds) more often than mandible. As a result, the AOT is commonly referred to as the tumors of "two thirds." Radiographically, AOT presents as a well-circumscribed radiolucency that may contain opaque foci and is associated with an impacted tooth, most commonly, the maxillary cuspid. Histologically, a thick capsule contains the nodular epithelial proliferation and calcified material. Ductal-like structures (pseudoducts) and rosettes are pathognomonic histologic findings for this tumor whereas the amount of calcified material dictates its degree of mixed radiographic appearance. Simple enucleation and curettage is adequate treatment because AOT has no recurrence potential.[6,7,12,15–17]

Philipsen in 1956 described the *odontogenic keratocyst* as a distinct entity for jaw cysts exhibiting keratinization and a specific pattern of characteristics of the epithelial lining.[18] These lesions may appear as early as the teen years, have a strong predilection for the posterior mandible, and are more common in male patients. They are usually asymptomatic but may cause bony expansion, tooth mobility, or tooth or root displacement. Radiographically, the presentation is that of a small or large unilocular or multilocular, well-demarcated radiolucency (Figure 37-3). The tissue of origin is either the dental lamina (60%) or the reduced enamel epithelium (40%). Two histologic types are commonly described: the *parakeratotic type*, (Forsell and Sainio group Ia), which is considered more aggressive, and the *orthokeratotic type* (Forsell and Sainio groups II and III). The aggressive nature of this entity, as well as the high recurrence potential, has been clearly demonstrated in clinical practice and supported by molecular evidence (Table 37-3). In 2005, the World Health Organization (WHO) classification of odontogenic lesions made the following change for this entity: "odontogenic keratocyst (OKC) is recognized as neoplasm and the term *keratocystic odontogenic tumor (KCOT)* is recommended to replace the previous terminology reflecting the neoplastic nature and characteristics of this entity."[19] KCOT in children is often associated with nevoid basal cell carcinoma syndrome (NBCCS) (Table 37-4). Several KCOTs

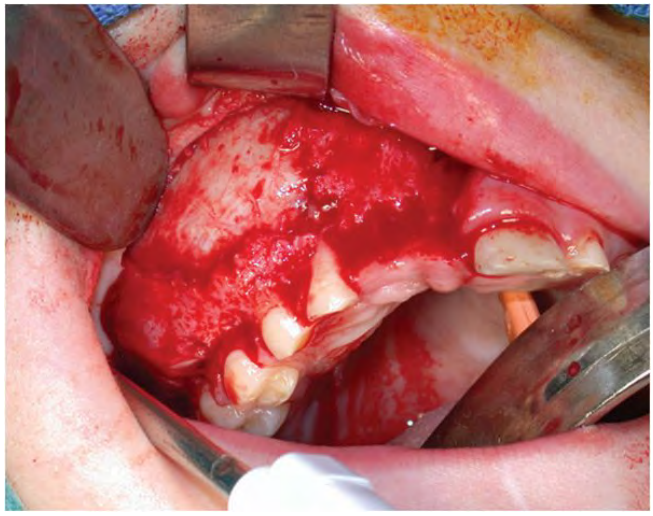


FIGURE 37-3. Keratocystic odontogenic tumor (KCOT) right maxilla causing expansion in an 11-year-old boy with nevoid basal cell carcinoma (NBCC) syndrome.

TABLE 37-3. **Characteristics and Molecular Markers Identified in Keratocystic Odontogenic Tumor That Support Growth, Neoplastic Nature, and High Recurrence Potential**

Growth and Recurrence Potential	Neoplastic Nature and Recurrence Potential
Intraluminal hyperosmolality	Ki67
Active epithelial proliferation	PCNA
Collagenolytic activity (cell wall)	p53
IL-1 and IL-6 (keratynocytes)	Bcl-2
Tumor necrosis factors (keratynocytes)	G-p38
Increase levels of prostaglandin	Cytokeratin 10
Increased level of parathyroid hormone–related protein	CEA
	EMA

CEA = carcinoembryonic antigen; EMA = epithelial marker antigen; IL = interleukin; PCNA = proliferating cell nuclear antigen.

TABLE 37-4. **Characteristics of Nevoid Basal Cell Carcinoma Syndrome**

Clinical	Skeletal/Radiographic
Frontal/temporoparietal bossin	Bifid ribs
Increased cranial circumference	Spayed ribs
Hypertelorism	Spina bifida
Mandibular prognathism	Calcified falx cerebri
Basal cell carcinomas	Shorten metacarpals
Palmar and plantar pitting	Multiple KCOTs that appear in association with the crowns of teeth and resemble dentigerous cysts and have a high recurrence rate
Ovarian fibromas	
Medulloblastomas	
Kyphoscoliosis	

KCOT = keratocystic odontogenic tumor.

appear at any one time in NBCCS patients and tend to behave more aggressively. NBCCS-associated KCOTs (and some sporadically occurring KCOTs) demonstrate *PCTH* gene (a tumor suppressor gene) and *SUFU* gene (involved in the hedgehog pathway) mutations that may explain, in part, their aggressive behavior. Several treatment modalities have been recommended for KCOT lesions, but special consideration should be given toward decompression of KCOTs, followed by enucleation and curettage, especially in children with or without NBCCS (Figure 37-4). Recurrence rates reported in the literature range from 0% in cases treated with en block resection to 56% in those treated with simple enucleation.[20] The choice of treatment, especially in children, should take into consideration not only the size and location or degree of soft tissue involvement but also the age of the patient at diagnosis as well as the specific histologic variant. The goal of therapy is to choose the treatment modality that carries the lowest risk for recurrence and the least morbidity while ensuring eradication of the lesion. Recent studies have found certain positive prognostic changes in the KCOT after treatment with decompression, including thickening of the cystic wall, inhibition of interleukin-1a (IL-1a), epithelial dedifferentiation, loss of cytokeratin-10 production, and transformation from a parakeratotic to an orthokeratotic type. These changes may be responsible for the less aggressive behavior and decreased recurrence seen after employing such a treatment strategy. After the decompression procedure, allowing time for these changes to occur as well as shrinkage of the lesion radiographically (months to years), enucleation and curettage should always be performed because remnants of the epithelial lining, even with complete resolution radiographically, are almost always present and may contribute to recurrence. The need for patient compliance and cooperation with close follow-up, if decompression is used as the treatment modality, is imperative, and parental involvement is essential for the pediatric patient. Long-term follow-up is recommended because recurrences have been reported as late as 10 years after initial successful management of KCOTs.[1,8,21–34]



FIGURE 37-4. Decompression tube placed in a large KCOT of the right mandible.

Mesenchymal Odontogenic Tumors

Odontogenic myxoma is a rare mesenchymal origin tumor of the jaws, but it is more commonly encountered in childhood, as early as 10 years of age. There is no gender or jaw predilection. Histologically, this tumor resembles the dental papilla or follicular connective tissue and is believed to be derived from the periodontal ligament or pulpal tissue. Bony expansion that causes facial asymmetry, or merely an incidental finding on radiographic examination, warrants further investigation in order to diagnose this particular tumor (Figure 37-5). The myxoma is composed of a bland, relatively acellular, myxomatous connective tissue and does not produce or contain calcified material. The residual bony islands in the jaws are responsible for the classic radiographic appearance of a "soap bubble" or "honeycomb" pattern, but the lesions are always radiolucent, not mixed in nature. Myxomas by themselves are not aggressive per se, but the production of apoptotic proteins in addition to the lack of capsule and the tumor consistency may result in recurrence or persistence if adequate resection with complete eradication is not achieved surgically. Clinical challenges result when the diagnosis of myxoma is provided via biopsies of developing dental pulp or hyperplastic follicular tissue because histologic differentia-

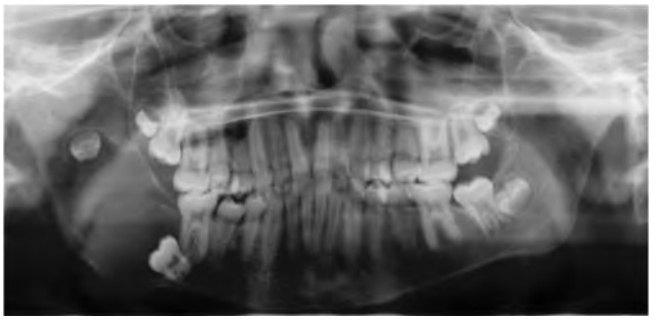


FIGURE 37-5. Odontogenic myxoma in the right mandible of a 11-year-old boy with displacement of the second and third molars.

tion is, therefore, not possible. Clinical and radiographic correlations are essential in order to prevent inappropriate management of these patients.[8,11,12,17,35–37]

Cementoblastoma or *true cementoma* is a rare benign neoplasm occasionally encountered in children or young adults. There is a predilection for the molar regions of the mandible, but no gender difference exists. Cementoblastomas are intimately related to the roots and may even replace the roots of the teeth radiographically. Cortical expansion may be present, and patients often report intermittent pain associated with these lesions. Classically, radiographs will reveal an opaque lesion surrounding and replacing the root(s) of the involved teeth. The presence of a radiolucent rim representing the periodontal ligament space, and potentially the advancing front of the lesion, along with the radiographic appearance described previously are pathognomonic for this entity. Unfortunately, removal of the lesion typically necessitates sacrificing the involved tooth.

Similarly, *cementifying fibroma* (or *cemento-ossifying fibroma [COF])* occurs most often in the premolar-molar region in the mandible and may be histologically and radiographically indistinguishable from fibrous dysplasia. Typically, COF has been described radiographically as a "disease in bone" such that a margin may be present between the lesion and the normal surrounding bone, whereas fibrous dysplasia is a "disease of bone" radiographically with indistinct radiographic borders with adjacent normal bone. The COF may demonstrate rapid growth and cause bony expansion. It generally affects young individuals younger than 15 years of age. Other terms used often for these lesions include *COF*, *ossifying fibroma (juvenile)*, and *psammomatoid ossifying fibroma*. The histologic examination will reveal cementum or bonelike substance and fibrous connective tissue with no findings indicative of aggressive behavior. Treatment is, therefore, based upon the clinical behavior of the lesion, and not the radiographic or histologic appearance. Clinical differentiation from fibrous dysplasia is important and, as mentioned, is based mainly upon the fact that COF is well demarcated and can be easily separated from the surrounding normal bone as opposed to fibrous dysplasia. Enucleation and curettage is the treatment of choice for the COF, and these lesions have no recurrence potential.

The term *cementoma* refers to a variety of odontogenic lesions, all of which are derived from the periodontal ligament tissue and occur more commonly in the adult patient. The most common lesion in this category is periapical cemental dysplasia. In these cases, the lesions may begin as periapical radiolucencies that can progress to radiopaque or mixed lesions and may involve multiple vital teeth; no treatment is usually required.[12,17,38]

Mixed Odontogenic Tumors

Ameloblastic fibroma is a benign odontogenic neoplasm that occurs more commonly than ameloblastoma in childhood and is composed of both epithelial and mesenchymal tissues. Similar to ameloblastoma, it may be associated with a tooth and involves the mandible more often that the maxilla, although the location of occurrence favors the parasymphysis region. Radiographically, the ameloblastic fibroma may present as a unilocular or multilocular radiolucency with sclerotic borders, without evidence of root resorption or bone destruction. Histologically, the lesion reveals fibromyxoid connective tissue with interposed strands, or "cords," of odontogenic epithelium. Enucleation and curettage is adequate treatment because these neoplasms have a relatively low recurrence potential (≤20%). When calcifications or toothlike structures, representing a mineralized component similar to odontoma, are present in the lesion and cause a radiographic appearance of a mixed radiolucent-radiopaque lesion, the lesion is referred to as an *ameloblastic fibro-odontoma*. The treatment involves enucleation and curettage similar to ameloblastic fibroma, and some data have demonstrated that these specific lesions are hamartomatous in nature, essentially preceding the development of complex odontomas.[12,17,38,39]

Odontoma is the most common odontogenic neoplasm in children and is typically found incidentally on radiographic surveillance or upon investigation of disturbance of tooth eruption. The odontoma is mixed in origin because it contains enamel, dentin, and pulpal tissue elements. The specific organization of these elements may result in the formation of either a lesion that appears like a tooth (compound odontoma) or a lesion with a haphazard arrangement of these toothlike structures (complex odontoma). The specific histologic structure of the lesion will result in the radiographic appearance, which is usually representative of the odontoma subtype (Figures 37-6 and 37-7). Tooth displacement by the

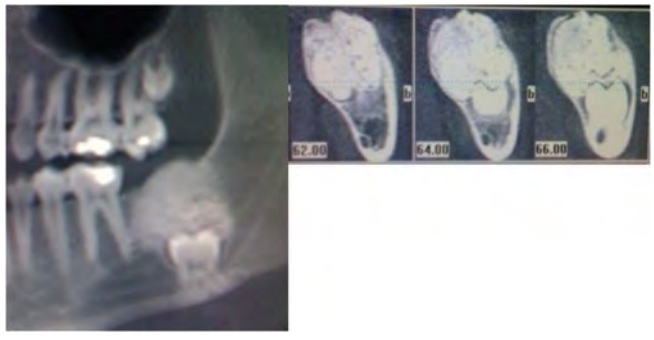


FIGURE 37-6. Complex odontoma of the left mandible with displacement of the second molar.

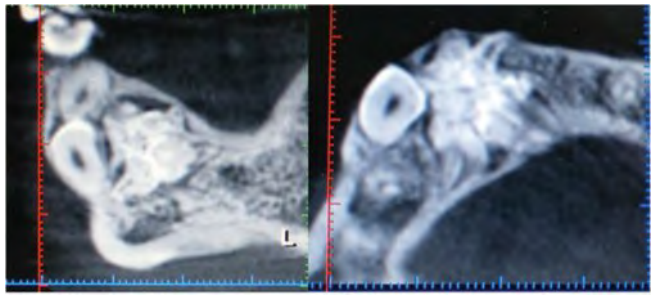


FIGURE 37-7. Compound odontoma of the anterior mandible.

SECTION 4

odontoma or the presence of an odontoma in place of a missing tooth is the common clinical presentation. Odontomas usually do not cause bony expansion or root resorption. "Extraction" or enucleation of the odontoma is the treatment of choice and is easily performed because these lesions are well encapsulated and can be easily separated from the surrounding bone.[12,17,38,40,41]

NONODONTOGENIC PATHOLOGY

Benign Mesenchymal Tumors

The most common benign mesenchymal lesions encountered in the pediatric patient population include giant cell lesions, fibro-osseous lesions, and the desmoplastic fibroma. In the giant cell lesion group, three entities are usually included: *the central giant cell granuloma (CGCG)*, *the brown tumor of hyperparathyroidism*, and the *giant cell tumor*. The latter is not common in children and is not discussed in this chapter. Among the fibro-osseous lesions, those most commonly encountered in the pediatric population are *fibrous dysplasia*, *cherubism*, *ossifying fibroma juvenile (aggressive)*, and *osteoblastoma*. Ossifying fibroma, also referred to as *cementifying fibroma* or *COF*, has been discussed in this chapter. Odontogenic myxoma is included under benign mesenchymal tumors by some authors but has also been discussed in this chapter under mesenchymal origin odontogenic tumors.

Giant Cell Lesions

CGCG occurs almost exclusively in the jaws, predominantly affects children and young adults, and female more often than male patients. Clinically, there is a painless expansion involving the anterior or premolar regions mainly in the mandible (Figure 37-8). On radiographic examination, the lesion is usually mutliloculated, is well-demarcated with scalloping borders, and may cause tooth or root displacement. Classically, it is described as the principal lesion that crosses the midline of the jaws. In some cases, CGCG may behave more aggressively with pain and rapid growth, root resorption, cortical perforation, and a higher recurrence potential. On histologic examination, a proliferation of fibroblasts, with various amounts of collagen, create a uniform connective tissue stroma with multinucleated giant cells dispersed throughout, as seen typically with all giant cell lesions. Several treatment modalities have been proposed for the CGCG lesion. Traditionally, enucleation and curettage has been the preferred treatment method, whereas resection is reserved for more aggressive, or recurrent, lesions. Several authors and clinicians have utilized intralesional corticosteroid injections (with local anesthetic agents), exogenous calcitonin (subcutaneous or intranasal), and most recently, the use of interferon-alfa (2a or b) with variable success rates.[38] The use of corticosteroids is based upon the assumption that these lesions are predominantly inflammatory in nature; however, the lack of consistent successful clinical outcomes indicates that this hypothesis may not be valid. Calcitonin administration, via subcutaneous injections or nasal administration, has been employed by some and proposed primarily for multiple, recurrent, or particularly aggressive lesions. The use of calcitonin is based upon the theory that the cells of origin of the CGCG are osteoclasts, but again, the unpredictable results as well as the recurrence of some lesions treated with this approach do not fully support this theory. Most recently, the suggested hypothesis is that these lesions should be considered proliferative vascular in origin, or at least angiogenesis-dependent, and therefore, the use of interferon-alfa is clinically indicated. Excellent results have been reported with 3,000,000 units/m^2 injected subcutaneously, 48 to 72 hours after enucleation and curettage, and for a period of 6 to 8 months for large, aggressive, high-risk, or recurrent lesions.[42–56]

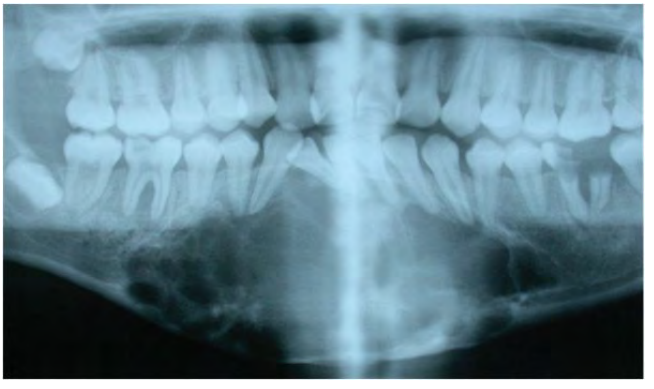


FIGURE 37-8. Central giant cell granuloma of the anterior mandible.

Brown tumors may represent a clinical manifestation of secondary, or hereditary, hyperparathyroidism in children, and the lesions are consistent with the expected osseous changes associated with the disease process (Table 37-5).

TABLE 37-5. **Types of Hyperparathyroidism Etiology and Associated Findings**

Primary	Secondary	Tertiary
Etiology		
Parathyroid adenoma (90%) MEN Hyperplasia (3%) Parathyroid carcinoma (3%)	Response of parathyroid glands to hypocalcemia and/or hypesphosphatemia usually due to renal failure, malabsorption or vitamin D deficiency	Owing to longstanding secondary hyperparathyroidism. Parathyroid glands become autonomously functioning = continuous production of PTH even without the presence of a stimuli
Symptoms		
Asymptomatic (rare and mostly primary) Fatigue, depression, constipation, body aches, decreased appetite, nausea, vomiting, polyuria, polydipsia, kidney stones, osteoporosis, arrhythmias, fibroblastic/giant cell tumors		

MEN = multiple endocrine neoplasia; PTH = parathyroid hormone.

Bone demineralization and fibrous replacement occurs in long bones. In the jaws, these appear as multiple radiolucent lesions that histologically are identical to CGCG lesions. Jaw lesions that show typical radiographic findings such as loss of the lamina dura or the presence of pulpal calcifications may be among the first indicators that further laboratory studies (e.g., calcium, phosphorus, parathyroid hormone [PTH]) are required for definitive diagnosis of the disease process. Management of the associated underlying disease process should result in resolution of jaw lesions, but occasionally enucleation and curettage, similar to the CGCG, may be required.[11,50,57–60]

Fibro-osseous Lesions

Fibrous dysplasia, a disease of bone, is a condition most commonly encountered up to and through the teen years, with a tendency for stabilization at puberty and slow growth, or regression, thereafter. Fibrous dysplasia results from the replacement of normal medullary bone by abnormal fibrous connective tissue and new bone formation. The clinical forms of fibrous dysplasia include monostotic, polyostotic, McCune-Albright syndrome, and Jaffe-Lichenstein syndrome (with no bone involvement) (Table 37-6). The maxilla is most commonly involved, and in the polyostotic form, the ribs, femur and tibia may also be affected. There is a slow painless unilateral expansion that radiographically appears as a diffuse "ground glass" or "peau d'orange" (orange peel) opacity of the involved bones, usually confined to the craniofacial skeleton. This radiographic description, however, is not pathognomonic because lesions may appear as unilocular or multilocular radiolucencies, especially when the long bones are involved (Figure 37-9). Histologic examination reveals a cellular fibrous connective tissue stroma with irregularly shaped trabeculae of immature bone in a relatively constant

TABLE 37-6. **Clinical Forms of Fibrous Dysplasia and Characteristics**

Monostotic FD	Polyostotic FD
One bone involvement	Two or more bones
Juvenile (Aggressive) Up to and through teens Stabilizes at puberty Maxilla > mandible Equal gender distribution	*Craniofacial* Bone involvement ***only*** (maxilla, zygoma, sphenoid, orbital floor)
Adult Reflection of insidious, asymptomatic nature of FD Usually present early in life, but discovered later owing to lack of symptoms	*McCune-Albright Syndrome* Hormonally regulated Bone involvement (all bones), melanotic skin pigmentations, endocrine abnormalities
	Jaffe-Lichtenstein Syndrome Melanotic skin pigmentations ***only*** Bone involvement = ***none***

FD = fibrous dysplasia.

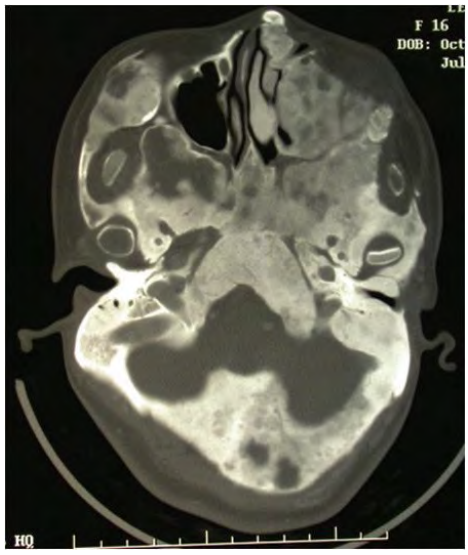


FIGURE 37-9. Computer tomography imaging (axial cuts) of fibrous dysplasia involving the craniofacial skeleton (McCune-Albright syndrome).

ratio. Classically, the term "Chinese characters" has been used to describe the irregular immature woven bone that appears to arise directly within the fibrous stroma. Characteristically, there are no inflammatory cells or osteoclasts, and only a few osteoblasts may be found. Prominent uniformly distributed capillaries are present in the connective tissue matrix. Normal laboratory values are expected for serum calcium, phosphorus, and alkaline phosphatase in the monostotic and polyostotic forms of fibrous dysplasia.

Treatment of fibrous dysplasia may include surgical recontouring of the involved bones in the facial skeleton for cosmetic reasons. It is best to delay this surgery until after cessation of the growth spurt, or after puberty, unless the condition causes such a severe deformity that earlier intervention is required for psychosocial reasons. There is typically a 25% failure of recontouring in cases of fibrous dysplasia treated too early, and in fact, the surgery itself may induce exacerbation of the disease process with overgrowth and/or extension beyond the involved areas. Low-dose radiation has been used in the past for treatment of polyostotic disease; however, there is a suggested association of radiation with malignant transformation of the lesion. In general, malignant transformation is a rare complication reported in 1% of fibrous dysplasia cases and, more commonly, in patients with the polyostotic form of the disease. Osteosarcomas may rarely occur in these patients, possibly years or decades after the initial diagnosis of fibrous dysplasia, and these malignant tumors require aggressive treatment and have a poor prognosis.[8,12,50]

McCune-Albright syndrome is a triad that includes polyostotic fibrous dysplasia, endocrine dysfunction (precocious puberty), and cutaneous hyperpigmentations (café au lait spots). It is a less common variant of polyostotic fibrous dysplasia representing only 1% to 3% of the cases, but it tends to

occur up to and through the teen years. A mutation in the gene for the alpha subunit of Gs (a G protein that stimulates cyclic adenosine monophosphate [cAMP] formation) is found in this syndrome. This results in substitution of arginine (at position 201) by either cysteine or histidine. These mutations lead to increased activation of adenylyl cyclase and elevation of cAMP. High concentrations of cAMP lead to increased proliferation and inappropriate differentiation of the mutated cells. This genetic alteration may ultimately affect the proliferation and differentiation of fibroblasts that form the disorganized immature fibrotic matrix. The lesions are hormonally regulated and undergo alternating phases of intense activity and quiescent asymptomatic periods. During active phases, patients may report throbbing pain or discomfort, while clinical edema is evident, and the lesions appear "hot" on radionuclide bone scans. Unlike other forms of fibrous dysplasia, in McCune-Albright syndrome, laboratory values demonstrate elevated serum alkaline phosphatase and urinary hydroxyproline, indicative of intense bone metabolic hyperactivity.

The use of bisphosphonate medications, specifically pamidronate, has been recently employed in the management of polyostotic fibrous dysplasia and McCune-Albright syndrome in both children and adults. Pamidronate inhibits osteoclastic function and has shown some promising therapeutic results with a significant decrease in bone pain and incidence of bony fractures from the disease. Furthermore, laboratory metabolic indices of cessation of bone turnover such as a decrease in serum alkaline phosphatase and urinary collagen type I *N*-telopeptide (elevated before treatment) have been demonstrated clinically. Although no significant adverse side effects were reported, clear evidence that the use of pamidronate has a positive effect on the dysplastic lesions is lacking, and further research studies are warranted.[5,11,12,50,61–69]

A benign hereditary condition of the jaws usually found in children younger than 5 years of age is *cherubism*, which occurs as an autosomal dominant disorder. There is a 2:1 male predominance with 100% genetic penetrance. The condition is self-limiting and usually stabilizes around the time of puberty. The classic presentation is bilateral painless expansion of the posterior regions of mandible, including the alveolar process, which occurs more often than in the maxilla. When the maxilla is involved, the classic "cherubic (angelic) face" is evident with increased scleral show between the inferior limbus of the iris and superior positioning of the globes, with possible dystopia, from involvement of the orbital floor. The process may involve any one site of the maxilla or mandible, although changes in all four quadrants of the jaws can be found as well. Radiographic examination will reveal multiple well-defined multilocular radiolucencies and often with displacement of the inferior alveolar canal. Additional radiographic findings include involvement of the coronoid processes, while the condyles are often spared; the teeth may appear to be "floating" in cystlike spaces. Histologically, a vascular connective tissue stroma with multinucleated giant cells can resemble the CGCG lesion. The symmetrical presentation as well as the radiographic findings will assist with the diagnosis. No treatment is required for this condition because spontaneous regression generally begins in puberty, and resolution is often achieved by age 30. In rare cases of severe deformity and functional impartment, curettage of these lesions and bony recontouring may be required (Figure 37-10).[70–73]



FIGURE 37-10. Lateral ceph film of a child with cherubism and bimaxillary involvement.

Osteoblastomas are uncommon lesions of the bone that occur in the posterior mandible or in the maxilla during the patient's twenties. Other common sites include the vertebra, skull, sinonasal tract, and temporal bone. The etiology remains unknown, but osteoblastomas are considered benign neoplasms that demonstrate a 2:1 male-to-female predilection. Clinical characteristics include localized swelling, bony expansion, and pain. Radiographically, a mixed lucent and opaque pattern exists that may be predominantly lucent or predominantly opaque (sclerotic) and, occasionally, may appear as a sun-ray pattern. These characteristics, together with the histologic findings of occasional hyperchromatic osteoblasts and irregular osteoid with variable degrees of maturation, may lead to a misdiagnosis of osteosarcoma. The lack of cytologic atypia, abnormal mitotic figures, and a heterogeneous pattern that are present in osteosarcoma will assist in establishing the appropriate diagnosis. These lesions are adequately treated with enucleation and curettage or complete local excision when found to infiltrate surrounding tissues. Recurrences, although rare, have been reported, as well as rare cases of malignant transformation to osteosarcoma.

The o*steoid osteoma* is considered a smaller version of an osteoblastoma, although certain differences make these lesions clinically distinct. Osteoblastomas are larger lesions, typically over 1.5 cm, and the pain experienced does not follow a circadian rhythm (e.g., nocturnal predilection) nor is the pain relieved with nonsteroidal anti-inflammatory medications or aspirin. Treatment is the same as for osteoblastoma and includes enucleation and curettage.[8,50,74]

Desmoplastic fibroma is a rare benign, but locally aggressive, lesion of bone that involves the long bones and pelvis and may occasionally be found in the mandibular ramus. This lesion is considered the central, or intraosseous, counterpart of fibromatosis that arises in young patients, with a mean age of 14 years. The origin of these tumors is not clear. Progressive, asymptomatic growth eventually results in swelling of the jaw. Radiographic examination is not helpful in identification of this lesion because a variety of presentations may be found. Desmoplastic fibromas may appear as unilocular or multilocular radiolucencies, with ill-defined, or well-defined, borders and can cause cortical perforation or root resorption. Histologically, there is an even distribution of benign-appearing fibroblasts in a collagenous stroma. No cytologic atypia or mitotic figures are seen, thereby confirming the benign nature of this neoplasm. The treatment of choice is en bloc resection, and teeth in the area must be sacrificed in order for the lesion to be completely eradicated. High recurrence rates, up to 25%, have been reported with simple curettage, and there appears to be a correlation between recurrence and tumor cellularity as well as inadequate initial surgical removal.[12,50,75]

Hematopoietic Reticuloendothelial Tumors

The hematopoietic reticuloendothelial malignancies that involve the head and neck in children discussed here include Burkitt's lymphoma (BL), Langerhans' cell histiocytosis (LCH), Hodgkin's disease, and non-Hodgkin's lymphoma. The latter two lesions combined account for 50% of all pediatric malignancies with an equal frequency. A discussion of leukemias that are extremely common in children is beyond the scope of this chapter, and extensive available literature and textbooks are devoted to these pediatric blood cell malignancies.

BL is an undifferentiated (non-Hodgkin's) lymphoma, first described in 1958 and typically classified into two forms, endemic and nonendemic, that frequently results in extranodal involvement. The *endemic (African)* form is associated with the Epstein-Barr virus and affects children in Africa. The *nonendemic* BL has no clearly established association with the virus and is also found in other parts of the world. The endemic form involves the jaws more frequently, with the maxilla affected twice as often as the mandible, although a bimaxillary presentation is not uncommon. The patient may present with swelling, mild pain, and premature loss of teeth with no associated neurologic findings. When the maxillary sinus is involved, proptosis of the globe may be evident. On radiographic examination, a nonspecific radiolucency without distinct cortical outlines and loss of lamina dura around the teeth will be seen. Diagnosis is made by histologic examination of lesional tissue that will reveal the classically described "starry sky" appearance due to numerous small round cells in a background fibrous connective tissue stroma. Aggressive chemotherapy, with high doses of alkylating agents, will lead to remission in approximately 90% of the cases. Surgical debulking to reduce tumor burden has been used, mainly in abdominal involvement, but the benefit of such an approach remains controversial. In addition, surgical complications may impact on the timely initiation of chemotherapy with devastating results. At present, surgery for head and neck disease is limited to biopsy for diagnostic purposes. If relapse occurs after adequate treatment, the tumor generally will respond well to additional chemotherapy, whereas untreated disease has a very poor prognosis.[8,11,76–82]

LCH is a very common neoplastic disorder in children and may present in one of the following three forms: eosinophilic granuloma, generalized or chronic disseminated histiocytosis (previously known as "Hand-Schüller-Christian disease"), and Letterer-Siwe (acute disseminated) disease. Eosinophilic granuloma represents an isolated bone disease and has the best response to treatment. The chronic disseminated form involves punched-out bone lesions, diabetes insipidus, and exophthalmos. The most aggressive acute disseminated form is highly lethal and is characterized by multiorgan involvement. LCH is a proliferation of cells exhibiting phenotypic characteristics of Langerhans' cells. The similar microscopic appearance of all three forms provides the rationale for grouping these lesions together, despite the diverse clinical presentation between them.

All forms of LCH have their onset early in childhood (peak between 1 and 4 yr), and often, symptoms from involvement of the maxillofacial region warrant further workup and diagnostic testing. Lytic lesions in the jaws or other bones are found commonly and appear as "punched-out" radiolucencies. In the tooth-bearing regions of the jaws, the involved teeth give the characteristic appearance of "floating in empty space." Definitive diagnosis of the disease process via biopsy, as well as a thorough investigation for the extent of involvement of the rest of the skeleton and other organs, is crucial. Laboratory evaluation should include a complete blood count to investigate for bone marrow involvement, and electrolytes, urinalysis, urine and serum osmolality, and bone marrow biopsy should be considered when indicated.

Prognosis and response to treatment are mainly dependent on the organs involved and their functional status. Chemotherapy with alkylating agents, folate antagonists, and steroids alone or in combination usually has a good response. Localized lesions, however, may respond well to radiotherapy, but this form of therapy is usually avoided owing to the devastating impact of radiation on growth in children. Eosinophilic granuloma lesions are treated with enucleation or intralesional steroids and have a good prognosis. Recurrences of LCH are encountered commonly and remain difficult treatment challenges. New chemotherapeutic agents are continuously introduced, and good responses are been reported with bone marrow transplantation for refractory cases.[8,11,83–88]

Approximately 80% to 90% of *Hodgkin's disease* cases involve the neck, most commonly the submandibular (90%) and, less often, the supraclavicular (10%) lymph nodes.

Adolescent boys are affected more often than girls. The lymph nodes involved appear as firm, enlarged, rubbery masses accompanied by constitutional symptoms such as night sweats and weight loss. *Non-Hodgkin's lymphoma* more commonly affects extranodal sites and, when present in children, occurs typically at a younger age than does Hodgkin's disease. Various regions of the maxillofacial skeleton are involved including Waldeyer's ring, nasopharynx, mandible, and orbit. Cervical lymph nodes lower in the neck are involved more often than the submandibular nodes. For both groups, prognosis and outcome depend on the stage and histologic pattern, although Hodgkin's disease has an overall better prognosis. Fine-needle aspiration biopsy (FNAB) of a persistent cervical mass that does not respond to antibiotic therapy, is accompanied by constitutional symptoms, and has no clear etiology should be considered immediately. Once the diagnosis is established, an appropriate staging workup should be undertaken. Currently, computed tomography (CT) remains the gold standard for staging, whereas magnetic resonance imaging (MRI) and positron-emission tomography (PET) are useful alternatives. The Ann Arbor staging system for lymphomas and the International Prognostic Index (IPI) for each subcategory, combined, are shown to provide valuable information not only for staging purposes but also for critical decision making with regard to treatment recommendations.[89–92] Also, various chemotherapeutic regimens and radiotherapy are available and employed in the treatment of Hodgkin's disease and non-Hodgkin's lymphomas. Radiotherapy is obviously reserved for localized disease. Failures and relapses occur often years after initial treatment, and usually the new tumor responds to the same chemotherapeutic regimen as the original lesion. If relapse occurs during treatment, it is usually due to the development of drug-resistant clones and is a sign of poor prognosis.[93–98]

Neurogenic Tumors

Congenital gingival granular cell tumor (congenital epulis) is a relatively rare tumor of neural origin that appears on the anterior gingiva of newborns. The lesion generally presents as a noninflamed pendunculated mass, occurring more often in the maxillary anterior gingival regions, and it affects girls more often than boys. The cell of origin is believed to be the Schwann cell, as with the granular cell tumor, based on immunohistochemical studies (S-100 positivity and actin negativity). If the lesion does not spontaneously regress with eruption of the deciduous tooth, then surgical excision is adequate treatment because there is no recurrence potential.

Mucosal neuromas appear early in life in children with multiple endocrine neoplasia syndrome (MEN) type 3. MEN type 3 is an autosomal dominant disorder that consists of medullary carcinoma of the thyroid, pheochromocytoma of the adrenal gland, and mucosal neuromas. The neuromas are small discrete nodules and appear on the lips, tongue, buccal mucosa, larynx, or conjunctiva. Of significant clinical importance is the fact that these lesions may be the first manifestation of the MEN type 3 syndrome that can be fatal if not diagnosed early. Surgical excision of the neuromas is usually adequate treatment.

Neurofibromas in the oral cavity in children may be familial owing to neurofibromatosis (von Recklinghausen's disease of the skin) type 1 (NF1) or occur sporadically. Histologic examination reveals haphazardly arranged spindle cells that represent Schwann cells in a delicate connective tissue stroma. The plexiform neurofibroma subtype is thought to be characteristic for NF and most often is found to involve the parotid and, less commonly, the submandibular or sublingual glands. Treatment is surgical excision that can be challenging for the plexiform type because it frequently has multiple extensions. Superficial parotidectomy with sparing of the facial nerve can be used in order to accomplish tumor debulking.[11,99–103]

Melanotic neuroectodermal tumor of infancy is thought to originate from neural crest tissue and is a rare benign neoplasm found in infants younger than 6 months of age, usually occurring in the maxilla. The tumor cells produce melanin that imparts a dark (brown or dark blue/black) pigmentation to the lesion, while the overlying epithelium remains intact. Radiographically, it will appear as poorly defined radiolucency often containing multiple deciduous teeth. Histologically, the tumor demonstrates an alveolar pattern with nests of tumor cells and small amounts of connective tissue that are found between the tumor nests, and there is typically a well-defined margin around the tumor. Surgical excision, en bloc resection, and enucleation are curative treatment measures; the recurrence potential is relatively low with a reported 15% from a few case series.

Vascular Pathology

Vascular anomalies are very common in the head and neck and frequently occur in the pediatric age group. Their management can be challenging, and evidence-based decisions about treatment are complicated by an inconsistency in the nomenclature for these lesions found in the literature. In 1982, Mulliken and Glowacki[101] proposed a biologic classification for these lesions based on their clinical and histologic findings. Based upon this classification scheme, which has been supported by subsequent radiographic and biochemical studies, vascular anomalies are classified as *hemangiomas* and *vascular malformations* (VMs; Table 37-7). The *aneurysmal bone cyst* is discussed here because it is believed to originate from a VM that is usually initiated by an unrelated bone lesion, ultimately resulting in the formation of a pseudocyst.

Hemangiomas are the most common tumors in infancy, especially in premature babies weighing less than 1000 g, with girls affected three to five times more often than boys. Caucasians are more prone to the development of hemangiomas, whereas the incidence in African Americans and Asians is low (1.4% and 0.8%, respectively). Hemangiomas are proliferative lesions characterized by increased turnover

TABLE 37-7. **Classification of Vascular Anomalies**

Vascular Tumors	Vascular Malformations Based on Involved Vessels	Vascular Malformations Based on Flow Characteristics
Hemangioma Hemangioendothelioma Angiosarcoma Aneurysmal bone cyst	Capillary Venous Arterialå Lymphatic Mixed	*High Flow* 1. Arteriovenous malformations 2. Arteriovenous fistulas *Low Flow* 1. Capillary VM 2. Venous VM 3. Lymphatic VM

VM = vascular malformation.

of endothelial cells. They appear as solitary lesions in 80% of children, and 60% of the tumors are found in the cervicofacial region. They occur sporadically, although some tumors may follow an autosomal dominant inheritance pattern in familial cases. Hemangiomas may be superficial, deep, or visceral, and although the location does not alter the biologic behavior, it does affect the clinical manifestations.

Hemangiomas typically appear during the first 2 years of life, if they are not present or evident at birth, as an erythematous patch, telangiectasia, or blanched area, when they are located superficially. Deep lesions may appear bluish, or a deep purple color, whereas the visceral ones will generally not be evident on clinical examination. Classically, a proliferative phase of rapid enlargement occurs that follows and outpaces the child's body growth, which may last for 4 to 8 months before it plateaus to be consistent with body habitus growth. During this phase, the bright coloration exhibited by the lesions gives the characteristic "strawberry" appearance. Clinical appearance, however, may be misleading, and Mulliken and Young[102] emphasize that "not all strawberries are hemangiomas, and not all hemangiomas look like strawberries." By age 12 months, the involution phase starts and usually continues for the next 5 to 7 years. Roughly half of the lesions become involuted by age 5, and 70% by 7 years of age, but the process may continue until adolescence. Even with complete involution, some clinical signs of the tumor may still be evident on physical examination.

A derangement of angiogenesis characterized by an uncontrolled proliferation of vascular elements results in the formation of a hemangioma. Various tumor factors and markers of angiogenesis (e.g., fibroblastic growth factor, vascular endothelial cell growth factor, proliferating cell nuclear antigen) are increased during the rapid proliferative phase and begin to decrease with the initiation of involution. None of these factors can be identified in lesions that have become completely involuted. Histologic examination will reveal abnormal plump endothelial cells with increased mitotic activity, increased number of mast cells in the extracellular matrix, and these histopathologic findings will differ during the various stages of progression of the lesion.

Clinical examination is adequate for the diagnosis of hemangioma in 95% of the cases, and if additional information is required to study the extent of the lesion or the level of involvement of adjacent anatomy, MRI is the preferred diagnostic modality. In T_2-weighted MRI images, the lesions will have similar signal intensity to adjacent muscle tissue, whereas gadolinium enhancement will reveal a mass with a high signal intensity. Treatment is not required for lesions that involute completely unless there is scarring or excessive overlying skin remaining that may need to be removed for cosmetic reasons. Lesions that undergo only partial involution and those that may cause severe cosmetic deformities may require earlier surgical intervention. For periorbital lesions that may interfere with visual axis development and with a risk of amblyopia development, treatment should be considered in a more prompt time frame. Intralesional steroids or surgical resection has been the proposed treatment modalities based upon the size and location of the hemangioma. Intrabony lesions and deep visceral involvement may cause uncontrolled life-threatening bleeding or iron deficiency anemias owing to occult blood loss. The most common complication of hemangiomas is overlying skin ulceration that is managed with local wound care and topical or systemic antibiotics with surgical excision in recurrent cases.

The second group of vascular lesions, the *VMs*, represent an abnormal proliferation of blood vessels. Further subclassification of VMs is based either on the type of vessels involved (capillary, venous, arterial, lymphatic or combined forms) or the flow characteristics of the lesion (low- vs. high-flow VMs). High-flow VMs are arteriovenous malformations (AVMs) or arteriovenous fistulas, whereas low-flow lesions are capillary, venous, or lymphatic VMs. VMs are present at birth because they represent errors in morphogenesis but, unlike hemangiomas, may go undetected until later in life, even into the teenage years. The lesion "grows" with the child and is usually affected by hormonal changes with occasional accelerated growth during puberty (Figure 37-11). The clinical appearance and characteristics vary according to the type of VM lesion. A palpable thrill or audible bruit may be evident in high-flow lesions, whereas diascopy can help to distinguish VMs from subcutaneous or deep hemangiomas because both appear bluish on clinical examination. Diascopy is performed by pressing a glass slide over the lesion and observing for blanching of the bluish/deep purple color as blood is evacuated from the VM; in contrast, no color change is noted when diascopy is performed on a hemangioma.

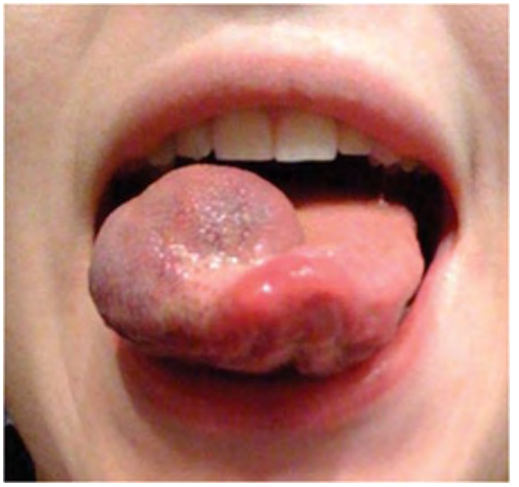


FIGURE 37-11. Low-flow vascular malformation in the tongue of a 16-year-old that demonstrated a sudden increase in size with puberty.

Angiography will provide valuable diagnostic and treatment planning information for AVMs and assist in identification of any major feeding vessel(s) that can be subsequently embolized as the sole treatment or as a treatment to attempt to shrink the lesion before resection. Intralesional sclerosing agents (e.g., sodium tetradecyl sulfate, bleomycin) can be used for VMs with good results, whereas capillary malformations may be treated with any one of several commercially available pulsed-dye lasers. Lymphatic malformations can be more challenging to treat, especially those composed mainly of small cystic spaces. When large cystic spaces are present, injection of streptococcal OK-432 into the large spaces has proved to be successful in shrinkage of these lesions. Excision may be difficult when extensive lymphatic malformations involve the neck (cystic hygromas), because vital structures may be encompassed by the mass and there may be no clear plane for surgical dissection resulting in inadvertent sacrifice of associated strutures.[104–115]

Aneurysmal bone cyst (ABC), more appropriately termed a *pseudocyst*, represents a benign lesion of bone that can involve the jaws as well as any other bone in the body. The involvement of the craniofacial skeleton is reported to be approximately 5%, with the majority (40%) of cases involving the mandible (Figure 37-12). ABC is generally considered to be a reactive process, and some underlying bone condition, such as fibrous dysplasia, CGCG, or ossifying fibroma, is usually identified as the antecedent lesion to the formation of an ABC. There is a female predilection, and pain is described in 50% of the cases. Nonpulsatile bony expansion is evident on clinical examination without a bruit or a thrill. Radiographic examination may reveal tooth displacement due to the presence of a unilocular or multilocular radiolucency that may appear to be destructive in nature. On histologic examination, sinusoidal blood spaces devoid of endothelial lining may contain a large number of multinucleated giant cells, extravasated blood cells, and hemosiderin

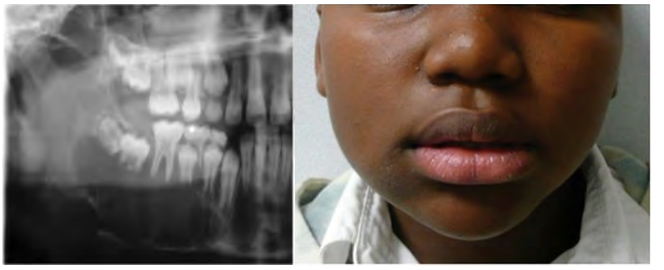


FIGURE 37-12. Aneurysmal bone cyst involving the right mandible and causing expansion and facial asymmetry in an 11-year-old boy.

breakdown products. Reactive bone formation may be noted as well. Simple curettage of the lesion may contribute to recurrence, and additional modalities such as cryotherapy have been proposed as an adjunctive therapeutic measure.[6,116]

Congenital Head and Neck Masses and Cysts

Congenital masses (some representing cystic lesions) are classically divided based upon their location in the neck as midline or lateral and include a wide variety of specific cysts and tumors (Table 37-8). The dermoid and thyroglossal duct cyst from the midline group and the branchial (cleft) cyst (BCC) from the lateral group are discussed here because they occur most commonly in the pediatric patient population.

Approximately 7% of all *dermoid cysts* occur in the head and neck region, and 20% of these are located in the cervical region. They are considered developmental in origin owing to entrapment of pluripotential cells or epithelium implantation during embryogenesis. Clinical features are dependent upon the location of the mass with regard to the relationship to the mylohyoid muscle. Those located superior to the mylohyoid muscle may cause displacement the tongue superiorly and posteriorly and appear as a midline swelling in the floor of the mouth. Those dermoid cysts located inferior to the myohyoid muscle will present externally as a midline neck mass. These cysts are painless, do not demonstrate constitutional symptoms, exhibit a slow growth pattern, generally remain small (<2 cm), and although present at birth, may go unrecognized until later in life. Palpation of the dermoid cyst

TABLE 37-8. **Congenital Neck Masses Based on Location in the Neck**

Midline Congenital Neck Masses	Lateral Congenital Neck Masses
Ranula	BCC
TDC	Lymphangioma
Dermoid cyst	Hemangioma
Epidermoid cyst	Laryngocele
Teratoma	Fibromatosis coli
Thymic cyst	

BCC = branchial cleft cyst; TDC = thyroglossal duct cyst.

reveals a soft doughy mass due to the keratin and sebum contained within the cyst. Histologic examination will reveal various secondary skin structures contained within the lumen of the cyst, which is lined by stratified squamous epithelium. If all three germ layers (ectoderm, mesoderm, and endoderm) are present in the lesion, the mass is designated as *teratoma*. Surgical excision is the treatment of choice, and recurrence is unusual.[6,117,118]

Thyroglossal duct cyst is the most common developmental cyst in the neck and accounts for two thirds of the lesions in this group. They can be present as early as age 5, and appear with equal frequency among boys and girls as a midline neck mass anywhere between the tongue and the pyramidal lobe of the thyroid gland. The etiology is related to the embryology and formation of the thyroid gland and involves failure of the thyroglossal duct to involute, which should occur between the fifth and the eighth week of gestation. The cyst is lined with respiratory epithelium that undergoes a squamous metaplasia with repeated infections. Occasionally, thyroid glandular tissue may be identified within the cyst as well. Owing to the fact that the descent of the thyroglossal duct occurs before formation of the hyoid bone, the cyst is usually located posterior to the hyoid bone but can also be found within the hyoid bone. A classic clinical finding that helps narrow the differential diagnosis is that the thyroglossal duct cyst moves during swallowing as well as tongue protrusion. Once a thyroid scan confirms that a normal thyroid gland is present, that there is no associated pathology with the gland itself, and that the cyst does not contain the majority of functional thyroid gland or parathyroid gland tissue, the thyroglossal duct cyst can be surgically removed via a trancervical approach (Sistrunk's procedure).[6,117,119,120]

BCCs are the most common congenital masses located in the lateral compartment of the neck. The intraoral counterpart, the *lymphoepithelial cyst*, is most commonly found in the floor of the mouth or the lateral tongue and contains, as the name implies, both lymphoid tissue and epithelial tissue. The BCC is thought to be the result of incomplete obliteration of a branchial arch, cleft, or pouch during embryogenesis. Five branchial arches, all of which are mesodermal in origin, are responsible for the formation of specific structures (bone, muscles, cartilage, nerves, and vessels) in the head and neck (Table 37-9). The BCC will generally follow a pathway between the derivatives of the responsible branchial arches, and this is critical information that will dictate the technique for surgical removal. Fistulas and sinus tracts associated with the branchial apparatus can also form and may be present at birth but are much less common than BCCs. The most common BCC is the one derived from the second branchial arch and is referred to as a *cervical lymphoepithelial cyst*. The other BCCs account for approximately 95% of the branchial apparatus anomalies. These appear as asymptomatic masses anterior to the sternocleidomastoid muscle, with equal frequency among males and females. On occasion, upper respiratory tract infections may cause an increase in the size of a BCC owing to the respiratory epithelial component of the cyst. Histologically, the BCCs are epithelium-lined and contain a nodular or lymphoid infiltrate often containing germinal centers. Treatment is surgical excision.

Epithelial Neoplasms

Epithelial neoplasms of the head and neck, specifically squamous cell carcinomas, are extremely rare in children. Lesions suspicious for malignancy should undergo a standard incisional biopsy following basic surgical principles. All cases of malignancy should be presented and discussed at an institutional multidisciplinary head and neck tumor board, and

TABLE 37-9. **Five Branchial Arches and the Structures Derived from Each Arch**

Arch	Artery	Crania Nerve	Muscles and Ligaments	Bone and Cartilage
First BA	None	Third division trigeminal N.(V3)	Tensor tympany m, Tenson palatini m, masticatory mm, anterior belly digastric m, mylohyoid m, anterior malleal ligament, sphenomandibular ligament	Malleus (h & n), incus (short process & body), mandible
Second BA	Stapedial A.	Facial N (VII)	Platysma m, mms of facial expression, stapedius m, posterior belly of digastrics m, buccinator m, stylohyoid m and ligament	Malleus (manubrium), incus (long process), stapes, hyoid (lesser cornu & part of body), styloid process
Third BA	Common carotid A and internal carotid A	Glossopharyngeal N (IX)	Stylopharyngeus m, superior and middle pharyngeal constrictor m	Hyoid (greater cornu and remaining body)
Fourth BA	Aorta (left), subclavian A (right)	Superior laryngeal N (X)	Inferior pharyngeal constrictor m, cricopharyngeus m, cricothyroid m	Thyroid c, cuneiform c
Fifth BA	Ductus arteriosus	Recurrent laryngeal N (X)	Intrinsic laryngeal mm (not cricothyroid m)	Trachea, cricoid c, arytenoid c, corniculate c

A = artery; c = cartilage; BA = branchial arch; h = head; m = muscle; mm = muscles; N = nerve; n = neck.

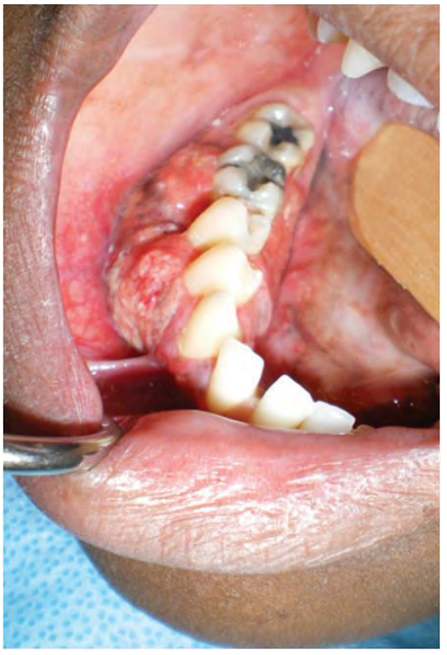


FIGURE 37-13. Squamous cell carcinoma of the mandibular gingival in a 17-year-old girl.

appropriate consultations with internal medicine colleagues and radiation oncologists should be obtained. If these malignancies are encountered in childhood, they are treated as for adults following the basic oncologic principles, with treatment consisting of resection with adequate tissue margins (1 cm linear margins, with resection to the next anatomic barrier) and neck dissection, when indicated. Appropriate reconstruction, with consideration for use of free vascularized hard and soft tissue flaps, when indicated, should take into account the child's continued growth and the future need for dental implants and prosthetic reconstruction (Figure 37-13).

Mesenchymal Neoplasms

Ewing's sarcoma (ES), first described by James Ewing in 1921, is a lethal round cell sarcoma of unknown etiology that represents approximately 6% of all bone malignancies. It has been related to primitive neuroectodermal tumor (PNET) owing to the presence of the same karyotype translocations that are evident in 90% of ES cases. One percent of the head and neck tumors involve the jaws, most commonly the mandibular ramus region. The mean age of occurrence of these tumors is approximately 11 years of age, and over 50% of the cases affect male patients. The classic presentation is the rapid onset of pain and facial swelling that may be confused with an infectious process. An elevated white blood cell count, increased erythrocyte sedimentation rate, and C-reactive protein may further support an infectious process and delay diagnosis and treatment. There is rapid bone destruction and loosening of teeth with an overlying soft tissue mass present in some cases. Radiographically, a moth-eaten destructive process of the medullary bone with cortical erosion is present, and a periosteal reaction with proliferation or "onion-skinning" may also be evident. On histologic examination, a uniform distribution of tightly packed cells is identified. The cells will stain positively for periodic acid–Schiff (PAS) stain that indicates the presence of glycogen in the tumor. The anaplastic and undifferentiated nature of this tumor is encountered in various other childhood malignancies, and clinical differentiation is essential in order to confirm the diagnosis of ES. The differential diagnosis should include lymphoma, osteosarcoma (small cell type), mesenchymal chondrosarcoma, and metastatic carcinoma. In addition, facial bone involvement may represent a metastatic process from other distant sites. Special immunohistochemical studies as well as electron microscopy may be used in difficult diagnostic dilemma cases so that appropriate treatment can be initiated without delay.

Treatment of ES requires control of the local disease as well as treatment of the metastatic process (often pulmonary) that occurs commonly with this malignancy. In fact, the majority of ES cases will present with micrometastasis at the time of initial presentation, and a comprehensive metastatic workup is essential. The various protocols developed that include intense chemotherapy with aggressive surgical resection have dramatically improved survival. In the past, only a 10% 5-year actuarial survival rate was reported, but recent developments have resulted in significant improvement, with a reported 80% 2-year and 60% 5-year disease-free survival. When chemotherapy fails, and surgery is not possible, radiotherapy has been advocated with a modest response, but with serious potential effects on the growth of the maxillofacial skeleton in the pediatric patient. Poor prognostic and treatment indicators for ES include a presentation before 10 years of age, metastatic disease present at the time of initial presentation, and lack of response to aggressive chemotherapy regimens.[8,11,121–125]

SALIVARY GLAND PATHOLOGY

Inflammatory Salivary Gland Disease

Mumps is the most common viral nonsuppurative condition that involves the parotid glands and usually occurs bilaterally. Since the introduction and routine administration of an attenuated vaccine for measles-mumps-rubella (MMR) in 1967, the yearly incidence of mumps in the United States has decreased from 76 to 2 cases per 100,000. The usual cause is an influenza/parainfluenza-related paramyxovirus with epidemic outbreaks occurring in spring and winter months. Other potential viruses include coxsakievirus A and B, Ebstein-Barr virus, influenza and parainfluenza, as well as human immunodeficiency virus (HIV). Fever, chills, headache, and preauricular pain occur 1 to 2 days before the appearance of unilateral or bilateral swelling of the parotid glands that may last between 5 and 10 days. Supportive therapy and hydration without excessive stimulation of the

salivary glands are usually adequate because spontaneous resolution occurs in the majority of uncomplicated cases.

Chronic, recurrent bacterial infections are usually of multifactorial etiology and may involve a sequence of events that make the glands vulnerable to recurrent infections due to fibrosis and microcyst formation. In children, chronic recurrent juvenile (parotid) sialoadenitis is caused either by a congenital abnormality of Stensen's duct (enlarged with a poor punctual seal) or a problem with the gland itself that may have baseline poor function due to repeated episodes of viral parotitis. Long-term antibiotic use with systemic steroids have anecdotally been reported to be successful treatments of this condition; however, sialography, which shows a "snowstorm" pattern of sialectasis; is not only diagnostic but also frequently curative. In addition, parasitic infections or tuberculous mycobacterial or nontuberculous mycobacteral diseases can be found in young or immunocompromised children and may mimic salivary gland tumors, and clinical differentiation is warranted.[126–139]

Cystic Conditions of the Salivary Glands

The most common "cystic" condition of the minor salivary glands in children is the *mucocele,* which represents a mucous extravasation phenomenon and involves the lower lip labial minor salivary glands. The etiology of mucocele formation is usually trauma to the glands from accidental lip biting and trauma to the underlying minor salivary gland tissue (Figure 37-14). The escape of mucin into the submucosal tissues triggers an inflammatory response by neutrophils and macrophages. The granulation tissue that forms creates a capsule around the free mucin, whereas the injured glands undergo associated inflammatory changes. With repeated injuries, the capsule ruptures and the mucocele regresses, but maintains the potential to reform after the mucosa heals. Over time, scarring and fibrosis may occur and the contributing glands may undergo atrophy and form a fibroma in the previous location of the mucocele. The treatment of a mucocele, or mucous-retention cyst, is surgical removal along with the contributing minor salivary glands to prevent recurrence at the same site. "Recurrent" cases, with an incidence of 15% to 30%, are usually due to incomplete initial removal of the lesion and associated minor salivary glands or repeated trauma. A carbon dioxide (CO_2) laser for treatment of the surgical bed (minor salivary glands) with expected healing by secondary intention has been used with some success for recurrent mucocele lesions.

Ranulas, common in children, are mucous extravasation phenomena (mucoceles) of the floor of the mouth associated with the sublingual gland. Trauma to the sublingual gland or dysgenetic development of the sublingual ductal system appears to be the most accepted causative theories for this condition, although there seems to be an association with sarcoidosis ("sarcoid ranula"). The accumulation of saliva under the thin mucosa of the floor of the mouth causes the pathognomonic appearance of swelling under the tongue or "frog's belly" (*rana* = frog) (Figure 37-15). The mylohyoid muscle distinguishes ranulas that are contained above the muscle in the floor of the mouth from those that extend through the mylohyoid muscle inferiorly into the neck as "plunging ranulas" that may compromise the airway. CT or MRI will provide the necessary information for accurate diagnosis and treatment planning in cases of plunging ranulas because other neck masses or cysts must be ruled out. Marsupialization has been shown to be successful as initial treatment of ranulas contained in the oral cavity; however, definitive treatment (either primary or for recurrent lesions) is surgical removal of the sublingual gland, along with the ranula, with protection of the lingual nerve and Wharton's duct.[133,135,136,140–142]

Salivary Gland Neoplasms

Salivary gland tumors are rare in the pediatric population, and additional details regarding the pleomorphic adenoma



FIGURE 37-14. Mucocele of the lower lip of a 9-year-old boy.



FIGURE 37-15. Ranula in an 11-year-old boy.

and mucoepidermoid carcinoma can be found in Chapter 35. The diagnostic methods used in the evaluation of salivary gland tumors in children is the same as in adults. FNAB, CT, and MRI are useful studies for establishing an appropriate diagnosis.[143–145]

Benign Salivary Gland Tumors

Pleomorphic adenoma (PA) is the most common benign salivary gland tumor in children. It originates either from dual proliferation of cells with ductal or myoepithelial features or from proliferation of a single cell with the potential to differentiate into either cell type. The myoepithelial features are responsible for determining the composition and histologic appearance of these tumors. Virtually all PAs of the major glands occur in the parotid gland. The most common site for minor salivary gland PA is the palate. Surgical treatment is the same as in adults, including wide local excision to prevent recurrence due to unpredictable extensions of the PA capsule peripherally. The periosteum serves as an excellent anatomic barrier if not involved; otherwise excision of the periosteum and underlying bone excision is recommended.[133,136,146–155]

Malignant Salivary Gland Tumors

Mucoepidermoid carcinoma (MECA) is the most common malignant salivary gland tumor in children for both minor and major salivary gland neoplasms. MECAs represent 16% of all salivary gland tumors and 51% of the malignant neoplasms. The intraosseous variant is believed to originate from embryonically entrapped salivary gland elements, may occasionally be found in children, and appears as a well-defined radiolucency of the mandible. On the basis of histologic appearance and degree of differentiation, this tumor is classified by grade. A low-grade or well-differentiated MECA is composed largely of mucous-secreting cells, often forming large glandular spaces; a high-grade or poorly differentiated MECA is characterized by a preponderance of squamous cells with a scant mucous component. An intermediate-grade MECA has been described as well, with a mixture of both mucous and epidermoid elements. Treatment is based upon the grade of differentiation and location of the tumor and varies from wide local excision of low-grade lesions to a more aggressive surgical resection with margins for high-grade lesions (Figure 37-16). Low- and intermediate-grade tumors in childhood have a better prognosis and response to treatment than high-grade lesions.[156–158]



FIGURE 37-16. Mucoepidermoid carcinoma of the right buccal mucosa in a 16-year-old girl.

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CHAPTER

Principles of Management of Maxillofacial Infections

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The incidence, severity, morbidity, and mortality of odontogenic infections have declined dramatically over the past 60 years. In 1940, Ashbel Williams published a series of 37 cases of Ludwig's angina in which 54% of the subjects died.[1] Only 3 years later, he and Dr. Walter Guralnick published the first prospective case series in the field of head and neck infections, in which the mortality rate of Ludwig's angina was reduced to 10%.[2] This dramatic reduction in mortality from 54% to 10% was not due to the first use of penicillin in the treatment of these infections. Rather, Dr. Guralnick applied the principles of the initial establishment of airway security, followed by early and aggressive surgical drainage of all anatomic spaces affected by cellulitis or abscess. Since then, with the use of antibiotics and advanced medical supportive care, the mortality of Ludwig's angina has been further reduced to 4%.[3]

Dentistry has made great progress in the prevention and early intervention of odontogenic infections. Oral and maxillofacial surgeons, as noted previously, have made great strides in managing and preventing mortality in severe odontogenic infections. These accomplishments, however, impose upon the oral and maxillofacial surgeon the obligation to remain intellectually prepared for the always unscheduled occurrence of severe odontogenic infections by keeping fresh one's knowledge of the relevant anatomy and surgery and by remaining abreast of current developments in the microbiology and antibiotic therapy of odontogenic infections.

The late Dr. Larry Peterson, who brought the first edition of this text to fruition, articulated the principles of management of odontogenic deep fascial space infections. These are eight sequential steps that, if followed with thoroughness and good judgment, will ensure a high level of care for these increasingly uncommon, yet occasionally life-threatening infections.

These principles outline the structure of this chapter. The eight steps in the management of odontogenic infections are

1. Determine the severity of infection.
2. Evaluate host defenses.
3. Decide on the setting of care.
4. Treat surgically.
5. Support medically.
6. Choose and prescribe antibiotic therapy.
7. Administer the antibiotic properly.
8. Evaluate the patient frequently.

This chapter examines each of these principles in order and discusses and relates current knowledge to them.

STEP 1: DETERMINE THE SEVERITY OF INFECTION

Within the first few minutes of the presentation of a patient with a significant odontogenic infection, the surgeon should have accomplished the first three steps listed previously. A careful history and a brief but thorough physical examination should allow the treating surgeon to determine the anatomic location, rate of progression, and the potential for airway compromise of a given infection. The host defenses, including immune system competence and the level of systemic reserves that can be called upon by the patient to maintain homeostasis, are largely determined by history. Given this initial database, the surgeon must then decide upon the setting of care, which will have a great influence on the outcome.

The clinical presentation and relevant surgical anatomy of infections of the various deep fascial spaces of the head and neck have been well described in other texts.[4,5] The borders,

TABLE 38-1. **Borders of the Deep Spaces of the Head and Neck**

Space	Anterior	Posterior	Superior	Inferior	Superficial or Medial*	Deep or Lateral†
Buccal	Corner of mouth	Masseter m, pterygomandibular space	Maxilla, infraorbital space	Mandible tissue and skin	Subcutaneous	Buccinator m
Infraorbital	Nasal cartilages	Buccal space	Quadratus labii superioris m	Oral mucosa	Quadratus labii superioris m	Levator anguli oris m, maxilla
Submandibular	Anterior belly of digastric m	Posterior belly of digastric, stylohyoid, stylopharyngeus mm	Inferior and medial. surfaces of mandible	Digastric tendon	Platysma m, investing fascia	Mylohyoid, hyoglossus superior constrictor mm.
Submental	Inferior border of mandible	Hyoid bone	Mylohyoid m	Investing fascia	Investing fascia	Anterior bellies of digastric m†
Sublingual	Lingual surface of mandible	Submandibular space	Oral mucosa	Mylohyoid m	Muscles of tongue*	Lingual surface of mandible†
Pterygomandibular	Buccal space	Parotid gland	Lateral pterygoid m.	Inferior border of mandible	Median pterygoid muscle*	Ascending ramus of mandible†
Submasseteric	Buccal space	Parotid gland	Zygomatic arch	Inferior border of mandible	Ascending ramus of mandible*	Masseter m†
Lateral pharyngeal	Superior and middle pharyngeal constrictor mm	Carotid sheath and scalene fascia	Skull base	Hyoid bone	Pharyngeal constrictors and retropharyngeal space*	Medial pterygoid m†
Retropharyngeal	Superior. and middle pharyngeal constrictor mm.	Alar fascia	Skull base	Fusion of alar and prevertebral fasciae at C6–T4	—	Carotid sheath and lateral pharyngeal space†
Pretracheal	Sternothyroid-thyrohyoid fascia	Retropharyngeal space	Thyroid cartilage	Superior mediastinum	Sternothyroid-thyrohyoid fascia	Visceral fascia over trachea and thyroid gland

m = muscle; mm = muscles.
*Medial border.
†Lateral border.
Adapted from Flynn TR. Anatomy of oral and maxillofacial infections. In Topazian RG, Goldberg MH, Hupp JR, editors. Oral and Maxillofacial Infections. 4th ed. Philadelphia: WB Saunders; 2002; pp. 188–213.

contents, and relations of the various anatomic deep spaces that are likely to be invaded by odontogenic infections are described in Tables 38-1 and 38-2.

Three major factors must be considered in determining the severity of an infection of the head and neck: anatomic location, rate of progression, and airway compromise.

Anatomic Location

The anatomic spaces of the head and neck can be graded in severity by the level to which they threaten the airway or vital structures, such as the heart and mediastinum or the central nervous system. The buccal, infraorbital vestibular, and subperiosteal spaces can be categorized as having *low severity* because infections in these spaces do not threaten the airway or vital structures. Infections of anatomic spaces that can hinder access to the airway owing to swelling or trismus can be classified as having *moderate severity.* Such anatomic spaces include the masticator space, whose components may be considered separately as the submasseteric, pterygomandibular, and superficial and deep temporal spaces, and the perimandibular spaces (submandibular, submental, and sublingual). Infections that have *high severity* are those in which swelling can directly obstruct or deviate the airway or threaten vital structures. These anatomic spaces are the lateral pharyngeal and retropharyngeal, the danger space, and the mediastinum. Cavernous sinus thrombosis and other intracranial infection also have high severity. Table 38-3 lists the severity level of each of the various deep fascial spaces.

Rate of Progression

Upon interviewing the patient with an infection, the surgeon can appraise the rate of progression by inquiring about the onset of swelling and pain and comparing those times with the current signs and symptoms of swelling, pain, trismus, and airway compromise. In a study of 37 severe hospitalized odontogenic infections, Flynn and colleagues[6] found trismus in 73% and dysphagia in 78% of patients at presentation.

Odontogenic infections generally pass through four stages before they resolve, the characteristics of which are

TABLE 38-2. **Relations of Deep Spaces in Infections**

Space	Likely Causes	Contents	Neighboring Spaces	Approach for Incision and Drainage
Buccal	Upper bicuspids Upper molars Lower bicuspids	Parotid duct Anterior facial a and v Transverse facial a and v Buccal fat pad	Infraorbital Pterygomandibular Infratemporal	Intraoral (small) Extraoral (large)
Infraorbital	Upper cuspid	Angular a and v Infraorbital n	Buccal	Intraoral
Submandibular	Lower molars	Submandibular gland Facial a and v Lymph nodes	Sublingual Submental Lateral pharyngeal Buccal	Extraoral
Submental	Lower anteriors Fracture of symphysis	Anterior jugular v Lymph nodes	Submandibular (on either side)	Extraoral
Sublingual	Lower bicuspids Lower molars Direct trauma	Sublingual glands Wharton's ducts Lingual n Sublingual a and v	Submandibular Lateral pharyngeal Visceral (trachea and esophagus)	Intraoral Intraoral-extraoral
Pterygomandibular	Lower third molars Fracture of angle of mandible	Mandibular division of trigeminal n Inferior alveolar a and v	Buccal Lateral pharyngeal Submasseteric Deep temporal Parotid peritonsillar	Intraoral Intraoral-extraoral
Submasseteric	Lower third molars Fracture of angle of mandible	Masseteric a and v	Buccal Pterygomandibular Superficial temporal Parotid	Intraoral Intraoral-extraoral
Infratemporal and deep temporal	Upper molars	Pterygoid plexus Internal maxillary a and v Mandibular division of trigeminal n Skull base foramina	Buccal Superficial temporal Inferior petrosal sinus	Intraoral Extraoral Intraoral-extraoral
Superfical temporal	Upper molars Lower molars	Temporal fat pad Temporal branch of facial n	Buccal Deep temporal	Intraoral Extraoral Intraoral-extraoral
Lateral pharyngeal	Lower third molars Tonsillar infection in neighboring spaces	Carotid a Internal jugular v Vagus n Cervical sympathetic chain	Pterygomandibular Submandibular Sublingual Peritonsillar Retropharyngeal	Intraoral Intraoral-extraoral

a = artery; n = nerve; v = vein.
Adapted from Flynn TR. Anatomy and surgery of deep fascial space infections. In Kelly JJ, editor. Oral and Maxillofacial Surgery Knowledge Update 1994. Rosemont, IL: American Association of Oral and Maxillofacial Surgeons; 1994; pp. 79–107.

listed in Table 38-4. During the first 1 to 3 days, the swelling is soft, mildly tender, and doughy in consistency. Between days 2 and 5, the swelling becomes hard, red, and exquisitely tender. Its borders are diffuse and spreading. Between the fifth and the seventh days, the center of the cellulitis begins to soften and the underlying abscess undermines the skin or mucosa, making it compressible and shiny. The yellow color of the underlying pus may be seen through the thin epithelial layers. At this stage, the term *fluctuance* is appropriately applied. Fluctuance implies the palpation of a fluid wave by one hand as the abscess is compressed by the other hand. The final stage of odontogenic infection is *resolution*, which generally occurs after spontaneous or surgical drainage of an abscess cavity. The swelling then begins to decrease in size, redness, and tenderness. The resolving swelling may stay firm for some time, however, as the inflammatory process is involved in removing necrotic tissue and bacterial debris.

A special note should be made of an especially rapidly progressive infection called *necrotizing fasciitis*. Occasionally found in the head and neck, frequently due to odontogenic sources, necrotizing fasciitis is a rapidly spreading infection that follows the platysma muscle down the neck and onto the anterior chest wall. Diabetes and alcoholism have been shown to be significant predisposing factors, whereas medical compromise, delay in surgery, and mediastinitis

TABLE 38-3. **Severity Scores of Fascial Space Infections**

Severity Score	Anatomic Space
Low	Vestibular
	Subperiosteal
	Space of the body of the mandible
	Infraorbital
	Buccal
Moderate	Submandibular
	Submental
	Sublingual
	Pterygomandibular
	Submasseteric
	Superficial temporal
	Deep temporal (or infratemporal)
High	Lateral pharyngeal
	Retropharyngeal
	Pretracheal
	Danger space (space 4)
	Mediastinum
	Intracranial infection

are associated with increased mortality.[7] It can rapidly result in necrosis of large amounts of muscle, subcutaneous tissue, and skin, resulting in severe reconstructive defects (Figure 38-1). Similar processes may be involved in descending necrotizing infections of the neck, which frequently progress to the mediastinum. The earliest signs of necrotizing fasciitis are small vesicles and a dusky purple discoloration of the involved skin (Figure 38-2). Soon thereafter, the skin may become anesthetic. Thereafter, frank necrosis occurs.[8]

A suspicion of necrotizing fasciitis is a surgical emergency, requiring broad-spectrum antibiotics, repeated surgical drainage, antiseptic wound packing, and intensive medical supportive care, including fluids, calcium, and possibly blood transfusion.[8] Repeated surgical débridement is the rule, not the exception. Hyperbaric oxygen therapy may also be of benefit.[9]

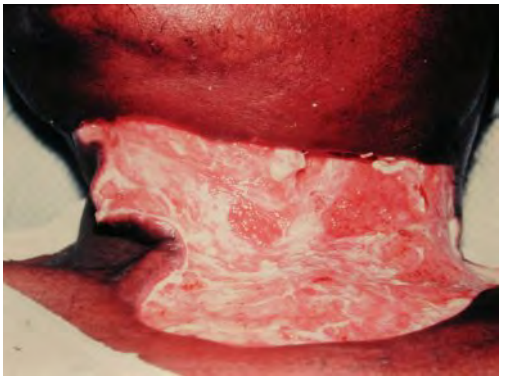


FIGURE 38-1. Necrotizing fasciitis. Large granulating skin defect extending from the inferior border of the mandible to the clavicle, 2 weeks after débridement. (Reproduced with permission from Flynn TR. Odontogenic infections. Oral Maxillofac Surg Clin North Am 1991;3:311–329.)



FIGURE 38-2. Necrotizing fasciitis. Early stage, with swelling extending from the inferior border of the mandible onto the anterior chest wall in a 7-year-old boy. The chalky material on the neck is calamine lotion that his mother used to treat the vesicles of presumed contact dermatitis due to poison ivy. (Reproduced with permission from Flynn TR. Odontogenic infections. Oral Maxillofac Surg Clin North Am 1991;3:311–329.)

TABLE 38-4. **Stages of Infection**

Characteristic	Inoculation	Cellulitis	Abscess
Duration	0–3 days	3–7 days	>5 days
Pain	Mild-moderate	Severe and generalized	Moderate-severe and localized
Size	Small	Large	Small
Localization	Diffuse	Diffuse	Circumscribed
Palpation	Soft, doughy, mildly tender	Hard, exquisitely tender	Fluctuant, tender
Appearance	Normal coloration	Reddened	Peripherally reddened
Skin quality	Normal	Thickened	Centrally undermined and shiny
Surface temperature	Slightly heated	Hot	Moderately heated
Loss of function	Minimal or none	Severe	Moderately severe
Tissue fluid	Edema	Serosanguineous, flecks of pus	Pus
Level of malaise	Mild	Severe	Moderate-severe
Degree of seriousness	Mild	Severe	Moderate-severe
Predominant bacteria	Aerobic	Mixed	Anaerobic

Adapted from Flynn TR, Piecuch JF, Topazian RG. Infections of the oral cavity. In Feigin RD, Cherry JD, editors. Textbook of Pediatric Infectious Diseases. 4th ed. Vol 1. Philadelphia: WB Saunders; 1998; pp. 134–148.

Airway Compromise

The most frequent cause of death in reported cases of odontogenic infection is airway obstruction. Therefore, the surgeon must assess current or impending airway obstruction within the first few moments of evaluating the patient with a head and neck infection.

Complete airway obstruction is, of course, a surgical emergency. In such cases, insufficient or absent air movement in spite of inspiratory efforts will be apparent. In highly skilled hands, one brief attempt at endotracheal intubation may be made, but a direct surgical approach to the airway by cricothyroidotomy or tracheotomy is more predictably successful. In such extreme circumstances, the presence of infection overlying the trachea is less important than the absence of ventilation. Therefore, infection in the region of surgical airway access is not a contraindication to an emergency cricothyroidotomy or tracheotomy.

In partial airway obstruction, abnormal breath sounds will be evident, consisting of stridor or coarse airway sounds suggestive of fluid in the upper airways. The patient may assume a special posture that straightens the airway, such as the "sniffing position," in which the head is inclined forward and the chin is elevated, as if one were sniffing a rose. Other such postures include a sitting patient with the hands or elbows on the knees and the chest inclined forward with the head thrust anterior to the shoulders (tripod position), which also straightens the airway and pulls the tongue forward and allows secretions to drool outward onto the floor or into a pan. Occasionally, a patient with a lateral pharyngeal space infection will incline the neck toward the opposite shoulder in order to position the upper airway over the laterally deviated trachea (Figure 38-3).

Trismus is an ominous sign in the patient suspected of odontogenic infection. A maximum interincisal opening that has decreased to 20 mm or less in a patient with acute pain should be considered an infection of the masticator space until proved otherwise. Infections of the pterygomandibular space are sometimes missed because trismus hinders the examiner's view of the oropharynx. Therefore, it is important for the examiner to position the patient's occlusal plane parallel to the plane of vision and to orient a light coaxial to that plane of view. Then the patient is asked to maximally open the mouth in spite of pain, and the tongue is depressed with a mirror or tongue blade. This should allow the examiner to get at least a glimpse of the position of the uvula and the condition of the anterior tonsillar pillars. The affected tonsillar pillar will usually be edematous and reddened, and it will displace the uvula to the opposite side (Figure 14-4). If the suspected site of infection is touched with the mirror or tongue blade, acute pain may be elicited, especially as compared with the opposite side. The patient's report of pain should be distinguished from the gagging that is likely to occur.



FIGURE 38-3. Left lateral pharyngeal space abscess. Note the swelling just anterior to the sternocleidomastoid muscle above the level of the hyoid bone and the deviation of the head toward the right shoulder, in an attempt to place the upper airway directly over the deviated trachea. (Reproduced with permission from Flynn TR, Piecuch JF, Topazian RG. Infections of the oral cavity. In Feigin RD, Cherry JD, editors. Textbook of Pediatric Infectious Diseases. 4th ed. Vol 1. Philadelphia: WB Saunders; 1998; pp. 134–148.)



FIGURE 38-4. Right pterygomandibular space abscess. Note the swelling of the anterior tonsillar pillar and the deviation of the edematous uvula to the opposite side. (Reproduced with permission from Flynn TR, Topazian RG. Infections of the oral cavity. In Waite D, editor. Textbook of Practical Oral and Maxillofacial Surgery. 3rd ed. Philadelphia: Lea & Febiger; 1987; pp. 273–310.)

Various clinical tests have been proposed with the aim of predicting difficult intubation. The Mallampati test has been correlated with difficult intubation by its initial proponent, as have trismus of less than 20 mm and decreased thyromental distance.[10,11] These results, however, have not been confirmed by independent examiners, although the combination of an abnormal Mallampati test and a thyromental distance of less than 5 cm has been correlated with difficult intubation in one study.[11]

In airway obstruction, the respiratory rate may be increased or decreased; yet one functional method of assessing the effectiveness of respiratory efforts readily available to the oral and maxillofacial surgeon is the pulse oximeter.[12]

An oxygen saturation of below 94% in an otherwise healthy patient is indeed an ominous sign because it indicates insufficient oxygenation of the tissues due to hypoperfusion or hypooxygenation. Given the patient with clinically apparent partial airway obstruction, an abnormally low oxygen saturation is an indication for immediate establishment of a secure airway.

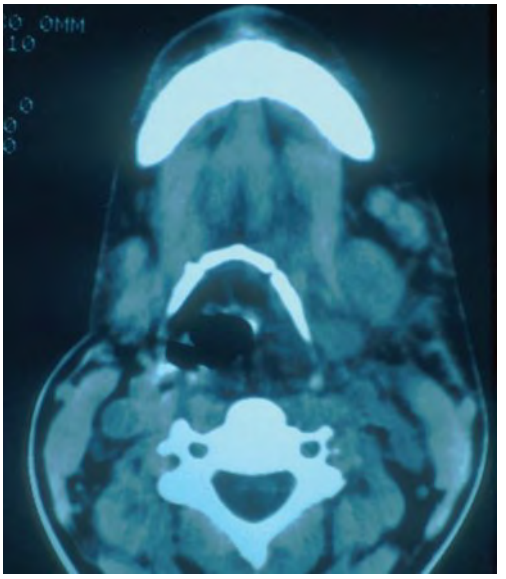


FIGURE 38-5. Axial computed tomography image at the level of the hyoid bone demonstrates a cellulitis of the left lateral pharyngeal space that is deviating the airway to the opposite side and spreading into the retropharyngeal space. (Reproduced with permission from Flynn TR. Anatomy of oral and maxillofacial infections. In Topazian RG, Goldberg MH, Hupp JR, editors. Oral and Maxillofacial Infections. 4th ed. Philadelphia: WB Saunders; 2002; pp. 188–213.)

Soft tissue radiographs of the cervical airway and chest can be quite valuable in identifying deviation of the airway laterally on a posteroanterior film or anterior displacement of the airway on a lateral view. These films can be taken fairly quickly, which can be an advantage for radiographic examination of the patient with a significant cervical swelling. During prolonged periods in the supine position, as required by the older generation of computed tomography (CT) scanners, an infected swelling may obstruct the airway. Conversely, the newer high-speed CT scanners can obtain a computerized CT examination within seconds to minutes, which, if available, would make conventional soft tissue radiographs obsolete (Figure 38-5). In a prospective study, Miller and coworkers[13] found 89% accuracy, 95% sensitivity, and 80% specificity in identifying "drainable pus" by the combined use of contrast-enhanced CT and clinical examination. By "drainable pus," the authors meant a collection of 2 mL or more of pus. The high diagnostic yield therefore of contrast-enhanced CT and clinical examination makes this combination the method of choice for evaluation of potential airway obstruction, as well as characterizing the location and quality of infections in the head and neck.[13]

STEP 2: EVALUATE HOST DEFENSES

Immune System Compromise

Table 38-5 lists the medical conditions that can interfere with proper function of the immune system, which is, of course, essential to the maintenance of host defense against infection. Diabetes is listed first because it is the most common immune-compromising disease. Diabetics have the combination of a white blood cell migration defect, which inhibits successful chemotaxis of white blood cells to the infected site from the blood stream, and a vascular defect that impairs blood flow to small vessel tissue beds, especially in end organs such as the foot. Orally, diabetics have an increased susceptibility to periodontal infections. This disease also appears to decrease host resistance to more severe odontogenic infections such as necrotizing fasciitis and deep fascial space infections.

TABLE 38-5. **Factors Associated with Immune System Compromise**

Diabetes
Steroid therapy
Organ transplants
Malignancy
Chemotherapy
Chronic renal disease
Malnutrition
Alcoholism
End-stage AIDS

AIDS = acquired immunodeficency syndrome.

The iatrogenic use of steroids has increased over recent years with the use of these medications to treat asthma, skin conditions, autoimmune diseases, cancer, and other inflammatory conditions. Corticosteroids appear to stabilize the cell membranes of immunocompetent cells, thereby decreasing the immune response. Patients with organ transplants are often treated with corticosteroids, as well as other immunosuppressive medications such as cyclosporine, tacrolimus, and azathioprine, to suppress organ rejection reactions.

It has been postulated that every patient with malignant disease has some defect of the immune system. The mechanisms of immune compromise in malignancy are variable and not well identified, but the surgeon treating the patient with ongoing cancer should assume that there is some defect of the immune system. Cancer chemotherapy directly suppresses the immune system along with rapidly dividing cancer cells. Therefore, all patients who have received cancer chemotherapy within the past year should be considered immunocompromised.

Other conditions that impair immune function include malnutrition, alcoholism, and chronic renal disease. The role of human immunodeficiency virus (HIV) infection in diminishing host resistance to odontogenic infections is somewhat unclear and paradoxical. HIV infection first and primarily damages the T cell. Conversely, most odontogenic infections are due to extracellular bacteria, which are attacked by B cells, the white blood cells that elaborate antibodies. Although HIV infection may damage B cells early in the course of the disease, its most devastating effects are seen on the T cells, which explains the increased rate of cancers and infections by intracellular pathogens in patients with acquired



FIGURE 38-6. **A,** Lateral view 6 weeks after débridement of osteomyelitis and necrotizing fasciitis with skin graft and vascularized free pectoralis muscle flap reconstruction, in a patient with previously untreated human immunodeficiency virus (HIV) infection. **B,** Intraoral view demonstrating healed resection of the left mandibular alveolar process and extraction of multiple teeth in treatment of osteomyelitis. (**A,** Reproduced with permission from Flynn TR. Antimicrobial treatment of head and neck infections. In Bagheri SC, Bell RB, Khan HA, editors. Current Therapy in Oral & Maxillofacial Surgery. Philadelphia: CV Mosby, in press.)

immunodeficiency syndrome (AIDS) and pre-AIDS. Although patients with HIV seropositivity may suffer a more intense and/or prolonged hospital course than other patients, HIV seropositivity does not seem to increase the incidence of severe odontogenic infections.[14] Figure 38-6 illustrates the postoperative condition of a man with previously untreated HIV infection complicated by necrotizing fasciitis and osteomyelitis of the mandible.

Systemic Reserve

The host response to severe infection can place a damaging physiologic load on the body. Fever can increase sensible and insensible fluid losses and caloric requirements. A prolonged fever may cause dehydration, which can, therefore, decrease cardiovascular reserves and deplete glycogen stores, shifting the body metabolism to a catabolic state. The surgeon should also be aware that elderly individuals are not able to mount high fevers, as often seen in children. Therefore, an elevated temperature at an advanced age is not only a sign of a particularly severe infection but also an omen of decreased cardiovascular and metabolic reserve owing to the demands placed on the elderly patient's physiology.[15]

In a prospective study of 37 consecutive patients admitted to the hospital with severe odontogenic infection, the statistically significant predictors of the lengh of stay, based on multivariate analysis, were indicators of the severity of infection, such as number of infected anatomic spaces, time in the operating room, and complications, such as therapeutic antibioitic failure, and the need for reoperation.[16] In other studies, number of involved spaces,[17] diabetes,[17] neck swelling, lower facial swelling, C-reactive protein greater than 100, and trismus also have been associated with an increased hospital stay.[18,19] Odontogenic infections, however, had a shorter length of hospital stay than other sources of head and infection in one study.[17] Therefore, evaluation of the anatomic extent and source of the infection, medical compromise, and possibly C-reactive protein are important in determining the severity of infection as well as in estimating the length of hospital stay.

The physiologic stress of a serious infection can disrupt previously well-established control of systemic diseases such as diabetes, hypertension, and renal disease. The increased cardiac and respiratory demands of a severe infection may deplete scarce physiologic reserves in the patient with chronic obstructive pulmonary disease or atherosclerotic heart disease, for example. Thus, an otherwise mild or moderate infection may be a significant threat to the patient with systemic disease, and the surgeon should be careful to evaluate and manage concurrent systemic diseases in conjunction with direct management of the infection.

STEP 3: DECIDE ON THE SETTING OF CARE

Table 38-6 lists the indications for hospital admission of the patient with a severe odontogenic infection. As previously stated, an elevated fever increases metabolic needs and fluid losses, which can lead to dehydration. In addition to the clinical signs of dry skin, chapped lips, loss of skin turgor, and dry mucous membranes, dehydration can be assessed in

TABLE 38-6. **Indications for Hospital Admission**

Temperature > 101°F (38.3°C)
Dehydration
Threat to the airway or vital structures
Infection in moderate or high severity anatomic spaces
Need for general anesthesia
Need for inpatient control of systemic disease

the presence of normal serum creatinine by an elevated urine specific gravity (>1.030) or an elevated blood urea nitrogen (BUN) with a normal serum creatinine, which indicates prerenal azotemia.

Infections in anatomic spaces that have a moderate or high severity (see Table 38-3) can hinder access to the airway for intubation by causing trismus, directly compress or deviate the airway by swelling, or threaten vital structures directly. Thus, an odontogenic infection involving the masticator space, the perimandibular spaces, or the deep neck spaces indicates hospital admission.

Occasionally, general anesthesia is required for patient management owing to inability to achieve adequate local anesthesia, the need to secure the airway, or the inability of the patient to cooperate, as in a young child. Sometimes, concurrent systemic disease indicates hospital admission and may even delay surgery, as in the need to reverse warfarin anticoagulation.

In deciding whether to admit the patient with a serious odontogenic infection, it is generally safer to err on the side of hospital admission. The inpatient setting affords the patient with continual professional monitoring, supportive medical care, the availability of radiologic and medical consultative services, and, most important, a team that can rapidly secure the airway should it become compromised.

STEP 4: TREAT SURGICALLY

Airway Security

The dramatic reduction in the mortality of Ludwig's angina from 54% to 10% in only 3 years, afforded by Williams and Guralnick,[2] was made possible by their changed surgical policy of immediate establishment of airway security by intubation or tracheotomy, followed by aggressive and early surgical intervention. No antibiotics were used in their patients, except sulfa drugs in some cases. In the antibiotic era, mortality has been further reduced to about 4%.[3] It is, therefore, apparent that immediate establishment of airway security and early aggressive surgical therapy are the most important intervention steps in the management of severe odontogenic infections.

Table 38-7 lists the indications for an operating room procedure. The paramount indication is, of course, to establish airway security. The involvement of moderate- or high-severity anatomic spaces generally necessitates a definitive airway management procedure, as well as surgical intervention in anatomic locations that are not amenable to profound local anesthesia. An infection that is rapidly progressing through the anatomic fascial planes, as in necrotizing fasciitis, indicates the prompt establishment of a secure airway, even if for anticipatory reasons, as well as the possible need to extend the anatomic dissection into regions that had not been contemplated preoperatively. Sometimes, general anesthesia is required for patient management reasons alone, especially in the patient who is not able to cooperate, such as a young child or a mentally handicapped individual. It is wise to maintain the endotracheal intubation postoperatively, because of the risks of continued spread of the infection and additional swelling due to the trauma of surgery.

TABLE 38-7. **When to Go to the Operating Room**

To establish airway security
Moderate to high anatomic severity
Multiple space involvement
Rapidly progressing infection
Need for general anesthesia

Successful airway management in difficult situations requires a team approach. Preoperatively, the surgeon should communicate with the anesthesiologist to establish the airway management plan. The anesthesiologist should be interested in understanding the anatomic location of the infection as well as its implications for airway management. The anesthesiologist will value the opportunity to see any effacement, displacement, or deviation of the airway as demonstrated on clinical examination and CT. The airway management plan should include the projected initial management as well as secondary procedures should the initial approach fail.

An infrequently used surgical technique that may aid in protecting the airway during intubation or tracheotomy is needle decompression. In this technique, under local anesthesia, an abscess of the pterygomandibular, lateral pharyngeal, submandibular, or sublingual space is aspirated with a large-bore needle in order to decompress the surrounding tissues. This maneuver may decrease the risk of abscess rupture through taut, distended oropharyngeal tissues during instrumentation of the airway. Additional benefits of this procedure are the redirection of pus drainage into the oral cavity or onto the skin, where it can easily be removed, and obtaining an excellent specimen for culture and sensitivity testing.

Surgical Drainage

In general, surgery for management of severe odontogenic infections is not difficult. Given a thorough knowledge of the anatomy of the deep fascial spaces of the head and neck, the surgeon should be able, by using appropriate anatomic landmarks, to use small incisions and blunt dissection without direct exposure and visualization of the entire infected anatomic space. Figure 38-7 illustrates the appropriate locations for extraoral incision placement for drainage of the various anatomic deep spaces. In addition, an intraoral vertical incision over the pterygomandibular raphe can be used to drain the pterygomandibular and submasseteric spaces as well as the anterior compartment of the lateral pharyngeal space, as illustrated in Figure 38-8. Lest the surgeon crush a vital structure within the beaks of a hemostat during blunt dissection, it is crucial to insert the instrument closed, then open it at the depth of penetration, and then withdraw the instrument in the open position. A hemostat should never be blindly closed while it is inside a surgical wound.

Another important principle of surgical incision and drainage is the need to dissect a pathway for the drain that includes the locations where pus is most likely to be found.

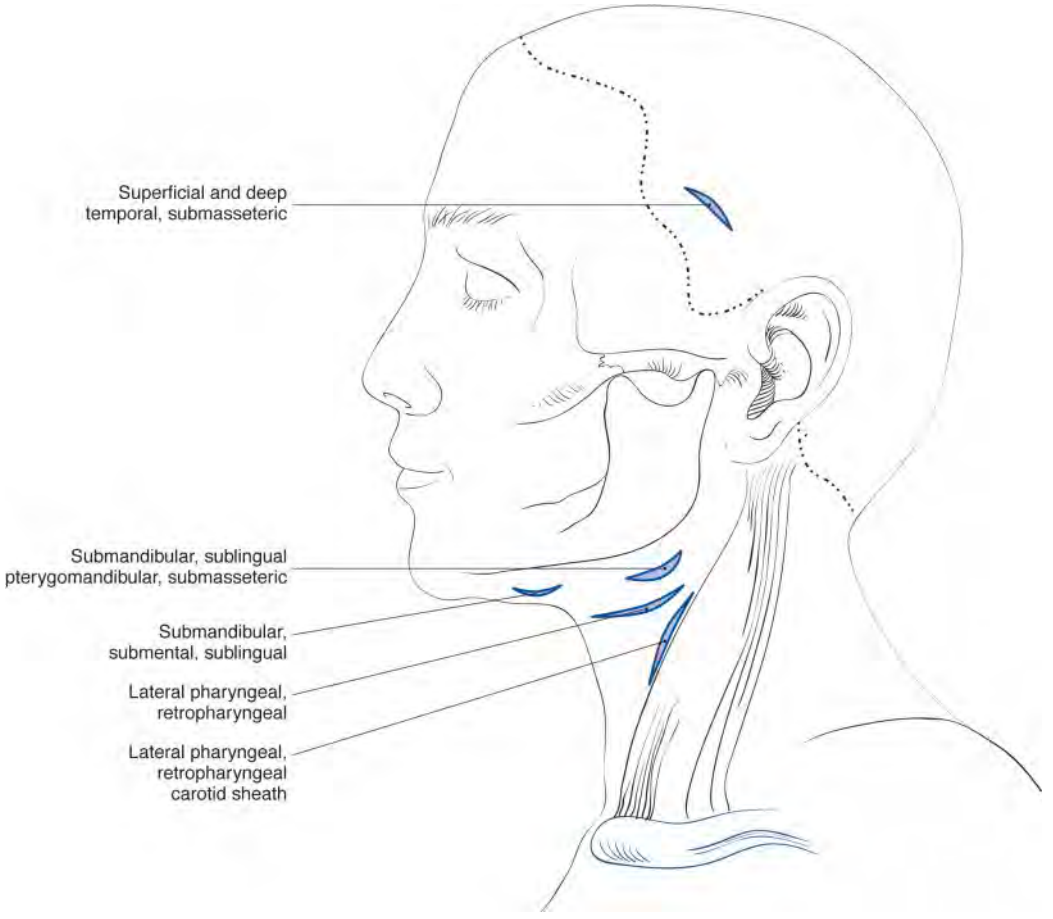


FIGURE 38-7. Incision placement for extraoral drainage of head and neck infections. Incisions at the following points may be used to drain infections in the indicated spaces: superficial and deep temporal, submasseteric; submandibular, submental, sublingual; submandibular, sublingual, pterygomandibular, submasseteric; and lateral pharyngeal, retropharyngeal; lateral pharyngeal, retropharyngeal, carotid sheath. (Adapted from Flynn TR. Surgical management of orofacial infections. Atlas Oral Maxillofac Surg Clin North Am 2000;8:77–100.)

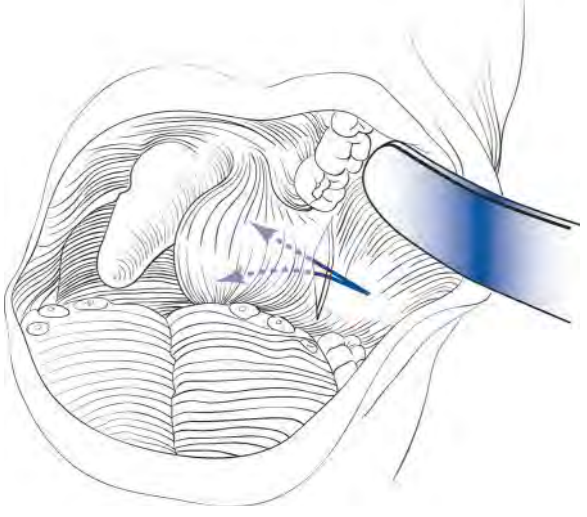


FIGURE 38-8. Intraoral incision placement for drainage of the anterior compartment of the lateral pharyngeal space *(curved arrow)* and the pterygomandibular space *(straight arrow)*. (Adapted from Flynn TR. Surgical management of orofacial infections. Atlas Oral Maxillofac Surg Clin North Am 2000;8:77–100.)

This can be guided by the preoperative CT examination and by knowledge of the pathways that odontogenic infection is most likely to take. For example, in drainage of the submandibular space, if incisions are placed over the anterior and posterior bellies of the digastric muscle at the submandibular, submental, and sublingual location and at the submandibular, sublingual location as shown in Figure 38-7, then the dissection must pass superiorly and medially until the medial (lingual) plate of the mandible is contacted. The most likely pathway for odontogenic infections to enter the submandibular space is through the thin lingual plate of the mandible, which also approximates the root apices of the lower molar teeth. By exploring this location, the surgeon may find a collection of pus that would otherwise have been missed. In order to pass a drain through the submandibular space effectively, the surgeon should, therefore, pass a large curved hemostat from one incision upward to the medial side of the mandible and then down to the other incision. A Penrose drain can then be grasped in the tip of the hemostat and pulled through the dissected pathway from one incision to the other, thus draining the entire submandibular space.

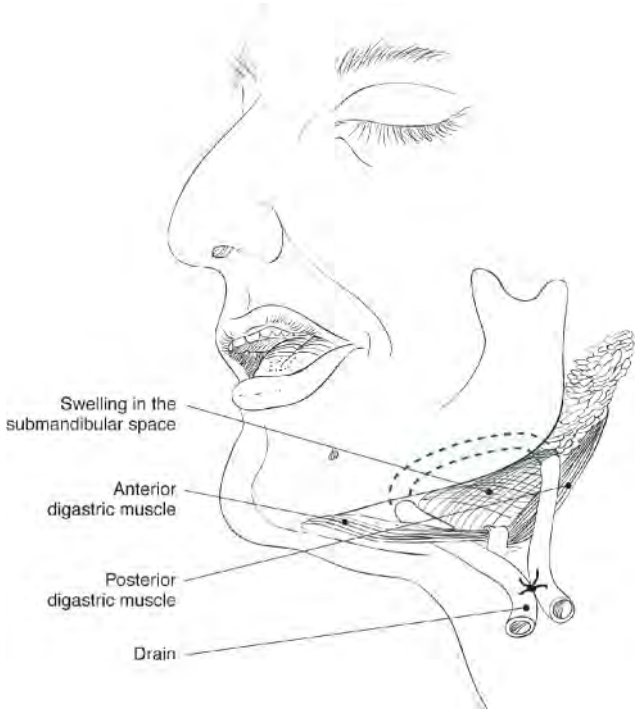


FIGURE 38-9. Pathway of a through-and-through drain of the submandibular space. Note that the drain passes deep to the medial surface of the mandible, below the attachment of the mylohyoid muscle. (Adapted from Flynn TR. Surgical management of orofacial infections. Atlas Oral Maxillofac Surg Clin North Am 2000;8:77–100.)

The resulting pathway for a through-and-through drain in the submandibular space is illustrated in Figure 38-9.

The advantages of through-and-through drainage are the provision of two pathways for the egress of pus, placement of the incisions in healthy tissue in cosmetically acceptable areas, and the ability to irrigate the infected wound with unidirectional flow from one incision to the other. Wound irrigation is facilitated especially by the use of a Jackson-Pratt–type drain, which is noncollapsible and perforated. Such unidirectional superior-to-inferior drainage of the pterygomandibular space using intraoral and extraoral incisions and a Jackson-Pratt drain is illustrated in Figure 38-10.

There is little evidence to indicate that frequent wound irrigation hastens the resolution of infection. However, it does make clinical sense to remove by irrigation bacteria, pus, clots, and necrotic tissue from infected wounds as they accumulate. Similarly, the use of bulky occlusive dressings has not been shown to substantially alter the outcome of cases of odontogenic infection. Nonetheless, the use of such a dressing, as illustrated in Figure 38-11, may be more comfortable over the long run than a dressing that is taped to the skin, and it certainly helps to prevent the contamination of the hospital by pathogenic organisms. The need for this type of hygiene is bound to increase in coming years, as both antibiotic-resistant organisms and critically ill, sometimes immunocompromised patients increasingly inhabit hospitals.

Drains should be discontinued when the drainage ceases. They may be advanced gradually or removed all at once.

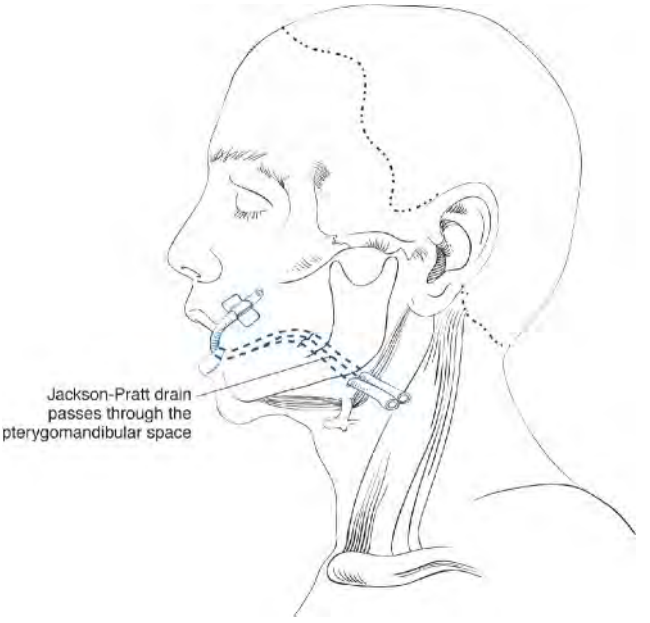


FIGURE 38-10. Jackson-Pratt irrigating drain placed from an intraoral incision through the pterygomandibular space to an extraoral incision, allowing unidirectional irrigation and drainage. (Adapted from Flynn TR. Surgical management of orofacial infections. Atlas Oral Maxillofac Surg Clin North Am 2000;8:77–100.)

There is no evidence in favor of either technique. Pus usually stops flowing from surgically drained abscesses in 24 to 72 hours, but this process may take somewhat longer when only cellulitis has been encountered. It should be kept in mind, however, that latex Penrose drains can be antigenic,



FIGURE 38-11. A properly placed Barton dressing, which avoids taping of the skin. It can occlude and absorb the drainage of a maxillofacial infection. (Reproduced with permission from Flynn TR. Odontogenic infections. Oral Maxillofac Surg Clin North Am 1991;3:311–329.)

and after several days, they may cause exudation owing to foreign body reaction alone.

Timing of Incision and Drainage

Much of the surgical literature on the management of deep fascial space infections of the head and neck advocates an expectant approach to surgical drainage of deep neck infections. The overall strategy of this approach is to use parenteral antibiotic therapy as a means of controlling, localizing, or even eradicating the soft tissue infection. Failure of the medical approach is determined by patient deterioration, impending airway compromise, and the identification of an abscess by CT or clinical examination or both. Only then is surgical drainage undertaken.[20–22] The expectant approach to management of severe odontogenic infections has not been supported by empirical investigation.

The alternative strategy, successfully demonstrated by Williams and Guralnick,[2] is the immediate establishment of airway security, as necessary, and aggressive early surgical intervention. Identification of an abscess is not required before surgical intervention. The approach by Williams and Guralnick[2] is predicated on the concept that early incision and drainage aborts the spread of infection into deeper and more critical anatomic spaces, even when it is in the cellulitis stage. In a prospective case series of 37 patients hospitalized with severe odontogenic infections, Flynn and colleagues[6] performed surgical drainage on all patients as soon as possible after admission. In none of their cases did incision and drainage seem to hasten the spread of infection. The need for reoperation and length of hospital stay were not significantly different between those patients in whom abscess and those in whom cellulitis was found.[6] On multivariate analysis, when all potentially confounding variables were controlled for, the only significant predictor of abscess formation was the negatively associated identification of peptostreptococci in culture. Thus, peptostreptococci were associated with cellulitis significantly more than with abscess.[16]

Culture and Sensitivity Testing

Infections that present in the low-severity anatomic spaces (see Table 38-3) are not in an anatomic position that is likely to threaten the airway or vital structures. In the absence of immunologic or systemic compromise, such infections are very unlikely to become serious or life-threatening. Straightforward treatments, such as removal of the involved teeth, intraoral incision and drainage, and empirical antibiotic therapy, are almost always successful. In this setting, it can be hard to justify the increased cost of routine culture and antibiotic sensitivity testing. Furthermore, because most odontogenic pathogens are slow-growing species, identification can become an expensive and time-consuming task for the microbiology laboratory. This expense is hard to justify, given the fact that, at least until recently, the oral flora is routinely sensitive to penicillin. Therefore, most microbiology laboratories, when given a specimen that grows out α-hemolytic streptococci mixed with short, anaerobic, weakly gram-negative rods will report the growth of normal oral flora, thus avoiding the necessity for species identification and subsequent antibiotic sensitivity testing. For these reasons, routine culture and sensitivity testing for minor oral infections does not appear to be justified.

When an infection involves anatomic spaces of moderate or greater severity or when there is significant medical or immune system compromise, culture and sensitivity testing as early as possible in the course of infection is important because the final result of antibiotic sensitivity testing can be delayed for as much as 2 weeks when fastidious or antibiotic-resistant organisms are involved.

Culture and sensitivity testing is also justified when the surgeon is dealing with infections that have been subjected to multiple prior courses of antibiotic therapy or in chronic infections that are recalcitrant to therapy. Immunocompromised patients also tend to harbor unusual pathogens, such as *Klebsiella pneumoniae* in diabetics, methicillin-resistant *Staphylococcus aureus* in intravenous drug abusers, and intracellular pathogens, such as mycobacteria in HIV/AIDS. In summary, culture and sensitivity testing should be performed in unusual infections, the medically and immunocompromised, and certainly in all cases severe enough to require hospitalization.

Proper culture technique involves the harvesting of the specimen in a manner that minimizes contamination by normal oral or skin flora. Ideally, the skin or mucosa should be prepared with antiseptic and isolated, and the culture should be obtained by aspiration from the point of maximum inflammation, where abscess is most likely to be found. If this is not possible, then at surgery, a swab and culturette system can be used, although the surgeon must be careful to avoid contamination of the specimen by saliva or skin flora. Furthermore, the culture transport system should be designed to maintain the viability of anaerobic organisms, which do not survive in commonly available aerobic culturette systems. Even though the surgeon may not encounter pus during aspiration attempts or surgical drainage, fluid aspirates and swab cultures of infected sites do yield valid cultures with readily interpretable results. Therefore, specimens should be sent for culture and sensitivity testing even when pus is not obtained. Molecular methods using genetic material from infecting pathogens are in development. In the foreseeable future, these techniques will allow rapid identification of both culturable and unculturable pathogens, as well as possibly the identification of antibiotic resistance genes in the specimen.[23,24]

STEP 5: SUPPORT MEDICALLY

Medical supportive care for the patient with a severe odontogenic infection is composed of hydration, nutrition, and control of fever in all patients. Maintenance or reestablishment of electrolyte balance and the control of systemic diseases may also be a crucial part of the necessary supportive medical care for some cases, and the reader is referred to appropriate texts for a more comprehensive discussion of these matters.[25,26]

Initial temperature has been shown to be a significant predictor of the length of hospital stay with severe odontogenic infections.[27] Fever below 103°F (39.4°C) is probably beneficial. Mild temperature elevations promote phagocytosis, increase blood flow to the affected area, raise the metabolic rate, and enhance antibody function. Above 103°F, however, fever can become destructive by increasing metabolic and cardiovascular demands beyond physiologic reserve capacity. Energy stores can be rapidly depleted and the loss of fluid is significantly increased.

Adequate hydration is perhaps the best method for controlling fever. Daily sensible fluid loss, consisting primarily of sweat, is increased by 250 mL per degree of fever. Insensible fluid loss, consisting mainly of evaporation from lungs and skin, is increased by 50 to 75 mL per degree of fever per day. Therefore, a 70-kg patient with a fever of 102.2°F would have a daily fluid requirement of about 3100 mL. This would translate to a required intravenous infusion rate of approximately 130 mL/hr, assuming no oral intake and no other extraordinary fluid losses.[28]

The next approach to controlling fever is usually the administration of acetaminophen or aspirin. Fevers are often exaggerated in children and decreased in the elderly. Thus, an older patient with a relatively mild elevation of temperature may have a fairly significant infection. At the same time, the surgeon may wish to control fever in the elderly at a lower temperature level than in the younger patient because of a fever's increased cardiovascular and metabolic demands.[28] Fever can be controlled or reduced by a variety of other methods when necessary. These include cool water or alcohol sponge baths, chilled drinks when practical, or even an immersion bath using tepid water.

Fever also increases metabolic demand by 5% to 8% per degree of fever per day.[28] Therefore, it may be necessary to supplement the infected patient's oral intake, which is likely to be significantly inhibited by the local effects of the infection and surgery, by using supplementary feedings or even enteral nutrition via a feeding tube.

STEP 6: CHOOSE AND PRESCRIBE ANTIBIOTIC THERAPY

It is beyond the scope of this chapter to discuss the topic of antibiotic selection for head and neck infections comprehensively. This matter has been recently covered in detail elsewhere.[29] The empirical antibiotics of choice for odontogenic infections are, however, listed in Table 38-8.

These antibiotic choices are separated by severity of infection. Mild or outpatient infections have been shown in a number of studies to respond well to the oral penicillins. There was no significant difference in pain or swelling at 7 days of therapy between penicillin and various other antibiotics, including clindamycin, amoxicillin, amoxicillin-clavulanate, and cephradine, although these parameters improved more rapidly during the first 48 hours of therapy with the alternative antibiotics.[30–32] In one pediatric study, pain and swelling were significantly better at 7 days with amoxicillin.[33] In all of the previously referenced studies, the involved tooth or teeth were treated with extraction or root canal therapy. Incision and drainage was performed as necessary. Therefore, a penicillin continues to be a highly effective antibiotic for uncomplicated odontogenic infections, owing to its low cost and low incidence of unwanted side effects.

TABLE 38-8. **Empirical Antibiotics* of Choice for Odontogenic Infections**

Severity of Infection	Antibiotic of Choice
Outpatient	Amoxicillin
	Clindamycin
	Azithromycin
	Penicillin allergy:
	Clindamycin
	Azithromycin
	Moxifloxacin
Inpatient	Ampicillin + sulbactam
	Clindamycin
	Ampicillin + metronidazole
	Penicillin allergy:
	Clindamycin
	Ceftriaxone
	Moxifloxacin (especially for ***Eikenella corrodens***)
	Vancomycin + metronidazole ± moxifloxacin

*Empirical antibiotic therapy is used before culture and sensitivity reports are available. Cultures should be taken in severe infections that threaten vital structures.

Since amoxicillin is administered orally only three times per day, compliance with the prescribed regimen should be better than with penicillin V, at four times per day. A recent price decline has made amoxicillin cheaper than penicillin. Therefore, amoxicillin has been given a slight preference to penicillin in Table 38-8.

In penicillin allergy, clindamycin becomes the antibiotic of first choice for odontogenic infections. An alternative antibiotic, azithromycin, one of the newer macrolides, has been shown to be effective in odontogenic infections in one trial,[34] and it has a significant safety advantage over the other macrolide antibiotics. Erythromycin and clarithromycin, for example, are metabolized in the liver by the cytochrome P-450 CYP3A4, which is responsible for 50% of drug interactions. Azithromycin is metabolized by a different pathway and, therefore, has a significantly fewer drug interactions. Further, although in vitro antibiotic sensitivity testing has recently shown that the macrolide antibiotics are generally ineffective against oral anaerobes and even streptococci, the fact that azithromycin is concentrated in phagocytic macrophages by 10 to 15 times the serum concentration may explain its clinical effectiveness for oral infections when combined with appropriate surgical therapy.

For severe infections warranting hospital admission, the antibiotics of choice for odontogenic infections do not include penicillin G or V. Flynn and coworkers[6] found a 21%

failure rate of penicillin G when used empirically in a series of 37 hospitalized cases of odontogenic infection. Of the 33 patients who were placed on penicillin, 7 experienced clinical therapeutic failure of penicillin, which was determined by failure of improvement in swelling, temperature, and white blood cell count after adequate surgical drainage was verified by postoperative CT. This high clinical failure rate of penicillin in hospitalized odontogenic infections is clinically unacceptable because of the seriousness of these cases.

Clindamycin resistance rates among the oral streptococci are rising recently to as high as 17% of cases.[6] Therefore, clindamycin has largely been replaced as the empirical antibiotic of choice for severe odontogenic infections, except in penicillin allergy. Ampicillin-sulbactam is now the antibiotic of choice in odontogenic infections that are serious enough to warrant hospital admission.

Most resistance to penicillin that occurs among the oral pathogens is due to synthesis of β-lactamase. Approximately 25% of the strains of the *Prevotella* and *Porphyromonas* genera are able to synthesize this enzyme. β-Lactamase can also be found in some strains of *Fusobacterium* and *Streptococcus* species. Importantly, however, the oral strains of streptococci that synthesize β-lactamase are generally among the *Streptococcus mitis, S. sanguis,* and *S. salivarius* species. These species are members of the *Streptococcus viridans* group that are responsible for many cases of endocarditis. They are not frequently found in odontogenic abscesses. *Streptococcus anginosus, S. constellatus,* and *S. intermedius* are the viridans streptococci that comprise the *Streptococcus milleri* group. The *S. milleri* group is most commonly found in odontogenic abscesses, and fortunately, it remains sensitive to the natural and semisynthetic penicillins, such as penicillin V and amoxicillin. Therefore, it is reasonable to use penicillin plus a β-lactamase inhibitor such as ampicillin-sulbactam or a penicillin plus metronidazole as alternative antibiotics for serious odontogenic infections. The penicillins and metronidazole have the advantage of crossing the blood-brain barrier when the meninges are inflamed. Clindamycin, conversely, does not cross the blood-brain barrier. Therefore, it is appropriate to use penicillin plus metronidazole or ampicillin-sulbactam when there is a risk of an odontogenic infection entering the cranial cavity.[29]

Few cephalosporins are able to cross the blood-brain barrier. Some third-generation cephalosporins, such as ceftriaxone, can do so. In addition, ceftriaxone is effective against the oral streptococci and most oral anaerobes. Among the cephalosporins, therefore, ceftriaxone is an alternative antibiotic of choice. Because ceftriaxone can cause pseudocholelithiasis owing to sludging of bile salts, it should be used with caution or avoided in patients with hepatobiliary disease.

A fourth-generation fluoroquinolone, moxifloxacin has great promise in the treatment of head and neck infections. Its spectrum against oral streptococci and anaerobes is excellent. The occasional oral pathogen, *Eikenella corrodens,* is always resistant to clindamycin and often resistant to the penicillins. The fluoroquinolones are the antibiotics of choice for *E. corrodens.* Their absorption is virtually complete via either the oral or the intravenous routes, and they penetrate bone readily. It can be given orally to treat osteomyelitis, for example, when a peripherally inserted central catheter (PICC line) is to be avoided, as in intravenous drug abusers. Therefore, moxifloxacin is a significant addition to the oral and maxillofacial surgeon's armamentarium.

Even though metronidazole is active only against obligate anaerobic bacteria, its use alone in the treatment of odontogenic infections, when combined with appropriate surgical therapy, may be effective. In one study, ornidazole, a member of the nitroimidazole family, was effective when used alone in the management of odontogenic infections.[35] Thus, the use of metronidazole alone may be an appropriate stratagem when all of the other appropriate antibiotics are contraindicated. As with all antibiotics, the surgeon should be aware of the side effects and drug interactions of the antibiotics he or she uses. Metronidazole has a disulfiram-like reaction with alcohol; it should be avoided in patients that may have difficulty abstaining from alcohol.

STEP 7: ADMINISTER THE ANTIBIOTIC PROPERLY

The tissue level of antibiotics determines their effectiveness. Those tissue levels are, of course, dependent on the antibiotic's level in serum, through which the antibiotic must pass in order to achieve therapeutic levels in soft tissues, bone, brain, and abscess cavities. Administration of antibiotics by the oral route requires that the drug successfully navigate the vagaries of the highly acidic stomach, the basic intestinal tract, and chemical interference by ingested foods. Once an antibiotic is absorbed by the gastric or intestinal mucosa, it may then be subject to first-pass metabolism in the liver and subsequent excretion though the bile. Part of the excreted antibiotic may then be reabsorbed by the intestine, resulting in enterohepatic recirculation. For these reasons, orally administered antibiotics achieve much lower serum levels at a slower rate than when they are injected directly into the vascular system intravenously.

Some antibiotics, however, are equally well absorbed intravenously and orally. The fluoroquinolones, such as ciprofloxacin and moxifloxacin, are the best examples of this. For this reason, the fluoroquinolones are not given intravenously unless use of the oral route is contraindicated.

The minimum inhibitory concentration (MIC) is the concentration of an antibiotic that is required to kill a given percentage of the strains of a particular species, reported as 50% or 90% of strains (MIC_{50} or MIC_{90}, respectively). The effectiveness of some antibiotics is determined by the ratio of the serum concentration of the antibiotic to the MIC required to kill a particular organism. For example, with the fluoroquinolones and the aminoglycosides, if the serum concentration achieved is three to four times the MIC for the organisms involved, then maximum killing power will be

achieved. These are examples of concentration-dependent antibiotics.[29]

With time-dependent antibiotics, such as the β-lactams and vancomycin, antibiotic effectiveness is determined by the duration for which the serum concentration of the antibiotic remains above the MIC. With time-dependent antibiotics, it is necessary to know the serum elimination half-life ($t^1/_2$) of the antibiotic in order to determine its proper dosage interval. The dosage interval can then be designed in order to maintain the serum concentration above the MIC for at least 40% of the dosage interval.[29]

Fortunately, the mathematics involved in these calculations have already been determined by the drug manufacturer. Dosage intervals should not be changed from published guidelines by the surgeon. Nonetheless, the surgeon must be aware of the greater effectiveness of intravenous antibiotics over their oral counterparts. For example, when penicillin G is given every 4 hours intravenously, a peak serum blood level of 20 µg/mL is achieved. Because the serum elimination $t^1/_2$ of penicillin G is 0.5 hours, after 3 hours (6 half-lives) the serum concentration will be approximately 0.3 µg/mL. Because the MIC_{90} of *Streptococcus viridans* is 0.2 µg/mL, the serum concentration of penicillin G after an intravenous dose of 2 million units will remain above the MIC_{90} for approximately 75% of the dosage interval. Therefore, penicillin G, 2 million units given intravenously every 4 hours, should be highly effective against the viridans group of streptococci, especially the abscess-forming *S. milleri* group.

By the same method, the peak serum level that can be achieved with an oral dose of 500 mg of amoxicillin is 7.5 µg/mL, and its $t^1/_2$ is only 1.2 hours. Because amoxicillin's MIC_{90} for viridans streptococci is 2 µg/mL, the serum concentration of amoxicillin will fall below the MIC_{90} at approximately 2 hours after the peak serum level has been achieved, which is only 25% of the 8-hour dosage interval. Therefore, oral amoxicillin, even though it is considered by many to be a more effective antibiotic, is less likely to be effective against the viridans streptococci than intravenous penicillin G.

In a randomized clinical trial of outpatient odontogenic infections comparing amoxicillin, cephalexin, and surgery alone (all patients received extraction and/or incision and drainage), there was no statistically significant difference in cure rate between the groups. The time to resolution was nearly significantly shorter in the antibiotic-assisted groups. This study indicates that surgical treatment is primary, and antibiotic therapy as adjunctive.[36]

Another practical matter that must always be considered in administering antibiotics is their cost, especially the out-of-pocket cost to the patient. When a patient does not have prescription drug insurance coverage, such as the working poor and the elderly, the retail cost of the antibiotic can be a significant factor in whether the prescribed antibiotic is indeed followed. In 2010, the retail cost of 1 week's supply of amoxicillin 500 mg taken three times per day was $11.99 at a large pharmacy chain in the northeastern United States. The retail cost of 1 week's supply of clindamycin 300 mg taken four times per day was $59.99. These prices reflect generic medications, not brand name antibiotics, which are significantly more expensive. Thus, an indigent patient may not be able to pay for a more expensive antibiotic, and therefore, she or he may be forced to either take reduced amounts of the antibiotic, extend the dosage interval, or forego taking the antibiotic entirely. Accordingly, the astute clinician will take the cost factor into account. When appropriate, a frank discussion of the cost of the antibiotic relative to the patient's means appears to be the best policy.

STEP 8: EVALUATE THE PATIENT FREQUENTLY

In outpatient infections that have been treated by tooth extraction and intraoral incision and drainage, the most appropriate initial follow-up appointment is usually at 2 days postoperatively for the following reasons:

1. Usually, the drainage has ceased and the drain can be discontinued at this time.
2. There is usually a discernible improvement or deterioration in signs and symptoms allowing the next treatment decisions to be made.

For odontogenic deep fascial space infections that are serious enough for hospitalization, daily clinical evaluation and wound care are recommended. By 2 to 3 postoperative days, the clinical signs of improvement should be apparent, such as decreasing swelling, defervescence, cessation of wound drainage, declining white blood cell count, decreased malaise, and a decrease in airway swelling such that extubation can be considered. Also at this time, preliminary Gram stains and/or culture reports should be available, which may provide some guidance as to the appropriateness of the empirical antibiotic therapy.

If the previous signs of clinical improvement are not apparent, then it may be necessary to begin an investigation for possible treatment failure. The causes of treatment failure in odontogenic infections are listed in Table 38-9. One of the best methods of reevaluation is the postoperative CT. A postoperative CT can identify continued airway swelling that may preclude extubation or further spread of the infection into previously undrained anatomic spaces or it may confirm

TABLE 38-9. **Causes of Treatment Failure**

Inadequate surgery
Depressed host defenses
Foreign body
Antibiotic problems
Patient noncompliance
Drug not reaching site
Drug dosage too low
Wrong bacterial diagnosis
Wrong antibiotic

Adapted from Flynn TR. Principles of management and prevention of odontogenic infections. In Ellis E, Hupp JR, Tucker MR, editors. Contemporary Oral and Maxillofacial Surgery. 5th edition. St. Louis: Mosby; 2008; pp. 291–315.

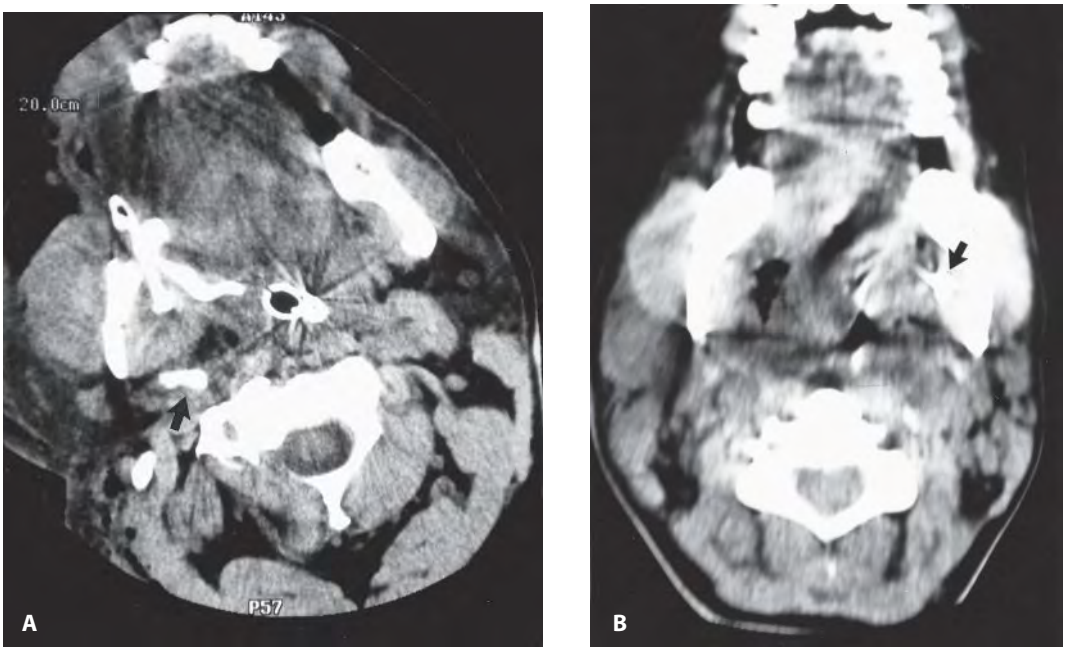


FIGURE 38-12. **A,** Four days postoperative computed tomography (CT) image of a patient with a right pterygomandibular and lateral pharyngeal space abscess. Note the intraoral drains in the pterygomandibular and anterior compartments of the lateral pharyngeal space and the extraoral drain in the posterior compartment of the lateral pharyngeal space *(arrow)*. **B,** Five days postoperative axial CT of a patient with a previously placed drain in the left pterygomandibular space *(arrow)*. Note the extension of the infection into the right and left lateral pharyngeal spaces and the retropharyngeal space, with constriction and deviation of the airway. (**A** and **B,** Reproduced with permission from Flynn TR. Surgical management of orofacial infections. Atlas Oral Maxillofac Surg Clin North Am 2000;8:77–100.)

adequate surgical drainage of all the involved anatomic spaces by the visualization of radiopaque drains in all of the involved fascial spaces.

Sometimes, it is difficult to determine whether the inability to extubate a patient is due to antibiotic resistance or inadequate surgical drainage. Figure 38-12 illustrates two such cases in which a postoperative CT was able to identify the most likely cause for the lack of clinical improvement. In Figure 38-12A, oropharyngeal swelling surrounds the endotracheal tube in spite of the presence of surgical drains in all of the infected spaces. This lack of improvement at 4 postoperative days was due to therapeutic failure of penicillin, which was treated by changing this patient's antibiotic to clindamycin. Subsequently, the patient improved. In Figure 38-12B, there is continued oropharyngeal swelling at 5 postoperative days. Conversely, the infection has progressed from the successfully drained left pterygomandibular space to the left and right lateral pharyngeal spaces as well as the retropharyngeal space. This patient was taken back to the operating room for repeated drainage of all of the infected spaces.

It should be noted, however, that in this author's experience, the use of CT scanning to determine whether a patient can be extubated gives a late positive signal. The best available clinical test for the ability to extubate in the case of upper airway swelling is the air leak test (Figure 38-13). The air leak test is performed in the following manner in the spontaneously ventilating patient:

1. The endotracheal tube and trachea are suctioned.
2. The oxygen supply is reconnected and any coughing that was stimulated by the tracheal suctioning is allowed to subside.
3. The oropharynx and oral cavity are suctioned free of debris, hemorrhage, and secretions.
4. The cuff of the endotracheal tube is deflated while the oxygen supply is maintained.



FIGURE 38-13. Air leak test, performed by occluding the endotracheal tube with a finger, to determine whether the patient can breathe around the outside of the endotracheal tube. (Reproduced with permission from Bennett JD, Flynn TR. Anesthetic considerations in orofacial infections. In Topazian RG, Goldberg MH, Hupp JR, editors. Oral and Maxillofacial Infections. 4th ed. Philadelphia: WB Saunders; 2002; pp. 439–455.)

5. After waiting for any coughing to subside, the oxygen supply is disconnected and the surgeon's thumb is placed to occlude the opening of the endotracheal tube.
6. The patient is then instructed to breathe spontaneously around the endotracheal tube, and if this can be done, a positive air leak test is obtained. If the patient cannot breathe around the occluded endotracheal tube, then a negative result is obtained and extubation should be delayed.

Given a positive air leak test result, the best method for patient extubation involves extubation over a stylet or preferably an endotracheal tube changer. Consideration may be given to performing the extubation procedure in an operating room, where the best facilities for handling an airway emergency are available. One method for extubation over a tube changer is described as follows:

1. The patient is preoxygenated for 3 to 5 minutes.
2. The endotracheal tube and trachea are suctioned.
3. Five milliliters of 1% lidocaine without epinephrine is administered via the endotracheal tube, followed by oxygenation and then repeated tracheal suctioning.
4. The oral cavity and oropharynx are suctioned free of debris, hemorrhage, and secretions.
5. The oxygen supply is disconnected and a tube changer is introduced into the trachea via the endotracheal tube.
6. The cuff of the endotracheal tube is deflated and the endotracheal tube is withdrawn over the tube changer until its tip is in the oropharynx.
7. If the patient is able to breathe around the tube changer as it remains in the trachea, extubation can be completed.
8. If the patient is not able to breathe around the tube changer, the endotracheal tube is re-inserted over the tube changer into the trachea.
9. The endotracheal tube cuff is re-inflated, the tube changer is withdrawn, and oxygen is reconnected.

After extubation, the patient is closely monitored clinically and with pulse oximetry. Arterial blood gases may be drawn 1 hour after extubation in order to verify adequate oxygenation and ventilation.

Occasionally, the infecting flora, especially in a particularly severe infection with a prolonged course, will change during the course of treatment. This may be due to the selection pressure exerted by intensive antibiotic therapy or to the subsequent introduction of hospital-acquired pathogens, resulting in a nosocomial infection. Therefore, in prolonged treatments and in especially severe cases, it may be prudent to reculture infected sites so that any new or previously undetected pathogens can be identified.

In cases in which there is continued chronic drainage from an infected site, such as in diagnosed or suspected osteomyelitis, the surgeon's mnemonic for the causes of a fistula can be used. "FETID" stands for *f*oreign body, *e*pithelium, *t*umor, *i*nfection, and *d*istal obstruction. In the maxillofacial region, this mnemonic can be used to provide a differential diagnosis for the chronic drainage of pus. Foreign bodies may be represented by bone plates and screws or dental or cosmetic facial implants. Epithelium may cause chronic drainage simply because an epithelialized fistulous tract has not been completely excised or because an epithelium-lined cyst has drained externally. Tumors (especially malignant ones) that become infected do not heal, which may result in chronic drainage. Infection can, of course, drain chronically, which should alert the surgeon to suspect osteomyelitis or a chronic periapical abscess that is draining onto the skin, as in Figure 38-14. *Distal obstruction* classically refers to intestinal obstructions, but the concept can still be applied to the salivary ducts and to the natural sinus drainage pathways, such as the ostium of the maxillary sinus. When these openings for natural drainage of saliva or mucus become obstructed, infection may result and drainage may occur by an alternate pathway, such as proximal fistulization of the submandibular salivary duct due to a salivary stone blocking the natural opening of Wharton's duct.



FIGURE 38-14. A draining sinus tract onto the face resulting from an untreated periapical abscess. (Reproduced with permission from Flynn TR, Topazian RG. Infections of the oral cavity. In Waite D, editor. Textbook of Practical Oral and Maxillofacial Surgery. 3rd ed. Philadelphia: Lea & Febiger; 1987; pp. 273–310.)

If a thorough search for previously undetected pathogens turns up negative or if another cause for treatment failure cannot be found, the surgeon should consider the possibility of antibiotic failure, such as microbial resistance to empirical antibiotic therapy or the use of an incorrect dosage or route of administration for the antibiotic. The criteria for changing antibiotics are listed in Table 38-10. Because of the necessary time delay in obtaining culture and sensitivity reports, it is occasionally necessary to change from one empirical antibiotic to another. Ideally, the surgeon should consider another of the empirical antibiotics of choice listed in Table 38-8. The input of an infectious disease consultant may also be valuable in this situation.

TABLE 38-10. **Criteria for Changing Antibiotics**

Allergy, toxic reaction, or intolerance
Culture and/or sensitivity test indicating resistance
Failure of clinical improvement, given
Removal of odontogenic cause
Adequate surgical drainage (suggest postoperative imaging)
Other causes for treatment failure ruled out
48–72 hr of the same antibiotic therapy

SINUSITIS

Occasionally, the oral and maxillofacial surgeon will be called upon to diagnose and treat sinus infections, most often as a complication of odontogenic infection or surgical procedures. Examples include sinusitis due to oroantral fistula or bone augmentation procedures in the posterior maxilla.

The clinical diagnosis of acute sinusitis includes pain overlying the involved sinus(es), which may range from a dull ache to throbbing pain, usually constant but exacerbated by bending over or straining. Early on, the only presenting pain may involve the posterior maxillary dentition on the affected side, which may be tender to percussion. In acute sinusitis, there is profuse nasal discharge, which may range from clear to mucopurulent; in chronic sinusitis, the discharge is generally purulent and associated with nasal congestion. The discharge may be seen only in the middle meatus or, if profuse, in the naso- or oropharynx. Involvement of the eustachian tube orifice by swelling may result in otologic symptoms.

Other signs of sinusitis include tenderness to finger pressure over the involved sinus, unilateral postnasal discharge, edema of the middle and inferior nasal turbinates, polyp formation, and opacity on transillumination through the affected sinus(es). Aspiration of pus from the involved sinus is the gold standard of diagnosis.[37]

Sinusitis is considered acute and bacterial between 10 days and 4 weeks after onset of symptoms. It is considered chronic after at least 8 weeks of symptoms. Subacute sinusitis is diagnosed between 4 and 8 weeks. Recurrent sinusitis is four or more episodes within 1 year.[38]

CT is the radiologic examination of choice in confirming the diagnosis of sinusitis. The radiologic signs of sinusitis include radiopacity of the normally radiolucent air spaces, air-fluid level, thickening of the mucosal sinus lining (≥4 mm), mucocoeles, polyps, destruction of bony sinus walls, and foreign bodies within the sinus cavity, such as retained teeth or dental restorative materials, or large fungal colonies (fungus ball or aspergilloma). It is important to evaluate and treat all of the paranasal sinuses, because retained infection in the ethmoid sinus, for example, may cause relapse of a treated maxillary sinusitis.

Sinusitis shares essentially the same microbiologic flora with all head and neck infections. There are minor differences among odontogenic, respiratory, and intracranial infections, such as viruses in droplet-borne contagious upper respiratory infections and influenza, and *Staphylococcus* or *Haemophilus* in otitis and sinusitis. Odontogenic infections, conversely, are almost exclusively bacterial. The common pathogens of bacterial head and neck infections are the *Streptococci* and the anaerobes, such as *Peptostreptococcus, Fusobacterium, Prevotella,* and *Porphyromonas.*[39]

In acute sinusitis, viruses cause the vast majority of infections, but often, bacteria may colonize and infect the sinuses after a viral upper respiratory infection due to obstruction of drainage by edema and inhibited mucociliary function. Acute bacterial sinusitis cultures generally yield *Staphylococcus, Moraxella, Haemophilus,* and *Streptococcus;* chronic bacterial sinusitis yields predominantly the anaerobic species listed previously.[39]

Culture and sensitivity testing of sinus aspirates is an important diagnostic tool. It is especially important in identifying unusual pathogens of sinusitis, such as actinomycosis and fungal infections, or *Pseudomonas* in intubated or mechanically ventilated patients. Intranasal aspiration of the involved sinus is one method for obtaining a sinus culture, but a technique oral and maxillofacial surgeons can easily adopt is to use a large-bore needle to aspirate the maxillary sinus intraorally under local anesthesia through mucosa that has been prepared with an antiseptic.

Antibiotic therapy is not indicated in the first 7 days of acute sinusitis, unless signs of invasive infection and severe systemic reaction are present, because nearly all early sinusitis is viral. Various organizations have published guidelines for the treatment of bacterial sinusitis, and their common elements include first-line, inexpensive antibiotics such as amoxicillin, doxycycline, and trimethoprim-sulfamethoxazole. When antibiotics are indicated by infectious invasion of surrounding tissues, such as the face and orbit, antibiotic treatment until 7 days after symptoms have resolved helps to prevent relapse.[38]

Second-line antibiotics are indicated for complicated sinusitis, such as when bacterial resistance is likely, as with prior antibiotic therapy; in moderate to severe sinusitis, as in recurrent or prolonged sinusitis or pansinusitis; with comorbidities such as diabetes or immunocompromise; and at the extremes of age (<2 or > 55 yr). Second-line antibiotics include amoxicillin-clavulanate, clindamycin, a macrolide plus metronidazole, or fluoroquinolones such as moxifloxacin. Antibiotic therapy is continued for 3 to 10 weeks.[38]

Surgical treatment of acute sinusitis is usually not indicated in uncomplicated sinusitis. However, the cases an oral and maxillofacial surgeon is called upon to treat usually do require surgery to remove the underlying cause of the sinusitis, such as dental infection, oroantral fistula, or foreign bodies, such as displaced implants, bone grafts, or restorative materials.

Most cases of uncomplicated chronic sinusitis are probably due to the inflammatory response to allergens or fungi instead of bacterial pathogens. Thus, the largely anaerobic flora of chronic sinusitis may be commensal rather than pathogenic. Intranasal or systemic steroids are usually more effective than antibiotic therapy for chronic sinusitis without

an identifiable cause amenable to surgery, such as oroantral fistula or retained foreign body.[37] A recent systematic review of functional endoscopic sinus surgery (FESS) for treatment of chronic rhinosinusitis found no convincing scientific evidence to demonstrate that FESS is superior to medical treatment with or without sinus irrigation in patients with chronic rhinosinusitis.[38]

Rarely, recurrent or persistent sinusitis leads to intracranial infection, such as brain abscess, subdural empyema, or cavernous sinus thrombosis, generally by direct extension through thin layers of bone or via emissary veins passing from the involved sinus into the intracranial structures. The treatment of intracranial extension of sinusitis is surgical débridement, if possible, and prolonged aggressive antibiotic therapy.[39,40]

SUMMARY

Severe odontogenic infections can be the most challenging cases that an oral and maxillofacial surgeon will be called on to treat. Often, the patient with a severe odontogenic infection has significant systemic or immune compromise and the constant threat of airway obstruction due to infections in the maxillofacial region raises the risk of such cases incalculably. Furthermore, the increasing rarity of these cases and the ever-changing worlds of microbiology and antibiotic therapy make staying abreast of this field difficult for the busy surgeon. Therefore, the eight steps in the treatment of severe odontogenic infections, first outlined by Dr. Larry Peterson, remain the fundamental guiding principles that oral and maxillofacial surgeons must use in successful management of these cases. The application of the eight steps must be thorough and the surgeon's mind must always remain open to the possibility of treatment failure, an error in initial diagnosis, antibiotic resistance, and previously undiagnosed medically compromising conditions. Although adherence to these principles cannot always guarantee a successful result, it can assure the oral and maxillofacial surgeon that he or she is practicing at the highest standard of care.

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39

CHAPTER

Osteomyelitis, Osteoradionecrosis, and BRONJ

George M. Kushner, DMD, MD and Brian Alpert, DDS

OSTEOMYELITIS

Osteomyelitis is defined as an inflammation of the bone marrow *with a tendency to progression.* This is what differentiates it in the jaw from the ubiquitous dentoalveolar abscess, "dry socket" and "osteitis," seen in infected fractures. It involves adjacent cortical plates and often periosteal tissues.

In the preantibiotics era, osteomyelitis of the mandible was not uncommon. With the advent of antibiotics, it became a rare disease. In recent years antimicrobials have become less effective and there has been a re-emergence of the disease, presenting major diagnostic and therapeutic challenges for practicing surgeons. Despite modern therapy it can still remain a major source of morbidity to the patient, requiring multiple surgeries and resulting in prolonged treatment with loss of teeth and/or jawbone.

The incidence of osteomyelitis is much higher in the mandible due to the dense poorly vascularized cortical plates and the blood supply primarily from the inferior alveolar neurovascular bundle. It is much less common in the maxilla due to the excellent blood supply from multiple nutrient feeder vessels. In addition the maxillary bone is much less dense than the mandible.

Diminished host defenses, both local and systemic, can contribute significantly to the emergence and clinical course of the disease. Osteomyelitis has been associated with multiple systemic diseases including diabetes, autoimmune states, malignancies, malnutrition, and acquired immunodeficiency syndrome.[1] The medications linked to osteomyelitis are steroids, chemotherapeutic agents, and bisphosphonates.[1–3] Local conditions that adversely affect the blood supply can also predispose the host to a bony infection. Radiation therapy, osteopetrosis, and bone pathology can alter the blood supply to the area and provide a potential foothold for osteomyelitis to set in (Figure 39-1).

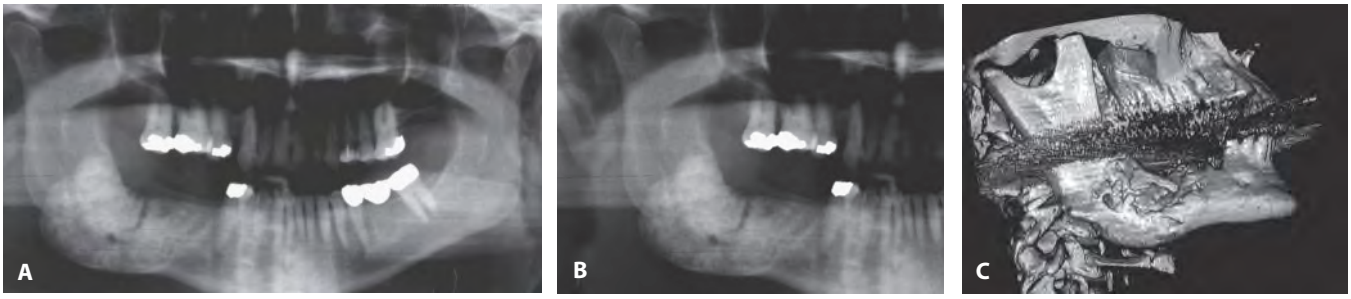


FIGURE 39-1. **A,** Panoramic view of cemento-ossifying fibroma of the right mandible, a poorly vascularized bone tumor. The patient had a transoral biopsy to establish the diagnosis. After the biopsy, the patient had repeated episodes of swelling and drainage. **B,** Close-up of panoramic view. Note the area of osteomyelitis seen within the center of the pathologic lesion. **C,** Three-dimensional computed tomography scan reconstruction showing multiple bony sequestrum from low-grade osteomyelitis within bony pathology.

Pathogenesis

In the maxillofacial region, osteomyelitis primarily occurs as a result of contiguous spread of odontogenic infections or as a result of trauma. Primary hematogenous osteomyelitis is rare in the maxillofacial region, generally occurring in the very young. The adult process is initiated by an inoculation of bacteria into the jawbones. This can occur with the extraction of teeth, root canal therapy, or fractures of the maxilla or mandible. This initial insult results in a bacteria-induced inflammatory process or cascade. In the normal healthy host, this process is self-limiting and is a component of healing. Occasionally, however, in the normal host, and certainly in the compromised host, there is the potential for this process to progress to the point where it is considered pathologic. With inflammation there is hyperemia and increased blood flow to the affected area. Additional leukocytes are recruited to this area to fight off infection. Pus is formed when there is an overwhelming supply of bacteria and cellular debris that cannot be eliminated by the body's natural defense mechanisms. When the pus and subsequent inflammatory response occur in the bone marrow, an elevated intramedullary pressure is created which further decreases the blood supply to this region. The pus can travel via haversian and Volkmann's canals to spread throughout the medullary and cortical bones. Once the pus has perforated the cortical bone and collects under the periosteum, the periosteal blood supply is compromised and this further aggravates the local condition. The end point occurs when the pus exits the soft tissues either by intraoral or extraoral fistulas.

Microbiology

More than 500 bacterial taxa have been identified in the mouth.[4–6] The mouth and the anus are opposing ends of the same alimentary tube, and many clinicians consider them to be the most highly contaminated areas of the human body. In the past, staphylococcal species were considered the major pathogen in osteomyelitis of the jaws. However, with refinements in the collection and processing of microbiologic specimens, we are able to get a true picture of the disease-causing organisms. As with most oral infections the prime pathogenic species are streptococci and anaerobic bacteria. The anaerobes responsible are generally bacteroides or peptostreptococci species. Often, the infections are mixed, growing several pathogens on final culture. The clinician must begin empiric antibiotic treatment based on the most likely pathogens. This could include penicillin and metronidazole as dual-drug therapy or clindamycin as a single-drug treatment. Definitive antimicrobial therapy should be based on the final culture and sensitivities for optimal medical management results.

Classification

Over the years many ways of classifying osteomyelitis have been presented. A rather complex classification system was proposed by Cierny and colleagues.[7] Osteomyelitis was classified as being either suppurative or nonsuppurative by Lew and Waldvogel.[8] This classification was modified by Topazian.[9] Additional authors classified osteomyelitis as being either hematogenous or secondary to a contiguous focus of infection.[10] Another system proposed by Hudson essentially divided the presentation of osteomyelitis into acute and chronic forms.[11] With the multitude of classification systems, the controversy involved in adequately classifying osteomyelitis is clearly evident.

However, for simplicity's sake, the classification system offered by Hudson is the most advantageous to the clinician. Osteomyelitis is divided into acute or chronic forms based on the presence of the disease for a 1-month duration.[11]

1. Acute osteomyelitis
   a. Contiguous focus (Figure 39-2)
   b. Progressive
   c. Hematogenous
2. Chronic osteomyelitis
   a. Recurrent multifocal (Figure 39-3)
   b. Garré's (Figure 39-4)
   c. Suppurative or nonsuppurative (Figure 39-5)
   d. Sclerosing (Figure 39-6)

Clinical Presentation

Very often, as with any infection, the patient with osteomyelitis of the maxillofacial region will present with classic symptoms:

- Pain
- Swelling and erythema of overlying tissues
- Adenopathy
- Fever
- Paresthesia of the inferior alveolar nerve
- Trismus
- Malaise
- Fistulas

The pain in osteomyelitis is often described as a deep and boring pain, which is often out of proportion to the clinical picture. In acute osteomyelitis it is very common to see swelling and erythema of the overlying tissues, which are indicative of the cellulitic phase of the inflammatory process of the underlying bone. Fever often accompanies acute osteomyelitis, whereas it is relatively rare in chronic osteomyelitis. Paresthesia of the inferior alveolar nerve is a classic sign of a pressure on the inferior alveolar nerve from the inflammatory process within the medullary bone of the mandible. Trismus may be present if there is inflammatory response in the muscles of mastication of the maxillofacial region. The patient commonly has malaise or a feeling of overall illness and fatigue, which would accompany any systemic infection. Lastly both intraoral and extraoral fistulas are generally present with the chronic phase of osteomyelitis of the maxillofacial region.

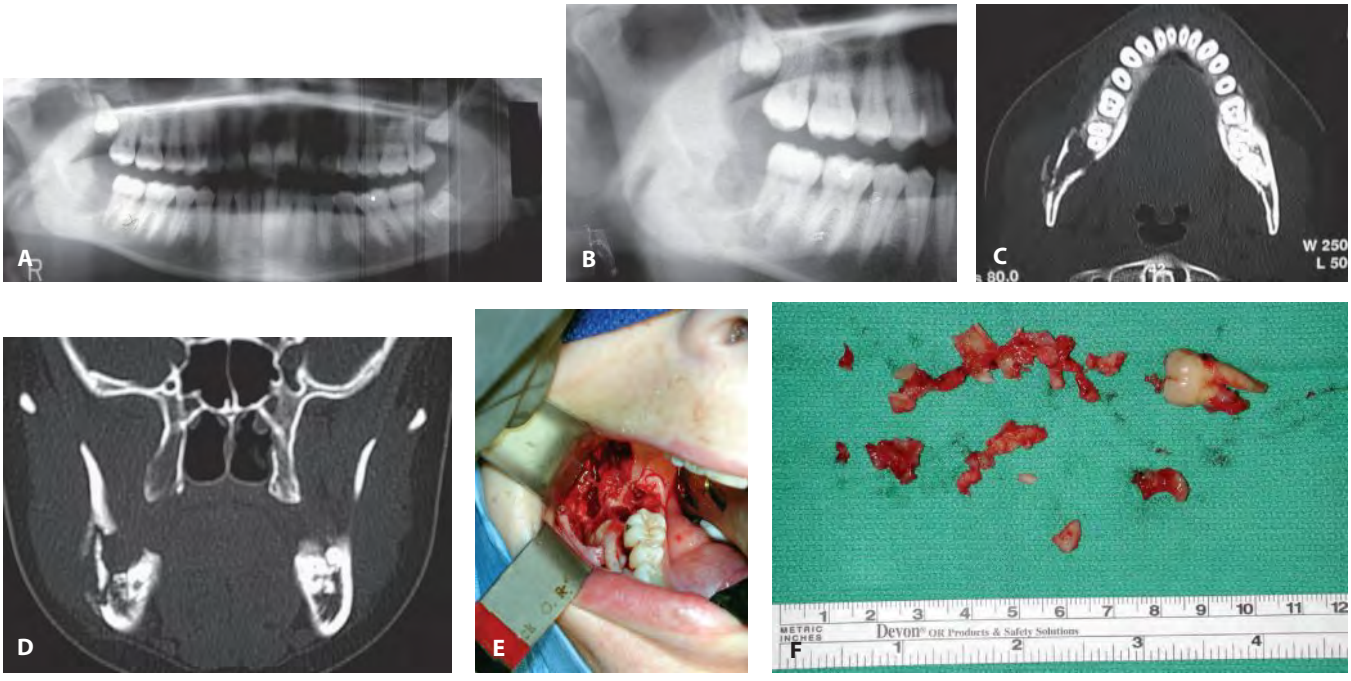


FIGURE 39-2. **A,** Panoramic view of extraction site of tooth no. 32 in an otherwise healthy 32-year-old patient. The patient experienced multiple episodes of pain and swelling in the right posterior mandible after tooth no. 32 was removed. **B,** Close-up of the panoramic view of the no. 32 site. **C,** Axial computed tomography scan of the no. 32 site. **D,** Coronal computed tomography scan of the no. 32 site. Note the moth-eaten bone and bone sequestrum. **E,** Transoral débridements of the right posterior mandible. **F,** Bone débrided and adjacent tooth no. 31 removed. Tissue eas sent for culture and sensitivity and histopathology.

Often these patients will have a laboratory work-up as part of their initial examination. In the acute phase of osteomyelitis it is common to see a leukocytosis with left shift, common in any acute infection. Leukocytosis is relatively uncommon in the chronic phases of osteomyelitis. The patient may also exhibit an elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). Both the ESR and CRP are very sensitive indicators of inflammation in the body and they are very nonspecific. Therefore, their main use is to follow the clinical progress of the osteomyelitis.

Nearly all patients will have some form of maxillofacial imaging. The orthopanoramic view is indispensable in the initial evaluation of osteomyelitis. This view is easily obtainable in most dental offices and can yield valuable information as to the radiographic changes with osteomyelitis, potential sources of the disease, and predisposing conditions such as fractures and underlying bone disease. One must bear in mind that radiographic images lag behind the clinical presentation since cortical involvement is required for any change to be evident. Therefore, it may take several weeks before the bony changes appear radiographically. Hence, it is possible to see a patient with acute osteomyelitis that has a normal-appearing orthopantomogram. However, one can often see the appearance of "moth-eaten" bone or sequestrum of bone, which is the classic appearance of osteomyelitis.

Computerized tomography (CT) scans have become the standard in evaluating maxillofacial pathology such as osteomyelitis. They provide three-dimensional imaging not available on an orthopanoramic view. The CT scan can give very detailed images as to early cortical erosion of bone in osteomyelitis. One can often see the extent of the lesion and bony sequestra along with pathologic fractures. CT scanning, like plain films, requires 30 to 50% demineralization of bone before changes can be seen, thus presenting an essential delay in diagnosis of osteomyelitis.[12]

Magnetic resonance imaging (MRI) is generally considered more valuable in the evaluation of soft tissue lesions of the maxillofacial region. However, MRI can assist in the early diagnosis of osteomyelitis by loss of the marrow signal before cortical erosion or sequestrum of the bone appears. Thus, MRI may benefit in identifying the earlier stages of osteomyelitis.[12]

Nuclear medicine has evolved to aid in the diagnosis of osteomyelitis. Technetium 99 has been the workhorse of nuclear medicine imaging of the maxillofacial region. The technetium 99 bone scan is very sensitive in highlighting areas of increased bone turnover; however, the scan is not very specific to areas of infection. With the addition of gallium 67 or indium 111 as contrast agents, one can differentiate areas of infection from trauma or postsurgical healing as these agents specifically bind to white blood cells.

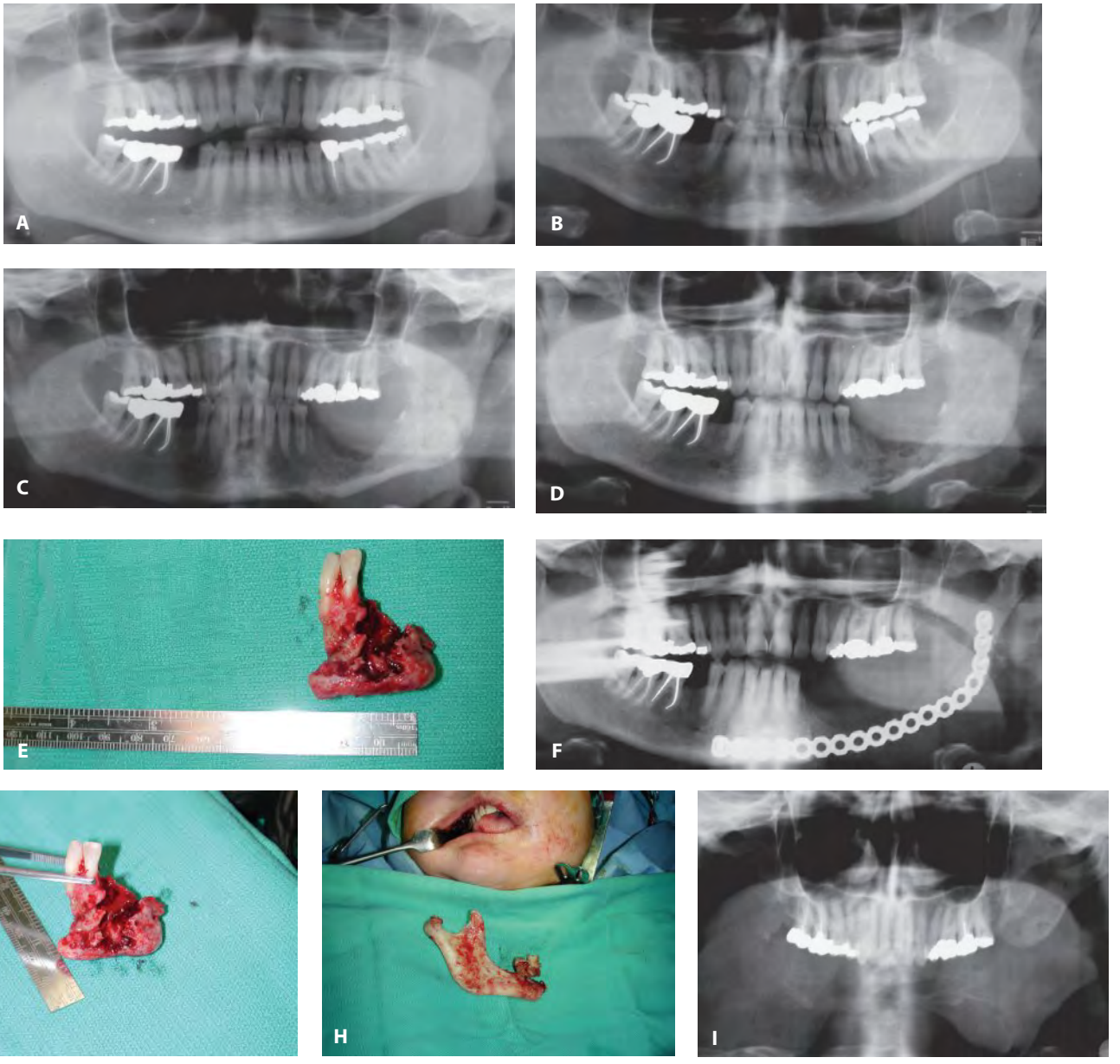


FIGURE 39-3. **A,** Panoramic view taken of a 55-year-old female before extraction of symptomatic tooth no. 17. The patient had a history of unusual infections and recurrent infections without a specific diagnosis. The patient began having pain and swelling in the left mandible after tooth no. 17 was extracted. **B,** Panoramic view of no. 17 site postoperatively. **C,** Panoramic view after intraoral débridements of the left mandible and extraction of teeth no. 18, 29, 20. Histopathology confirmed diagnosis of osteomyelitis. The patient was treated with antibiotics based on culture and sensitivity reports. **D,** Panoramic view shows radiographic worsening of disease. Note the classic appearance of moth-eaten bone and impending pathologic fracture of the left mandible. Medical work-up revealed hypogamma globulinemia, a chronic immunocompromised state. **E,** Bone specimen showing osteomyelitis resected. **F,** Panoramic view after left mandible resection of osteomyelitis with pathologic fracture. Rigid internal fixation with a reconstruction plate allowed maintenance of space and facial form with continuous jaw function and mobility. **G,** The patient was asymptomatic for 2 years before having pain and swelling in the anterior mandible. Débridement revealed necrotic moth-eaten bone. **H,** The patient eventually required removal of the remainder of the right mandible due to uncontrollable osteomyelitis. The patient was hospitalized and received intravenous antibiotics based on multiple specific culture and sensitivity reports. She also received intravenous gamma globulin to correct hypogammaglobulinemia. Hyperbaric oxygen treatments were also used to treat refractory osteomyelitis. The patient had a prolonged in-patient hospital course with multiple surgeries. **I,** Panoramic view with subtotal mandibulectomy for osteomyelitis. Only the left ramus and condyle remain intact. The patient is currently on daily antibiotic immunosuppressive therapy for life, as well as monthly infusions of gamma globulin. Despite aggressive medical management by infectious disease experts, she still has bouts of recurrent pneumonia.

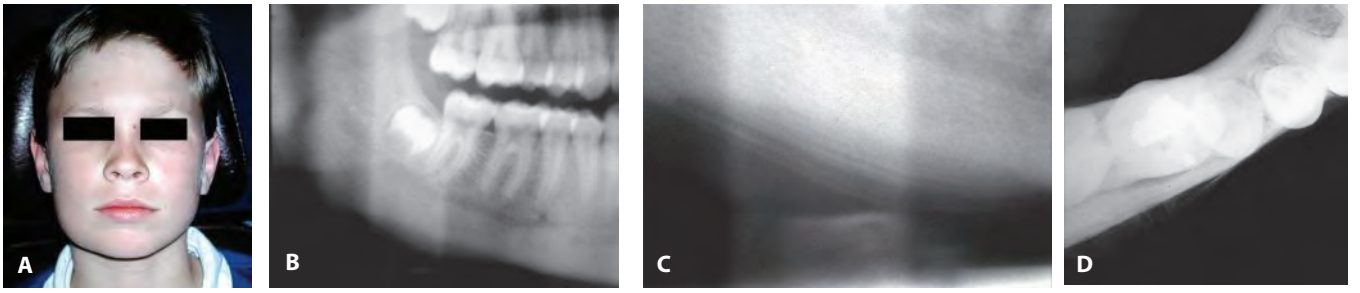
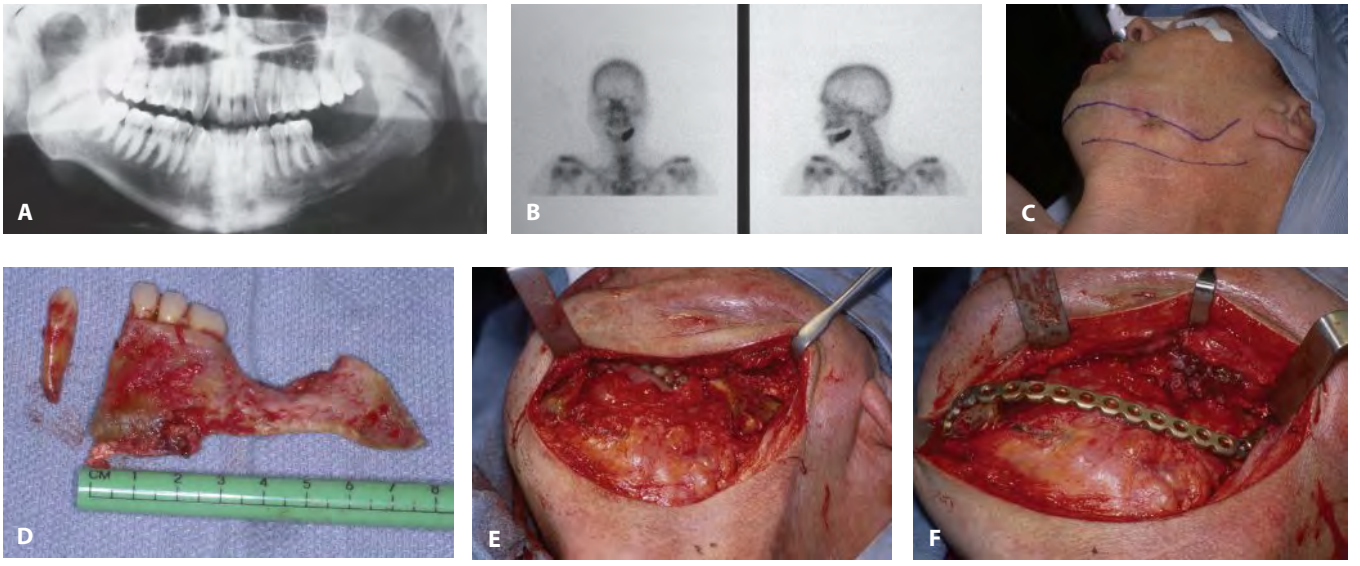


FIGURE 39-4. **A,** Facial view of a 13-year-old male, otherwise healthy. Note the swelling of the right mandible posterior body. **B,** Close-up of the panoramic view of the right mandible. Note the proliferative periostitis at the inferior border that is characteristic of Garré's osteomyelitis. **C,** Close-up of the right mandible inferior border with classic "onion skin" appearance. **D,** Occlusal view of the right mandible showing "onion skin" appearance. (Courtesy of Dr. Mark Bernstein)



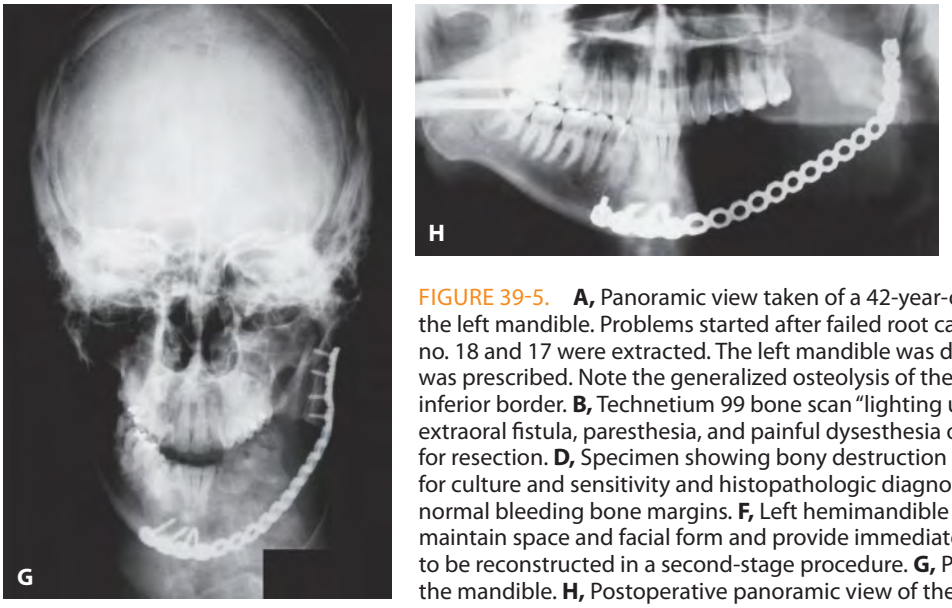


FIGURE 39-5. **A,** Panoramic view taken of a 42-year-old male with pain and swelling of the left mandible. Problems started after failed root canal treatment on tooth no. 18. Teeth no. 18 and 17 were extracted. The left mandible was débrided and oral antibiotic treatment was prescribed. Note the generalized osteolysis of the left mandible with dissolution of the inferior border. **B,** Technetium 99 bone scan "lighting up" the left mandible. **C,** Patient with extraoral fistula, paresthesia, and painful dysesthesia of the left mandible that was scheduled for resection. **D,** Specimen showing bony destruction of the left mandible. Tissue was sent for culture and sensitivity and histopathologic diagnoses. **E,** Surgical site showing defect and normal bleeding bone margins. **F,** Left hemimandible with reconstruction plate in place to maintain space and facial form and provide immediate function. The patient's mandible was to be reconstructed in a second-stage procedure. **G,** Postoperative anteroposterior view of the mandible. **H,** Postoperative panoramic view of the mandible.

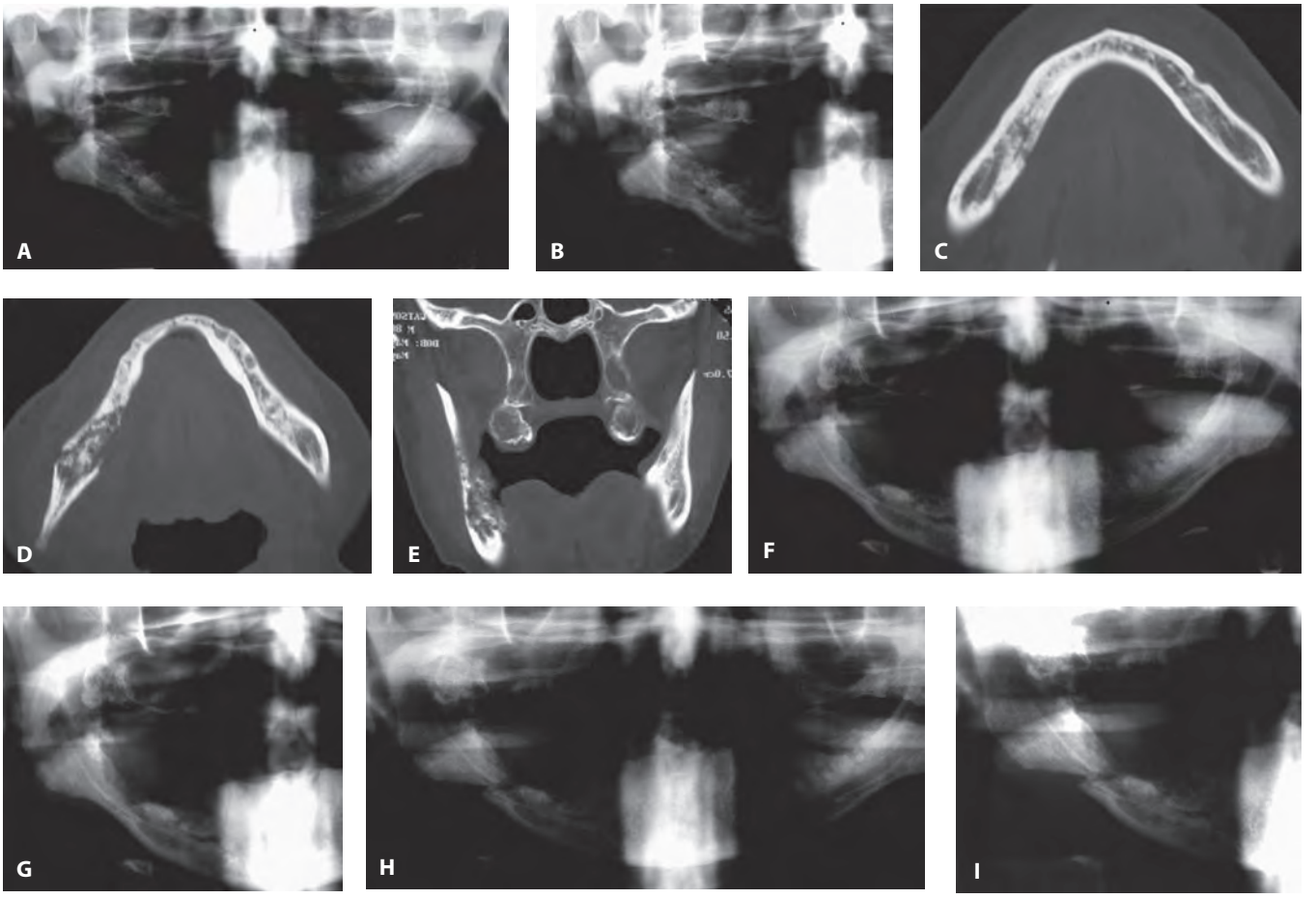


FIGURE 39-6. **A,** Panoramic view taken of a 70-year-old male with pain and swelling in the right mandible. Note the sclerotic lesion in the right mandible. **B,** Close-up of a panoramic view showing sclerotic lesion in the right mandible. Incisional biopsy revealed osteomyelitis. **C,** Axial computed tomography (CT) scan showing sclerotic lesion of the right mandible. **D,** Axial CT scan showing lesion of the right mandible. **E,** Coronal CT scan showing sclerotic lesion of the right mandible with areas of "moth-eaten" bone. **F,** Panoramic view of the right mandible after débridement back to good bleeding bone. **G,** Close-up of a panoramic view showing a weakened area of the right mandible. **H,** Panoramic view of the mandible 3 months postoperatively. The patient had heard a "pop" while chewing. **I,** Close-up of a panoramic view showing pathologic fracture of the right mandible. **J,** Open reduction and rigid internal fixation of pathologic fracture of the right mandible.

Treatment

The management of osteomyelitis of the maxillofacial region requires both medical and surgical interventions. In rare cases of infantile osteomyelitis, intravenous antibiotic therapy alone may eradicate the disease. Antibiotic therapy is rarely curative in later-onset cases, and the overwhelming majority of osteomyelitis cases require surgical intervention.

Clearly the first step in the treatment of osteomyelitis is diagnosing the condition correctly. The tentative diagnosis is made from clinical evaluation, radiographic evaluation, and tissue diagnosis. The clinician must be aware that malignancies can mimic the presentation of osteomyelitis and must be kept in the differential diagnosis until ruled out by tissue histopathology (Figure 39-7). Tissues from the affected site should be sent for Gram stain, culture, sensitivity, and histopathologic evaluations. The clinical response to the treatment of any patient will be compromised unless altered host factors can be optimized. Medical evaluation and management in defining and treating any immunocompromised state is indicated and often helpful. For example, glucose control in a diabetic patient should be stabilized for best response to therapy.

Empiric antibiotic treatment should be started based on Gram stain results of the exudate or the suspected pathogens likely to be involved in the maxillofacial region. Definitive culture and sensitivity reports generally take several days or longer to be obtained but are valuable in guiding the surgeon

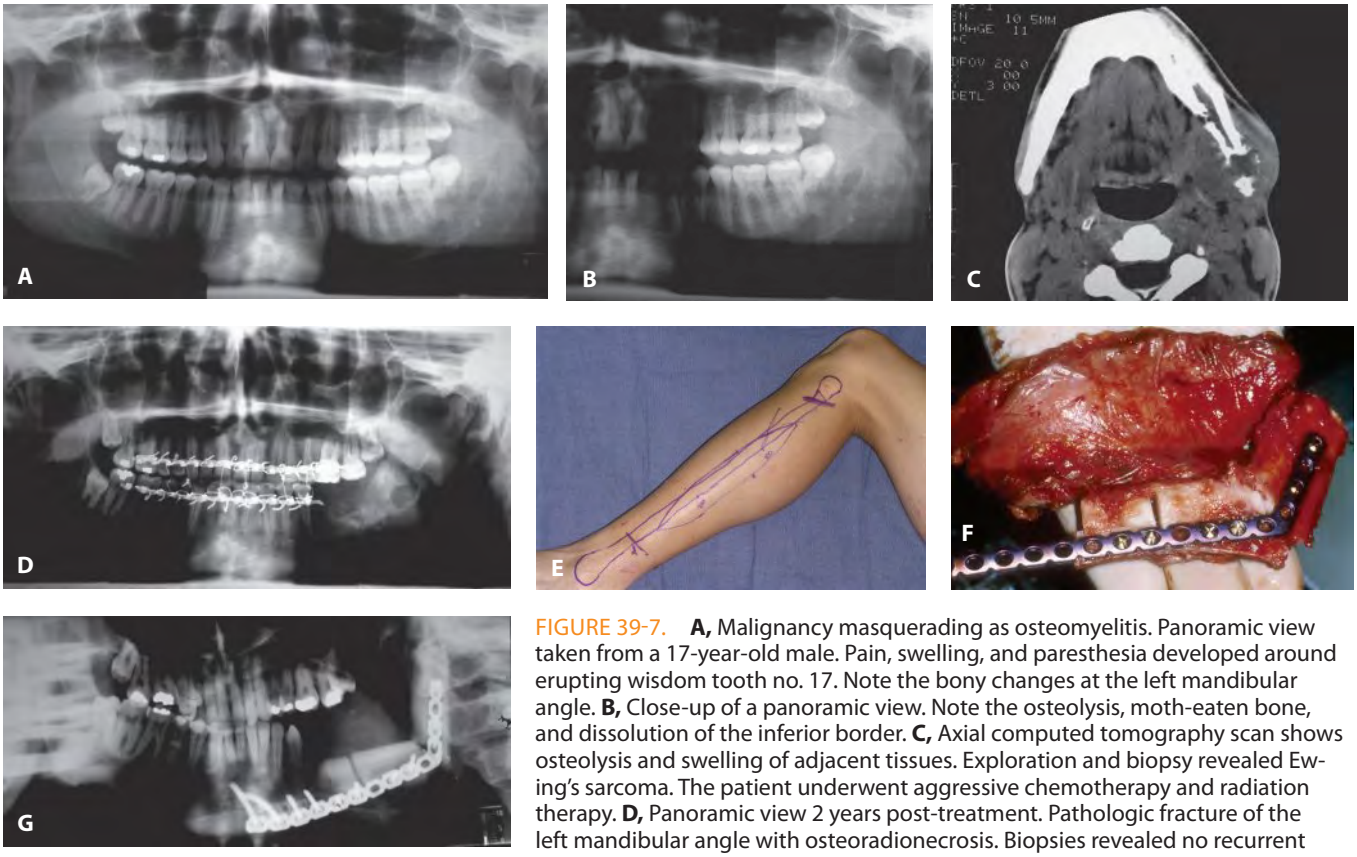


FIGURE 39-7. **A,** Malignancy masquerading as osteomyelitis. Panoramic view taken from a 17-year-old male. Pain, swelling, and paresthesia developed around erupting wisdom tooth no. 17. Note the bony changes at the left mandibular angle. **B,** Close-up of a panoramic view. Note the osteolysis, moth-eaten bone, and dissolution of the inferior border. **C,** Axial computed tomography scan shows osteolysis and swelling of adjacent tissues. Exploration and biopsy revealed Ewing's sarcoma. The patient underwent aggressive chemotherapy and radiation therapy. **D,** Panoramic view 2 years post-treatment. Pathologic fracture of the left mandibular angle with osteoradionecrosis. Biopsies revealed no recurrent malignancy. **E,** Fibula being prepared for free tissue transfer after resection of the left mandibular angle region. **F,** Fibula with osteotomies to create mandibular contour. Note the healthy soft tissue skin paddle attached. **G,** Panoramic view of the free fibula flap reconstruction of the left mandible.

to the best choice of antibiotics based on the patient's specific causative organisms.[13] Infectious disease consultation may illustrate the most current antimicrobials and/or regimens.

Surgical Options

Classic treatment is sequestrectomy and saucerization. The aim is to débride the necrotic or poorly vascularized bony sequestra in the infected area and improve blood flow. Sequestrectomy involves removing infected and avascular pieces of bone—generally the cortical plates in the infected area. Saucerization involves the removal of the adjacent bony cortices and open packing to permit healing by secondary intention after the infected bone has been removed. Decortication involves removal of the dense, often chronically infected and poorly vascularized bony cortex and placement of the vascular periosteum adjacent to the medullary bone to allow increased blood flow and healing in the affected area. The key element in the above procedures is determined clinically by cutting back to good bleeding bone. Clinical judgment is crucial in these steps but can be aided by preoperative imaging that shows the bony extent of the pathology. It is often necessary to remove teeth adjacent to an area of osteomyelitis. In removing adjacent teeth and bone the clinician must be aware that these surgical procedures may weaken the jaw bone and make it susceptible to pathologic fracture (see Figure 39-6).

Supporting the weakened area with a fixation device (external fixator or reconstruction type plate) and/or placing the patient in maxillomandibular fixation is frequently used to prevent pathologic fracture. Indeed, we have primarily grafted such areas when the sequestrectomy and saucerization have been deemed adequate.

Some authors have proposed adjunctive treatment methods that deliver high doses of antibiotic to the area using antibiotic impregnated beads or wound irrigation systems.[14–16] This therapy works on the premise that high local levels of antibiotics are made available and the overall systemic load is very low, thus reducing the possible side effect and complication rate.

Hyperbaric oxygen (HBO) treatment has also been advocated for the treatment of refractory osteomyelitis. This treatment method works by increasing tissue oxygenation levels that would help fight off any anaerobic bacteria present in these wounds. The widespread use of HBO treatment of osteomyelitis still remains controversial.

Resection of the jaw bone has traditionally been reserved as a last-ditch effort, generally after smaller débridements

have been performed or previous therapy has been unsuccessful or to remove areas involved with pathologic fracture. This resection is generally performed via an extraoral route, and reconstruction can be either immediate or delayed based on the surgeon's preference. Rigid internal fixation has simplified the postoperative course by providing a means for immediate function of the jaws.

We believe that early resection and reconstruction shorten the course of treatment. Once the patient develops paresthesia in mandibular osteomyelitis, resection and immediate reconstruction are indicated. At this point preservation of the mandible is highly unlikely and one should attempt to shorten the course of the disease and treatment (Figure 39-8).

OSTEORADIONECROSIS

Radiation therapy is a valuable treatment modality in treating cancer of the maxillofacial region. Radiation therapy can be used alone or as adjunctive therapy in combination with surgery and chemotherapy. Radiation therapy like any treatment modality has deleterious side effects, including mucositis and xerostomia. One of the most dreaded side effects is osteoradionecrosis (ORN). Historically, ORN was felt to represent a radiation-induced osteomyelitis. However, Marx has shown that osteoradionecrosis represents a chronic nonhealing wound that is hypoxic, hypocellular, and hypovascular.[17] In years past, the radiation therapist used orthovoltage therapy and there was a high incidence of ORN. However, the modern radiation therapists use megavoltage, which is felt to be kinder to the bone and soft tissues. In addition, collimation and shielding of tissues in conjunction with careful dental evaluation preoperatively have greatly decreased the incidence of ORN. The effects of radiation last a lifetime and do not decrease over time.

ORN is generally caused by trauma to the radiated area, usually by dental extraction, but it can also occur spontaneously. The clinical picture of ORN is most commonly seen with pain and exposed bone in the maxillofacial region (Figures 39-9 and 39-10). ORN is more common in the mandible than in the maxilla for reasons described earlier in this chapter. A dosage of radiation above 5,000 to 6,000 rads is generally felt to make the mandible susceptible to ORN.

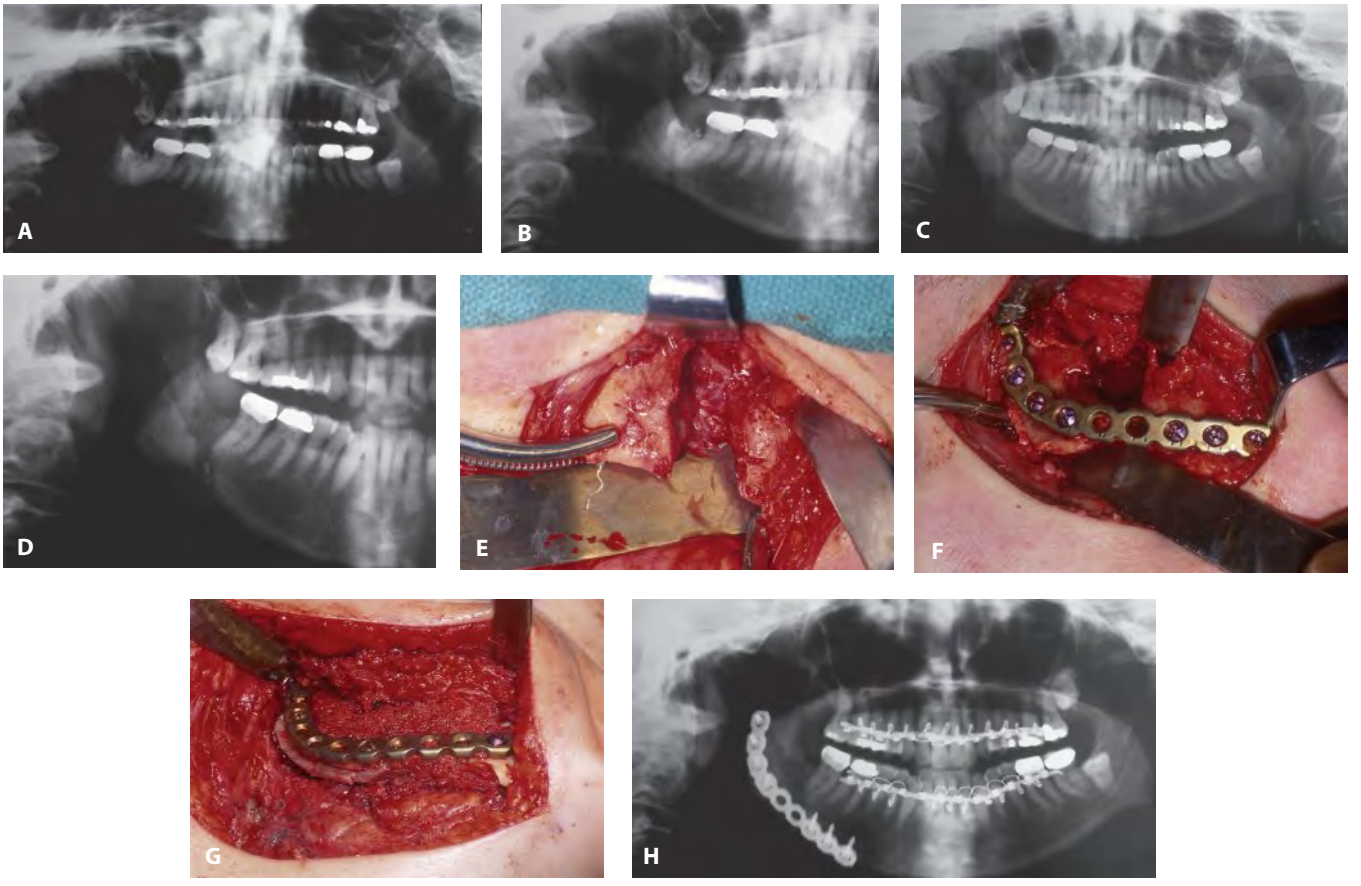


FIGURE 39-8. **A,** Panoramic view taken of a 64-year-old female with symptomatic tooth no. 32 scheduled for extraction. **B,** Close-up of a panoramic view showing decay in partially impacted tooth no. 32. **C,** Panoramic view of the mandible with pain, swelling, and paresthesia of the right mandible. **D,** Close-up of a panoramic view showing pathologic fracture with bone sequestrum at the right mandibular angle region. **E,** Right angle débrided via an extraoral approach. **F,** Rigid fixation applied to a "defect fracture." No bony contact is present after osteomyelitis is débrided to normal bleeding time. **G,** The patient receives an autogenous bone graft as part of primary surgery. **H,** Panoramic view of débridements and reconstruction as a one-stage procedure.



FIGURE 39-9. **A,** Panoramic view of the mandible post-radiation in a patient with oral squamous cell carcinoma. Note the large bony sequestrum. **B** and **C,** Intraoral views of the right and left mandible showing exposed bone. **D,** Transoral débridements of osteoradionecrosis. **E,** Specimen of the mandible, essentially "lifted out" of the tissue bed. **F** and **G,** Lateral and frontal views after removal of the mandible involved with osteoradionecrosis. The remaining deformity is commonly known as "Andy Gump" deformity.

Radiographically, the appearance on the orthopantomogram or CT scan resembles conventional osteomyelitis with areas of osteolysis and bony sequestrum. Often there is an appearance of moth-eaten bone present on these films.



FIGURE 39-10. Rare case of maxillary osteoradionecrosis. Clinical presentation is one of exposed bone and pain.

The treatment of ORN is aimed at removing the nonviable (necrotic) tissue and allowing the body to heal itself. The clinician must always be aware that tissue removed in a prior cancer patient should be sent to pathology to rule out occult or recurrent malignant disease that is masquerading as a bony infection. Minor débridements of exposed bone may work in the most minor cases of ORN. Current therapy calls for augmentation of tissue healing response by the use of HBO. HBO therapy consists of 100% oxygen delivered in a pressurized manner. Tissues treated with HBO have increased levels of oxygen, which has a negative effect on bacteria and a positive effect on angiogenesis and increased blood flow to the area. HBO has been used effectively to treat ORN and as an adjunctive treatment with maxillofacial reconstructive procedures such as dental extractions, dental implants, and jaw reconstruction in the radiated patient.

HBO treatment consists of dives or treatment sessions for 90 minutes based at 2.4 atm of pressure. Twenty to 30 dives are given preoperatively before any surgical intervention is

performed. The area of ORN is then débrided and followed with 10 additional HBO treatments. Reconstruction of the maxillofacial region is based on the patient's response to the treatment protocol. HBO treatments are expensive and facilities are often scarce, available only in larger cities with medical centers or academic health science centers.

With the addition of microvascular surgery to the surgical armamentarium, there now exists an excellent surgical option in treatment of the patient with ORN. Microvascular surgery (free flaps) allows the surgeon to bring in hard and soft tissues that have their own independent blood supply. The fibula, iliac crest, scapula, and radius are all considered applicable donor sites.[18,19] The fibula is very popular in maxillofacial reconstruction as the surgeon can bring an excellent length of bone which can be osteotomized and fabricated into a new mandible.[20,21] There is an excellent skin paddle to provide soft tissue coverage (see Figure 39-7). The microvascular flap is plugged into the facial vessels or the carotid artery and jugular vein system for blood supply and drainage. The clinical advantage of microvascular surgery is that the surgeon does not have to rely on a compromised host bed from radiation therapy or a lack of soft tissue, which very often occur in ablative cancer surgery. In addition HBO treatments are not necessary with microvascular surgery. Lastly dental implant reconstruction has been used with free tissue transfer techniques and has proven successful in the dental reconstruction of these patients.[22]

BISPHOSPHONATE-RELATED OSTEONECROSIS OF THE JAWS

A new pathologic entity was discovered in 2001–2002 when clinicians noticed an increased incidence of exposed alveolar bone and refractory osteomyelitis in patients taking medications in a new class of drugs named *bisphosphonates* (Figure 39-11). Dental patients had common procedures such as extraction of teeth with the postoperative complication of nonhealing, exposed jawbone.[2] Spontaneous exposure of the alveolar jawbone was also reported. Bisphosphonates were used to treat bony sequelae of malignant disease such as multiple myeloma and metastatic disease, very typically, breast cancer. These patients commonly had growing destructive lesions in bone that caused two major problems. The hypercalcemia of malignancy is seen when bone is destroyed by the bony lesions. The weakened bones are also susceptible to pathologic fracture. Bisphosphonates acted by decreasing bony resorption. Patients who were treated with bisphosphonates had the progression of their bony lesions either halted or slowed. This left patients with less pain, fewer pathologic fractures, and a better quality of life. This was certainly a huge benefit to the medical oncology community and their patients. Bisphosphonate medications can also be used to treat Paget's disease of bone and osteogenesis imperfecta.



FIGURE 39-11. Exposed bone and nonhealing extraction site of the mandible in patient receiving IV Zometa for multiple myeloma.

With the excellent response seen by the medical oncology community, the use of bisphosphonates became more common.[23–26] Patients went about their day-to-day living, which included routine dental care. The dental profession began seeing the complications in bisphosphonate patients after seemingly routine procedures such as extractions and implant placement. The medical and dental clinicians were first alerted to this growing problem in 2003.[2]

The new pathologic entity evolved to the name of bisphosphonate-related osteonecrosis of the jaws and used the acronym BRONJ. To establish a diagnosis of BRONJ, patients must have exposed jawbone for at least 8 weeks, a prior or current history of bisphosphonate treatment, and no history of radiation therapy to the jaws (Table 39-1).[27,28]

Bisphosphonates alter normal bone metabolism. Bone is constantly undergoing remodeling via bone resorption and bone deposition. Bisphosphonates inhibit osteoclast activity and, thus, prevents bone breakdown. Bisphosphonates also affect osteoblast activity, which in turn, secondarily affects osteoclast activity. The combined effect of bisphosphonates significantly alters the normal process of bone deposition and resorption. In addition, recent findings suggest that bisphosphonates also affect soft tissue wound-healing cells.[29] The combined hard and soft tissue healing alterations may lead to the complex process of BRONJ, which is still not completely understood. In addition, the half-life of this medication is long, often uncertain, but measured in years.

TABLE 39-1. **List of Bisphosphonates**

A. Etidronate (Didronel)
B. Tiludronate (Skelide)
C. Pamidronate (Aredia)
D. Zolendrenic acid (Zometa, Reclast)
E. Alendronate (Fosamax)
F. Risedronate (Actonel)
G. Ibandronate (Boniva)

Intravenous bisphosphonates had a well-established clinical efficiency in treating hypercalcemia of malignancy and bony metastases. Oral bisphosphonates were used to treat osteoporosis with worldwide acceptance. Osteoporosis has significant adverse health care implications that include hip fracture and vertebral fractures and is a major public health care initiative.[30,31] It became clear to clinicians that the incidence of BRONJ was higher with intravenous bisphosphonate therapy than with oral therapy. Many studies have tried to establish the incidence of BRONJ with both oral and intravenous therapy but have been hampered by underreporting of the disease.

The American Association of Oral and Maxillofacial Surgeons (AAOMS) developed a task force to provide the best recommendations to clinicians in management of BRONJ. The recommendations were meant to give clinicians a guide in management of bisphosphonate patients.[32] The recommendations were expected to be modified as scientific data were accumulated and studied.

Patients about to Initiate Bisphosphonate Therapy

The goal in this group is to prevent the development of BRONJ. If at all possible, the initiation of bisphosphonate therapy should be delayed until oral/dental health is optimized. This will mandate cooperation and communication between medical and dental colleagues. Non-restorable teeth and teeth with poor prognoses should be extracted. It is recommended to allow 14 to 21 days to allow adequate healing before starting bisphosphonate therapy. Other necessary dentoalveolar surgery, such as tori removal or alveoloplasty, should be done at this time. Restorative dentistry, scaling, and prophylaxis are best completed before initiation of bisphosphonate therapy. Patients with complete or partial dentures should be evaluated for areas of potential mucosal trauma or irritation. Patients should be educated about the importance of good oral and dental hygiene during bisphosphonate therapy, similar to instructions given to patients undergoing radiation therapy to the jaws. Routine dental examinations and evaluations are important to monitor patient compliance and to detect problems early rather than at a later stage.

Patients Receiving Intravenous Bisphosphonates

Patients should maintain good oral and dental hygiene in order to prevent problems that require osseous surgery. However, there will always be patients who have situations arise that are unexpected and unpredictable. Dental procedures that involve bony surgery should be avoided. Endodontic therapy is preferable to extraction of teeth. Placement of dental implants should be avoided. The incidence of developing BRONJ in patients receiving intravenous bisphosphonates is higher than in patients receiving oral bisphosphonate therapy.

Patients Receiving Oral Bisphosphonates

The risk of BRONJ appears to be associated with a longer duration of therapy, 3 years or greater. Scientifically based recommendations are lacking at this point. Recommendations are based on expert opinion and will be modified as the scientific data become available and are analyzed. In patients who have taken oral bisphosphonates for less than 3 years and have no clinical risk factors, no alteration in the planned dentoalveolar treatment is required. Risk factors include conditions that may adversely affect healing, such as steroid treatment, diabetes and other medications, and medical conditions that would make the patient immunocompromised.

In patients who have taken oral bisphosphonates for less than 3 years but have clinical risk factors, a "drug holiday" of at least 3 months should be considered before dentoalveolar surgery. This will require communication between the dentist and the physician to make the judgment if the systemic condition permits this action.

In patients who have taken oral bisphosphonates for greater than 3 years (with or without risk factors), a drug holiday of 3 months should be considered before dentoalveolar surgery. This will again require communication between the dentist and the physician to evaluate the overall systemic condition of the patient.

In addition, informed consent of possible complications of BRONJ should be discussed with the patient before any dentoalveolar surgery. Many surgeons have evolved to using written informed consent forms with their patients, and customized forms specific to BRONJ are now available.

Despite clinicians' and patients' best efforts, there is still the possibility of developing BRONJ in our patient population. BRONJ may occur after a dentoalveolar surgery, denture irritation, or spontaneously (Figure 39-12). The usual presenting symptoms will be exposed necrotic bone, with or without pain. The AAOMS has proposed a clinical staging protocol in their 2009 position paper (Table 39-2).



FIGURE 39-12. Exposed symptomatic bone on maxillary torus. The torus should have been removed before starting bisphosphonate therapy.

TABLE 39-2. **Patients at Risk**

No apparent necrotic bone in asymptomatic patients who have been treated with IV or oral bisphosphonates.

Stage 0
Patients with no clinical evidence of necrotic bone, but have non-specific clinical and radiographic findings such as pain and change in the bony trabecular pattern of the jaws.

Stage 1
Patients have exposed and necrotic bone who are asymptomatic and show no sign of infection.

Stage 2
Patients have exposed and necrotic bone who are symptomatic with pain and infection.

Stage 3
Patients have exposed and necrotic bone who are symptomatic with pain and infection and one or more of the following:

- Exposed and necrotic bone extending beyond the alveolar bone.
- Pathologic fracture.
- Extraoral fistula.
- Oral-antral or oral-nasal communication.
- Osteolysis extending to the inferior border of the mandible or maxillary sinus floor.

Treatment strategies are based on the stage of the disease. The guidelines are meant to provide recommendations and not dictate treatment. The personal experience of the clinician and overall condition of the patient will determine the ultimate treatment plan. As more scientific data become available, treatment recommendations will be modified to reflect the evidence-based decision-making process that is most desirable. In patients who are at risk for BRONJ, no specific treatment is required other than educating the patient of the risks involved and the signs and symptoms of the disease. Routine dental examinations and good oral/dental hygiene are emphasized.

In stage 0 patients, clinical follow-up is recommended with conservative treatment. Clinicians should provide symptomatic treatment such as pain control and manage any treatable condition such as dental caries and periodontal disease.

In stage 1 patients, oral anti-microbial rinses, such as chlorhexidine 0.12%, are indicated. Close monitoring of the oral condition and maintaining good oral hygiene are recommended. Surgical treatment is generally not recommended. Sharp bony edges that are irritating oral tissues may be smoothed down.

In stage 2 patients, treatment is more aggressive. Oral antimicobial rinses are recommended in conjunction with antibiotic therapy. BRONJ is not due to a primary infectious etiology. However, secondary infections are seen. Penicillin is still effective in the management of BRONJ with infection. Quinolones, metronidazole, clindamycin, doxycycline, and erythromycin have been used empirically in penicillin-allergic patients. Culture and sensitivity of infected bone may be considered to more appropriately direct antimicrobial therapy. Refractory cases may require intravenous antibiotic treatment along with maintenance or suppressive treatment to manage the disease.



FIGURE 39-13. Panorex film of a patient with stage 3 Bisphosphonate-related osteonecrosis of the jaws (BRONJ) with pathologic fracture of the right mandible.

In stage 3 patients, surgical débridement, including resection, is used in conjunction with antibiotic treatment. Resection is generally reserved for extensive, late presentation of disease and pathologic fractures (Figures 39-13 to 39-17).

Mobile segments of bony sequestrum should be removed regardless of the disease stage. Care should be taken not to expose the uninvolved bone for fear of extending the disease process. Symptomatic teeth in exposed necrotic bone should be extracted. Clinical controversy arises with regard to stopping the bisphosphonate medication during active treatment for BRONJ. Stopping intravenous bisphosphonates appears to offer no short-term benefit. However, long-term cessation of bisphosphonate therapy may be beneficial in patient healing. Each case requires careful consultation between medical and dental colleagues who can establish the risk/benefit ratio of bisphosphonate therapy in that specific patient, based on the overall systemic condition and co-morbidity factors. Stopping

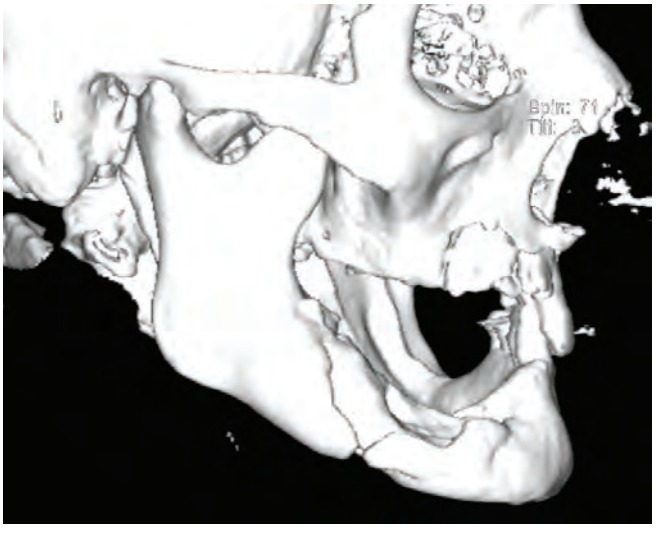


FIGURE 39-14. Three-dimensional computed tomography (CT) scan of a patient with stage 3 BRONJ with pathologic fracture of the right mandible.



FIGURE 39-15. Extraoral fistula of a patient with stage 3 BRONJ with pathologic fracture of the right mandible.

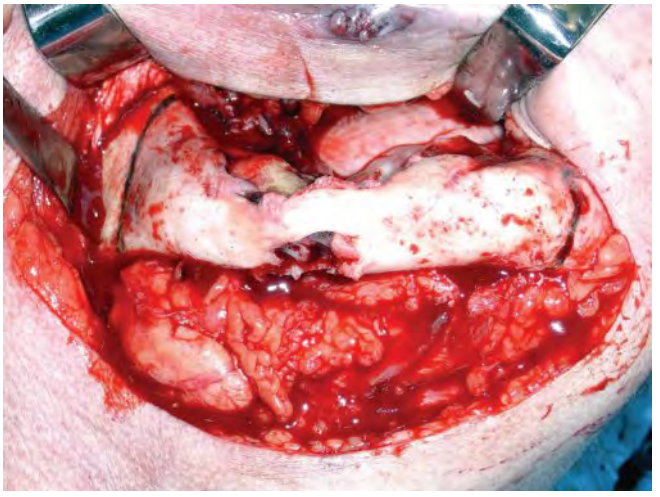


FIGURE 39-16. Planned resection of a patient with stage 3 BRONJ with pathologic fracture of the right mandible.



FIGURE 39-17. Resected right mandible of a patient with stage 3 BRONJ and pathologic fracture.

oral bisphosphonate treatment in patients with BRONJ has shown gradual improvement in the disease process.[32]

Researchers are actively studying BRONJ to further elucidate the etiology and possibly direct effective treatments or preventive strategies. Hyperbaric oxygen (HBO) is currently being evaluated.[33,34] Extracorporeal shockwave therapy is also being evaluated. Clinicians are evaluating new techniques such as using platelet-rich plasma to aid in healing after dental extractions in bisphosphonate therapy patients.[35] The scientific knowledge base will surely continue to grow and we will gain a better understanding of BRONJ.

Recently, oral bisphosphonate therapy patients were found to have an unusual complication. Long-term patients were noted to have spontaneous fractures in their long bones, specifically the femur. Long term is felt to be greater than 5 years but some fractures occurred after 2 years of oral bisphosphonate therapy. These fractures occurred with walking and low-energy injuries. There was generally a prodromal symptom of pain in the affected area. This has caused intense scrutiny as bisphosphonate medications were prescribed to prevent fractures.[36–38] This has caused clinicians to reevaluate their recommendations for oral bisphosphonate therapy treatment. Proposals have included drug holidays in which patients do not receive oral bisphosphonate medications for a period of time and allow the bone to resume a more normal state of resorption and deposition. The final therapeutic regimen of oral bisphosphonate therapy has yet to be determined.

CONCLUSION

Osteomyelitis and osteoradionecrosis present an ongoing and potentially difficult clinical scenario to manage. Many patients will receive a combination of surgery and medical management to adequately heal from these diseases. Some patients will be required to undergo extensive and potentially disfiguring surgery to manage their disease. The medical management, including antibiotic therapy and HBO treatment, may be expensive, time consuming, and disruptive to the patient's life. Both of these conditions can be started with something as innocuous and common as a *dental extraction*.

Clinicians must always be vigilant for post-treatment complications, including osteomyelitis and osteoradionecrosis. Despite advances in both medical management and surgical therapy, the absolute answer to the prevention and/or oral management of osteomyelitis and osteoradionecrosis has yet to be found.

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SECTION 5

Maxillofacial Reconstruction

40

CHAPTER

Local and Regional Flaps

Alan S. Herford, DDS, MD, and G. E. Ghali, DDS, MD

FLAP PRINCIPLES

Over the past 50 years, the development and application of several different flaps has led to reliable reconstruction of facial defects. Most defects can be reconstructed immediately, leading to better restoration of form and function with early rehabilitation.[1] Reconstructing facial defects can be both challenging and rewarding. Missing tissue most often results from either trauma or oncologic surgery. Commonly, a wide range of options is available for repairing a given defect, including healing by secondary intention, primary closure, placement of a skin graft, or mobilization of local or regional tissue. Compared with skin grafts, local flaps often produce superior functional and aesthetic results.[2–6] A great advantage of local tissue transfer is that the tissue closely resembles the missing skin in color and texture. These flaps can be rotated, advanced, or transposed into a tissue defect. Regional tissue can also be recruited to repair facial defects.

When deciding which option to use, there should be a progression from simple to complex treatments. Consideration should be given to primary closure or the use of skin grafts first, followed by local, then regional, and finally, distant pedicled or microsurgical free tissue transfer. Flaps require additional incisions and tissue movement, which increase the risks of postoperative bleeding, hematoma, pain, and infection. Confirmation of tumor-free margins should be done before flap reconstruction if a malignant lesion has been excised.[7]

Some defects are amenable to closure with a single flap, but others require a combination of flaps for optimal results.[8] An advantage of using multiple flaps is that they can be harvested from separate aesthetic units. This decreases the size of the secondary defect and may allow placement of scars between aesthetic units, thus improving scar camouflage, leading to better cosmesis. Often, separated repair of individual facial subunits with separate flaps provides a better cosmetic result than if a single flap is used to reconstruct the entire defect.

Flaps differ from grafts in that they maintain their blood supply as they are moved. Abundant dermal and subdermal plexus allow for predictable elevation of random cutaneous flaps. A cutaneous flap may also have its arterial supply based on a dominant artery in the subcutaneous layer. Muscular perforating arteries are important contributors to the cutaneous vascular bed. The most important variable for flap viability is not the length-to-width ratio but, rather, the perfusion pressure and vascularity at the pedicle base.[9] Because local flaps provide their own blood supply, they are particularly useful in patients with compromised recipient sites such as those that have been irradiated.

As local flaps heal, regaining of blood flow and cutaneous sensibility increases. The rate of blood flow and two-point discrimination on the surface of local flaps is statistically no different when compared with the corresponding area of the unoperated side.[10] The recovery of sensory nerve function in facial flaps is dependent on the intimacy of contact between the flap and the recipient bed and on the viability of the type of restoration.

Relaxed skin tension lines (RSTLs) result from vectors within the skin that reflect the intrinsic tension of the skin at rest. They are due to the microarchitecture of the skin and represent the directional pull on wounds. The RSTLs are generally parallel to the facial rhytids. Lines of minimal tension (rhytids) result from repeated bending of the skin from muscular contraction. A permanent crease results from the adhesions between the dermis and the deeper tissues. These natural skin creases run perpendicular to the direction of muscle pull and can guide incision orientation for optimal scar camouflage and cosmesis.

The face is composed of aesthetic subunits.[11,12] The areas where these subunits meet are referred to as *anatomic borders.* The aesthetic subunit principle is based on the fact

that our eyes see objects as a series of block images that are spatially organized. Scars that are located at the junction of two adjacent anatomic subunits are inconspicuous because one expects to see a delineation between these areas.

FLAP NOMENCLATURE

Many methods are described for classifying cutaneous flaps: by the arrangement of their blood supply, their configuration, location, tissue content, and method of transferring the flap.

Blood Supply

Cutaneous flaps consist of skin and subcutaneous tissue and can be characterized by their predominant arterial supply. These include random pattern, axial pattern, and pedicle flaps (Figure 40-1). *Random flaps* are supplied by the dermal and subdermal plexus alone and are the most common type of flap used for reconstructing facial defects. *Axial pattern flaps* are supplied by more dominant superficial vessels oriented longitudinally along the flap axis. *Pedicle flaps* are supplied by large named arteries that supply the skin paddle through muscular perforating vessels. *Free tissue transfer* refers to flaps that are harvested from a remote region and have the vascular connection reestablished at the recipient site.

Location

Another means of classification is by the region from which the tissue is mobilized. This includes local, regional, and distant flaps. *Local flaps* imply use of tissue adjacent to the defect, whereas *regional flaps* refer to those flaps recruited from different areas of the same part of the body. *Distant flaps* are harvested from different parts of the body.

Configuration

Flaps are often referred to by their geometric configuration. Examples of these flaps include *bilobed, rhombic,* and *Z-plasty.*

Tissue Content

The layers of tissue contained within the flap can also be used to classify a flap. *Cutaneous flap* refers to those flaps that contain the skin only. When other layers are incorporated into the flap they are classified accordingly. Examples include *myocutaneous* and *fasciocutanous flaps.*

Method of Transfer

The most common method of classifying flaps is based on the method of transfer. *Advancement flaps* are mobilized along a linear axis toward the defect (Figure 40-2). *Rotation flaps* pivot around a point at the base of the flap (Figure 40-3). Although most flaps are moved by a combination of rotation and advancement into the defect, the major mechanism of tissue transfer is used to classify a given flap. *Transposition flap* refers to one that is mobilized toward an adjacent defect over an incomplete bridge of skin. Examples of transposition flaps include rhombic flaps and bilobed flaps (Figure 40-4). *Interposition flaps* differ from transposition flaps in that the incomplete bridge of adjacent skin is also elevated and mobilized. An example of an interposition flap is a Z-plasty. *Interpolated flaps* are those flaps that are mobilized either over or beneath a complete bridge of intact skin

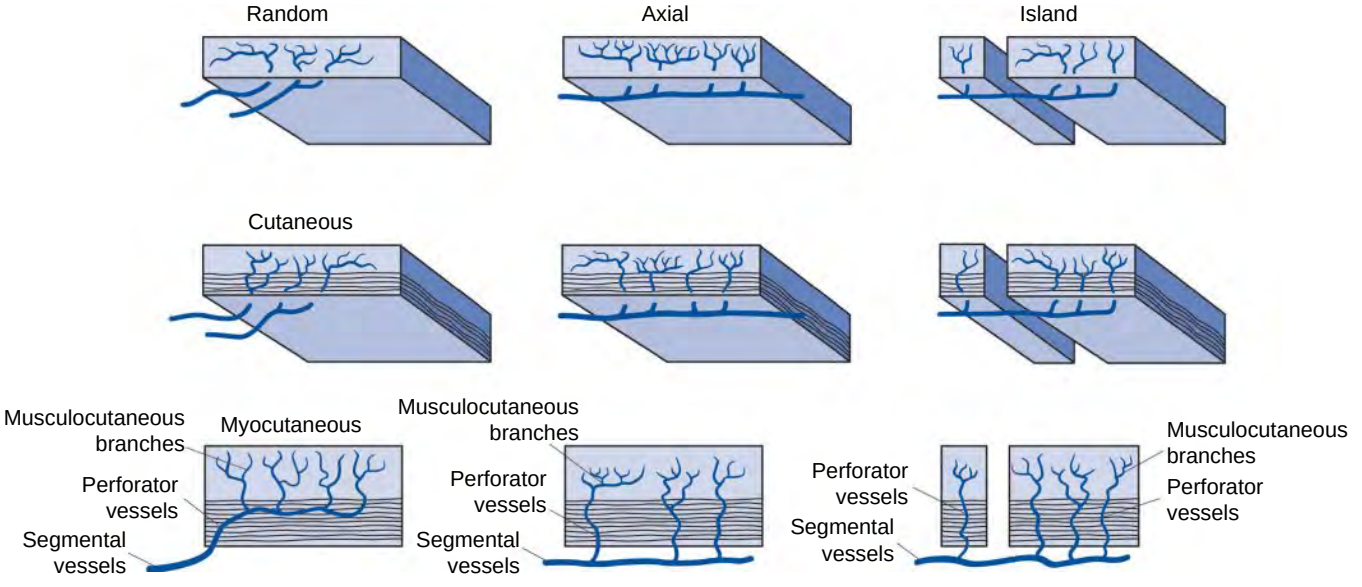


FIGURE 40-1. Diagrammatic representation of cutaneous blood supply in skin and myocutaneous flaps. (Adapted from Ariyan S. Pectoralis major, sternomastoid, and other musculocutaneous flaps for head and neck reconstruction. Clin Plast Surg 1980;7:89–109.)

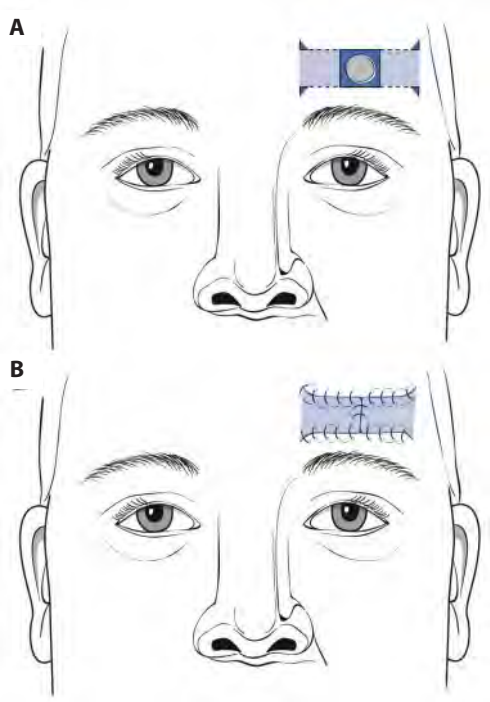


FIGURE 40-2. **A,** Double advancement flaps with Burow's triangles. **B,** Closure of the defect.

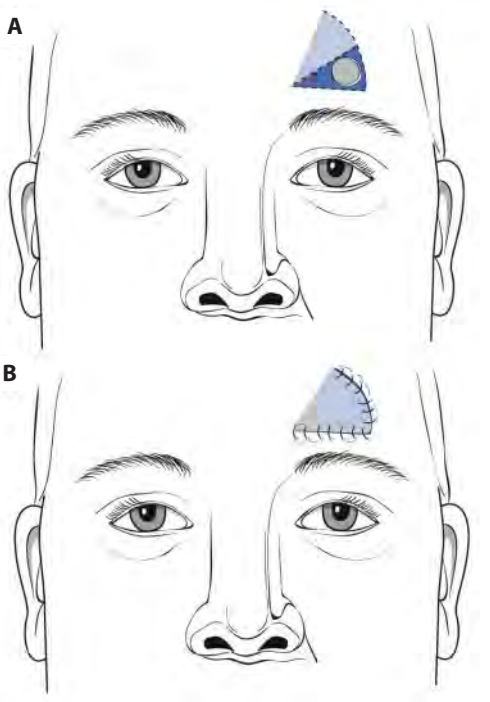


FIGURE 40-3. **A,** Rotation flap for closure of a forehead defect. **B,** Closure of the defect.

via a pedicle. These flaps often require a secondary surgery for pedicle division. Microvascular free tissue transfer from a different part of the body relies on reanastomosis of the vascular pedicle.

DESIGNING THE FLAP

Many options are available for reconstructing facial defects. Often, the optimal method is not readily apparent. A stepwise approach can be helpful in selecting and designing a flap. The characteristics of the defect and adjacent tissue must be analyzed. These include color, elasticity, and texture of the missing tissue. The defect size, depth, and location are evaluated as well as the availability and characteristics of adjacent or regional tissue. It is important to determine the mobility of adjacent structures and to identify those anatomic landmarks that must not be distorted. The orientation of the RSTLs and aesthetic units should by analyzed closely.

Potential flap designs should be drawn on the skin surface, being careful to avoid those designs that obliterate or distort anatomic landmarks. The final location of the resultant scar should be anticipated by previsualizing suture lines and choosing flaps that place the lines in normal creases.

The secondary defect created as the tissue is transferred into the primary defect must be able to be closed easily. When designing a flap, it is important to avoid secondary deformities that distort important facial landmarks or affect function. Avoid obliterating critical anatomic lines that are essential for normal function and appearance.

Proper surgical technique involves gentle handling of the tissue by grasping the skin margins with skin hooks or fine-toothed tissue forceps. Avoid traumatizing the vascular supply by twisting or kinking the base of the flap. Deep pexing sutures minimize tension on the flap and eliminate dead space. Excessive tension on the flap may decrease blood flow and cause flap necrosis. Meticulous hemostasis should be achieved before final suturing so that a hematoma does not develop beneath the flap. It is important to adequately mobilize and extend the flap, which should be of adequate size to remain in place without tension to minimize the chance of dehiscence, scarring, or ectropion.

TYPES OF FLAPS

Local Flaps

Advancement Flaps

Advancement flaps have a linear configuration and are advanced into the defect along a single vector. These flaps can be single or double. Advancement flaps are often chosen when the surrounding skin exhibits good tissue laxity and the resulting incision lines can be hidden in natural creases. Advancement flaps limit wound tension to a single vector with minimal perpendicular tension. They are often helpful in reconstructing defects involving the forehead, helical rim, lips, and cheek. In these areas, advancement flaps capitalize on the natural forehead furrows without causing vertical distortion of the hairline superiorly or the eyebrow inferiorly (Figure 40-5).

Advancement flaps are created by parallel incisions approximately the width of the defect. Standing cutaneous

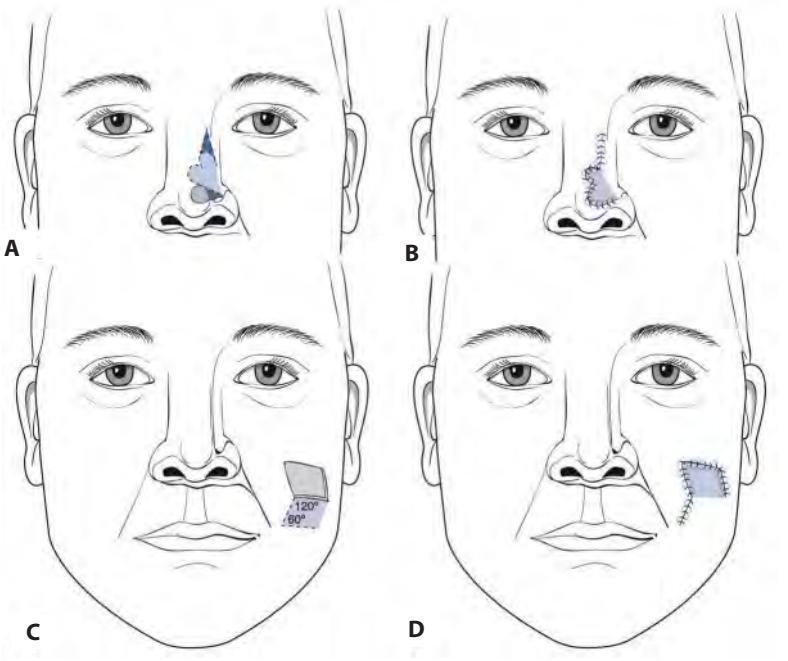


FIGURE 40-4. **A,** Bilobed flap for closure of a nasal tip defect. **B,** Closure of the defect. **C,** Rhombic flap for closure of a check defect. Note the 120- and 60-degree angles. **D,** Closure of the defect.

deformities ("dog-ears") are usually created and are managed with excision. A Z-plasty incision or Burow's triangle may be performed at the base of the flap, reducing the standing cutaneous deformities.

A variation of the advancement flap is the V-Y flap. A triangular island of tissue adjacent to the defect is isolated and attached only to the subcutaneous tissue. It relies on a subcutaneous pedicle for blood supply. As it is advanced into the defect, the secondary defect is closed primarily in a simple V-Y manner. These flaps are especially amenable for cheek defects along the alar facial groove and are generally avoided where there are superficial nerves because of the depth of the incisions.

Local flaps are also useful for staged recostruction of ear defects. Advancement of local tissue can be used to rebuild the missing tissue including the helical rim (Figure 40-6).

Intraoral uses of advancement flaps include covering oroantral fistulae and alveolar clefts. A disadvantage of buccal advancement flaps is the decrease in vestibular sulcus depth (Figure 40-7).

Rotation Flaps

Rotation flaps have a curvilinear configuration. Defects reconstructed with rotation flaps should be somewhat triangular or modified by removing normal tissue to create a triangular defect. These flaps have a large base and are usually

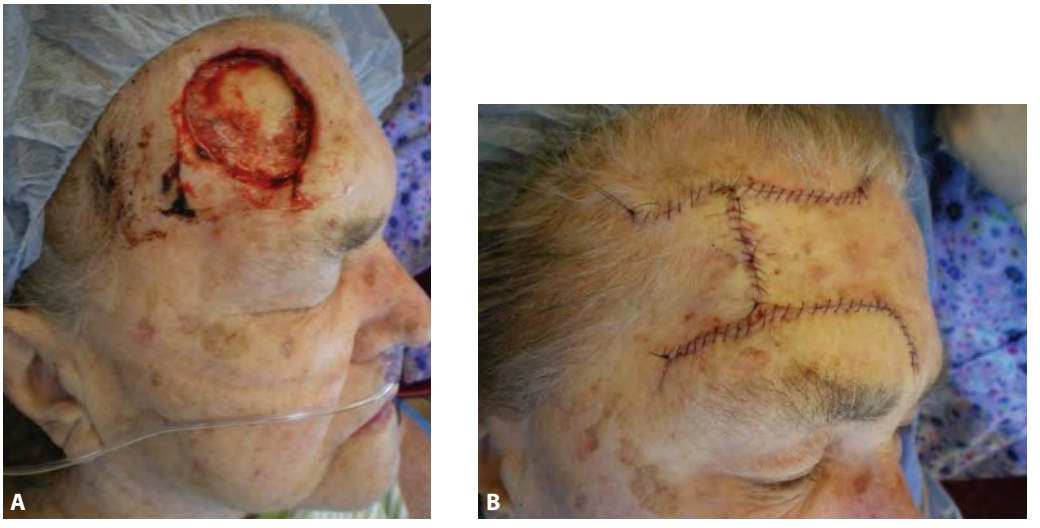


FIGURE 40-5. **A,** Large forehead defect. **B,** Closure utilizing bilateral advancement flaps combined with Burow's triangles.



FIGURE 40-6. **A,** Missing portion of posterior ear. **B,** A cartilage graft is placed. **C,** Coverage of the cartilage graft with advancement of tissue. **D,** Advancement of tissue to form the new helical rim. **E,** Healed ear.

random in their vascularity but may be axial. One or more rotation flaps are often used to reconstruct scalp defects. Because of the relative inelasticity of the scalp tissue, these flaps must be large relative to the size of the defect. Scoring of the galea is helpful in gaining additional rotation and advancement (Figure 40-8).

The axial frontonasal flap is a modified simple rotation flap with a back cut.[13–16] It is useful for closing nasal defects (Figure 40-9). The flap is based on a vascular pedicle at the level of the medial canthus. This pedicle consists of a branch of the angular artery and the supraorbital artery.

Rotated palatal flaps are helpful for closing large oroantral fistulae.[8,17] Fistulae less than 5 mm in diameter usually close spontaneously.[18,19] Local flaps or grafts can be used to close larger fistulae. Two-layer closures are less prone to developing recurrence of oroantral fistulae. Approximately 75% of the palatal soft tissue can be rotated to cover adjacent defects.

Transposition Flaps

These flaps are rotated and advanced over adjacent skin to close a defect. Examples of transposition flaps include rhombic flaps and bilobed flaps. These flaps are advantageous in areas where it is desired to transfer the tension away from closure of the primary defect and into the repair of the secondary defect. Transposition flaps have a straight linear



FIGURE 40-7. Buccal advancement flaps can be used to cover an oroantral fistula. **A,** A Moczair buccal sliding trapezoidal flap is slid *(arrow)* to use the papilla of the adjacent tooth to rotate into the defect. **B,** Rehrman's buccal advancement flap uses a flap that has vertical extensions. To adequately mobilize this flap to cover the defect without tension, the periosteum must be incised *(broken line)* along its base and the flap advanced *(arrow)* over the defect. **C,** If the fistula is present along an edentulous region, a transverse flap or bipedicle flap can be used.

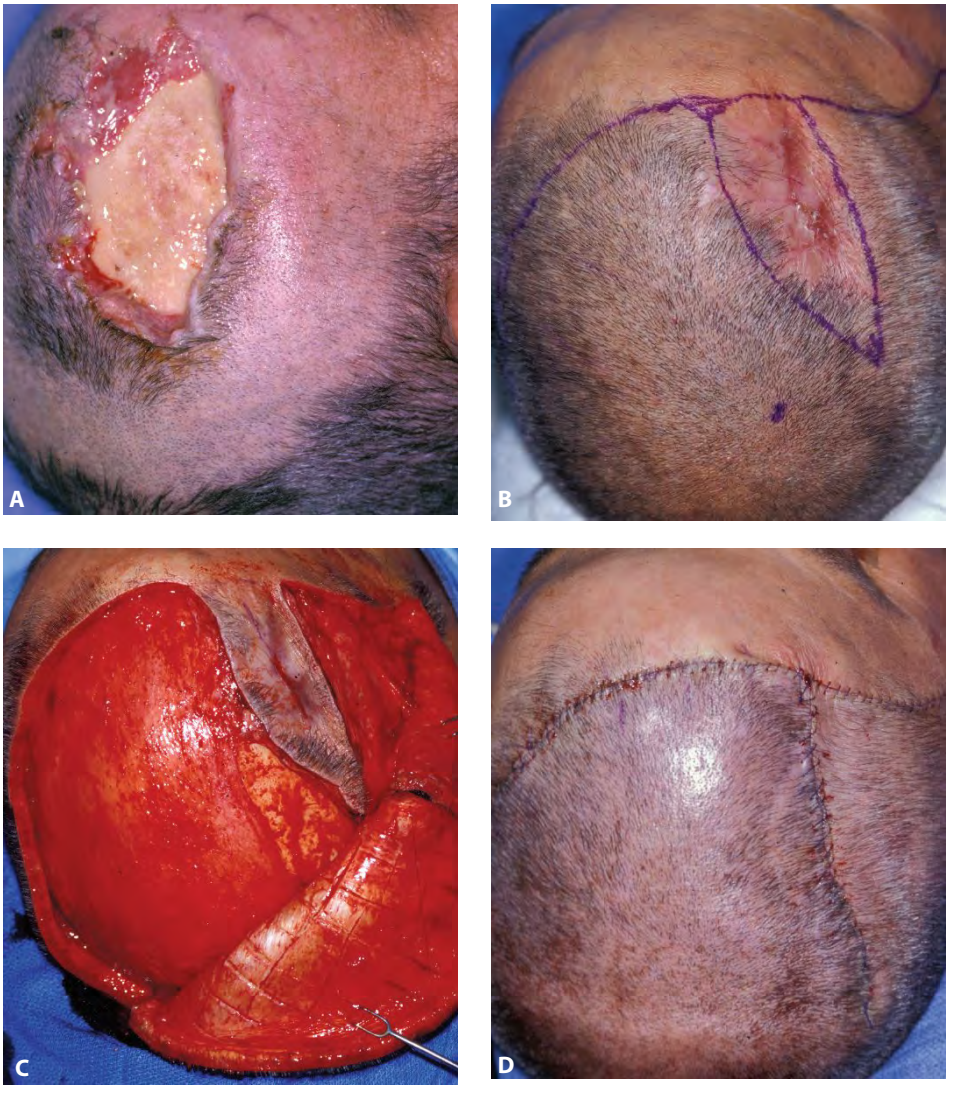


FIGURE 40-8. **A,** A large scalp defect secondary to trauma. **B,** Outline of scalp flaps. **C,** Elevation of flaps with scoring of the galea. **D,** Closure of the defect.



FIGURE 40-9. **A,** Axial frontonasal flap for repair of a nasal defect. **B,** Elevation of the flap with thorough undermining. **C,** Closure of the defect

axis and are usually designed so that one border of the flap is also a border of the defect. An advantage of this type of flap is that it can be developed at variable distances. Areas where these flaps are often used include the nasal tip and ala, the inferior eyelid, and the lips.

The rhombic flap is a precise geometric flap that is useful for many defects of the face.[20,21] The traditional rhombic ("Limberg") flap is designed with 60- and 120-degree angles and equal-length sides. The angle of the leading edge of the rhombic flap is approximately 120 degrees but may vary. The flap is begun by extending an incision along the short axis of the defect that is equal to the length of one side of the rhombic defect. Another incision is then made at 60 degrees to the first and of equal length (Figure 40-10). Disadvantages of the rhombic flap are the significant tension at the closure point as well as the amount of discarded tissue to transform a circular defect into a rhombus.

The bilobed flap is a transposition flap with two circular skin paddles (see Figure 40-4).[22,23] Esser is credited with the design of the bilobed flap in 1918. It is useful for skin



FIGURE 40-10. **A,** Nasal defect with missing skin and exposure of lower lateral cartilages. **B,** Inferior view of missing tissue. **C,** Use of Doppler to isolate the supratrochlear vessels to incorporate into the flap. **D,** The flap is secured in place with the blood supply intact. **E,** After the flap has been taken down and the incisions have healed.

repairing of lateral nose and nasal tip defects up to 1.5 cm. The bilobed flap has a random pattern blood supply. The flap is primarily rotated around a pivot point and the paddles are transposed over an incomplete bridge of skin. The second lobe allows the transfer of tension further from the primary defect closure. The bilobed design rotates around an arc that is usually 90 to 100 degrees. In the bilobed flap, the first lobe closes the defect and the second closes the first lobe defect. The flap is designed with a pivot point approximately a radius of the defect away from the wound margin. The first lobe is usually the same size as the defect, and the second lobe is slightly smaller with a triangular apex to allow for primary closure. The axis of the second flap is roughly 90 to 100 degreees from the primary defect and undermined widely to distribute the tension.

An advantage of the bilobed flap is that one can construct a flap at some distance from the defect with an axis that is independent of the linear axis of the defect. A disadvantage of this flap is that it leaves a circular scar that does not blend with the existing skin creases. During healing, the flap may become elevated ("pin cushioning") because of the narrow pedicle that is prone to congestion, scar tissue that impedes lymphatic drainage, and curvilinear scars that tend to bunch the flap up as they shorten.

Interpolation Flaps

Interpolation flaps contain a pedicle that must pass over or under intact intervening tissue. A disadvantage of these types of flaps is that for those passing over bridging skin, the pedicle must be detached during a second surgical procedure. Occasionally, it is possible to perform a single-stage procedure by deepithelializing the pedicle and passing it under the intervening skin. Advantages of interpolation flaps include their excellent vascularity and their skin color and texture match.

The forehead flap (median and paramedian) is a commonly used interpolation flap and remains the workhorse flap for large nasal defects.[24–27] It is a robust and dependable flap. The forehead flap is primarily based on the supratrochlear vessel, is relatively narrow, and uses a skin paddle from the forehead region. The flap is supplied by a rich anastomosis between the supratrochlear and the angular arteries. Because of the marked vascularity, it is possible to incorporate cartilage or tissue grafts for nasal reconstruction. The forehead flap has abundant tissue available, allowing resurfacing of the entire nasal unit with a single flap, and provides a good texture and color match to the native nose.

The technique for elevating the forehead flap is straightforward. The flap can be designed directly in the midline or in a paramidline location. A template of the defect is used to outline the flap. Elevation of the flap proceeds in either a subgaleal or a subcutaneous plane. The pedicle is always elevated in such a way as to incorporate the frontalis muscle. The width of the pedicle is usually 1.0 to 1.5 cm, which allows for easy rotation of the pedicle. Before inset, the skin paddle is selectively thinned to match the native skin thickness. The pedicle is divided approximately 3 weeks later, with the base of the pedicle inset into the glabellar area to reestablish brow symmetry. The incision, and resulting scar, is perpendicular to the RSTLs but tends to heal well (Figure 40-11).

The nasolabial flap (melolabial) is useful for reconstructing defects involving the oral cavity and those involving the lower third of the nose (Figure 40-12).[28–31] It can be used

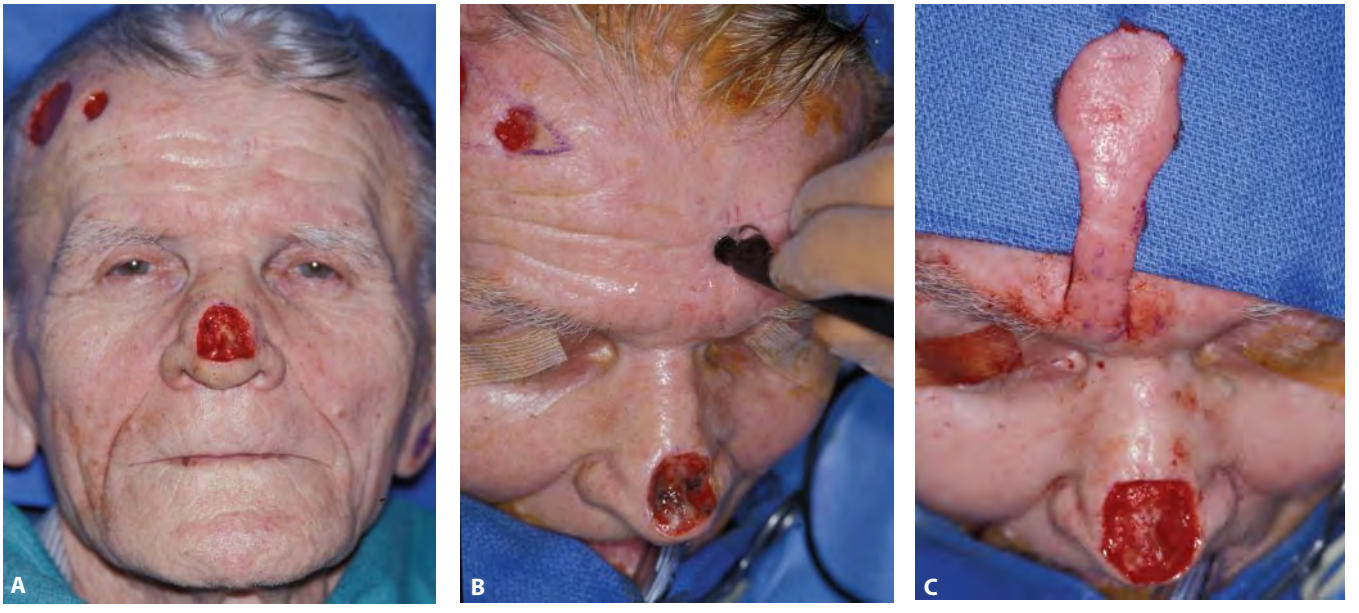


FIGURE 40-11. **A,** Nasal defect after excision of a squamous cell carcinoma lesion. **B,** Use of Doppler ultrasonography to locate the supratrochlear artery. **C,** The forehead flap has been elevated. *(Continued)*

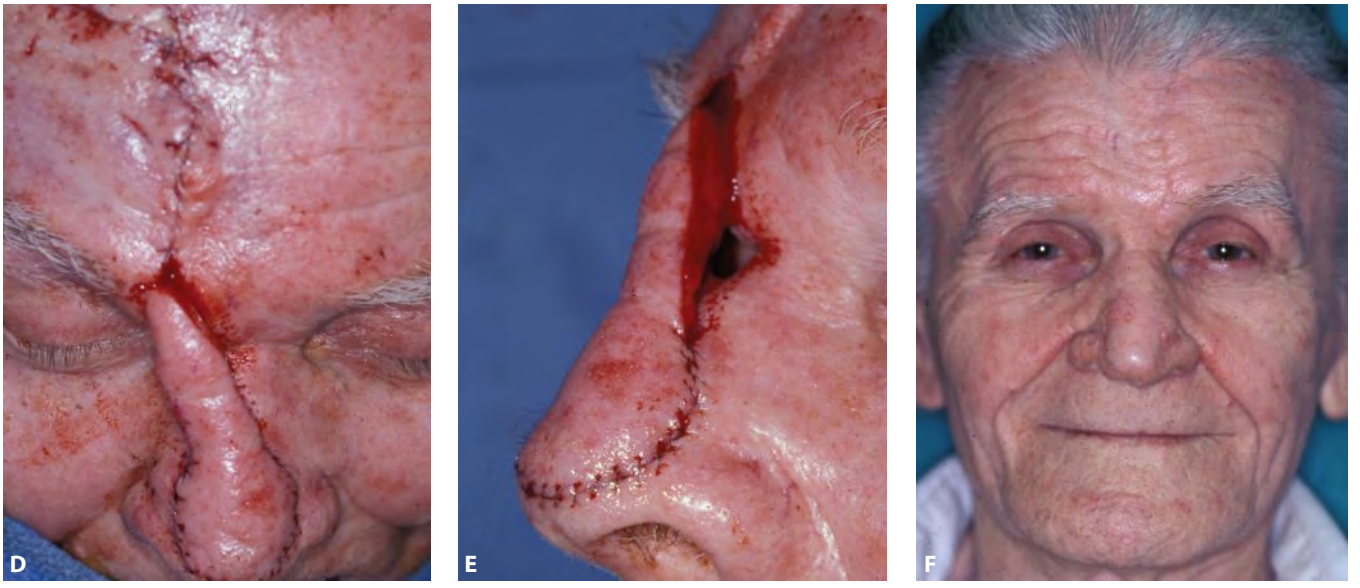


FIGURE 40-11. *(Continued)* **D,** The flap is turned 180 degrees and sutured into place. **E,** The pedicle is divided 2 to 3 weeks later. **F,** Postoperative result.



FIGURE 40-12. **A,** Outline of a nasolabial flap in a patient with a defect in the anterior floor of the mouth. **B,** The pedicle is deepithelialized and tunneled into the mouth. **C,** The flap is sutured into place to restore the missing soft tissue. **D,** The incision has been hidden in the nasolabial fold.

as an interpolation flap with either a single or a staged technique. The flap is supplied by the angular artery, intraorbital artery, and infratrochlear artery and can be based either superiorly or inferiorly. The area of recruitment for nasal reconstruction is in closer proximity to the primary defect than is the forehead flap. A disadvantage of the nasolabial flap is that there is a limited amount of tissue available, and asymmetry can occur along the nasolabial flap folds. When the pedicle is divided, the defect can be closed primarily by placing the scar in the nasal facial junction and the nasolabial flap fold.

The lip-switch flap (Abbé) can be taken from either lip, but it is most commonly switched from the lower to the upper lip.[32–34] This flap can be used to reconstruct as much as one third of the upper lip. The lower lip can supply a flap of one quarter of its length, and the Abbé flap offers immediate replacement of total lip anatomy (Figure 40-13). The labial artery supplies the flap and should be maintained with a small cuff of subcutaneous tissue and muscle surrounding the vascular pedicle. The pedicle is divided after approximately 2 to 3 weeks.

Tongue flaps are excellent flaps for intraoral reconstruction. They use adjacent tissue, have an excellent blood supply, and are associated with minimal morbidity. The tongue has excellent axial and collateral circulation, with the lingual artery providing the main blood supply. Up to one half of the tongue can be rotated for tissue coverage without compromising speech, mastication, or deglutition.[35] A variety of flap designs have been described including anterior- and posterior-based tongue flaps (Figure 40-14). Some indications include repair of oral defects and fistula closure. These flaps are helpful for providing closure of large oroantral fistulae.

Regional Flaps

For large facial defects, local flaps may not provide sufficient tissue to adequately restore the missing tissue. In these cases, consideration should be given to using a regional flap.[36,37] *Regional flaps* are defined as those located near a defect but not in the immediate proximity. They are frequently harvested from the neck, chest, or axilla and can provide coverage of large surface areas on the face. Selection of a specific regional flap depends on the size and location of the defect and also on the intrinsic properties of the flap. Advantages of regional flaps include the large amount of soft tissue and skin available. Disadvantages of these types of flaps include poor color and texture match, excessive bulkiness of the flap, and donor site morbidity.

Pectoralis Major Myocutaneous Flap

The pectoralis major myocutaneous flap remains a workhorse of reconstructive surgery[38–40] The flap was introduced by Ariyan[41] and has provided a reliable method of soft tissue reconstruction of bone and soft tissue defects of the mandible and maxilla. The pectoralis major myocutaneous flap can be rotated around a pivot point 180 degrees and is supplied by

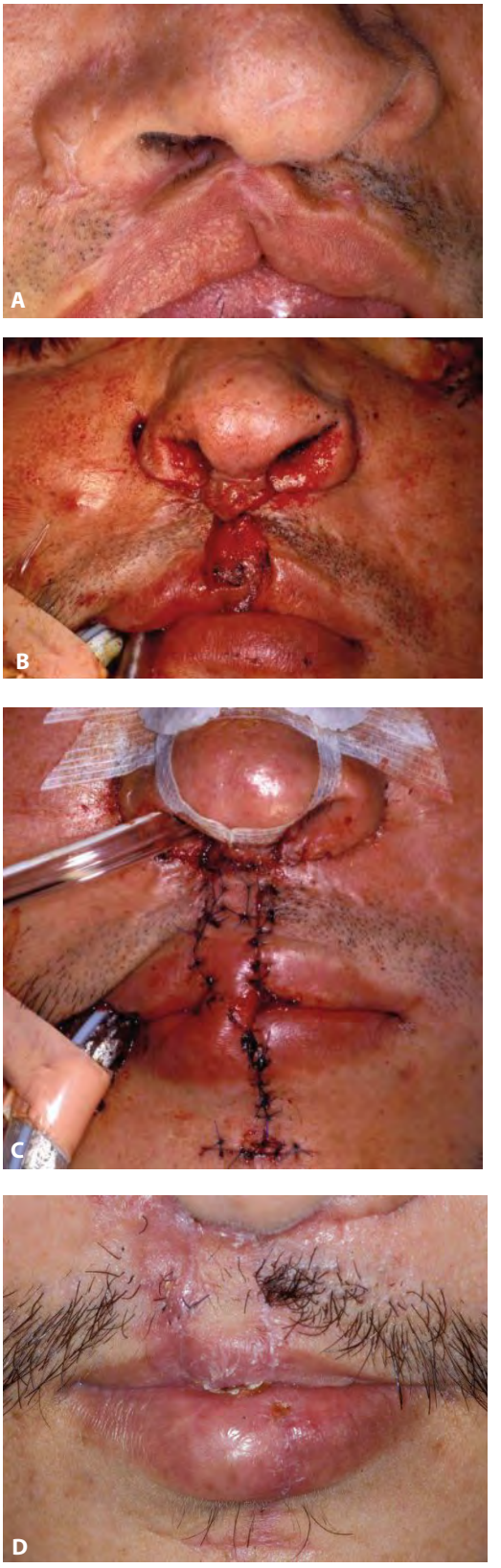


FIGURE 40-13. **A,** Patient with a traumatic lip deformity with avulsion of a portion of his upper lip. **B,** Reapproximation of the orbicularis oris muscles and perialar advancement flaps to reestablish upper lip length. **C,** An Abbé flap is used to restore the missing philtrum. **D,** Postoperative result.

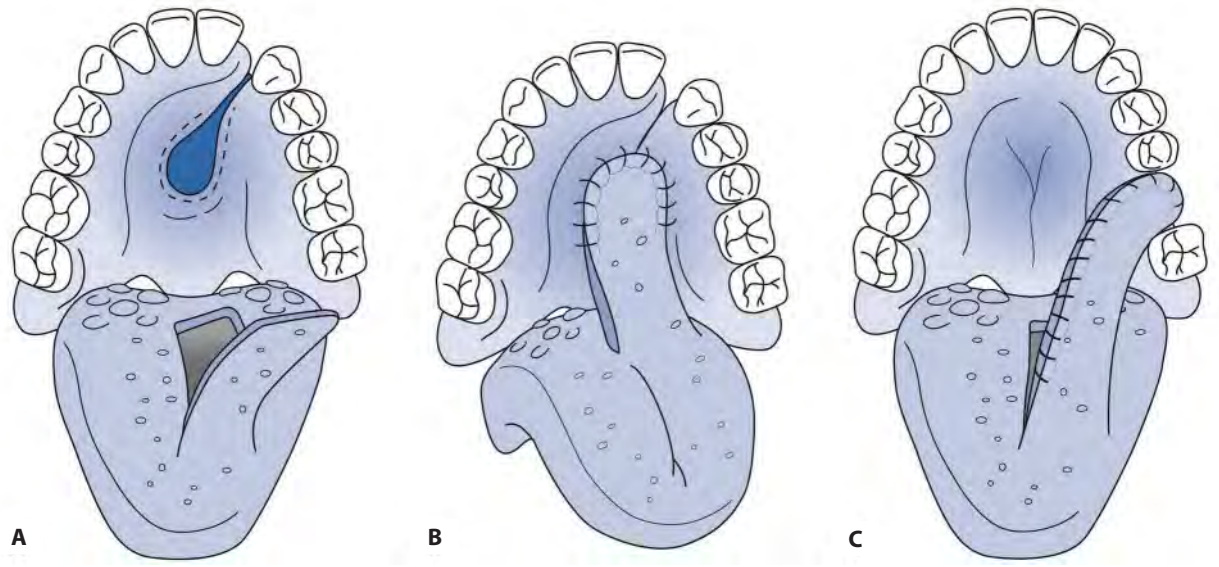


FIGURE 40-14. **A** and **B,** Use of an anteriorly based tongue flap to cover the soft tissue deficit resulting from an alveolar cleft. **C,** This type of flap is also useful for closing large oroantral fistulas.

two separate blood supplies (Figure 40-15). The thoracoacromial artery arises from the second portion of the axillary artery and forms four branches as it penetrates the fascia. The pectoral branch is the major artery that supplies the pectoralis major myocutaneous flap. The position of the vascular pedicle can be approximated by drawing a line from the shoulder point to the xiphoid. The pectoral branch descends at a right angle from the middle of the clavicle until it meets this line. Branches of the internal mammary artery supply the medial portion of the muscle and skin over the sternum. The flap provides good coverage for the carotid artery when combined with a neck dissection.

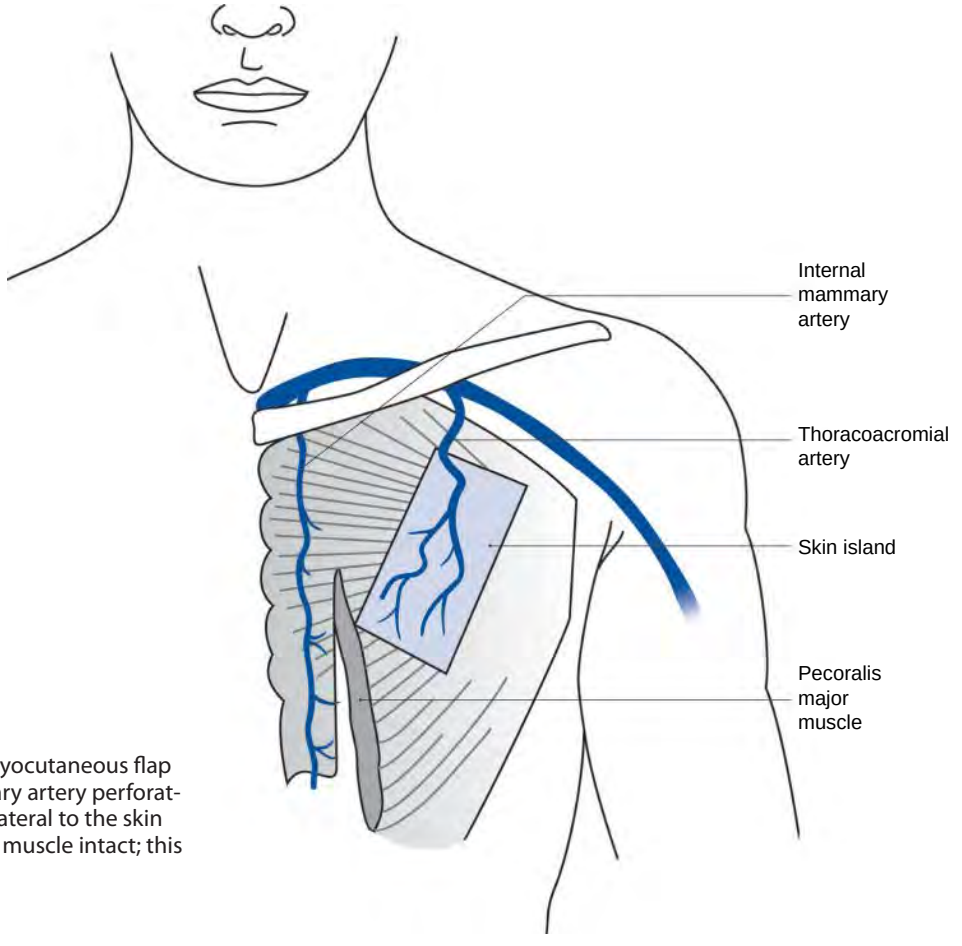


FIGURE 40-15. The pectoralis major myocutaneous flap is divided lateral to the internal mammary artery perforating vessels. The muscle can be divided lateral to the skin island to leave the lateral portion of the muscle intact; this preserves the axillary fold.

Deltopectoral Flap

The introduction of the deltopectoral flap by Bakamjian and colleagues represented a significant improvement for reconstructing large ablative resections for head and neck cancer.[42–44] Currently, it is used as an alternative to the pectoralis major myocutaneous flap for soft tissue reconstruction of the mandible and maxilla. This flap is composed of fascia, subcutaneous tissue, and skin but does not contain muscle (Figure 40-16). Perforators from the internal mammary artery provide vascular supply to the flap. The secondary defect is covered with a skin graft.

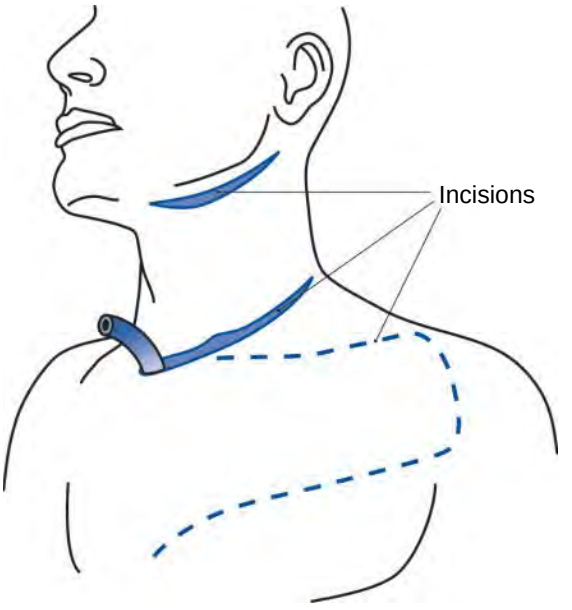


FIGURE 40-16. Incisions for a deltopectoral flap.

Temporalis Flap

The temporalis flap was introduced by Golovine in 1898 and remains useful for covering intraoral defects (Figure 40-17).[45–48] The outer portion of the muscle is invested by the deep temporal fascia. This fascia is supplied by the middle temporal vessel, which originates just below the zygomatic arch. The temporalis muscle is supplied by both the anterior and the posterior deep temporal arteries, which arise from the second portion of the internal maxillary artery. This dual

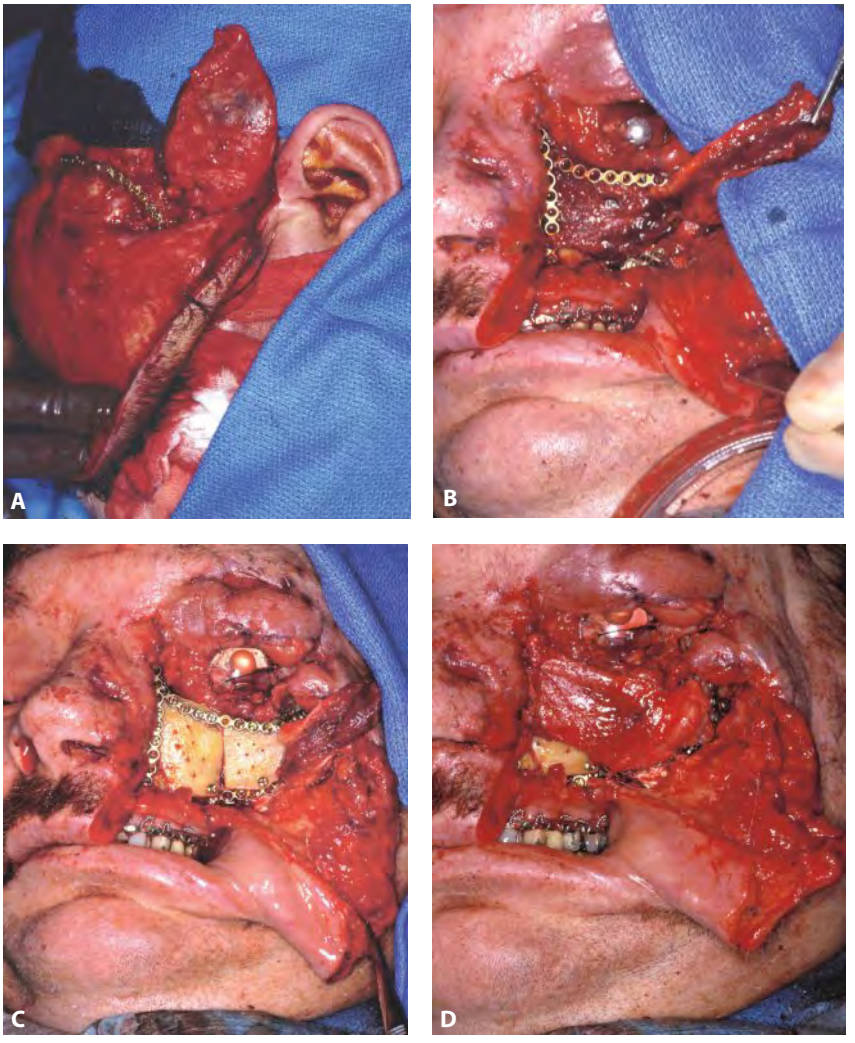


FIGURE 40-17. **A,** Temporalis muscle flap for repair of a midface defect caused by a shotgun wound. **B,** The temporalis muscle is divided, and the posterior portion is sutured into place. **C,** Cranial bone is used to restore the missing tissue. **D,** The anterior portion of the temporalis flap is sutured into place to "sandwich" the bone grafts.

blood supply allows for splitting of the muscle into anterior and posterior flaps.

When elevating the muscle, it is important to remain on the deep temporal fascia beneath the superficial temporal fascia to avoid damage to the frontal branch of the facial nerve. Elevation of the inferior portion of the flap is performed in a subperiosteal plane to avoid damage to the deep temporal arteries, which lie on the undersurface of the muscle. An osteotomy of the zygomatic arch is often helpful to facilitate placement of the muscle into the mouth. The arch can be put back into place and secured with plates and screws. A disadvantage of the temporalis flap is the minimal cosmetic deformity of hollowing in the temporal region; this can be corrected with autogenous or alloplastic materials and can be minimized by using either an anterior or a posterior flap.

Sternocleidomastoid Flap

First described by Jinau in 1909[49] for facial reanimation, the sternocleidomastoid flap was repopularized by Owens.[50–55] The muscle is invested by the deep cervical fascia and is supplied by three arteries. The dominant vessel is the occipital artery, which enters the muscle below the mastoid tip and supplies the superior portion of the muscle. The superior thyroid artery supplies the middle portion, and the thyrocervical trunk supplies the inferior third of the muscle.

The muscle is elevated over the deep cervical fascia superior to the carotid sheath. It is recommended to maintain two of the three vessels when elevating the flap to enhance the viability of the flap. The spinal accessory nerve enters the deep portion of the muscle approximately at the carotid bifurcation and should be preserved to prevent denervation atrophy of the muscle (Figure 40-18). Advantages of the sternocleidomastoid flap include its close proximity to the defect and minimal donor site defect (Figures 40-19 and 40-20).

Trapezius Myocutaneous Flap

The trapezius myocutaneous flap is supplied by three arteries, allowing several flaps to be used. The main vessel supplying the trapezius muscle is the transverse cervical artery, which is a branch of the thyrocervical trunk. The upper portion of the muscle is supplied by the occipital artery. The trapezius myocutaneous flap is a ready source of skin of uniform thickness without excessive muscle bulk.[56] The main disadvantage is the limited rotation and the short pedicle.

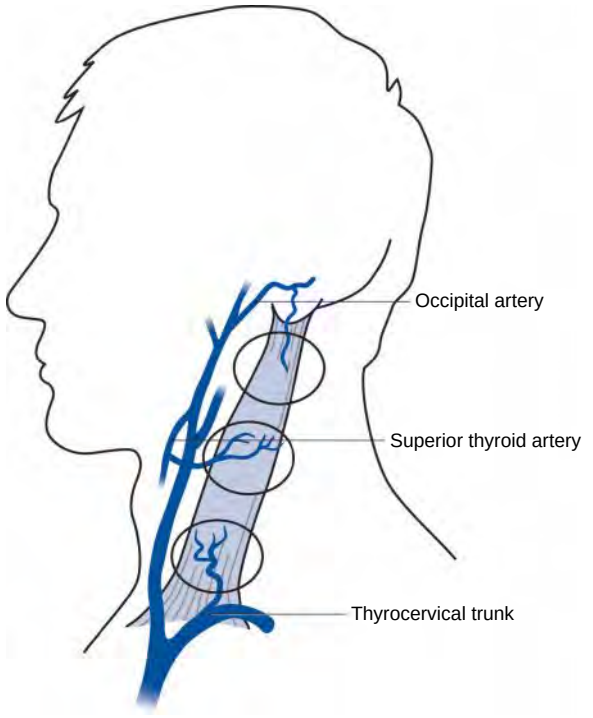


FIGURE 40-18. Blood to the sternocleidomastoid muscle is supplied through three arteries.

Latissimus Dorsi Myocutaneous Flap

Quillen and colleagues[57] first described the use of the latissimus dorsi myocutaneous flap for head and neck reconstruction in 1978.[57] The flap is not commonly used for head and

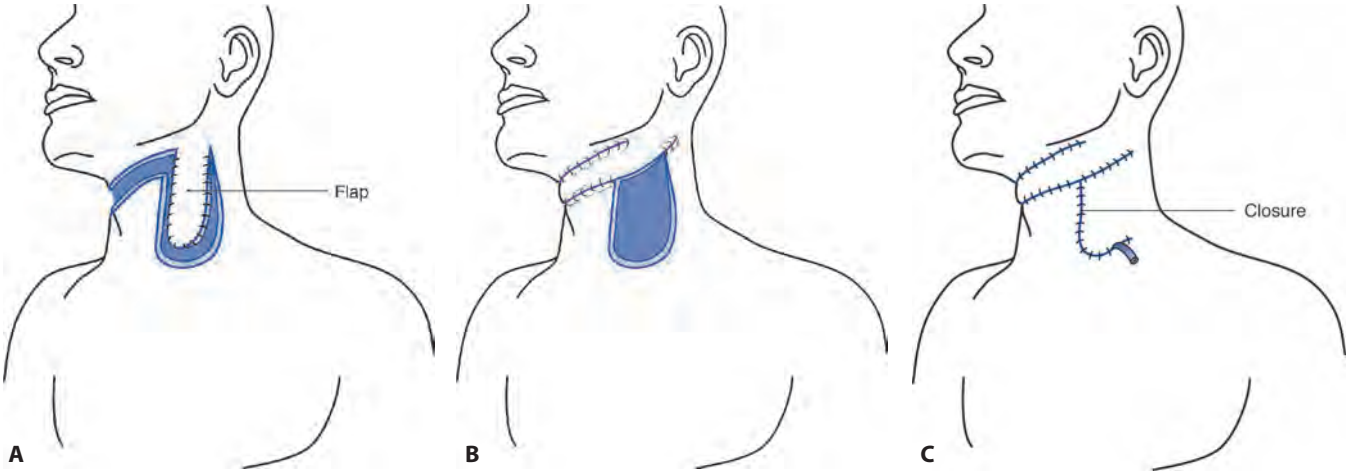


FIGURE 40-19. **A,** A superiorly based flap with a skin pedicle. **B,** Transposition of the flap. **C,** Closure of the donor defect.

SECTION 5

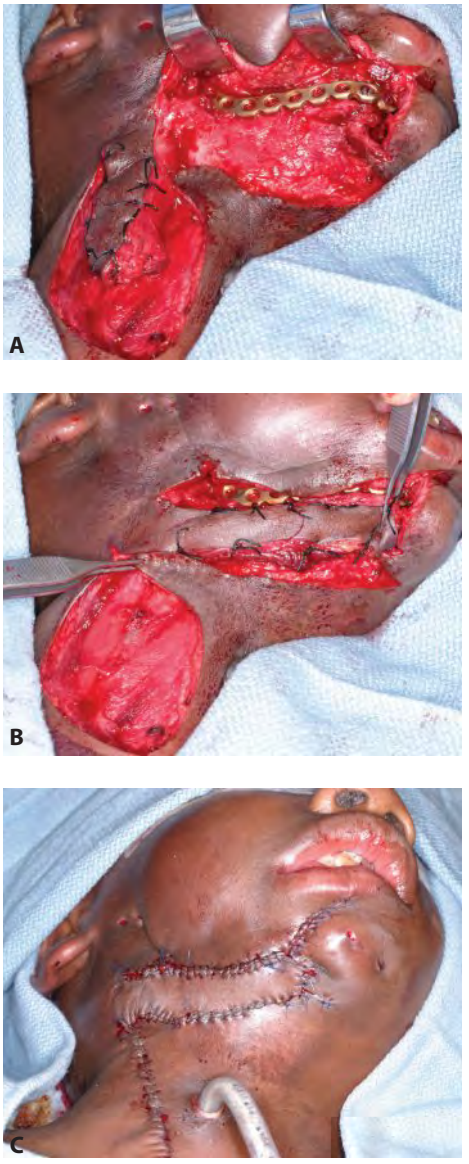


FIGURE 40-20. **A,** The sternocleidomastoid flap is elevated with a superior base. **B,** The flap is rotated in place to provide soft tissue coverage over the reconstruction plate. **C,** The flap is sutured into place and the donor site closed primarily.

neck reconstruction unless other flaps are unavailable or there are very large defects requiring coverage. The muscle is supplied by the thoracodorsal artery, which is the dominant vessel, and also by four to six perforators from the posterior intercostals and lumbar vessels. The main advantage of the latissimus dorsi flap is the large amount of skin provided. The main disadvantages are the need to reposition the patient during the operation and morbidity from the donor site.

COMPLICATIONS

Postoperative complications can be minimized with careful preoperative planning of flap design and by early recognition of problems.[59] A medical history can be used to identify patients with risk factors involving small vessels. These risk factors include smoking, diabetes, hypertension, previous radiation, and preexisting scars.[60,61] Complications may be reversible or irreversible. Early recognition and treatment can minimize complications and prevent them from becoming irreversible. Two main unwanted outcomes are flap failure and unacceptable cosmetic results.

Flap survival depends on early recognition of flap compromise. *Ischemia* is defined as an inadequacy of perfusion in providing tissue needs. Signs of arterial ischemia include a pale and cool flap that does not blanch with pressure and typically does not bleed with a pinprick. Flaps are somewhat ischemic initially because the original tissue perfusion has been compromised by flap elevation. Most tissue can survive on 10% of its average blood flow.[59] Whether the flap will undergo necrosis depends on patient-related and surgery-related factors that influence the risk of necrosis in facial flaps. Smoking is associated with an increased risk of flap failure. The deleterious effects of smoking on flap survival include hypoxemia and vasoconstriction. Patients should be advised to quit smoking during the perioperative period.

Common causes of bleeding in facial reconstruction with local flaps include inadequate hemostasis and drug-induced coagulopathy. Hematoma formation should be identified and decompressed within 24 hours.[62] Decompression can be accomplished with aspiration using a 22-gauge needle or by taking out one or two sutures and applying gentle compression on the flap. Hematoma formation may diminish tissue perfusion and can lead to ischemia or necrosis by inducing vasospasm, stretching the subdermal plexus, or separating the flap from its recipient bed. Patients should be questioned carefully about the use of medications that affect coagulation such as acetylsalicylic acid, nonsteroidal anti-inflammatory drugs, and vitamin E. If possible, these medications should be avoided for 2 weeks before and 1 week after surgery.

Congestion is the most common vascular problem associated with facial flaps. Signs of a congested flap include warmth, edema, and a purple color that blanches with pressure then immediately refills. A pinprick will cause release of dark venous blood. Venous congestion can lead to arterial compromise and flap necrosis. Management of congested flaps may include temporarily releasing sutures to allow decompression at the flap edges or possible impingement involving the flap pedicle. Tight bandages around the flap pedicle should be removed. Medicinal leeches *(Hirudo medicinalis)* may be useful in decompressing congested flaps.[63,64] Saliva from the leech contains an anticoagulant and a vasodilator that facilitate continued oozing from the site even up to 6 hours after they detach.

Hyperbaric oxygen (HBO) has been shown to be beneficial in improving the vascularity of marginal tissues.[65] Prophylactic HBO therapy in cutaneous flap surgery in the irradiated tissue bed may be particularly helpful to combat the hypoxia and hypocellularity. HBO is beneficial in treating both venous congestion and arterial ischemia by creating

a local arterial vasoconstriction through the rise in arterial oxygen content, which reduces the amount of inflow. The tissue oxygen levels continue to rise owing to the improved diffusion even though there is vasoconstriction and a reduction in vascular perfusion. The flap can maintain viability while continued neovascularization occurs. Other options include the use of heparin and dipyridamole to help increase the survival of an ischemic flap.[66]

Infection can complicate flap healing.[67] The postoperative infection rate for clean wounds in facial surgery is as low as 2.8%, with higher rates in facial reconstruction with local flaps.[68] Tissue oxygenation is an important factor in prevention of wound infection and is closely related to blood supply. Infections involving local flaps may result in flap failure or poor cosmetic outcome secondary to wound dehiscence and scarring.

CONCLUSION

A variety of facial flaps are available to the reconstructive surgeon for repairing facial defects. The goal of flap surgery is to restore form, function, and aesthetics. There are many advantages to using local and regional flaps, which can lead to optimal aesthetic results.

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41

CHAPTER

Vascularized and Nonvascularized Hard and Soft Tissue Reconstruction

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INTRODUCTION

Reconstruction of the oral cavity involves restoration of complex functional, anatomic, and aesthetic characteristics that can make this a daunting task. The reconstructive surgeon has many techniques at his or her disposal for realizing these goals; often, this requires complex decision making as to which techniques will yield the best outcome. These decisions should be made by evaluating the patient and the defect on a case-by-case basis. Clearly, the myriad of techniques available to the reconstructive surgeon each have their own merits and demerits and the most ideal technique for one patient may not be adequate for the next.

This chapter outlines many of the major options in soft tissue and bony reconstruction. Many smaller defects of the oral cavity may be amenable to simpler techniques for reconstruction such as primary closure, skin grafting, secondary intention, or random pattern local flaps. These techniques are described in numerous articles throughout our literature and are not covered in this chapter. Instead, the authors focus on the use of regional or distant soft tissue and bone for reconstruction of more complex defects in the head and neck. The authors have attempted to detail the procedures, advantages, and disadvantages in a concise and objective manner.

Flaps can be classified based on their proximity to the recipient site. This results in the clinically useful terms: local, regional, and distant flaps. Most regional and distant flaps are based on a vascular pedicle that provides the nutrient inflow and outflow of blood to the transferred tissue. Understanding the variations of the different classifications of muscle-containing flaps is extremely important for the reconstructive surgeon; the studies of the blood supply to muscles reported by Mathes and Nahai[1] are primary to this understanding (Table 41-1). Of course, this classification system involves only muscles, but the understanding of the blood supply to the skin has been elucidated Taylor and Palmer's

TABLE 41-1. **The Classification of Muscle Flaps**

Type 1	One vascular pedicle	Gastrocnemius, rectus femoris, tensor fascia lata
Type 2	One dominant vascular pedicle plus minor pedicles (most common pattern)	Abductor digiti minimi, abductor hallucis, biceps femoris, flexor digitorum brevis, gracilis, peroneus longus, peroneus brevis, platysma, semitendinosis, soleus, sternocleidomastoid, temporalis, trapezius, vastus lateralis
Type 3	Two dominant pedicles	Gluteus maximus, rectus abdominis, serratus anterior, semimembranosus
Type 4	Segmental vascular pedicles	Extensor digitorum longus, extensor hallucis longus, flexor digitorum longus, flexor hallucis longus, sartorius, tibialis anterior
Type 5	One dominant vascular pedicle and secondary segmental vascular pedicles	Pectoralis major, latissimus dorsi

Adapted from Mathes SJ, Nahai F. Classification of the vascular anatomy of muscles: experimental and clinical correlation. Plast Reconstr Surg 1981;67:177–187.

anatomic study[2] of the human body's angiosomes. The angiosome theory proposes that the human body is composed of approximately 40 blocks of composite tissue, each with its own cutaneous territory supplied by a discrete source vessel. Simply stated, all arteries to the skin travel either directly to their destination (direct perforators) or first travel through other tissues, usually muscle (indirect perforators). These studies allow the reconstructive surgeon to classify flaps according to the type of vascular supply and predictably determine the vascular territory of the flap's pedicle.

Reconstruction of the craniomaxillofacial complex has been a cornerstone of the specialty of oral and maxillofacial surgery, and the reconstructive options for bony defects continue to expand. Currently, the surgeon has a vast array of autologous and autogenous options. This chapter covers the available autogenous options.

The use of nonvascularized bone has always been within the everyday realm of maxillofacial surgeons. Recently, the use of microvascular free tissue transfer has become an established and routine reconstructive option in head and neck surgery. Furthermore, it has been embraced by oral and maxillofacial units worldwide wherein free flap reconstruction of such defects is now considered the gold standard of care.[3] The use of nonvascularized bone grafts continues to be the workhorse for the majority of defects encountered by maxillofacial surgeons. As such, this chapter also reviews the commonly used nonvascularized grafts and highlights the advantages as well as disadvantages of their use. Equally, the chapter highlights commonly used microvascular bone flaps and their associated advantages and disadvantages.

TECHNIQUES FOR SOFT TISSUE RECONSTRUCTION OF THE ORAL CAVITY

Regional Soft Tissue Flaps

Regional flaps have historically been the mainstay technique for head and neck soft tissue reconstruction. These techniques take advantage of the flap's proximity to the ablative or injured site and generally rely on an "arc of rotation" as the limiting factor for utilization of these flaps. Their popularity is undeniable owing to proximity, ease of harvest, general reliability, and elimination of the need for specialized instruments and skills for microvascular anastomosis. The main drawbacks of these flaps are the relative limited area of application due to the arc of rotation as well as a limited volume of soft tissue available for reconstruction. Some flaps such as the pectoralis major, temporalis, and latissimus flaps require removal of a relatively large volume of tissue with only a small portion of it used for the actual reconstruction. The combination of these drawbacks has led to an increase in utilization of free tissue reconstruction techniques for head and neck defects.

Submental Island Flap

The submental island flap was introduced by Martin and coworkers in 1993[4] and has rapidly increased in popularity in head and neck reconstruction owing to its ease of harvest, accessibility, quality of available tissue, and overall versatility. It can be raised as a cutaneous, musculocutaneous, fasciocutaneous, or osteocutaneous flap and can be utilized as a rotational flap for oral or facial defects as distant as the upper third of the face.[5] The flap may also be harvested as a free flap for more distant recipient sites. The submental flap is an axial patterned flap based on the submental branch of the facial artery and vein (Figure 41-1). This is a consistent branch of the facial artery and arises behind the submandibular gland and crosses anteriorly along the mylohyoid muscle. Here, the artery may pass superficially across the anterior belly of the digastric muscle but passes deep to this muscle 70% of the time.[6] The artery ends at the symphysis of the mandible where it sends terminal branches to the lower lip and sublingual gland. The artery gives off one tow four cutaneous perforators throughout its course,[7] which travels through the platysma muscle to the subdermal plexus where it anastomoses with the contralateral artery. This pattern of perfusion allows for reliable skin paddle design that can extend from one mandibular angle to the other. Many times, the dimensions of the flap may be limited by the remainder of the neck skin's mobility to achieve primary closure of the donor site defect. However, split- or full-thickness skin grafting may be utilized in cases in which primary closure cannot be obtained. The potential skin paddle for this flap has been described as large as 18 × 7 cm and the vascular pedicle length can be as long as 8 cm.[8]

Several descriptions of increasing the flap's vascular pedicle have been offered. The facial artery may be ligated distal to the take-off of the submental artery, providing a modest increase in pedicle length. The facial artery may also be

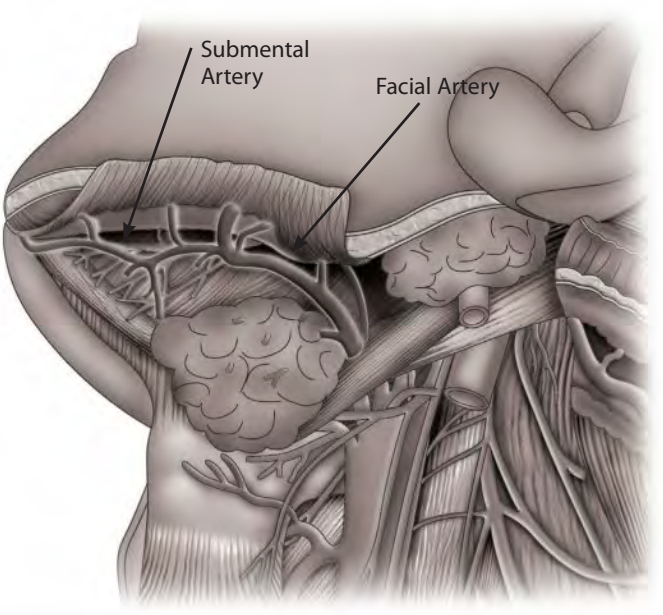


FIGURE 41-1. The course of the submental artery. (From Clement CD. Anatomy Regional Atlas of the Human Body. 3rd ed. Baltimore: Urban & Schwarzenberg; 1987; Fig. 581.)

ligated proximal to the take-off of the submental artery, relying on reversed flow from the distal facial artery, resulting in a reverse-flow flap. Finally, one or both of the submental artery or vein may be divided and anastomosed to a suitable recipient vessel nearer to the defect.[9]

The submental skin is very pliable and thin. The quality of this donor site is analogous to the skin of the radial forearm flap. Therefore, this flap is well suited for moderate-sized defects of the tongue, floor of mouth, and buccal mucosa. In facial reconstruction, the hair-bearing nature of the submental skin in males and some females must be considered before its use.

Owing to this donor site's location, its use for reconstruction of oncologic defects of the oral cavity may be limited. If a neck dissection is included in the treatment of an oral cavity malignancy, the vascular pedicle must be dissected through the primary lymphatic drainage basin of many of these, tumors which may compromise the oncologic safety of the resection. Some authors have advocated close dissection of the pedicle of this flap in these circumstances to avoid leaving a significant amount of fibroadipose tissue surrounding the vessels.[7] This flap has even been advocated in cases in which cervical node involvement is known or suspected.[10] Others have recommended a more prudent approach to donor site selection owing to the vast array of available reconstructive options; the risk of using this flap in this circumstance is difficult to justify.[11]

Flap Harvesting

With the patient in the supine position and the neck extended, the skin paddle is designed along the inferior border of the mandible at a distance of approximately 1 cm inferior to this landmark. This provides a relatively hidden scar and decreases the chance of pulling the lower lip down upon closure of the defect (Figure 41-2). The neck skin can be pinched to determine the maximum dimension of the flap that will facilitate primary closure; otherwise, skin grafting will be required.[10] Dissection of the flap may begin on the ipsilateral or contralateral side of the vascular pedicle to be utilized and also either the inferior or the superior border of the flap. The dissection is in the subplatysmal plane on the contralateral side of the skin paddle, and because this dissection proceeds toward the pedicle, it is carried over the contralateral anterior belly of the digastric muscle and the mylohyoid muscle in the midline of the neck. The ipsilateral anterior belly of the digastric muscle is detached at the inferior border of the mandible and at the common tendon and included in the flap. The ipsilateral portion of the mylohyoid muscle may be preserved by meticulous dissection of the submental vessels, or a portion of this muscle may be removed along with the pedicle, which may facilitate passage of the flap to an intraoral site (Figure 41-3). The facial artery is usually easily identified behind the submandibular gland as it passes behind the posterior belly of the digastric muscle. It can then be traced distally either through or posterior to the submandibular gland where numerous glandular branches will be encountered and ligated. The submental artery is then dissected off its deep attachments and the distal facial artery can be ligated.

Applications

The flap has many indications for head and neck reconstruction including coverage for any intraoral defect including maxillectomies. The nature of the submental skin makes it particularly useful for tongue, floor of mouth, and buccal mucosa defects. Facial skin can be reconstructed ranging from lips and cheeks as well as the temporal region.[7]

Temporalis Flap

The temporalis muscle flap has extensive applications for head and neck reconstructions. The flap is well described for use in facial reanimation as well as skull base and midfacial



FIGURE 41-2. Submental flap elevated in the subplatysmal plane until the ipsilateral anterior belly of the digastric. This muscle is incorporated into the flap by detaching from the mandible and at the common digastric tendon.



FIGURE 41-3. Elevated submental flap with ipsilateral anterior belly of digastric tendon.

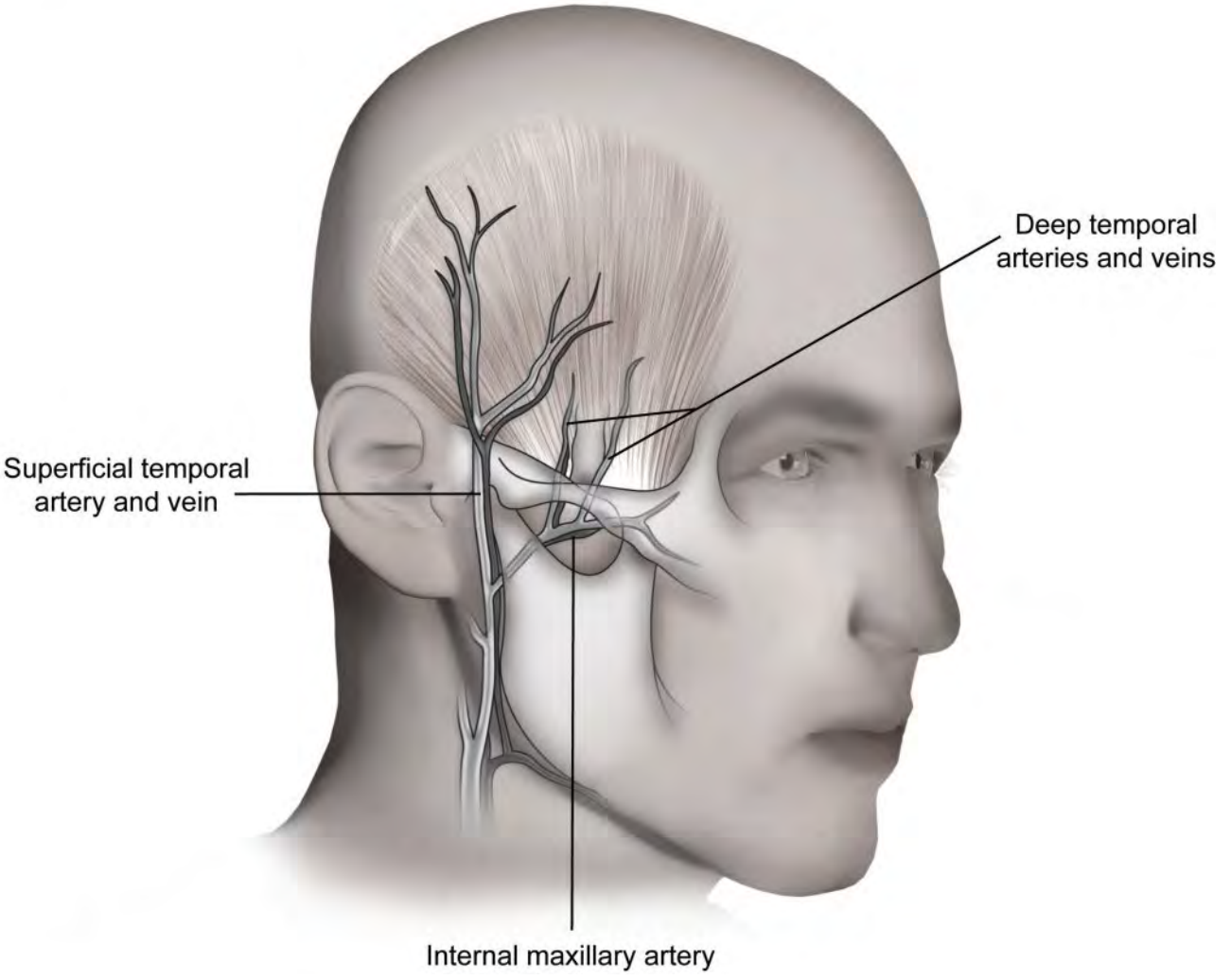


FIGURE 41-4. Blood supply to the temporalis muscle. Anterior and posterior deep temporal arteries and the middle temporal artery (branch off the superficial temporal).

reconstruction. First introduced in 1898 by Golovine,[12] it is known as one of the oldest muscular flaps in use today. The flap's popularity is bolstered by its relative ease of harvest, proximity to maxillofacial applications, and flexibility in harvesting all or only a portion of the muscle. The main disadvantages include a relative lack of soft tissue available for reconstruction, minimal application due to arc of rotation limitations, as well as a contour deformity of the donor site and over the zygoma. Of course, these last two concerns have been addressed by alloplastic implants or autogenous tissue to minimize the donor site defect and by osteotomy of the zygomatic arch to pass the temporalis muscle and prevent a contour deformity.

Based on the deep temporal arteries, the temporalis muscle is classified by Mathes and Nahai[1] as a type 2 muscle with one dominant pedicle plus minor pedicles. The second blood supply is from the middle temporal artery, which is a branch of the superficial temporal artery. The deep temporal arteries arise from the internal maxillary artery and enter the muscle from its deep surface and provide a segmental blood supply because there are usually separate anterior and posterior deep temporal arteries (Figure 41-4).

The muscle originates from the superior temporal line and coalesces to a dense tendon at its insertion to the coronoid process. In terms of the layers of the scalp, the temporalis muscle lies directly on the temporal bone and is covered by the deep temporal fascia, which also inserts into the superior temporal line and is analogous to the pericranium covering the remainder of the skull (Figure 41-5). Caudally, the fascia splits into superficial and deep layers approximately 2 cm above the zygomatic arch; a layer of fat exists between the two layers. The deep and superficial layers continue caudally to fuse with the periosteum of the zygomatic arch. The zygomatic and temporal branches of the facial nerve cross the zygomatic arch in the layer of the temporoparietal fascia, which is superficial to the superficial layer of the deep temporal fascia. By incising the superficial layer of the deep temporal fascia at the root of the zygoma and extending it anterosuperiorly at a 45-degree angle, the entire zygomatic arch can be exposed while adequately protecting the facial nerve.

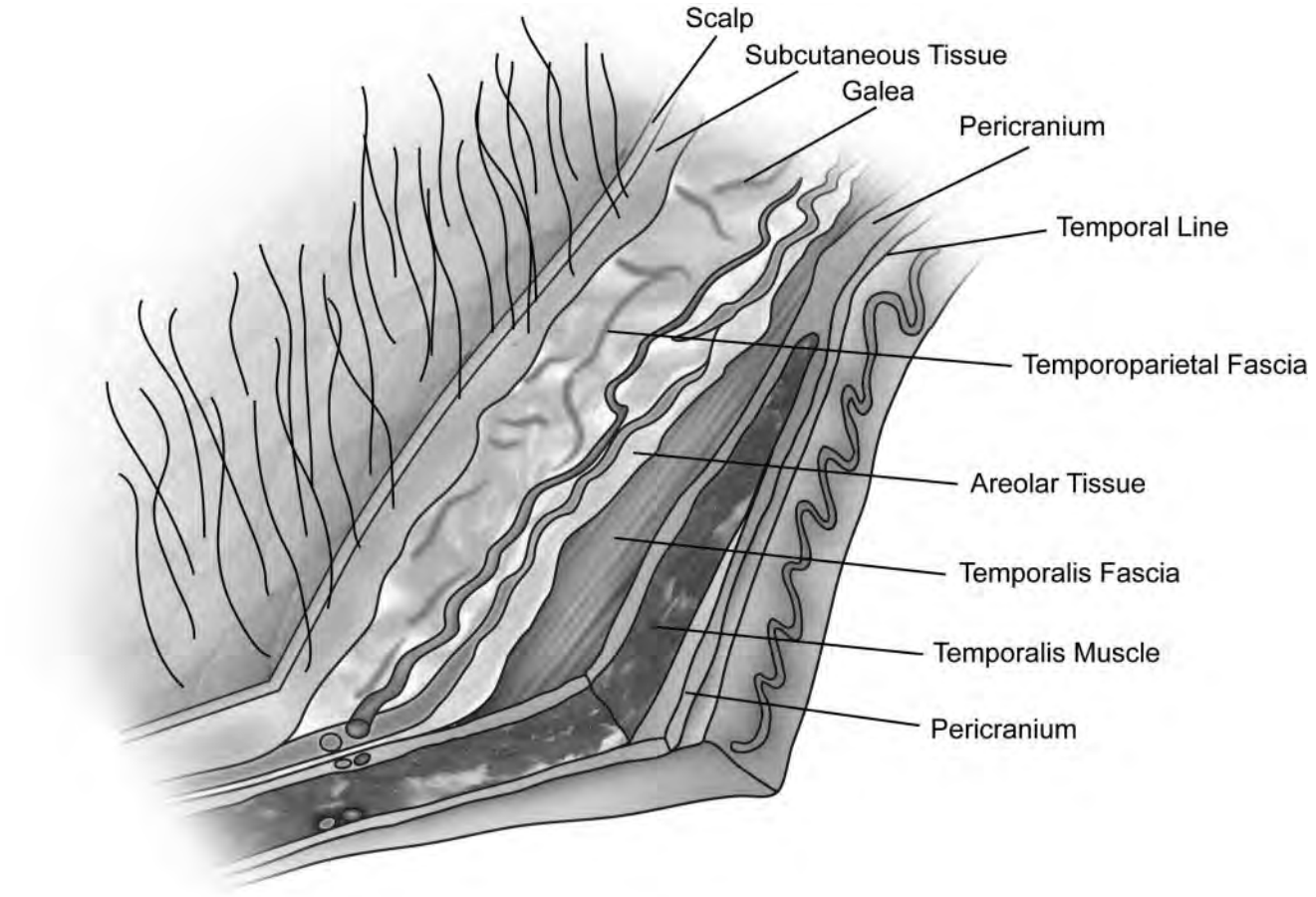


FIGURE 41-5. Layers of scalp at the superior temporal line. Note the confluence of the deep temporal fascia with the pericranium.

Flap Harvesting

Many techniques for exposing and harvesting a temporalis muscle flap exist for specific indications. Some of the more common uses include a small strip of temporalis muscle harvested for interpositional reconstruction of the temporomandibular joint (TMJ) after ankylosis release or diskectomy and temporalis muscle for facial reanimation surgery. This narrative deals with harvesting the entire temporalis muscle for intraoral reconstruction. With the patient in a supine position, the head should be placed on a Mayfield headrest to allow for easy access to the scalp. The authors do not recommend shaving the patient's head for this procedure, but long hair may be trimmed so it does not interfere with the dissection. A coronal or hemicoronal incision within the hair-bearing scalp can be planned based on the particular needs of the reconstruction or ablative defect location. Likewise, the planned incision can be combined with a preauricular, endaural, or retroaural incision and injected with 1% lidocaine with 1:100,000 epinephrine for hemostasis. The incision is carried through all the layers of the scalp except for the pericranium and deep temporal fascia. Raney clips may be used for hemostasis of the incised scalp. The scalp is bluntly elevated anteriorly above this layer until a point 2 cm above the lateral orbital rim is reached and posteriorly until the most posterior portions of the superior temporal line is identified. The root of the zygomatic arch is palpated and the deep temporal fascia is cut at a 45-degree angle upward from the root of the arch, anteriorly to the point approximately 2 cm above the lateral orbital rim. This exposes the temporal fat pad between the superficial and the deep layers of the deep temporal fascia and allows for exposure of the entire zygomatic arch in a subperiosteal plane from the root of the arch to the lateral orbital rim while adequately protecting the frontal branch of cranial nerve VII.

At this point, the entire temporalis muscle and deep temporal fascia is visualized and separated from its origin at the superior temporal line. The whole muscle may be raised and used if necessary. Alternatively, if a smaller defect is present, the muscle may be split into an anterior third and a posterior two thirds and utilizing the thinner and longer posterior portion of the muscle while leaving the anterior portion attached to the temporal fossa, thus decreasing the cosmetic deformity of the donor site depression (Figure 41-6). The dissection elevates the muscle off the temporal bone along with its fascia down to the infratemporal fossa. Although the superior portion of the deep temporal fascia is elevated with the flap, its blood supply is derived mainly from the middle temporal artery, which is usually sacrificed during this dissection. Therefore, this fascia may not be reliable for oral cavity

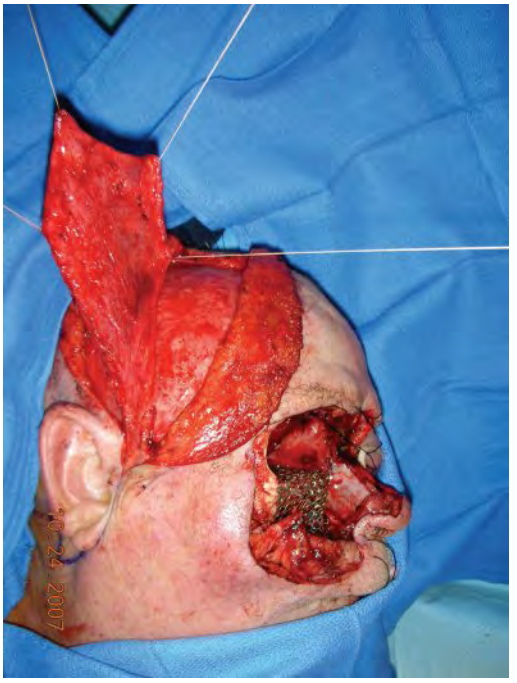


FIGURE 41-6. Temporalis muscle flap elevated.

relining as described by Wong and colleagues.[13] The muscle itself without a fascial covering is adequate for most oral reconstructions.

Several techniques for introducing the flap into the oral cavity have been offered. These include zygomatic arch osteomy, coronoidectomy,[14] and passage over or under the zygomatic arch. These are all viable techniques depending on the clinical indication and can be performed fairly easily without compromise of the vascular pedicle to the muscle flap. For many defects, the simplest method of transfer is to create a tunnel through the infratemporal fossa between the coronoid process and the remaining lateral wall of the maxilla by blunt dissection. A silk suture can be placed on the free edge of the temporalis muscle and grasped by a long clamp introduced through the tunnel from the oral cavity. Gentle passage through the tunnel can allow the muscle to unfold and adequately line the defect.

Applications

The temporalis muscle is well suited for relining defects of the maxilla, ipsilateral hard and soft palate, pharynx, retromolar trigone, and buccal mucosa. It has also been used for tongue and floor of mouth reconstructions.

Pectoralis Major Myocutaneous Flap

The pectoralis major myocutaneous flap (PMMF) has earned the title of the "workhorse flap" for head and neck reconstruction since its introduction in 1979 by Ariyan.[15] The popularity of this soft tissue flap has decreased significantly with the generalized acceptance of microvascular fasciocutaneous, musculocutaneous, and perforator flaps. However, the flap remains popular for reconstruction in patients who may have one or more contraindications to microvascular surgery, in facilities that lack microvascular capabilities, or as a rescue flap for failed microvascular reconstructions. The main reasons for this flap's popularity include its relative ease of harvest, proximity to the head and neck, hearty and consistent vascular supply, low donor site morbidity, and good versatility for head and neck defects.

The pectoralis major is a fan-shaped muscle that originates at the sternum, clavicle, and external oblique aponeurosis and inserts via a thick tendon to the greater tubercle of the humerus. The muscle medially rotates and adducts the arm with some flexion (upper fibers) and extension (lower fibers) of the arm. The loss of this muscle is tolerated well with most of the muscle's function compensated by the latissimus dorsi.

According to Mathes and Nahai,[1] the pectoralis major is a type 5 muscle with one dominant pedicle, the pectoral branch of the thoracoacromial artery, and a secondary segmental blood supply through the internal mammary artery's parasternal perforators. Both the lateral thoracic and the superior thoracic arteries provide some vascularity to the pectoralis major but both are usually sacrificed to obtain a greater arc of rotation. Notably, the lateral thoracic has been shown to provide a substantial vascular supply to the pectoralis major muscle and has a caliber as large as or larger than the pectoral branch of the thoracoacromial artery. Some authors have advocated the preservation of this artery to improve the flap's blood supply.[16] Cadaver injection studies suggest that the thoracoacromial artery supplies the lateral and proximal portions of the muscle and the inferior and medial sections of the muscle are dependent on the lateral thoracic artery.[17,18] Though these studies provide less than conclusive data for the preservation of the lateral thoracic artery, they do offer compelling evidence for further study regarding the optimum blood supply to this flap.

The use of the PMMF for head and neck reconstruction usually involves the harvest of a skin paddle along with the muscle. The skin of the anterior chest wall is adequately supplied by the thoracoacromial axis superiorly and laterally by the fourth intercostal space. However, inferior and medially, the skin is supplied by direct musculocutaneous perforators from the internal mammary vessels. The use of an inferiorly and medially placed skin paddle relies on a network of "choke" vessels that link the adjacent angiosomes (Figure 41-7). This skin paddle can be compromised when based on the thoracoacromial artery if these "choke" vessels are insufficient or become occluded. This may be the anatomic reason for skin paddle necrosis with intact blood supply to the underlying muscle.[19] An even more tenuous situation occurs when the skin paddle is extended onto the abdominal skin by including a portion of the underlying rectus fascia. This orientation introduces an area of "random" skin in an angiosome "once removed" from the angiosome of the main arterial pedicle. The use of a laterally placed skin paddle along the free edge of the pectoralis major muscle in the area of the inframammary fold may allow for more predictable



FIGURE 41-7. Patient markings for pectoralis major myocutaneous flap with a medially based skin paddle.

cutaneous blood supply. According to Reid and Taylor's ink injection studies,[18] this region derives its blood supply through direct fasciocutaneous perforators from the thoracoacromial axis (Figure 41-8). This benefit would come with a slightly decreased arc of rotation.

Flap Harvesting

The patient's chest should be prepared for surgery to include the upper abdomen, past midline of the chest, as well as the ipsilateral shoulder and axilla. The ipsilateral arm may be placed on an arm board and extended to facilitate dividing the humeral attachment. Design of the skin paddle should take into consideration the shape and extent of the defect as well as the anatomic position to maximize the vascularity of the skin paddle. The most common location of the skin paddle for head and neck reconstruction is a parasternal skin paddle, which utilizes the "choke" vessel system between the thoracoacromial artery and the internal mammary perforators (see Figure 41-7). Alternatively, a laterally positioned skin paddle takes advantage of the direct fasciocutaneous perforators from the thoracoacromial axis (see Figure 41-8). Next, the arc of rotation for the desired skin paddle should be estimated using a lap sponge or suture held at the junction of the middle and lateral third of the clavicle and estimating the length of pedicle necessary by extending it to the farthest point of the defect. This length of lap sponge can then be transposed to the chest by rotating around the point held at the clavicle. The point estimated on the chest represents the distalmost point of the necessary soft tissue reconstruction. Finally, access to the anterior chest wall can be designed in a curvilinear fashion from the ipsilateral axilla (see Figure 41-7) across the anterior chest wall to the medially placed skin paddle. This incision design allows for direct exposure of pectoralis major muscle with minimal undermining, and less tunneling is necessary to pass the flap over the clavicle. In female patients or those adverse to a scar across the chest, an inframammary skin incision can be utilized (see Figure 41-8), but this limits access and visualization of the upper portions of the muscle and requires significant undermining to pass the flap to the head and neck.



FIGURE 41-8. Laterally based skin paddle.

The skin is incised from the shoulder to the superior margin of the proposed skin paddle and extended down the lateral portion of the skin paddle. All layers are incised until the pectoralis major muscle is exposed. The skin is then undermined laterally to visualize the lateral and inferior borders of the pectoralis major muscle. This allows the surgeon to evaluate the position of the skin paddle's orientation over the muscle and can be adjusted as necessary to minimize the amount of "random" skin that is harvested. The circumference of the skin paddle is then incised down to the muscle and the skin should be sutured down to the muscle to prevent shearing of the perforators during harvest. The skin and fascia of the access incision are then undermined cephalad to communicate with the neck incision and a wide tunnel (four fingers wide) over the clavicle should be created for adequate passage of the flap pedicle without compression.

Separation of the pectoralis major from the anterior chest wall is then started at the lateral border of the muscle where a plane between the pectoralis major and minor muscles can be identified. This is continued inferiorly to detach the pectoralis major from the ribs. Care must be taken to identify and ligate the intercostal perforators, and monopolar cautery should be avoided. This dissection is carried cephalad by detaching the sternal origins of the muscle. The second and third intercostal perforators can be preserved by moving the dissection of the muscle at this point more lateral to avoid these arteries, which supply the deltopectoral flap. As this dissection progresses superiorly, the pectoral branch of the thoracoacromial artery will come into view on the undersurface of the pectoralis major along the medial aspect of the

pectoralis minor muscle. One or two branches of the pectoral nerve will also be seen in this area and should be divided to provide a greater arc of rotation.

Once the pedicle is in view, the humeral attachment can be divided with meticulous hemostasis to prevent postoperative hematoma formation. During this dissection, the lateral thoracic artery can be identified. The decision to preserve the lateral thoracic for additional blood supply to the flap should be made at this point. This dissection can be carried up to the clavicle while preserving the thoracoacromial artery. Once complete, the flap can be turned over the clavicle and passed through the tunnel to reach the head and neck site. The donor site should be evaluated for hemostasis and primary closure obtained over suction drains with wide undermining if necessary.

Applications

The usefulness of the pectoralis major myocutaneous flap is limited by its arc of rotation, character and quantity of skin available for reconstruction, and bulk of tissue in the neck. However, the flap is well suited for reconstruction of floor of mouth and lateral mandibular defects when a delayed bone reconstruction is considered. Essentially, any defect of the lower third of the face can be reached with this flap. However, it is not ideal for partial glossectomy defects owing to the relative immobility of the flap.

Latissimus Dorsi Myocutaneous Pedicled Flap

The latissimus dorsi myocutaneous flap (LDMF) is a very versatile flap for head and neck reconstruction. Owing to its flat, broad nature and large amounts of soft tissue available, this flap can cover huge defects of the head and neck. Indeed, the LDMF was the first musculocutaneous flap to be reported in the literature in 1896,[20] but it was not reported for use in the head and neck until 1978.[21]

The latissimus dorsi muscle arises from the thoracic spine (T6–12), the lower four ribs, the iliac crest, and the thoracolumbar fascia. The muscle converges around the teres major and inserts into the intertubular groove of the humerus. Like the pectoralis major, the latissimus dorsi is classified as a type 5 muscle by Mathis and Nahai[1] with its one dominant pedicle being the thoracodorsal artery and its segmental secondary pedicles consisting of the paraspinal perforators. The thoracodorsal artery is a branch of the subscapular artery, which is itself a branch of the third part of the axillary artery. The thoracodorsal artery also gives off branches to the teres major, serratus anterior, and subscapularis muscles as well as the angular branch to the inferior tip of the scapula. The vascular territories of the skin can be divided into three angiosomes. Zone I is the superior third of the muscle, which is supplied by perforators from the thoracodorsal artery. Zone II is the middle third of the muscle, which is mainly supplied by the paraspinal perforators. Zone III is the caudal third, which is fed by the lumbar arteries.[2] Placement of a skin paddle over this distal zone may be at risk of necrosis owing to this "once removed" relationship of that angiosome. It is the anterior free margin of zone II where an abundance of perforators exist and the skin paddle can be most ideally placed.

Flap Harvesting

The most cumbersome aspect of utilizing this flap is patient positioning. The flap can be approached in a supine position with the ipsilateral arm extended across the chest and rolls placed under the ipsilateral shoulder and hip to expose the back. However, a lateral decubitus position allows for easier access to this flap, although it requires multiple preparing and draping of the head and neck to avoid contamination. Once properly positioned, the surface landmarks should be identified and marked. These include the midpoint of the axilla, the midpoint between the anterior and the posterior superior iliac spines, the scapular tip, and the spinous processes of the vertebrae. A line connecting the midpoint of the axilla and the midpoint of the iliac crest approximates the anterior free border of the latissimus dorsi muscle. The thoracodorsal artery runs 3 to 4 cm posterior to the edge of the muscle and divides into a horizontal and vertical branch just below the scapular tip. A skin paddle can be designed over the anterior edge of the muscle overlying zone II as described previously (Figure 41-9). This design provides adequate perfusion of the overlying skin as well as good length of pedicle for rotation into the head and neck.

Incision of the anterior margin of the skin paddle and extension along the line estimating the anterior edge of the muscle up to the axilla allows for identification of the muscle's leading edge. At this point, an attempt to identify the pedicle can be made by elevating the muscle off the posterior chest wall. A branch off the thoracodorsal pedicle will be encountered supplying the serratus anterior muscle and can be traced proximally to identify the main thoracodorsal pedicle. Once the thoracodorsal artery is identified, the branch to the serratus is ligated. Alternatively, the remainder of the skin paddle can be incised down to the underlying muscle and can be sutured down to the underlying fascia to prevent shearing of the cutaneous perforators. The surrounding skin is then undermined to expose the origins of the muscle and can be



FIGURE 41-9. Patient markings for latissimus myocutaneous flap with the patient in the lateral decubitus position.

detached from its origins using electrocautery. Working from inferior to superior, the muscle is raised off the posterior chest wall with identification of the vascular pedicle on the undersurface of the muscle near the tip of the scapula. Once identified, the pedicle can be followed proximally ligating and dividing the muscular branches as well as the angular branch to the tip of the scapula. The humeral attachment of the latissimus dorsi muscle must be detached carefully to avoid damage to the underlying pedicle. Finally, the circumflex scapular vessels will be identified at the most proximal portion of the pedicle dissection and can be ligated to provide even more mobilization.

Once the flap is elevated, a tunnel must be prepared to pass the flap to the head and neck region. This is accomplished by identifying the lateral edges of the pectoralis major and minor muscles and creating a plane between them. This dissection continues until the level of the clavicle where a separate incision is necessary. The attachments of the pectoralis major to the clavicle must be incised while attempting to preserve the thoracoacromial vascular pedicle to the pectoralis major muscle. A generous passage into the neck should be achieved and can be estimated by the ease of passage of three or four fingers through the tunnel. The flap can then be passed through the tunnel with care not to twist the pedicle.

Applications

Like the pectoralis major myocutaneous flap, the utility of the latissimus dorsi flap is limited by its arc of rotation. However, the area that the latissimus can reach in the head and neck is quite broad, extending as far superiorly as the apex of the skull. Owing to the large surface area of available muscle and skin, this flap is well suited for use in defects where a large surface area of soft tissue is necessary for reconstruction.

Microvascular Soft Tissue Flaps

The continued penetration of microvascular reconstructive techniques into head and neck surgery has been astounding. Once thought to be radical, last-resort procedures that were fraught with failure and complications, these procedures have come to the forefront of head and neck reconstruction and have unsurpassed reliability and versatility. These reconstructive techniques have allowed for customized reconstruction options for tumor and trauma patients in whom previously a "one-size-fits-all" approach was the norm. The use of microvascular reconstruction often allows for more comprehensive ablative surgery without the limitations placed on the ablative surgeon that local or regional flaps have incurred in the past. Furthermore, in anatomic regions like the maxilla, midface, and skull base where reconstructive options were particularly limited and resulted in poor cosmetic and functional outcomes, free tissue transfer has proved to be invaluable in providing meaningful surgical outcome improvements. Finally, several studies have shown that microvascular reconstruction does not incur higher cost for treatment compared with conventional reconstructive techniques.[22,23]

Though the benefits of microvascular surgery are numerous, the limitations of these techniques are also clearly delineated. The need for specialized training and instrumentation are certainly obstacles that are becoming decreasingly prevalent, with more training facilities exposing residents and fellows to microvascular reconstruction. Also, patient factors such as hypercoagulability states or vessel depletion after ablative surgery must be taken into consideration. Finally, the increased operative time required for these procedures may make medically compromised patients poor candidates for this type of reconstruction.

It is difficult to classify many microvascular flaps into soft tissue or bone flaps because many have variations in harvesting that can easily incorporate composite tissue. However, this section attempts to describe some of the more common flaps used in soft tissue reconstruction of the head and neck.

Radial Artery Fasciocutaneous Flap

In many institutions, the radial artery fasciocutaneous flap (RAFF) or radial forearm free flap has supplanted the pectoralis major myocutaneous flap as the "workhorse" soft tissue flap for head and neck reconstruction. Popularized by Soutar and associates in 1983,[24] the RAFF has become one of the most commonly used and reliable soft tissue free flaps for head and neck reconstruction. This versatile flap offers thin, pliable skin that can be as large as nearly the entire forearm from the flexor crease of the wrist to the antecubital fossa except for a small strip over the ulnar aspect of the forearm. The flap may also be harvested with partial-thickness radial bone, palmaris longus tendon, brachioradialis muscle, and lateral antebrachial cutaneous nerve. The characteristics of the skin of the forearm allows for this flap to be nearly ideal for reconstruction of areas in the head and neck that require thin, mobile tissue such as the tongue, floor of mouth, and soft palate. The skin paddle should be centered over the radial vessels, but the design of the flap can be tailored to the particular reconstructive situation. Bilobed, double paddle, folded, tubed, and fascia-only flaps have been described.

The radial and ulnar arteries are the terminal branches of the brachial artery and supply the hand and fingers through an arterial array known as the *superficial and deep palmar arches*. The radial artery ends in the deep palmar arch and the ulnar artery ends in the superficial palmar arch. In most individuals, the superficial palmar arch alone can supply adequate blood to the hand and fingers either directly by sending branches to all five digits or by communicating branches to the deep palmar arch to supply the thumb and forefinger. However, in an estimated 12% of individuals,[25] the superficial palmar arch is incomplete *and* no communication between the deep and the superficial palmar arches exists. It is in these patients that harvesting the radial artery along with a RAFF can have catastrophic consequences to the distal upper extremity. If the ulnar distribution is undisturbed, the third, fourth, and fifth digits should still remain viable but the thumb and forefinger are at risk for ischemia should the previously described circumstances be present.

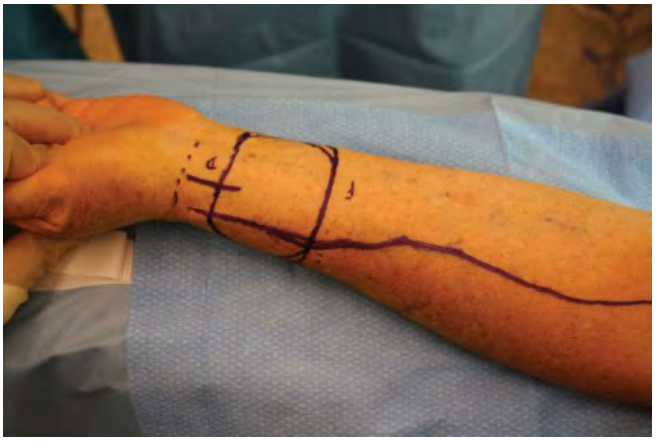


FIGURE 41-10. Skin markings for radial artery fasciocutaneous flap

To avoid this complication, the performance of an accurate Allen's test is essential and in cases in which this test is equivocal, radial and ulnar artery duplex studies may be performed to objectively observe the pattern of flow and the reversal of flow with total occlusion of the radial artery.

Flap Harvesting

The arm should be prepared circumferentially up to the axilla and draped accordingly for access to the entire arm. The distal portion of the radial artery should be palpated and marked as well as the distal location of the cephalic vein in the "snuffbox" region. Flap design should be carefully considered to approximately center the flap over the radial vessels as much as possible, with particular care in selecting the orientation of the pedicle relative to the skin paddle (Figure 41-10). Once marked, the arm is exsanguinated with an elastic wrap and the tourniquet inflated to 250 mmHg. The tourniquet time should be recorded, as should the overall ischemia time to the flap.

The initial incision is along the palmar crease of the forearm, and initial identification of the distal radial artery and venae commitantes as well as the cephalic vein is performed. These should then be isolated and ligated. The superficial branches of the radial nerve will also be encountered here and can be preserved or transected, leaving an area of anesthesia over the dorsum of the thumb and forefinger. Once the distal vasculature is secured, the remainder of the skin paddle incisions can be made. The flap is then raised off the deep forearm muscles and tendons from medial to lateral in a subfascial plane. This dissection will traverse the flexor tendons of the forearm, which must maintain a thin layer of paratenon to facilitate skin graft healing. This dissection continues until the lateral intermuscular septum is approached. Similarly, on the radial side, the flap is elevated from lateral to medial in the same subfascial plane. Here the superficial branches of the radial nerve can again be preserved by breaking the subfascial plane of dissection or sacrificed. Once complete, the flap can then be elevated from distal to proximal by carefully elevating the radial artery and veins along the intermuscular septum. Vascular clips are necessary to control the numerous branches to the surrounding muscles and radial bone.

The remainder of the vascular pedicle is dissected by first making a curvilinear incision from the antecubital fossa to the proximal portion of the skin paddle. This incision is taken to subcutaneous tissue only and the skin is undermined in this subcutaneous plane medially and laterally. Care must be taken not to injure the proximal cephalic vein during this dissection. The cephalic vein is identified as it exits the skin paddle and circumferentially dissected. A vessel loop is passed around the vein and the entire length of vein is dissected up to the desired length. Finally, the proximal radial pedicle is accessed by separating the brachioradialis and the flexor carpi radialis muscles and retracting the brachioradialis laterally. The remainder of the pedicle can then be elevated up to the brachial artery if necessary (Figure 41-11).

The flap is then allowed to reperfuse for 15 to 20 minutes by releasing the tourniquet. Hemostasis can also be achieved at this time. The flap is then harvested and passed to the ablative field. A suction drain should be placed in the deep tissues in the proximal forearm and the curvilinear incision can be closed primarily. A full- or split-thickness skin graft is normally required to close the skin paddle site. The skin graft is covered with a nonadherent dressing, the arm is then padded generously with cast padding, and a volar splint is fabricated to immobilize the arm during skin graft healing.

Applications

The quality of the skin of the forearm lends itself for use in a wide variety of head and neck reconstruction applications. Most commonly used for reconstruction of tongue, floor of mouth, and buccal mucosa defects, this flap also has the versatility for use in facial skin or scalp reconstructions. The generous pedicle length allows for placement in areas quite remote from recipient vessels, and the relatively low morbidity of the procedure make the radial artery fasciocutaneous flap a workhorse in microvascular soft tissue reconstruction.

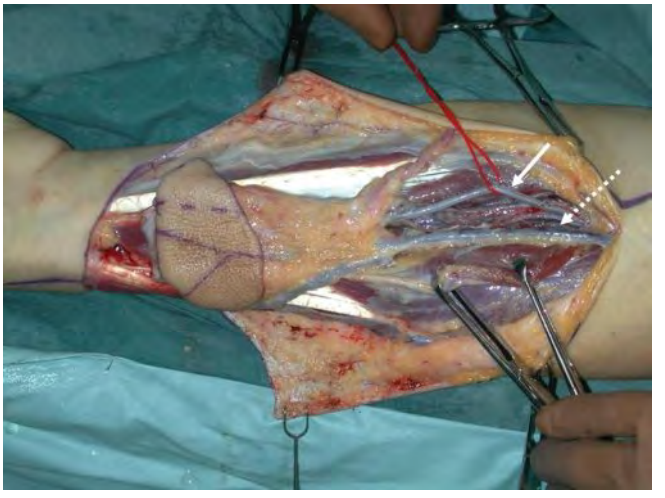


FIGURE 41-11. Radial artery fasciocutaneous flap elevated up to antecubital fossa.

Anterolateral Thigh Flap

The anterolateral thigh flap (ALTF) is based on the cutaneous perforators of the descending branch of the lateral circumflex femoral artery, a branch of the profunda femoris. The pedicle travels in the intermuscular septum between the rectus femoris and the vastus lateralis muscles along with the motor nerve to the vastus lateralis. The cutaneous perforators may travel through the intermuscular septum between these muscles (septocutaneous perforators) or through a portion of the vastus lateralis (musculocutaneous perforators). This latter variation appears to be the more common configuration. The skin of the lateral thigh may also be supplied by the transverse branch of the lateral circumflex femoral artery or directly from the deep femoral artery. Though the flap has enjoyed a recent explosion in popularity, it is these variations in vascular anatomy that have detracted from the flap's overall acceptance in head and neck reconstruction.

The pedicle to this flap can support a large area of relatively thin and flexible skin, depending on the patient's overall body habitus. Up to 800 cm^2 has been reported,[26] encompassing an area from the greater trochanter of the femur to a line 3 cm above the patella. No preoperative evaluation is required before flap harvesting[27]; however; the use of a handheld Doppler can identify the dominant perforator to the thigh skin. The most likely position of this perforator can be estimated by drawing a line from the anterior superior iliac spine (ASIS) to the superolateral corner of the patella. At the midpoint of this line, a 3-cm-radius circle is drawn and the most likely position of the skin perforator will be in the inferolateral quadrant of this circle. The skin paddle should be centered on this point (Figure 41-12).

The main disadvantage to this flap is the inconsistent size and location of the cutaneous perforators. These perforators have been reported to be completely missing.[27] Celik and coworkers[28] argue that many of these reports result in a lack of recognitions of small perforators or due to the surgeon's inability to dissect the musculocutaneous perforators through the vastus lateralis muscle.[25] The reconstructive surgeon should be prepared to convert to an anteromedial thigh flap or a tensor fascia lata flap if this situation arises.[28]

Flap Harvesting

The surface landmarks for the ALTF are drawn as described previously. A Doppler probe is used to confirm the location of this perforator. The flap's skin paddle is designed to be centered on this perforator. The initial incision is made on the medial aspect of the skin paddle down through the deep fascia to the rectus femoris muscle. The flap is elevated laterally off the muscle until a cutaneous perforator is identified. Once the perforator is identified, the remainder of the skin paddle can be incised. The perforator is followed through the intermuscular septum between the rectus femoris and the vastus lateralis (septocutaneous perforators) or through the vastus lateralis muscle (musculocutaneous perforators) until the source artery is identified (Figure 41-13). The descending branch of the lateral circumflex femoral artery can be dissected proximally for the desired pedicle length or up to 16 cm.[29] Running along with the source vessels is the nerve to the vastus lateralis muscle. This nerve must be preserved to maintain motor innervation to this muscle.

Once the flap is harvested, any muscular dissection should be reapproximated and a suction drain placed in the deep tissues. The area of the skin paddle can generally be closed primarily with a modest amount of undermining of the surrounding skin if the skin paddle's dimensions do not exceed 6 to 9 cm.

Applications

For many reconstructive surgeons, this flap has supplanted the radial forearm flap as the primary soft tissue flap for head and neck reconstruction. Indeed, the low morbidity and the ability to primarily close the donor site are key advantages to utilizing this flap. However, the inconsistent nature of the perforators remains as the primary deterrent for the widespread adoption of this flap. Furthermore, the nature of the thigh skin varies with the patient's overall body habitus

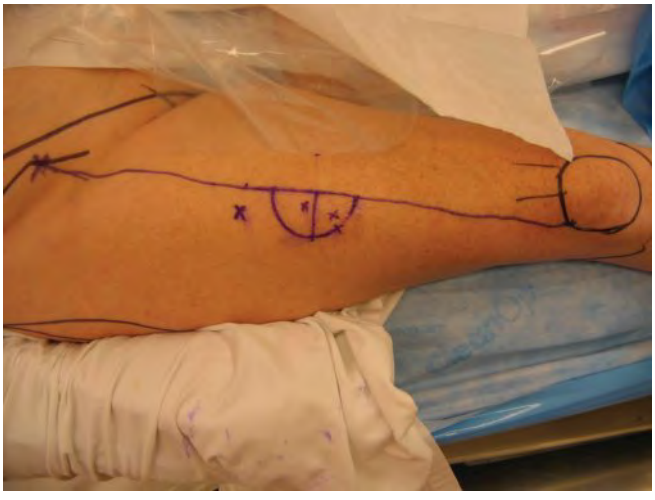


FIGURE 41-12. Skin markings for anterolateral thigh flap.

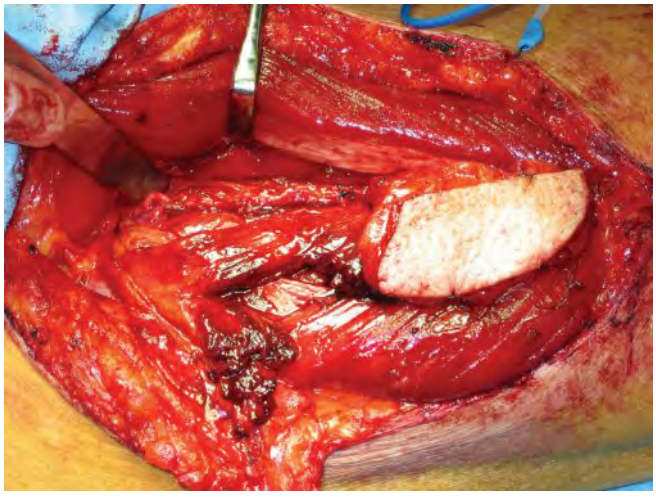


FIGURE 41-13. Dissection through vastus lateralis muscle for musculocutaneous perforators.

and is generally thicker and less pliable than the radial forearm skin. In patients who have had axillary node dissection, radial artery harvested for coronary bypass grafting, or previous trauma to the forearm, the ALTF can be an excellent alternative source of soft tissue for head and neck reconstruction.

Rectus Abdominis Myocutaneous Flap

The rectus abdominis myocutaneous flap (RAMF) is based on the deep inferior epigastric artery and vein (DIEA and DIEV) and has been a reliable flap for many areas of reconstruction. These vessels are of good caliber and a fairly long vascular pedicle can be obtained. The flap can transfer a large volume of skin, fat, muscle, and fascia, which is dependent on the patient's body habitus. This volume will decrease over time because the denervated muscle will atrophy significantly. The RAMF is well suited to cover defects that require a large volume of tissue such as total maxillectomy, total glossectomy, skull base, or scalp defects. Pedicled and free flaps based on the inferior or superior epigastric arteries have been well described for breast reconstruction. A variety of different skin paddle orientations are available for this flap depending on the needs of the reconstruction.

A perforator flap based on the DIEA and DIEV allows for harvesting the skin, subcutaneous tissue and fascia without the rectus muscle.[31] In this flap (DIEP), the cutaneous perforators are dissected through the rectus muscle to the vascular source, allowing for a much thinner flap.

The rectus muscle receives its blood supply from both the deep superior epigastric artery and vein (DSEA and DSEV) and the DIEA and DIEV. The DIEA and DIEV have approximately twice the diameter of the DSEA and DSEV and the paraumbilical musculocutaneous perforators are directly associated with the DIEA and DIEV, whereas they must anastomose to the DSEA and DSEV through a series of small vessels where flow can be reversed.[32]

The DIEA branches from the external iliac artery just cephalad to the inguinal ligament. It travels superiorly and medially to penetrate the transversalis fascia 3 to 4 cm caudal to the arcuate line on the undersurface of the rectus muscle. It courses superiorly through the muscle, giving off branches to the skin near the umbilicus.

The anatomy of the anterior abdominal wall is important when harvesting this flap because preservation of fascial sheaths is crucial to preventing postoperative hernia formation. The rectus sheath extends from the pubis to the xiphoid process and is formed by the fibrous aponeurosis of the abdominal muscles. The arcuate line is estimated by a line connecting the ASISs and indicates the change in composition of the posterior rectus sheath. Above the arcuate line, the posterior sheath is composed of the transversalis fascia and a portion of the internal oblique aponeurosis. This double layer of fascia is adequate to prevent hernias. Below the arcuate line, the posterior sheath is formed only by the transversalis fascia. If not reinforced, this area is prone to bulging or hernia formation. In this region, the anterior rectus sheath must be preserved and used to reinforce the transversalis fascia (Figure 41-14).



FIGURE 41-14. Elevated rectus abdominis myocutaneous flap. Note the bulging of the abdominal contents below the arcuate line.

Flap Harvesting

Surface landmarks for the RAMF include the midline of the abdomen and the estimated width of the rectus abdominis muscle as well as both ASISs with a line connecting them to estimate the arcuate line. The ipsilateral femoral vessels should be palpated and marked as well as the costal margin (Figure 41-15). A concentration of cutaneous perforators exists around the umbilicus and the skin paddle design should be centered in this area. Flap elevation begins by creating the superior and inferior skin paddle incisions down through the anterior rectus sheath to expose the rectus muscle. The rectus sheath is divided horizontally until the medial and lateral edges of the muscle are identified. The lateral edge of the muscle indicates the linea semilunaris and the medial edge of the muscle indicates the linea alba. Once these fascial divisions are identified, the lateral skin paddle incision can be created down to Scarpa's fascia and then undermined from lateral to medial to the linea



FIGURE 41-15. Skin markings for rectus abdominis myocutaneous flap.



FIGURE 41-16. Caudal portion of rectus abdominis muscle exposed.

semilunaris. The same can be performed medially. The rectus sheath is then incised along the linea semilunaris and the linea alba.

To access the DIEA and DIEV, a vertical incision is made from the inferior portion of the skin paddle down toward the femoral vessels down to the anterior rectus sheath. The rectus sheath is incised at the midpoint of the long axis of the muscle and undermined laterally and medially to expose the caudal portion of the muscle (Figure 41-16). This split anterior rectus sheath will serve as the hernia-preventing layer inferior to the arcuate line to preserve the integrity of the abdominal wall. Once the incision is completed and the caudal rectus muscle is exposed, the flap may be elevated from superior to inferior off the posterior rectus sheath. The mixed motor/sensory nerve supply from the intercostals nerves will be encountered laterally as the flap is elevated and can be ligated and divided. These nerves have been reported to be useful in segmentally reinnervating the rectus muscle. On the undersurface of the muscle, the DIEA and DIEV will come into view. The pedicle should be protected while the inferior portion of the rectus muscle is transected at any point inferior to the vascular pedicle. The pedicle is followed inferiorly until the desired length is achieved or the external iliac vessels are reached. The DIEVs usually join to become a single vein just before its take-off from the external iliac vein, which may be beneficial depending on the size of the recipient vein. The pedicle is then divided and the flap passed to the ablative field.

Closure of the donor site begins with reapproximation of the inferior portion of the cut anterior rectus sheath. Again, this layer will serve to restore the integrity of the abdominal wall inferior to the arcuate line. The superior portion of the anterior sheath that was harvested with the flap may also be reapproximated by large, slowly absorbable suture. Care should be exercised to prevent visceral injury with suture needles or other sharp instruments. Finally, a layered primary closure of the skin can be achieved with wide undermining with suction drains placed in the dead space.

Applications

Owing to the large volume of skin and muscle available for reconstruction, the RAMF is ideally suited for applications in which a large bulk of tissue is necessary. Total maxillectomy and total glossectomy defects are the most common indications, though it may also be useful in scalp or facial skin reconstructions. The potential for reinnervation of the muscle by anastomosis of the segmental nerves to a recipient nerve in the ablative field makes this flap feasible for facial reanimation surgery.

Latissimus Myocutaneous Free Flap

The latissimus myocutaneous free flap shares the anatomic and flap harvesting details with the latissimus rotational flap. Obviously, a tunnel through the axilla is not necessary for use as a free flap and the thoracodorsal vessels are divided. The flap is useful in head and neck reconstruction when a large surface area of soft tissue is necessary and the ability to anastomose the pedicle to local recipient vessels eliminates the bulk of the pedicle traversing the axilla and neck (Figure 41-17).

Lateral Arm Free Flap

Introduced in 1982 by and colleagues,[32] the lateral arm free flap derives its blood supply from several septocutaneous perforators through the lateral intermuscular septum from the posterior radial collateral artery and vein (PRCA and PRCV, respectively). The PRCA is itself a branch of the profunda brachii artery, which is a branch off of the brachial artery. The flap has the potential for dual outflow with the PRCV venae commitantes serving as the deep venous system and the cephalic vein as the superficial system. The flap may be harvested with skin, soft tissue, nerve (posterior cutaneous nerve of the arm [PCNA]), muscle (triceps), and bone (partial thickness humerus). Large amounts of skin and soft tissue have been reported , the largest being 18 × 11 cm,[33] but most are limited to one third of the upper arm

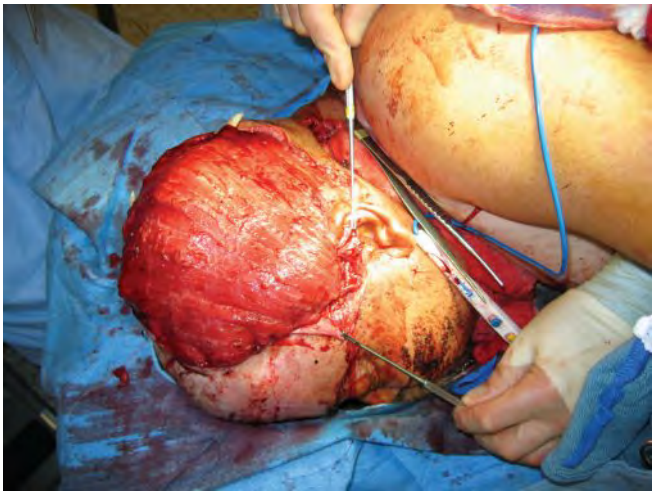


FIGURE 41-17. Latissimus muscle flap for near-total scalp reconstruction.

circumference in order to facilitate primary closure of the donor site defect.

The lateral arm flap's primary advantage is a potentially thin, pliable soft tissue paddle without the concern of ischemic injury to the upper extremity as with the radial forearm flap. Also, the donor site defect can often be closed primarily without the need for an additional skin graft donor site. The primary disadvantages to this flap are the relatively short vascular pedicle and the proximity of the profunda brachii artery to the radial nerve with potential for injury during dissection.

Flap Harvesting

The arm should be circumferentially prepared and draped exposing the upper forearm to the axilla. The surface landmarks include the lateral epicondyle of the humerus and the V-shaped insertion of the deltoid muscle. A line is drawn between the point of the V, and the lateral epicondyle represents an area 1 cm anterior to the lateral intermuscular septum (Figure 41-18). The skin paddle is designed in a fusiform shape with its long axis 1 cm posterior to this line.

The initial incision is made at the anterior margin of the skin paddle down to the brachioradialis and brachialis muscles. The posterior cutaneous nerve of the forearm (PCNF) may be encountered in the distal portion of this incision and may be preserved or sacrificed, creating an area of anesthesia in the upper forearm. The dissection continues in a subfascial plane posteriorly toward the intermuscular septum where septocutaneous perforators will be identified. The posterior skin paddle incision can then be made down to the triceps muscle and dissected in the subfascial plane anteriorly toward the intermuscular septum and identification of the septocutaneous perforators. Following these perforators proximally will lead to the source artery and vein (PRCA and PRCV). The same vessels are then identified from the anterior dissection by separating the brachioradialis from the intermuscular septum. The radial nerve will be encountered in this dissection and must be preserved.



FIGURE 41-18. Skin markings for lateral arm flap.



FIGURE 41-19. Elevated lateral arm flap with generous pedicle length.

The flap is elevated from distal to proximal by first ligating the continuation of the PCRA distally to become the recurrent interosseous artery and separating the septum from the humerus. The PCNA and the PCNF run along with the pedicle. The pedicle is followed proximally through the intermuscular septum toward the spiral groove of the humerus. Part of the deltoid attachment to the humerus may be divided to allow access to the proximal pedicle (Figure 41-19). The pedicle is severed when the desired pedicle length is achieved.

A suction drain may be placed in the deep tissues. The brachialis and triceps muscles can be lightly reapproximated and primary closure can be achieved in most cases.

Applications

The applications for the lateral arm flap mirror those of the radial forearm and ALTF. The skin quality of the upper arm is generally thin and pliable and a generous vascular pedicle is available for harvest. The main advantage of this flap over the radial forearm is that the distal circulation of the arm and hand is not dependent upon the PRCA and, like the ALTF, may serve as an effective alternative in patients in whom a radial forearm flap is contraindicated.

TECHNIQUES FOR HARD TISSUE RECONSTRUCTION OF THE ORAL CAVITY

Nonvascularized Bone Grafts

The most commonly used nonvascularized bone grafts in the head and neck are the anterior iliac crest, posterior iliac crest, tibial bone, and cranial bone. When the quantity of needed bone is small, intraoral sites can also be a source for harvesting. Sites in the oral cavity such as the ramus of the mandible, the anterior symphysis region, and the maxillary

tuberosity region are commonly used for these purposes. Owing to chapter length constraints, the intraoral donor sites are not covered and the readers are encouraged to review other chapters within this text covering dental implants to obtain details of intraoral bone harvesting. This chapter focuses more on larger bone grafts for use in maxillofacial reconstruction.

Iliac Crest (Anterior and Posterior)

The iliac crest system has for a number of years been the most popular site for the harvesting of nonvascularized bone grafts for use in the maxillofacial region. Reconstruction of defects measuring less than 5 cm in size can be accomplished with a single anterior iliac crest, whereas greater defects warrant either bilateral harvest or harvesting of the posterior iliac crest. An established guideline is that the anterior iliac crest allows for the harvest of approximately 50 cc of uncompressed bone, and the posterior iliac crest allows for 100 cc of bone.

The overview of the anatomy is divided into the anterior iliac regional anatomy and the posterior iliac anatomy.

Anterior Crest

The anterior ilium is located between the ASIS and the tubercle of the ilium. The inguinal ligament attaches to the ASIS laterally and to the pubic symphysis medially. Inferior to the attachment of the inguinal ligament is the sartorius muscle attachment; the muscle then travels in a diagonal fashion to insert along the medial aspect of the tibial head. Lateral to the sartorius is the origin of the tensor fascia latae muscle. This muscle attaches along the inferior aspect of the lateral crest for a few centimeters in a lateral direction. Directly lateral to the tensor fascia latae is the iliotibial tract. Behind the iliotibial tract is the gluteus medius muscle. The medial surface of the ilium is covered by the iliacus muscle.

The main blood vessel in this region is the deep circumflex iliac artery, which courses superficial to the iliacus muscle medially. Several sensory nerves traverse this area but only two are of significance; the lateral cutaneous branch of the iliohypogastric nerve (L1, L2), and the lateral cutaneous branch of the subcostal nerve (T12, L1).

Posterior Crest

The muscles attached to the posterior aspect of the iliac crest are fewer than those attached to the anterior. The main muscles are the gluteus medius and maximus. The gluteus maximus has the more medial attachment. The sensory nerves in this region are the superior and middle cluneal nerves. The superior cluneal nerve (L1–3) pierces the lumbodorsal fascia superior to the posterior iliac crest and innervates the skin over the posterior buttocks. The middle cluneal nerves (S1–3) emerge from the sacral foramina and course laterally to innervate the medial buttocks. The sciatic nerve is the only motor nerve in this region and it runs deep to the gluteus maximus. It emerges between the piriformis muscle and the superior gemellus muscle on its inferior course to the lower limb.

The blood vessels in the region are the superior and inferior gluteal arteries. The terminal branches of the superior gluteal artery may be encountered because they may be found between the gluteal maximus and medius.

Graft Harvesting

Anterior crest The patient is placed in a supine position with a bump under the hip to be harvested. This maneuver elevates the iliac crest and facilitates the palpation as well as the harvest of the bone. Once the patient is prepared and draped, the skin marking is made. Care should be taken to place the incision in a location where it will not interfere with wearing pants or skirts. This is accomplished by rolling the skin in a cephalad/cranial manner before making the mark. The incision should be a couple of centimeters lateral to the ASIS and should follow the curvature of the ilium. The skin and subcutaneous fascia and Scarpa's fascia are incised and the periosteum overlying the crest is incised and reflected. The medial aspect of the iliac is addressed by reflecting the iliacus. The exposure is maintained by placing a Taylor retractor. The amount of bone needed is then marked with an oscillating saw, taking care to make the medial cut in an oblique fashion away from the ASIS. This oblique cut away from the ASIS prevents the undermining of the ASIS and fracture. Several approaches to the harvest as it relates to the crest have been reported. Irrespective of the approach used, if the cortical bone is to be harvested, the amount needed is outlined and harvested. The cancellous bone is harvested with the aid of large curettes until the desired quantity is obtained.

Posterior crest Harvesting of the posterior iliac crest necessitates that the patient be placed in a prone position. Once in prone position and the airway secured, the hip ipsilateral to the donor site should be elevated using a bump made of folded linen or an intravenous bag. The patient is then prepared and draped. The hip is palpated and marked. An exact palpation is difficult in obese patients. In these cases, the palpation should begin at the inferior rib cage bilaterally and move caudally until the lateral projection of the iliac bone is felt. The midline is marked and the curvature of the posterior iliac is also marked. A linear incision is made closer to the medial aspect, and the dissection is continued through the subcutaneous fat until the fascia overlying the muscle is encountered. At this point, the periosteum is reflected and the bone is exposed. A self-retaining retractor is placed and the bone is harvested in a similar manner to that for the anterior iliac crest.

The closure of the site for either anterior or posterior harvesting is straightforward. The bleeding is often diminished when the cancellous bone is completely harvested at the site, causing the marrow bleed to stop. Use of hemostatic agents such as microfibrillar collagen is often done in order to maintain hemostasis. Some surgeons advocate the use of resorbable mesh to re-create the contour of the crest in cases in which this was harvested. A drain is placed and the closure is continued by reapproximating the fascia and dermis followed by skin. The posterior closure is done in a similar fashion (Figure 41-20).



FIGURE 41-20. **A,** Patient positioned in a prone fashion with markings made depicting the posterior iliac crest. **B,** Exposure of the posterior iliac crest bone with placement of a deep retractor. **C,** Closure of the donor site. **D,** Harvested bone consisting of both cortical and cancellous bone. **E,** Insetting of the harvested bone to re-create the mandibular contour. **F,** Postoperative computed tomography (CT) scan depicts the newly reconstructed mandible.

The anterior iliac crest has specific advantages and disadvantages that are not shared by the posterior iliac crest. Its main advantages are the ease of harvest and ability to have a two-team approach. The main drawback for the anterior iliac is the limited quantity of available bone. The possibility for gait disturbances exists if too much reflection of the tensor fascia lata is performed. This scenario results in pain on ambulation and possible delays in rehabilitation.

The posterior iliac crest has a distinct advantage of greater quantity of available bone to be harvested. The amount of bone on each side is around 100 cc, thus allowing for reconstruction of large defects. The main disadvantage of the posterior harvest is the need for repositioning of the patient. This fact increases the operative time while making it impossible to have a two-team approach. Another potentially serious problem associated with the posterior iliac crest is the possible displacement of the endotracheal tube when the patient is moved. This can lead to a disastrous outcome.

Tibial Bone Graft

The tibial bone graft was initially described for use in maxillofacial reconstruction by Catone and associates.[34] Since that description, it has become very popular and is routinely performed in the office setting under intravenous sedations.

The tibia is the main bone of the lower leg. It provides support for weight bearing, and it is medial to the fibula. The superior aspect of the bone has the tuberosity, which serves as the point of insertion for the quadriceps femoris; it is medial to the sartorius, gracilis, and semitendinosus muscles. Lateral to the tuberosity is the origin of the tibialis anterior muscle. The patellar ligament inserts superiorly. Anteriorly and laterally, the proximal tibia exists an oval protuberance called Gerdy's tubercle. This is the main target for the bone graft.

The main blood supply to the region is the inferior medial genicular artery and the inferior lateral genicular artery. Both of the genicular arteries are branches of the popliteal artery. The saphenous nerve is the only sensory nerve in the region.

Graft Harvesting

The patient's knees should be elevated with a bump so as to cause a moderate degree of flexion. The area is then prepared and draped. Local anesthesia is infiltrated along the palpated Gerdy's tubercle in an oblique line of approximately 3 cm. The skin is incised down to the subcutaneous fascia; the periosteum is then incised and reflected. A bone window is marked with a surgical drill and a fine bur. The cortical bone is removed, thus gaining access to the marrow space. Using a curette, the bone is harvested. Usually the amount of bone that can be harvested is around 20 to 40 cc. Once the harvest is completed, a hemostatic agent may be placed in the area, if so desired, followed by approximation and closure of the periosteum. The remaining closure is as usual.

Minimal complications exist with the harvesting of the tibial bone. A fairly common sequelae is the formation of ecchymosis along the lower leg extending to the ankle. Given that this is almost an expected sequelae, the patient should be informed of it before the surgery.

Cranial Bone Graft

Cranial bone grafts have long been used in maxillofacial reconstruction. Their use has been largely associated with the reconstruction of craniofacial defects. More recently, they have been used for mandibular and maxillary alveolar ridge augmentation in cases of severe atrophy. One of the major advantages of the cranial bone is its ability to withstand intraoral exposure and resist resorption.

The cranial vault is made up of several bones, each with a contralateral match. The bones that make up the skull are the frontal, parietal, temporal, sphenoid, and occipital. The sagittal sinus is directly inferior to the midline of the skull along the vertex. This area is to be avoided during cranial bone harvesting. The parietal bone has the greatest thickness and also the best location for ease of harvest.

Graft Harvesting

The cranial bone graft may be harvested in one of three ways: particulate, split thickness, or full thickness. The most commonly harvest method is the split thickness and, therefore, the one covered in this chapter.

The parietal bone is the most commonly harvested cranial bone. The approach is made either via a hemicoronal, a coronal, or a horizontal incision over the area to be harvested. Once the incision is made, it is extended to the pericranium. The scalp is retracted and the area to be harvested is marked with a thin bur, usually multiple strips are marked out. Following this, a round bur is used to feather the bone outside of the markings in order to create a bevel and facilitate the saw cut. Using a thin reciprocating saw, the strips are harvested, taking care not to break them.

The closure of the donor site is begun by first establishing hemostasis. The bone bleed can be controlled with the aid of bone wax and the soft tissue bleeds may be cauterized, taking care not to damage the hair follicles. A suction drain is placed and the incision is closed in a layered fashion. The scalp may be closed with staples or sutures (Figure 41-21).

The most commonly encountered complication is the formation of a hematoma. A hematoma is more often seen in cases in which a drain is not placed. The more feared complication of a cranial bone graft is an inadvertent cranial penetration with or without dural tear. This complication is rare if care is taken to harvest small strips and bevel the bone so as to have a less acute angle of harvest. A rare but dreaded complication is a subdural bleed. Given this, the patient should be monitored for altered mental status for several hours after cranial bone harvest.

Vascularized Flaps

Osteocutaneous Radial Forearm Flap

The radial forearm flap has enjoyed tremendous popularity since its initial description. The vast majority of radial forearm flaps (RFFFs) used in head and neck reconstruction are soft tissue–only flaps. The use of these flaps as bone-containing flaps was first described by Soutar and coworkers.[35] The use of the RFFF as an osteocutaneous flap is

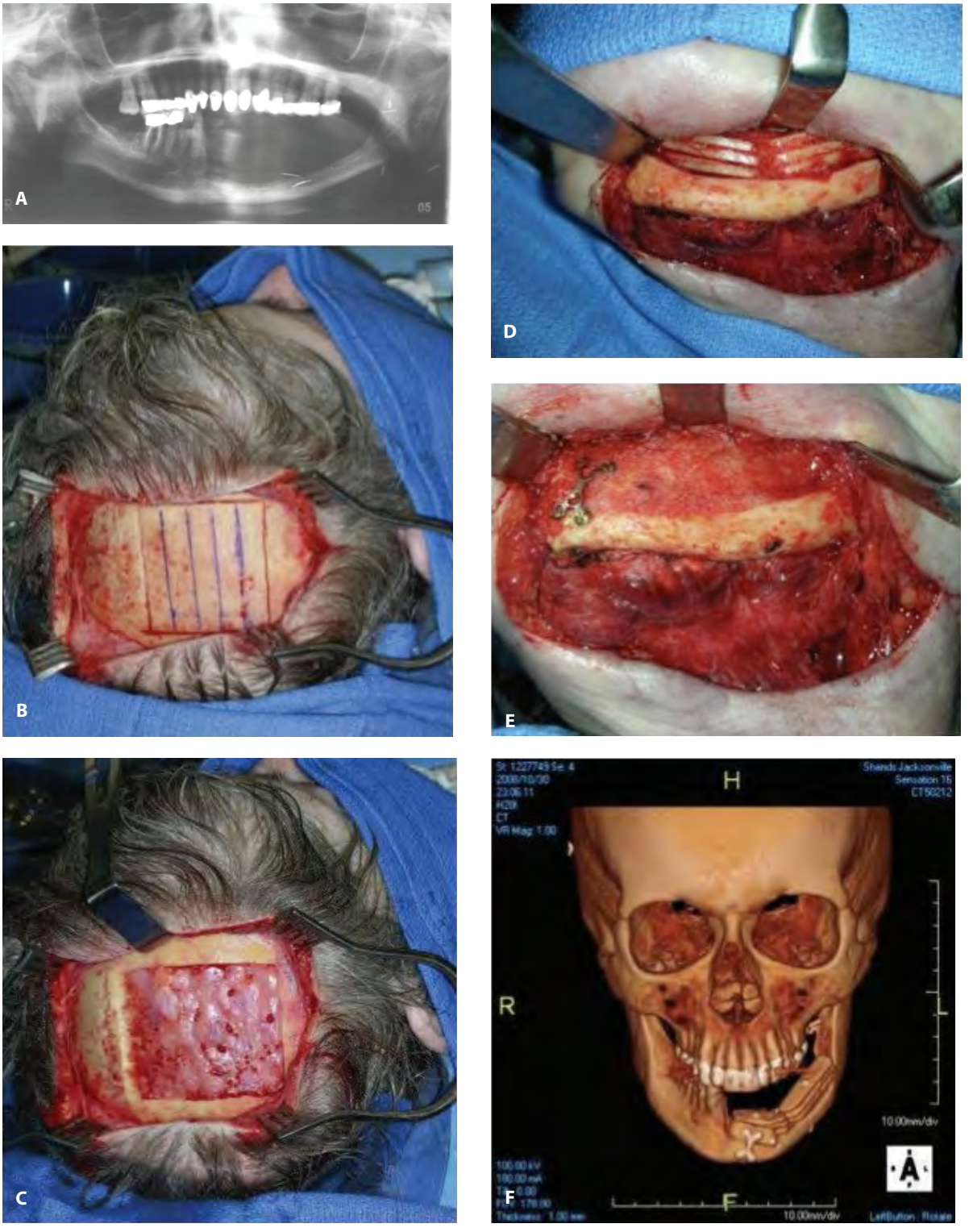


FIGURE 41-21. **A,** Panoramic radiograph of a mandible after a left marginal mandibulectomy was performed secondary to a squamous cell carcinoma. **B,** Harvesting of the cranial bone with markings of the planned strips. **C,** Use of bone wax to improve hemostasis after split calvaria bone harvest. **D,** Insetting of the harvested cranial bone in a stacked fashion. **E,** The cranial bone stack is secured to the native mandible using titanium plates and screws. **F,** Postoperative CT depicts the reconstructed left hemimandible using the harvested cranial bone. (**B–F,** Courtesy of Dr. Luis Vega.)

surpassed by other bone flaps such as the fibula and the deep circumflex femoral artery (DCIA) flap. The reason for the poor performance of the osteocutaneous RFFF in head and neck reconstruction has been twofold: (1) its poor bone quantity and (2) its potential for significant donor site morbidity.

The anatomy of the RFFF has been covered earlier in this chapter. The surgical anatomy of the bone-containing RFFF is related to the bone perforators from the radial artery. The nutrition to the bone is by the periosteal and direct bone perforating the flexor pollicis longus muscle.

Flap Harvesting

The harvest begins under tourniquet control distally and proceeds in a proximal fashion. An incision is made at the most distal point of the flap and is carried to the subcutaneous fascia, directly overlying the muscles and the tendons. The tendons of the flexor carpi radialis, the brachioradialis, and the palmaris longus are identified. A subfascial plane is dissected while maintaining the paratenon over the tendons. The radial artery and the accompanying venae commitantes are identified and isolated using an angled clamp. Two 2-0 silk sutures are passed under the vessels. The vessels are then ligated and divided. Dissection continues on the radial side and the cephalic vein is identified, ligated, and divided. The cephalic vein is commonly harvested by the author in order to increase the venous drainage to the flap. Continued subfascial dissection is carried out toward the radial pedicle while taking care to identify and preserve the sensory branches of the radial nerve. At this point, attention is turned to the ulnar aspect. The skin paddle on the ulnar side is incised to the fascia and a subfascial elevation of the flap is similarly carried out toward the radial vascular pedicle.

At this point, the proximal portion of the flap is incised and a subcutaneous flap is elevated toward the antecubital fossa. The cephalic vein is further dissected until the desired length is exposed. An Allis clamp is used to retract the flexor carpi radialis muscle and dissection of the vascular bundle is carried out between the flexor carpi radialis and the brachioradialis. Dissection in this region is performed in a very meticulous fashion, taking care to preserve the perforators to the muscle and the radius. A cuff of the muscle is then incised so as to maintain the perforators to the bone. Using a periosteal elevator, the bone is exposed from both sides. A measurement of the desired bone length is marked. Using the oscillating saw, the bone is cut from the opposite side of the pedicle and in a curved fashion so as to avoid a sharp angle and, therefore, stress risers. Once the bone cut is performed, the desired length of pedicle is dissected, the tourniquet is deflated, and the flap is allowed to reperfuse. The residual radius bone can be plated using the dynamic compression plate over the harvested bone site. Prophylactic plating diminishes the incidence of radius bone fracture. The arm is closed by reapproximating the proximal flap and closing it over a suction drain. The proximal donor site defect is repaired with a full-thickness skin graft. A volar splint is placed with the wrist in a 45-degree extension. The splint is left in place for at least 2 weeks.

This flap has rapidly lost popularity and has been superseded by superior bone flaps owing to its limited bone stock and significant incidence of postoperative radius fracture. Early reports showed an incidence of fracture as high as 28% to 43%,[36–39] whereas larger series reported incidences of 23%[40] and 31%.[41] Refinements in osteotomy technique and prophylactic internal fixation have reduced this functionally disabling complication to 15%.[42] Serious frequent sequelae of pathologic radius fracture include wrist deformity and reduced wrist and grip strength from impaired flexor pollicis longus function. Postoperative radius fracture can be minimized by strictly adhering to bony dimensions not exceeding 30% of its cross-sectional area and 40% of its circumference.[43] Additional radius protection can be offered by external support with an above elbow cast or by splint to ensure 6 to 8 weeks of immobilization. However, fracture rates remain as high as 19%.[44] Prophylactic internal fixation with dynamic compression plate (DCP) is the most effective method of increasing both the torsional and the bending strength of the osteotomized radius, supported by several large clinical series.[45–48]

Osteocutaneous Fibula Flap

Hidalgo in 1989[49] performed the first case of mandibular reconstruction with the fibula using multiple defined osteotomies to reproduce the shape of almost an entire mandible. Since then, the fibula free flap has enjoyed much popularity in mandibular reconstruction and has continued to undergo technical developments.

The fibula is a long, thin, non–weight-bearing bone of the lower extremity. It has a tubular shape with a thick circumference of cortical bone providing it with significant inherent strength. Approximately 22 to 25 cm of bone may be harvested, while preserving 6 to 7 cm of bone proximally and distally to maintain integrity and functional stability of both the knee and the ankle joints, respectively. Proximally, the common peroneal nerve is encountered as it wraps around the neck of the fibula.

The fibula can be harvested as a free osseous or free osteoseptocutaneous flap. The inclusion of an overlying skin paddle is possible because septocutaneous or musculocutaneous perforators from the peroneal artery and vein provide a viable blood supply to this area of skin.

The peroneal artery and vein compose the dominant blood supply and vascular pedicle to the fibula osteocutaneous flap. Classically, the popliteal artery divides into the anterior and posterior tibial arteries below the knee, with the latter vessel subsequently giving rise to the peroneal artery. The peroneal artery and its paired venae commitantes descend in the lower leg between the flexor hallucis longus and the tibialis posterior muscles as they course toward the foot.

The peroneal artery via a nutrient medullary artery provides a rich endosteal vascular supply to the fibula along with multiple periosteal feeding vessels. The vascular supply to the skin over the fibula arises from numerous fasciocutaneous perforators running in the posterior crural septum.

Their position and course may be highly variable. The amount of skin that can be harvested is usually limited by the ability to primarily close the defect, although skin grafting the donor site defect is also frequently performed successfully.

The sensory supply to the skin over the lateral calf is derived from the lateral sural cutaneous nerve, a branch of the common peroneal nerve, arising within or above the popliteal fossa. When harvested as part of the osteocutaneous fibula flap, it can provide variable sensation to the accompanying skin paddle.

The goal of preoperative arteriography with respect to the free fibula flap is to identify patients in whom the harvest of this flap would result in either a nonviable flap or a compromised extremity.

Noninvasive clinical assessment begins with a thorough patient history and general physical examination. Claudication with walking should alert the clinician of underlying arteriocclusive disease. A more detailed examination of the perfusion status of the lower leg and foot is performed looking for signs of limb deformity, previous surgery, or trauma. Stigmata of peripheral arterial and/or venous vascular disease include skin pallor or cyanosis, ulceration, cool skin temperature, sparse hair growth, and thickened nail beds. Palpation of popliteal, posterior tibial, and dorsalis pedis pulses is mandatory. Questionable or absent pulses should be investigated further with Doppler flow assessment.

Conventional angiography, computed tomography (CT) angiography, or magnetic resonance angiography (MRA), although more invasive, provides superior anatomic detail and functional assessment of limb perfusion adequacy and quality of the donor vessels (Figure 41-22).

Flap Harvesting

The fibula free flap can be harvested as an osseous or osteocutaneous flap. The latter is described first because the former does not require preservation of cutaneous perforator vessels.

The patient is positioned supine on the operating table with the hip and knee slightly flexed and internally rotated and maintained in that position. The entire lower extremity is prepared and draped in the standard fashion with circumferential exposure up to the groin. The sole of the foot is supported with a sandbag or a 1-L saline bag. Pertinent landmarks such as the head of the fibula, lateral malleolus, and peroneal nerve are outlined on the skin. A vertical mark joining the proximal and distal fibula represents the intermuscular septum. The necessary skin island is outlined over the junction of the middle and lower thirds of the fibula to capture the largest possible caliber septocutaneous perforators. A Doppler will frequently and reliably aid in identifying these perforators, ensuring the skin island is centered over them. A sterile tourniquet is applied, the lower extremity is exsanguinated, and the tourniquet inflated to 350 mmHg.

The incision begins at the anterior margin of the skin island and extends proximally and distally to within 6 cm of the fibula head and lateral malleolus, respectively, to a level below the superficial fascia. Subfascial dissection in the lateral compartment proceeds toward the intermuscular septum where attention focuses on identifying and ensuring incorporation of the relevant perforators. Exposure and anterior retraction of the peroneus longus and brevis muscles allows their dissection off the fibula directed toward the anterior crural septum. Incision through the anterior crural septum



FIGURE 41-22. **A,** CT angiogram used to evaluate the lower extremity vasculature. **B,** Magnetic resonance angiography (MRA) used to evaluate the lower extremity vasculature.

provides entry into the anterior compartment, exposing the anterior tibial vessels and deep peroneal nerve beneath, which are preserved and gently retracted anteriorly. The interosseous membrane is exposed and incised to reveal the tibialis posterior muscle and its typical chevron-appearing fibers. Further dissection through this muscle will reveal the peroneal vessels lying beneath running close to the medial aspect of the fibula. Attention now focuses on proximal and distal exposure of the fibula with subperiosteal dissection 6 cm below the fibula head and 8 cm proximal to the lateral malleolus. With the aid of curved periosteal elevators protecting the peroneal vessels immediately deep to the fibula, appropriate osteotomies can be made with the oscillating saw removing a 1-cm segment of fibula. The pedicle can now be ligated and divided distally, and the fibula is now able to be rotated laterally enabling easier and safer dissection of the pedicle in a distal to proximal fashion. Upon reaching the tibioperoneal trunk, the peroneal artery and venae commitantes are isolated. The posterior incision of the skin island can now be performed down to the soleus muscle where it is optional to include a small cuff of soleus and flexor hallucis longus in cases in which musculocutaneous perforators are encountered. With the composite flap now isolated on its pedicle, the tourniquet is deflated and hemostasis of the donor bed is achieved (Figure 41-23).

In instances in which a bone-only flap is required, one need only make a linear skin incision without inclusion of a skin island. When dissection is directed toward the intermuscular septum, the remainder of the flap harvest technique proceeds as described previously (Figure 41-24).



FIGURE 41-23. Harvesting of an osteocutaneous fibula flap. The proximal cuts were already done and plated, re-creating the mandible to be reconstructed. Note the skin paddle and the dissected vascular pedicle.

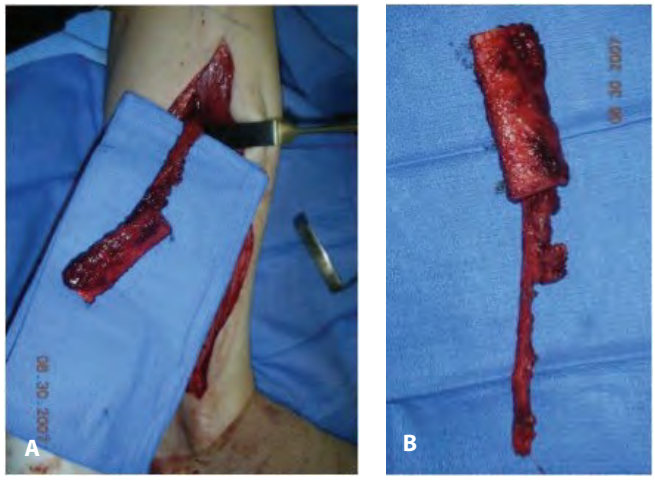


FIGURE 41-24. **A,** Harvesting of a bone-only fibula flap, flap in situ with dissected vascular pedicle. **B,** Harvested bone-only fibula flap. Note the length of the vascular pedicle.

Donor site closure is performed with loose approximation of muscles, with the flexor hallucis longus sutured to the tibialis posterior to optimize postoperative great toe flexion. A suction drain is placed and secured. A split-thickness skin graft harvested from the thigh is sutured to the skin paddle donor defect, followed by application of a bolster and posterior splint. While the patient is nonambulatory, leg elevation is advised to minimize dependent edema. The cast and bolster are removed after 7 days, at which time ambulation may commence.

Following division of the pedicle, the flap is transferred to the prepared recipient site. Preparation of the fibula at this stage is dependent upon the site of the mandibular defect. For reconstruction of straight segments, fibula preparation is minimal, often with no osteotomies required. For defects in which osteotomies are required to reproduce mandibular contour, one or more osteotomies may be necessary. The osteotomized fibula is then fixed to a preadapted reconstruction plate using fixation screws and subsequently fixed in situ to the native mandibular defect.

Acquired vascular insufficiency of the lower extremities is frequently associated with atherosclerosis particularly in elderly patients. Preoperative imaging with CT angiography, MRA, or routine angiography can avoid limb-threatening ischemia. Compartment syndrome from wound closure under excessive tension can produce disastrous ischemic complications.

Almost all patients develop limited flexion capability of the hallux consistent with inclusion of the flexor hallucis longus muscle with the flap.

The common peroneal nerve is the nerve at risk during fibula flap harvest. It has both sensory and motor functions, with sensory disturbance reported in up to 24% of cases in either the superficial or the deep peroneal nerve distribution.[50] Motor disturbances lead to weakness in dorsiflexion in about 7% of cases resulting from damage to peroneal nerve branches and associated equinovarus deformity.[51] Common

peroneal nerve damage is avoided by limiting proximal dissection to greater than 6 cm from the fibula head and avoidance of overzealous traction during proximal dissection.

Scapula Free Flap

The scapula free flap was first transferred by Gilbert in 1979.[52] This was after the anatomic description by Saijo.[53]

The scapula free flap has proved to be an extremely valuable reconstructive option in head and neck surgery. Its ability to carry multiple skin flaps, latissimus dorsi muscle, serratus anterior muscle, and scapula bone, all based on a single pedicle, makes this system of flaps uniquely suited for the complex three-dimensional sculpting necessary in the head and neck. A distinct advantage in the elderly population is the unimpeded early postoperative ambulation not shared by the fibula and iliac crest flaps.[54] The main disadvantage of this flap is the need to reposition the patient in order to harvest the flap, preventing a two-team approach.

The scapula is a triangular bone. A number of muscles attach and originate from the scapula. The triceps brachii muscle, teres minor, and teres major originate along the lateral aspect of the scapula from a superior to inferior direction. The medial aspect of the bone is the location of the insertion of the rhomboid major, rhomboid minor, and levator scapula in an inferosuperior direction. The infraspinatus occupies the regions inferior to the spine of the scapula and the supraspinatus is in the superior portion. The deltoid muscle originates along the inferior aspect of the spine of the scapula and the superior region is the location for the insertion of the trapezius muscle.

The blood supply to the region begins with the subscapular artery, a branch of the axillary artery. The circumflex scapular artery originates from the subscapular artery and divides into the superficial skin branches: a transverse and a descending branch and a deep periosteal branch. The deep periosteal branch travels along the lateral border of the scapula, giving perforators to the periosteum and bone.

Flap Harvesting

The harvesting of the scapula flap is done by placing the patient in a lateral decubitus position with the arm fully prepped.

In cases in which a scapular skin paddle is to be included in the harvest, the area is marked, taking advantage of the transverse cutaneous branch from the circumflex scapular artery, or a dual skin flap may be designed, taking advantage of the descending cutaneous branch in addition to the transverse.

The incision is begun medially and progresses laterally toward the lateral border of the scapula. The skin flap is raised in a suprafascial plane, taking care to identify and not injure the transverse branch. Once the lateral border of the scapula is approached, the dissection is directed in the triangular space to identify the deep branch of the circumflex scapular artery. The bone feeders are identified and preserved. The teres minor and major muscles are disinserted from the bone, taking care not to injure the periosteal feeders. The latissimus muscle is reflected from the inferior border of the scapula and the subscapularis muscle is freed. The area of bone to be harvested is marked out and the infraspinatus is reflected to expose the bone. A malleable retractor is placed under the scapula to protect the thoracic cavity and the bone cut is then made. Care should be taken superiorly to make sure that this cut does not encroach on the joint nor compromise the vascular pedicle to the bone or skin paddle. With the bone cut completed, the subscapularis muscle is then divided. The pedicle is then dissected toward the circumflex scapular artery and vein and the flap is mobilized and harvested (Figure 41-25).

The closure of the scapula demands special attention in order to prevent winging of the scapula. Several bone holes are placed along the lateral aspect of the scapula, and the teres muscles are then approximated to the scapula. The skin is mobilized and suction drains are placed followed by closure in a layered fashion.

Shoulder weakness results from division of the rotator cuff muscles teres major and minor, from the lateral border of the scapula during flap harvest. Restricted arm elevation, extension, and adduction are the most common impaired shoulder movements.[55] The most significant and feared complication associated with the scapula flap harvest is compromise of the joint space during the osteotomy. Another potential complication is the winging of the scapula, which can result from poor attention to closure with lack of approximation of the teres muscles to the bone using drilled holes or from injury to the long thoracic nerve. Intense postoperative physical therapy has also been shown to maximize shoulder mobility with return to premorbid function by 6 months after surgery.[56,57]

Iliac Crest Free Flap

The iliac crest free flap arguably provides the greatest quantity of bone stock for head and neck reconstruction. Its site distant from the ablative head and neck team permits synchronous harvest, and its inherent shape is well suited for reconstruction of the facial bones.[58] This flap is often avoided owing to an apparent technically demanding flap harvest, relatively short vascular pedicle, and small-caliber vessels. Others, however, have shown excellent outcomes with this flap.[59]

Taylor and colleagues[60] and Sanders and Mayou[61] in 1979 separately identified and reported the DCIA and DCIV as the most reliable and favorable pedicle for free transfer of the ilium. Taylor and associates[62] further elucidated the endosteal and periosteal blood supply of the ilium through dye injection studies. Experimental work by Ramasastry and coworkers in 1984[63] identified the ascending branch of the DCIA as the primary blood supply to the internal oblique muscle allowing for composite flap transfer.

The iliac crest possesses a natural curvature that makes it ideal for reconstruction of mandibular defects. The height of bone that can be harvested from this site allows for the restoration of the height of a native dentate mandible.

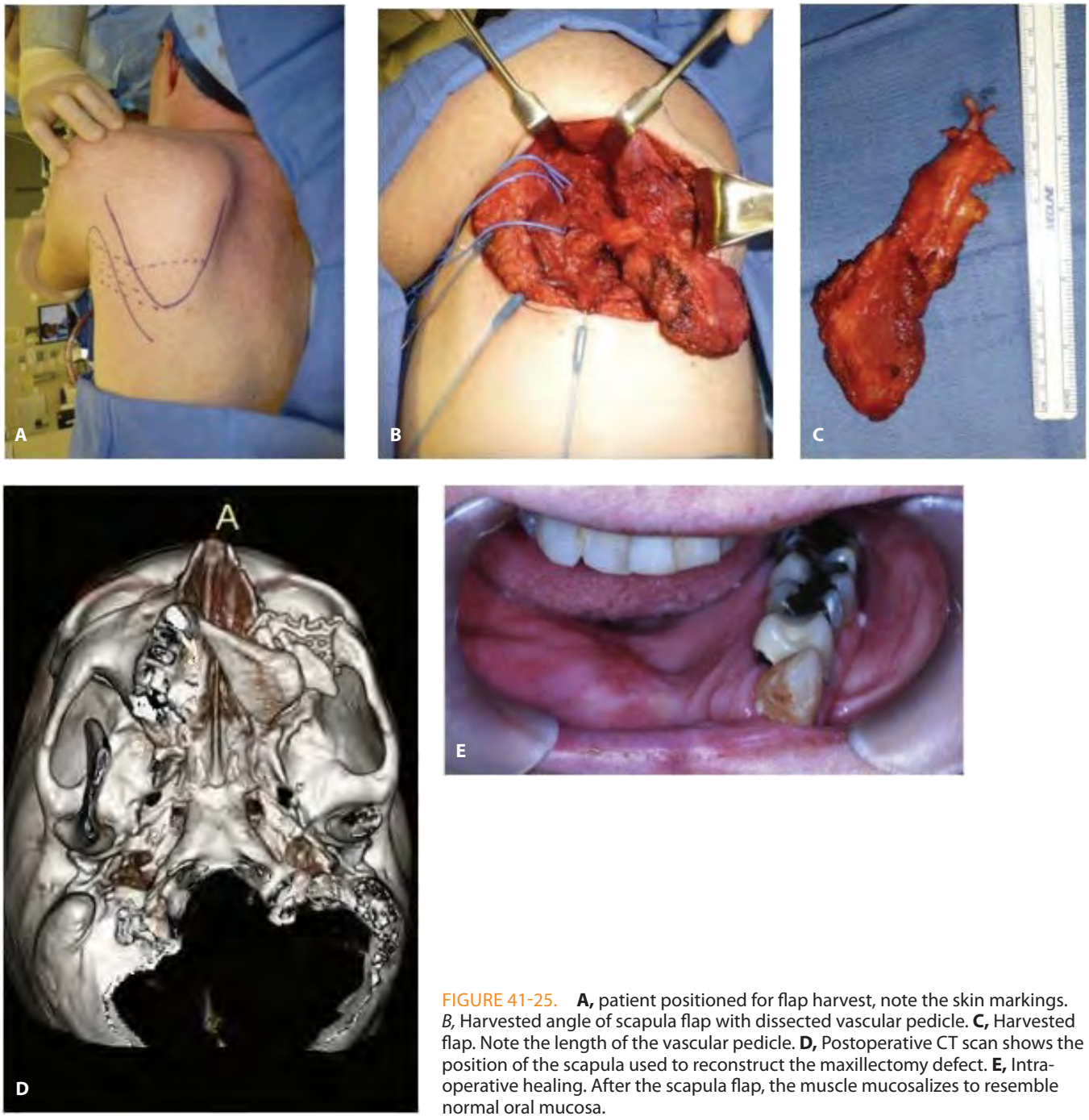


FIGURE 41-25. **A,** patient positioned for flap harvest, note the skin markings. *B,* Harvested angle of scapula flap with dissected vascular pedicle. **C,** Harvested flap. Note the length of the vascular pedicle. **D,** Postoperative CT scan shows the position of the scapula used to reconstruct the maxillectomy defect. **E,** Intraoperative healing. After the scapula flap, the muscle mucosalizes to resemble normal oral mucosa.

The anatomy of this region has been described previously in this chapter. Additional to the bony and muscular anatomy, the vascular pedicle to the flap is the deep circumflex iliac artery and vein. The lateral femoral cutaneous nerve crosses over the artery medially as it approaches its take-off from the external iliac vessels.

Flap Harvest

The patient is placed in a supine position with a bump under the hip to be harvested. The incision for the harvest of the DCIA flap is begun just lateral and above the palpable femoral pulse. The incision in then continued in a curved fashion, following the contour of the iliac bone in a postero-superior direction.

After the incision of the skin and subcutaneous fat, the external oblique muscle is incised allowing for the visualization of the internal oblique muscle. This muscle is readily identified owing to the direction of the muscle fibers, which run in an opposite direction from the external oblique muscle. The internal oblique is incised 2 cm cephalad and along the crest. Once the oblique muscles are transected, the iliacus

is visualized. The DCIA may be palpated at this time. Incision of the iliacus is done a safe distant inferior to the path of the DCIA. The muscles along the lateral/gluteal aspect are reflected. The planned amount of bone to be harvested is marked. The bone cuts are made while protecting the pedicle. The pedicle dissection is extended toward the external iliac vessels. Once adequate vessel length is dissected, the flap is harvested (Figure 41-26).

The bone harvest can be achieved in two forms, full thickness or split thickness. The advantage of the latter is decreased donor site deformity (Figure 41-27).

Ventral hernias result from weakening of the abdominal wall through harvest of the internal oblique muscle and denervation of the rectus muscle whose motor nerves run in the neurovascular plane between the internal oblique and the transversus abdominis.[64] Mesh repair to reinforce the abdominal wall can eliminate this phenomenon.

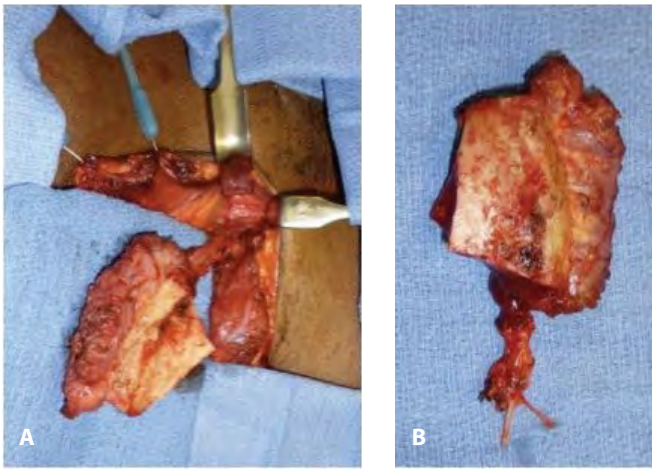


FIGURE 41-26. **A,** Harvesting of a deep circumflex femoral artery (DCIA) flap in situ with vascular pedicle still connected. **B,** Harvested DCIA; note the short pedicle length.

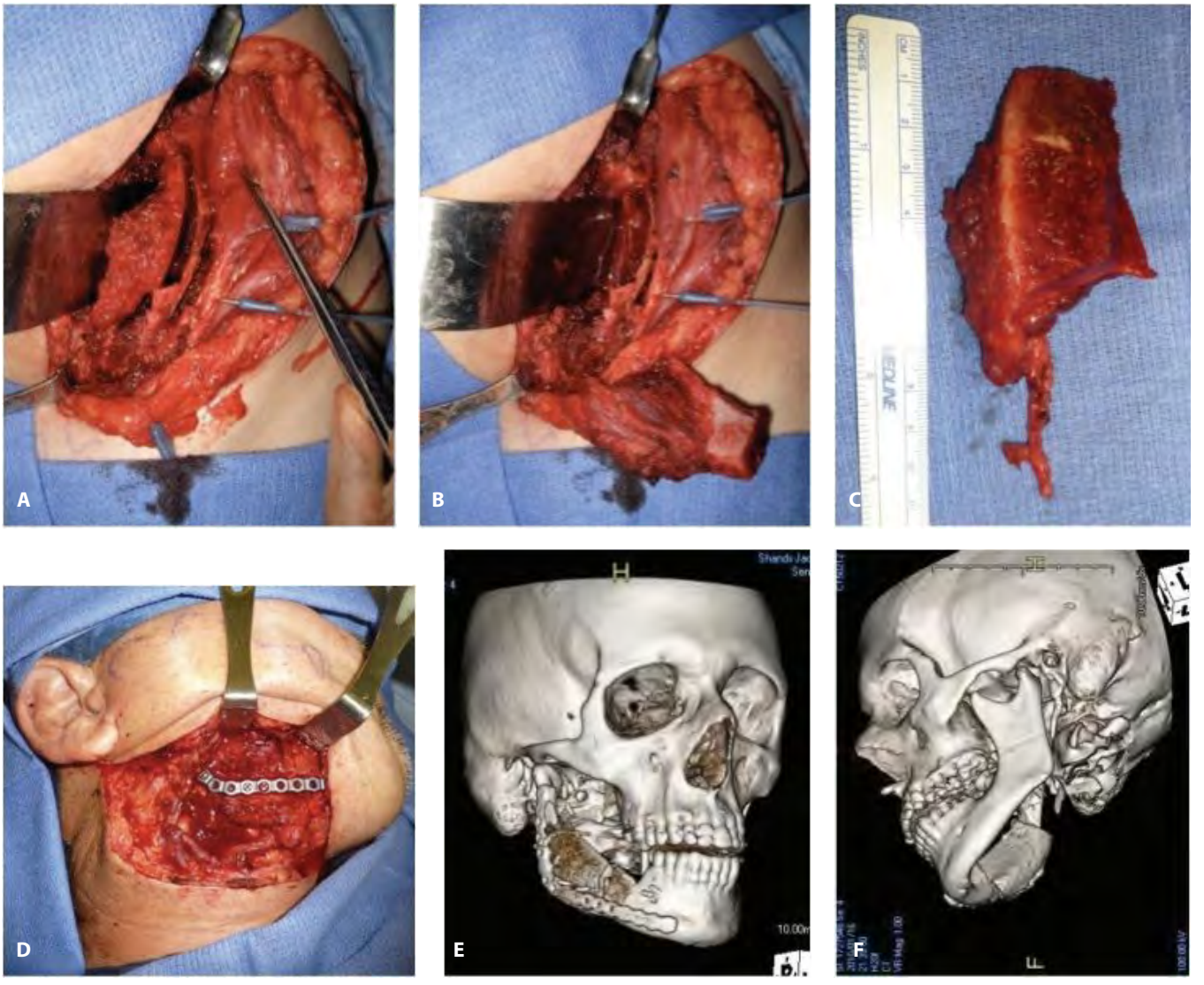


FIGURE 41-27. **A,** Markings for a harvesting of a split DCIA flap. **B,** Donor site defect after harvesting of a split DCIA flap. Note the preservation of the contour of the iliac crest. **C,** Harvested flap. **D,** Inset of the split DCIA flap. **E,** CT scan of the flap in place. **F,** CT scan depicting the lingual view of the reconstructed mandible. Note the amount of bone in terms of not only height but also width.

Immediate postoperative and short-term effects on ambulation are common following iliac crest free flaps, with antalgic gait and weakness of the operated hip being usual sequelae. Reports in the literature have demonstrated through controlled orthopedic objective testing that ambulation resolves by 6 months postoperatively.[65,66]

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42

CHAPTER

Microneurosurgery

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Injuries to the terminal branches of the trigeminal nerve may occur commonly after routine oral and maxillofacial surgical procedures, and the overwhelming majority of these injuries undergo spontaneous neurosensory recovery without intervention. Third molar surgery accounts for most of the injuries that occur to both the inferior alveolar nerve (IAN) and the lingual nerve (LN). The reported incidence of iatrogenic nerve injury varies in the literature, but both temporary and permanent rates of paresthesia must be considered. Potential for nerve injury involves mandibular and maxillary orthognathic surgery, maxillofacial trauma, dental implant placement, endodontic therapy, facial fractures, and treatment of benign and malignant head and neck pathologic lesions. The anatomy of the trigeminal nerve system is unique because it carries, in some branches, both general sensory information and special (e.g., taste) sensation. Injury to a nerve may result in neuroma formation, which can manifest with a wide spectrum of objective clinical signs and subjective patient symptoms. Nerve injuries are classified by two different classification schemes (Seddon and Sunderland, described later), which are based on the degree of axonal injury at the histologic light microscopic level but, more important, upon the likelihood that an injured nerve will recover spontaneously after injury. A basic understanding of nerve terminology (see Appendix) and normal neural wound healing anatomy and physiology is essential to most appropriately managed clinical situations.

The initial clinical evaluation of patients with nerve injuries should proceed in an structured and orderly fashion, with the performance of several levels of objective testing to most accurately determine the degree of individual nerve injury. A standardized clinical neurosensory test (CNT) may be used for most patients; however, some advanced testing is also available for special circumstances (e.g., gustatory testing). It should be remembered that there are many nonsurgical, including pharmacologic, treatments available for the patient who sustains a nerve injury, and for the majority of patients with dysesthesia or painful neuropathies, drug therapy is considered the mainstay of treatment and may involve other medical professionals, including a neurologist.

Once the decision is made that the patient is a surgical candidate to potentially improve sensation with microneurosurgery, an orderly sequence of surgical steps must be followed meticulously. Specific surgical techniques depend on which nerve is involved as well as the estimated extent of the injury. In general, microneurosurgical repair of a trigeminal nerve injury involves neurolysis (surgical manipulation of the nerve) and preparation of the nerve stumps to perform neurorrhaphy (nerve repair). The deleterious effects of tension on a nerve repair site have been well documented, so the inability to perform a primary, tension-free, repair warrants consideration for an autogenous nerve graft or another option for nerve gap management, such as entubulation (gap or conduit repair). Following microneurosurgery, postoperative sensory reeducation may play an important role in the neural regenerative process. The overall success rates of microneurosurgical repair of the trigeminal nerve vary considerably based upon individual surgeon reports and a lack of consensus regarding "success criteria." However, an important factor that affects the success of microneurosurgery is the length of time from injury to repair, because this affects the degree of trigeminal ganglion cell death, wallerian degeneration of the distal axon, and cortical somatosensory reorganization of the information from the target site (lower lip and chin or tongue). The American Association of Oral and Maxillofacial Surgeons Clinical Interest Group on Maxillofacial Neurologic Disorders has promulgated treatment time recommendations for the patient who sustains a trigeminal nerve injury, and these are based upon allowing sufficient time for spontaneous neurosensory recovery, while considering the impact of delayed treatment on final functional neurosensory outcome.[1]

The field of microneurosurgery is in its infancy considering the current rapid advances in technology and growth factor research, and as more surgeons become adept at the diagnosis and treatment of patients who sustain trigeminal nerve injuries, more clinical, laboratory, and radiologic information will become available to guide the therapeutic decision-making process. Also, postgraduate training programs in oral and maxillofacial surgery will become more capable of training surgical residents in the principles and practice of microneurosurgery, and this will hopefully foster access to this aspect of specialty care throughout the country and abroad.

DEMOGRAPHICS

Trigeminal nerve injuries result from a variety of routine oral and maxillofacial surgical procedures, such as third molar odontectomy, management of facial trauma, orthognathic surgery, endosseous dental implant placement, salivary duct and gland surgery, treatment of benign and malignant lesions of the head and neck, preprosthetic surgery, and endodontic and periradicular surgery. Complications of third molar removal are responsible for the majority of injuries to the terminal branches of the third division of the trigeminal nerve.[2] These can occur during any phase of third molar surgery, including local anesthetic injection, incision and flap design, the use of a high-speed drill for bone removal or tooth sectioning, elevation of the tooth with trauma to the lingual soft tissues, socket curettage with exposed neurovascular tissue, removal of remnants of an assumed "dental follicle" that may contain neural or vascular tissue, the use of medicaments in the extraction site to aid healing or prevent alveolar osteitis (e.g., tetracycline-containing compounds[3,4]), and the placement of sutures. The efficacy of LN retraction during lower third molar surgery has shown that, although the incidence of temporary LN paresthesia is increased owing to a slight stretching or manipulation (6.4% with retraction vs. 0.6% without retraction), the difference in long-term dysfunction is not significant (0.6% with retraction vs. 0.2% without retraction).[5] Other studies have indicated a temporary paresthesia rate of approximately 10% to 15% with LN retraction and protection, with a permanent paresthesia rate of less than 1%.

The incidence of trigeminal nerve injury may be estimated based on a review of the available literature. Overall, the incidence of IAN injury from third molar surgery is 0.41% to 7.5% and from sagittal split osteotomy is 0.025% to 84.6%, whereas the LN is affected 0.06% to 11.5% of the time after third molar removal. However, the more important clinical distinction is to differentiate temporary from permanent paresthesia rates. For sagittal split osteotomies, temporary IAN paresthesia may be as high as 80% to 100%, but permanent rates, which are age-dependent, are typically less than 1% to 5%. For third molar surgery, both IAN and LN temporary paresthesias range from 2% to 6%, whereas permanent rates are approximately one fourth of the temporary rates, or 0.5% to 2% overall. Many risk factors for nerve injury during third molar surgery have been reported in addition to the vulnerable position of the IAN and LN and include advanced patient age, female gender, depth of impaction, mesiodistal angulation of the tooth (distoangular), lingual angulation of the tooth, lack of integrity of the lingual cortex, the need for tooth sectioning, removal of bone distal to the third molar, longstanding pericoronitis that may result in scarring of the soft tissue and LN compression postoperatively, and surgeon experience. It has been postulated that a right-handed surgeon standing on the right side of the patient during third molar removal would experience more injuries to the LN on the right side (associated with the mandibular right third molar) because visualization of the LN is partially obstructed, and more IAN injuries on the left side (associated with the mandibular left third molar) owing to visual obstruction of the buccal aspect of the left posterior mandible. Certainly, the risk of an IAN injury may be influenced by so-called Rood and Shehab[6] radiographic predictors of potential tooth proximity to the inferior alveolar canal, although these were described earlier by Howe and Poynton in 1960.[7] These seven radiographic predictors on panoramic radiograph may indicate the potential for increased risk of injury to the IAN, and they are listed in Table 42-1. Of the seven radiographic predictors, Rood and Shehab[6] noted that three of these were more significant: root darkening, root deflection, and interruption of the white line of the canal. It has been suggested that the lack of any radiographic finding would correlate with a minimal risk of IAN injury (<1%), whereas a finding of one or more predcitors would increase the risk of IAN injury up to 10%. With the current increased use of cone beam computed tomography (CT) scanning, the assessment of the proximity of the third molar to the IAN has improved significantly. In cases with a high index of suspicion of nerve injury (e.g., deep impaction, advanced age), intentional coronectomy with close observation should be considered.[8] As opposed to the relatively consistent course of the IAN, the LN position is variable; and it is injured less often than the IAN after third molar surgery.[9–13] The position of the LN has been documented clinically,[13] in cadaveric dissections,[15,16] and radiologically.[17] On average, in the third molar region, the LN lies 2.5 mm medial to the lingual plate of the mandible and 2.5 mm inferior to the lingual crest. The LN may be in direct contact with the lingual plate in 25% of

TABLE 42-1. Rood's Radiographic Predictors of Potential Tooth Proximity to the Inferior Alveolar Canal

1. Darkening of the root
2. Deflection of the root
3. Narrowing of the root
4. Dark and bifid root apex
5. Interruption of the white line of the canal
6. Diversion of the canal
7. Narrowing of the canal

Adapted from Rood JP, Shehab AAN. The radiological prediction of inferior alveolar nerve injury during third molar surgery. Br J Oral Maxillofac Surg 1990;28:20.

cases (Kisselbach and Chamberlain reported 62%[14]) and may lie above the lingual crest in 10% to 15% of cases (Kisselbach and Chamberlain reported 17.6%[14]) based on an undisturbed radiographic assessment of the nerve.

Mandibular blocks may result in IAN and LN injuries; however, the incidence is unknown owing to a significant number of unreported cases. An estimated 1 in 100,000 to 1 in 500,000 blocks result in paresthesia. Perhaps the largest study of its kind, Harn and Durham's study of 9587 mandibular blocks[18] showed a 3.62% incidence of temporary paresthesia and a 1.8% incidence of long-term paresthesia lasting longer than 1 year. Several theories have been proposed to explain the mechanism of injury. Direct neural trauma is unlikely owing to abundant interfascicular neural components resulting in separation of the fascicles by a needle or suture without direct neural disruption.[19] The resultant edema may be responsible for the transient paresthesia that resolves spontaneously. Local anesthetic toxicity may be responsible for prolonged paresthesia after a mandibular block, especially if the solution is deposited within the confines of the epineurium. Several reports indicate that prilocaine and articaine may be associated with an increased risk of long-term paresthesia, especially with mandibular block anesthesia, compared with other local anesthetic solutions owing to a concentration gradient effect, but further investigation is warranted.[20–22] The third potential mechanism of injury involves the formation of an epineurial hematoma. The epineurium and perineurium contain a vast plexus of vessels that nurture the neural elements, and a needle may cause disruption of one or more vessels. The localized bleeding most certainly tamponades itself owing to the surrounding epineurium, and the pressure may impinge on select groups of fascicles contained within the nerve. The resultant clinical signs and symptoms of localized paresthesia, not involving the entire distribution of the IAN/mental nerve, nicely match the expected histologic situation, making this theory plausible. Also, lymphatic drainage of the localized hematoma over the few days to weeks after surgery coincides with the clinical resolution of symptoms in most cases. The final theory is that of the needle-barb mechanism of injury.[23] During a mandibular block injection, the needle may be advanced to the medial ramus where a small barb may form at the needle tip. On withdrawal, if the needle has passed through or in the vicinity of the LN or IAN, fascicular disruption may occur with potentially longstanding clinical consequences. Recent trends in our clinical understanding of injection-related nerve injuries include the following:

1. Injection nerve injuries are difficult to predict and prevent.
2. The classic electric shock sensation upon injection is reported uncommonly by patients who sustain these injuries.
3. Injection injuries are more likely to result in dysesthesia than are other causes of nerve injuries.
4. There may be a nonanatomic distribution of nerve involvement (including the second and third divisions of the trigeminal nerve) due to progressive demyelination along the nerve.
5. Persistent symptoms after injection injuries occur more commonly in female patients.
6. The LN, which is stretched more upon mouth opening than is the IAN, is more commonly affected.
7. The majority of cases resolve within 8 weeks, and if paresthesia persists for longer than 8 weeks, then only one third of those injuries resolve spontaneously.

Microneurosurgery is a poor option for patients with injection-related nerve injuries not only because most of these patients have a component of dyesthesia but also because surgical access is extremely difficult; therefore, most cases are managed with pharmacologic therapy. One of the clinical dilemmas is to differentiate a mandibular block injury to the IAN from a third molar injury to the IAN. On rare occasions, the third molar site of the IAN has been explored after third molar removal and resultant lower lip and chin paresthesia and the IAN is found to be normal at the third molar site, with the assumption at that point that the injury occurred as a result of injection rather than extraction.[24] However, again, it is difficult to estimate the incidence of mandibular block injuries to the IAN and LN or to precisely define the etiology.

It is well known that orthognathic surgery may result in nerve injury. The IAN is affected more often than is the LN, and rarely, the facial nerve may be affected (0.67% with sagittal split osteotomy in one study[25]). Certainly, a great deal is known about the risks of IAN injury associated with sagittal split osteotomy as well as screw overpenetration injury or manipulation injury to the LN.[26,27] Unfortunately, the reported incidence of immediate and long-term neurosensory deficit varies considerably (from < 5% to > 90%) owing to poorly controlled factors inherent in the study designs or case reports, such as individual surgeon experience, technical variability in the surgery, lack of standardization of neurosensory testing, lack of control sites for normal cutaneous facial sensibility (in bilateral cases), and variation in the time periods when neurosensory testing is performed. Several studies have examined the specific parameters for neurosensory recovery after bilateral sagittal split osteotomy by using objective and subjective assessment criteria.[28] One study found a 39% incidence of neurosensory dysfunction after sagittal ramus surgery,[29] and others have shown less than 15% dysfunction at 6 months.[30] Although the incidence of nerve dysfunction varies, there are well-known risk factors for nerve injury,[31] including patient age[32]; increased length of the surgical procedure; proximal or distal segment fractures ("bad splits"); concomitant third molar removal; concomitant genioplasty procedures; compression during fixation (e.g., lag screw technique); inadvertent use of chisels in the osteotomy site; nerve entrapment in the proximal segment; nerve manipulation in the area of the osteotomy as well as the region of the lingula during the medial dissection (based on intraoperative recordings of IAN somatosensory evoked potentials)[33]; the location of the inferior alveolar canal close to the inferior border; low mandibular body corpus height

and retrognathism in class II deformities (the IAN tends to be closer to buccal cortex and more vulnerable to injury)[34]; and frank nerve transection during surgery, especially with a large mandibular advancement. Unfortunately, long-term neurosensory dysfunction after orthognathic surgery is not generally amenable to microneurosurgical correction. However, most patients tolerate the paresthesia well after correction of a significant dentofacial deformity. Three caveats to remember regarding orthognathic surgery-related nerve injuries: (1) that the older the patient the more severe the potential paresthesia; (2) that patients tolerate "mild" paresthesia after "major" surgery well (with appropriate informed consent); and (3) that the magnitude of neurosensory dysfunction decreases as the time from injury increases.

Craniomaxillofacial trauma may result in injury to any of the terminal branches of the trigeminal nerve. Orbitozygomatic and zygomaticomaxillary complex fractures may involve the infraorbital canal and the second division of the trigeminal nerve. The small fibers of the infraorbital nerve as it exits the infraorbital foramen are less than ideal for surgical manipulation and repair. Mandible fractures that violate the IAN canal result in temporary or permanent paresthesia, depending upon the degree of fracture displacement. Treatment of mandible fractures with inadvertent placement of screws that traverse the inferior alveolar canal may cause iatrogenic transien or permanent paresthesia. In general, reduction and stabilization of the fracture segments aid in realigning the natural bony conduit (i.e., the IAN canal or the infraorbital canal) that will help to guide spontaneous neurosensory recovery, even in cases of partial or complete transection injuries.

Also, the presence and/or biopsy and treatment of head and neck pathologic lesions may result in nerve injury. The use of Carnoy's solution (ferric chloride 0.1 g/mL, absolute alcohol 6 mL, chloroform 3 mL, glacial acetic acid 1 mL) after treatment of pathology has been shown to have a critical exposure time in an animal model of 5 minutes, after which time there may be long-term irreversible neural injury.[35,36] After a resection procedure, consideration should be given to immediate or delayed neural reconstruction using autogenous or allogeneic nerve grafts or conduits.

Although preprosthetic surgery is performed less frequently today than in the past, procedures such as torus mandibularis reduction and maxillary or mandibular vestibuloplasty place the terminal branches of the mental nerve and infraorbital nerve at risk for injury. As mentioned, surgical repair of the small terminal nerve fibers of the infraorbital and mental nerves is difficult and often results in scarring of the nerve and adjacent soft tissues and a poor chance of neurosensory recovery. The maxilla and mandible are excellent sources of autogenous bone grafts; however, they are not performed without potential morbidity. The majority of patients who undergo genial bone graft harvest complain of desensitization of the mandibular anterior teeth, when present. Depending on the specific technique employed for posterior mandibular ramus bone graft harvesting, the IAN may be at risk of iatrogenic injury; the anatomic position of the IAN laterally within the ascending ramus contributes to its vulnerability. Mandibular endodontic therapy and periapical surgery may result in an injury to the IAN, depending on the proximity of the root apex to the canal. Some endodontic filling materials may be neurotoxic (paraformaldehyde-containing pastes [Sargenti paste, N2, AH26], or eugenol-containing cements [ZOE, PCS]); to prevent irreversible paresthesia that in many cases results in dysesthesia, consideration should be given to prompt exploration (12–24 hr) and débridement of medicaments that have permeated through the root apex and are in direct contact with the nerve. Distraction osteogenesis of the mandible has been shown to induce transient changes in neuronal conduction without significant long-term nerve dysfunction.[37,38] Because age is a factor in spontaneous neurosensory recovery after injury, a younger patient would certainly tolerate a "stretch-type" of injury to the nerve better than an adult. Some data indicate that with a true corticotomy (vs. osteotomy) and distraction rates of 1 mm/day, permanent neural changes are unlikely, but that distraction rates greater than this may be deleterious to nerve function. However, more studies are necessary.[39]

Finally, implant-related injuries to the IAN are common (30–40%) and difficult to manage appropriately; however, with current technqiues of advanced imaging and computer treatment planning with surgical implant guides, the incidence of iatrogenic nerve injury may be declining. Unfortunately, there is a lack of data regarding appropriate patient assessment and management, with a lack of consensus on treatment protocols. In the posterior mandible, the likely cause of nerve damage may be that the initial pilot (depth) drill penetrates the superior cortex of the canal and violates the IAN vein (or artery, which is less likely). This results in some bleeding that, on placement of the implant, will tamponade itself. However, the resultant increased pressure in the closed environment of the inferior alveolar canal creates a "compartment syndrome," with harmful effects on neurosensory function. This type of compression injury commonly results in long-term unpleasant altered sensation (dysesthesia) rather than simple decreased sensation (hypoesthesia). The recognition postoperatively that the patient has paresthesia and that the implant is within the confines of the canal warrants the clinician to consider removal of the implant, with or without immediate replacement with a shorter implant. If, however, the injury was due to a compartment syndrome effect, implant removal without replacement may be the more prudent option. For patients with persistent paresthesia, referral to a microneurosurgeon or neurologist may be warranted for consideration for pharmacotherapy. The procedure of IAN repositioning (lateralization and transpositioning) is an option that theoretically would induce a "controlled" injury to the nerve and protect it during implant preparation. With lateral decortication of the mandible and nerve exposure, a compartment syndrome would not be possible. Despite the potential advantages of nerve repositioning, there is a high incidence of long-term paresthesia

ranging from 0% to 77%, with a mean of approximately 30% to 40%.[39] With appropriate surgeon experience, proper patient selection, the presence of a strong indication for the procedure (e.g., lack of interocclusal clearance in the posterior mandible), and an adequate and informed consent discussion, this procedure remains an option for posterior mandibular implant reconstruction.

TRIGEMINAL NERVE ANATOMY AND PHYSIOLOGY

A review of trigeminal nerve anatomy and physiology is necessary in order to understand clinical patient presentation and diagnosis and management. The trigeminal nerve (Figure 42-1) is composed of a mesoneurium that suspends the nerve within the surrounding tissues and is continuous with the outer epineurium that defines and surrounds the nerve trunk. This epineurium contains a vast plexus of blood vessels called the *vasa nervorum* as well as lymphatic channels. The epineurium is divided into both outer and inner components; the inner layer is composed of a loose connective tissue sheath with longitudinal collagen bundles that protect against compressive and stretching forces imposed on the nerve. Individual fascicles are defined by the perineurium, which is a continuation of the pia-arachnoid layer of the central nervous system. This perineurium functions to provide structural support and act as a diffusion barrier, similar to the blood-brain barrier that prevents the transport of certain molecules. The individual nerve fibers and Schwann cells are surrounded by the endoneurium, which is composed of collagen, fibroblasts, and capillaries. In the peripheral nervous system, there are three types of neural fascicular patterns: monofascicular (one large fascicle), oligofascicular (2–10 fascicles), and polyfascicular (>10 fascicles) (Figure 42-2). The IAN and LN are, for the most part, polyfascicular in nature. Polyfascicular nerves have abundant interfascicular connective tissue; the importance of this fact is twofold: first, needle penetrations rarely cause direct neural trauma to the fascicles themselves because the needle would pass preferentially through the interfascicular spaces, and second,

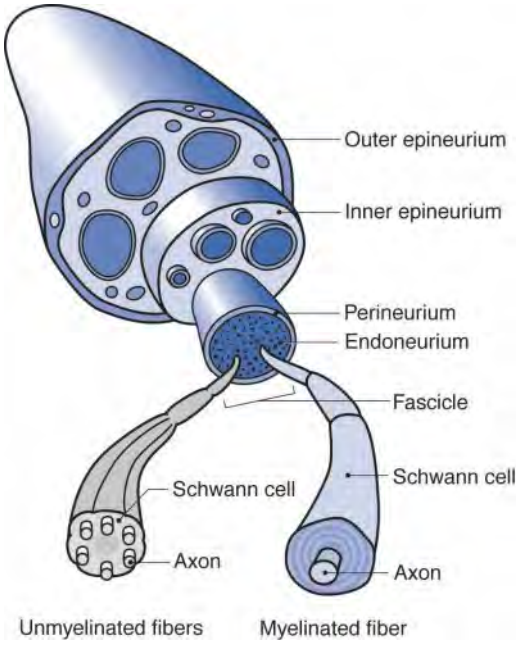


FIGURE 42-1. Trigeminal nerve anatomy.

nerve repair with realignment of the fascicles (coaptation) is challenging to perform on polyfascicular nerves.

The trigeminal nerve is composed of a functional unit with differing fiber types that transmit a variety of information (Table 42-2). The A-alpha fibers are the largest myelinated fibers with the fastest conduction velocity; they mediate position and fine touch through muscle spindle afferents and skeletal muscle efferents. The A-beta fibers mediate proprioception sense. The smallest myelinated fibers are the A-delta fibers that carry pain ("first" or "fast" pain) and temperature information. The smaller-diameter and slower-conducting unmyelinated C fibers mediate "second" or "slow" pain and temperature sensations. The Schwann cells surround both myelinated (one Schwann cell per nerve fiber) and unmyelinated (one Schwann cell per several nerve fibers) nerves, and they play a major role in nerve survival and regeneration after injury. Although the myelin sheath may not survive a nerve injury, the Schwann cells do, and

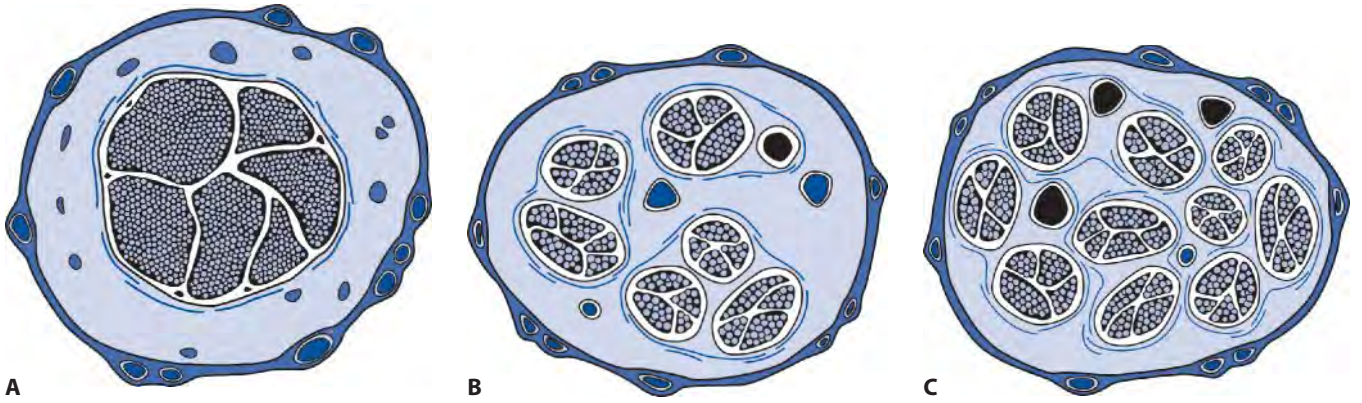


FIGURE 42-2. Three types of neural fascicular patterns. **A,** Monofascicular. **B,** Oligofascicular. **C,** Polyfascicular. (**A–C,** Adapted from Lundborg G. The nerve trunk. In Lundborg G, editor. Nerve Injury and Repair. New York: Churchill Livingstone; 1998; p. 198.)

TABLE 42-2. **Trigeminal Nerve Fibers**

Fiber	Size (μ)	Conduction Velocity (m/sec)	Function
A alpha (myelin)	12–20	70–120	Position, fine touch
A beta (myelin)	6.0–12	35–170	Proprioception
A delta (thin myelin)	1.0–6.0	2.5–3.5	Superficial (first) pain, temperature
C (unmyelinated)	0.5–1.0	0.7-1.5	Deep (second) pain, temperature

they provide a supportive role in the production of neurotrophic and neurotropic factors (such as nerve growth factor) that enhance and support neural recovery. In a myelinated axon, the nodes of Ranvier are the 0.3- to 2.0-μm unmyelinated segments between the myelin sheaths that are responsible for the diffusion of certain ions that cause nerve depolarization and repolarization as well as the rapid saltatory conduction of a nerve impulse along the nerve fiber.

Following nerve injury, many neural changes occur, but the basic process of nerve healing involves both degeneration and regeneration (Figure 42-3).[41,42] The nerve cell body responds with an increased metabolic phase with a heightened production of RNA (protein synthesis) and breakdown of Nissl's substance for export from the cell body. At the site

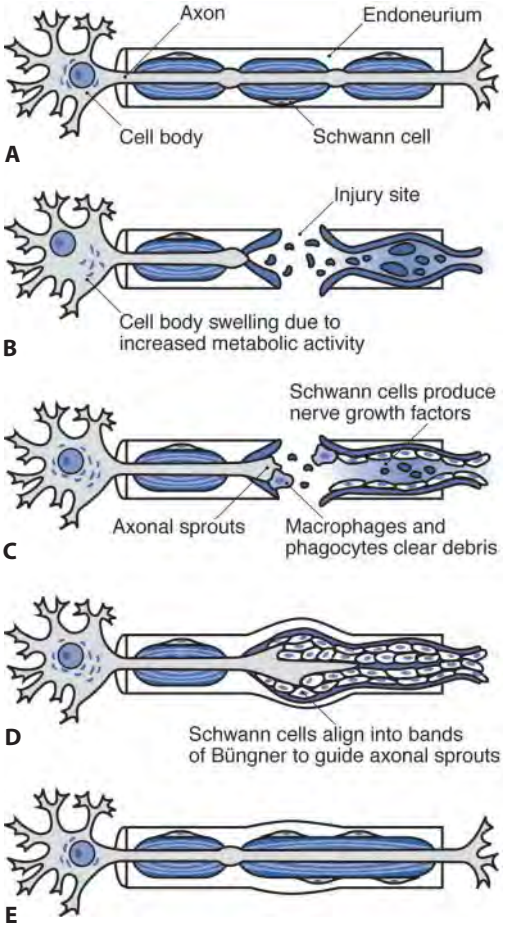


FIGURE 42-3. **A–E,** Neural wound healing mechanisms.

of injury, there is edema and accumulation of particulate cellular debris. In addition, there is a proliferation of phagocytes and macrophages that begin to clean the area of this debris. Within days, axonal sprouts begin to bud and extend from the proximal nerve stump in the area of injury. Each axon may have as many as 50 collateral sprouts from the proximal nerve stump with random orientation toward the distal nerve stump. There is proliferation and a high level of activity of Schwann cells as well. These Schwann cells begin to form new myelin "conduits" in anticipation of the arrival of the new axonal sprouts. In addition, nerve growth factors are produced that influence the direction of sprouting and guide the new axons into the newly formed myelin sheath conduits, known as the *bands of Büngner.* In the event that all of these interrelated processes occur appropriately, spontaneous neural regeneration will occur. If one or more of the reparative processes fail to occur at the appropriate time and location, there may formation of a neuroma and lack of spontaneous neurosensory recovery. This failure may be due to a variety of reasons including advanced local tissue scarring, insufficient neurotrophic and neurotropic factor production, or malaligned nerve stumps separated beyond the critical size defect to allow sponatenous reconnection. A neuroma is simply a disorganized mass of collagen fibers and randomly oriented small nerve fascicles (sprouts that could not find the distal target). Neuromas are classified by gross morphology into the following types (Figure 42-4): amputation (stump) neuroma, neuroma-in-continuity (central or fusiform neuroma), and lateral neuromas that are either lateral exophytic neuromas or lateral adhesive neuromas. From a surgical perspective, the neuroma must be excised completely both proximally and distally to allow the neurorrhaphy a chance for success rather than suturing a neuromatous proximal segment to a neuromatous distal stump with no chance of improvement in functional neurosensory recovery.

NERVE INJURY CLASSIFICATION

Two classification schemes are used to describe the anatomic and histologic changes that occur after nerve injury. Seddon described a three-stage classification system in 1943,[43] and Sunderland revised and further subclassified nerve injuries into five grades in 1951 (Figure 42-5 and Table 42-3).[44] A neurapraxia (Seddon) or first-degree (Sunderland) injury is characterized as a conduction block from transient anoxia due to acute epineurial/endoneurial vascular interruption resulting from mild nerve manipulation (traction or compression), with rapid and complete recovery of sensation and no axonal degeneration. Damage is confined to within the endoneurium. Sunderland[44] further subdivides first-degree injuries into types I, II, and III. Type I results from mild nerve manipulation with rapid (hours) return of sensation when neural blood flow is restored. Type II is due to moderate traction or compression with the formation of transudate or exudate fluid and intrafascicular edema, with return of sensation after resolution of edema (days). Type III injuries result

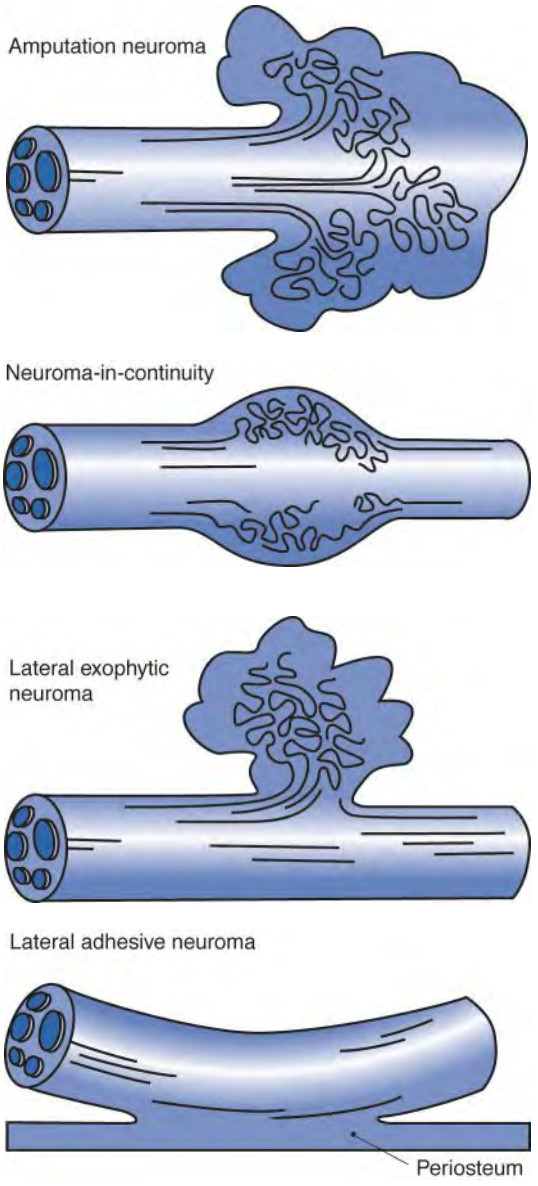


FIGURE 42-4. Neuroma types: amputation neuroma, neuroma-in-continuity, lateral exophytic neuroma, lateral adhesive neuroma.

from more severe nerve manipulation that may cause segmental demyelination, with recovery within days to weeks. An axonotmesis (Seddon) corresponds to second-, third-, and fourth-degree (Sunderland) injuries, with the difference being the degree of axonal damage. Second-degree injuries are due again to traction or compression that results in ischemia, intrafascicular edema, or demyelination. This damage extends through and includes the endoneurium with no significant axonal disorganization. Recovery is slow and may take weeks to months, and it may not be complete. Third-degree injuries continue the spectrum of more advanced neural injury due to more significant neural trauma with variable degrees of intrafascicular architectural disruption and damage extending to the perineurium. Recovery is variable; it may take months and be incomplete. Fourth-degree injuries result in damage to the fascicle that extends through the perineurium to the epineurium, but the epineurium remains intact as a near-complete transection injury. There is axonal, endoneurial, and perineurial damage with disorganization of the fascicles. Spontaneous recovery is unlikely, but minimal improvement may occur in 6 to 12 months. Finally, neurotmesis (Seddon) and fifth-degree (Sunderland) injuries result from complete transection of the nerve (traverses the entire width of the fascicle) with epineurial discontinuity and likely subsequent neuroma formation during attempted spontaneous regeneration of the nerve, making spontaneous neurosensory recovery unlikely. For completeness, in 1988, Dellon and Mackinnon[45] described a sixth-degree injury, which recognizes that many nerve injuries exhibit features of different degrees of injury according to Sunderland (Table 42-4). The Seddon and Sunderland classification schemes attempt to correlate histologic changes of nerve injury with expected clinical outcomes (see Table 42-3).

CLINICAL NEUROSENSORY TESTING

The patient who sustains an injury to the trigeminal nerve may present with a variety of signs and symptoms. These may be divided into nonpainful anesthesia, hypoesthesia, hyperesthesia or painful anesthesia (anesthesia dolorosa), hypoesthesia, or hyperesthesia (allodynia, pain from a nonpainful stimulus, or hyperpathia, increased pain due to a painful or nonpainful stimulus). The history usually indicates the etiologic event, and the chief complaint may include the following: “numbness,“ “itchy,” “crawling,” “stretched,” “drooling,” “painful,” “tingling,” “tickling,” “pulling,” “burning,” “stinging,” “pins and needles,” “hot sensation,” “cold sensation,” inability to feel food on lip, inability to taste, inability to shave, inability to smile, and loss of consortium. The history of present illness should be explored in depth with a comprehensive description of the onset and progression of symptoms, change in symptoms, treatment received and response to that treatment, any aggravating and alleviating factors, and current symptoms.

The McGill Pain Questionnaire (MPQ) may be used to assess pain and altered sensation, and it is a useful tool for monitoring progression of neurosensory recovery. The MPQ uses three classes to assess the level of dysfunction and interference with activity: sensory class (temporal, spatial, thermal, punctate, incisive, constrictive, traction pressure), affective class (tension, fear, autonomic properties, punishment), and evaluative class (patient perception). Perhaps the simplest and most reliable measure of subjective patient assessment is the use of a visual analogue scale. Typically, this is a 10-cm five-degree scale, with a degree marked every 2.5 cm (Figure 42-6). This is a useful tool for monitoring subjective improvement. It must be remembered that subjective and objective nerve test results are rarely at the same level. For example, in one study of nerve testing after sagittal split osteotomy, the subjective neurosensory deficit was 26.0%, whereas objective testing revealed an 89.5% deficit.[46]

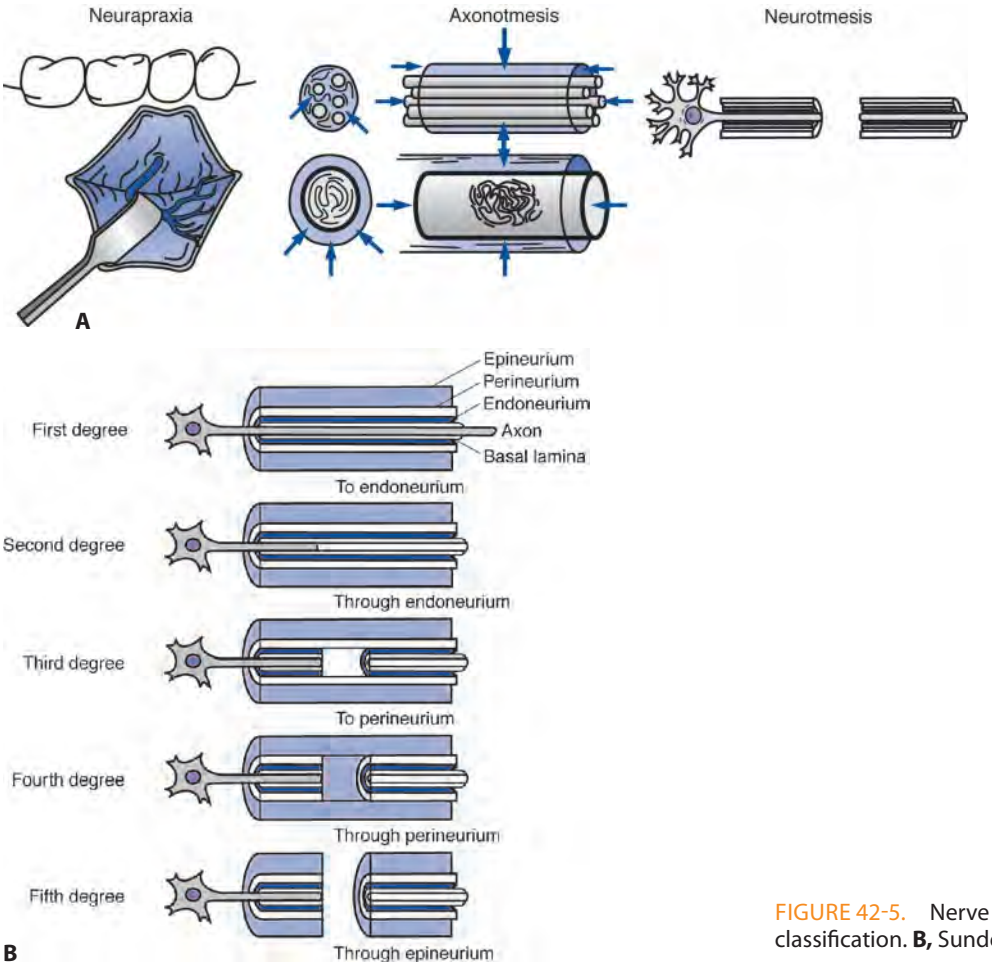


FIGURE 42-5. Nerve injury classifications. **A,** Seddon classification. **B,** Sunderland classification.

TABLE 42-3. **Nerve Injury Classifications: Seddon versus Sunderland**

Seddon	Sunderland	Histology	Outcomes
Neurapraxia	First degree	No axonal damage, no demyelination, no neuroma	Loss of sensation, rapid recovery (days to weeks), no microneurosurgery
Axonotmesis	Second, third, and fourth degrees	More axonal damage, demyelination, possible neuroma	Loss of sensation, slow incomplete recovery (weeks to months), possible microneurosurgery
Neurotmesis	Fifth degree	Severe axonal damage, epineurial discontinuity, neuroma formation	Loss of sensation, spontaneous recovery unlikely, microneurosurgery

TABLE 42-4. **Sunderland Grade and Recovery Patterns**

Degree of Injury	Recovery Pattern	Rate of Recovery	Treatment
First degree	Complete	Fast (days–wk)	None
Second degree	Complete	Slow (wk)	None
Third degree	Variable	Slow (wk–mo)	Possible nerve exploration
Fourth degree	None	Unlikely recovery	Microneurosurgery
Fifth degree	None	No recovery	Microneurosurgery
Sixth degree*	Varies†	Varies†	Varies†

*Sixth-degree injury data from Dellon AL, Mackinnon SE. Basic scientific and clinical applications of peripheral nerve regeneration. Surg Annu 1988;20:59.
†Depending on specific injury pattern.

Treatment planning decisions must be based on a thorough assessment of both the subjective and the objective testing results. Also, a radiographic assessment may reveal the presence of radiographic predictors of root proximity to the inferior alveolar canal, retained root fragments, evidence of distal bone removal, or the presence of foreign bodies in the extraction sites, which should be ruled out in the initial assessment of the patient's nerve injury.

The CNT begins with inspection of the oral cavity, which may show signs of self-induced trauma to the tongue or lower lip and chin, a lingually positioned third molar incision scar, or atrophic changes of the tongue fungiform papillae.[47] Palpation in the area of the suspected nerve injury (e.g., lingual aspect of the third molar extraction site) may induce Tinel's sign, which is a provocative test of regenerating nerve



FIGURE 42-6. Visual analogue scale.

sprouts that is performed by light palpation over the area of suspected injury. This maneuver elicits a distal referred "tingling" sensation at the target site of the nerve (tongue or lower lip). This Tinel's sign is thought to indicate small-diameter nerve fiber recovery; however, it is poorly correlated with functional recovery and is often confused with neuroma formation. To perform the CNT appropriately, the patient should be seated comfortably in a quiet room, and the specific testing procedures should be explained clearly to the patient, with confirmation that there is an understanding of what the patient is being asked to do and what possible responses are acceptable. The specific tests are performed with the patient's eyes closed, and the contralateral uninjured side serves as the control, when appropriate, to establish a baseline for assessment of the injured side.

The CNT is performed at three levels: levels A, B, and C (Table 42-5).[48] The CNT involves a dropout algorithm that attempts to correlate the results of the CNT with the level of nerve injury (Figure 42-7). This level of nerve injury may be correlated with the Sunderland classification and expected outcomes. If the results of level A testing are normal, the CNT is terminated and the patient is considered normal; this would correspond to a Sunderland first-degree injury. An abnormal result at level A indicates the need to proceed to level B testing. If the results of level B testing are normal, the patient is considered mildly impaired (Sunderland second-degree injury). If level B results are abnormal, level C testing is performed. If level C results are normal, the patient is considered to have a moderate impairment of the nerve (Sunderland third-degree injury). If level C results are abnormal, the patient is considered severely impaired (Sunderland fourth-degree injury). If the patient's test results are abnormal at levels A, B, and C and there is no response to any noxious stimulus at level C, the patient is considered completely impaired (Sunderland fifth-degree injury). Level A testing includes brushstroke directional and static two-point discrimination. These two tests assess function of the larger myelinated A-alpha and A-beta fibers. These fibers are the most sensitive to compression and traction injuries; therefore, the CNT is terminated if level A is normal, indicating that these fibers are uninvolved in the injury. Brushstroke directional discrimination is performed with a fine sable or camel hair brush. The brush is stroked gently across the area of involvement at a constant rate and pressure, and the patient is asked to indicate the direction of movement (i.e., to the left or to the right) and the correct number of patient responses out of 10 strokes is recorded. Two-point discrimination is performed in a static fashion (vs. a moving two-point discrimination test) and with blunt tips to avoid A-delta and C fiber stimulation. This test can be performed with any device capable of allowing the distance between two points to be measured consistently (e.g., a Boley gauge). The closest distance (in millimeters) at which the patient can consistently discern the two points is recorded at the control site for reference and then the affected area is tested. At level B testing, contact detection is performed with Semmes-Weinstein monofilaments or von Frey hairs, which also assess the A-beta fiber integrity and function. These devices are acrylic resin or plastic transparent/translucent rods with nylon filaments of varying diameters. The stiffness of each filament determines the force necessary to deflect or bend the filament. The narrowest diameter filament that requires the least amount of force to deflect that is detected consistently by the patient is recorded. At level C testing, pinprick nociception and thermal discrimination assess the smaller A-delta and C fibers, which are most resistant to injury. Pinprick nociception may be performed simply with a dental explorer tip or a 30-gauge needle; however, a pressure-sensitive device is more appropriate in order to quantify the test result rather than achieving an all-or-none response, which would vary depending upon depth of penetration of the needle. Thermal discrimination is usually performed with suprathreshold temperature devices such as an ice cube or ethyl chloride spray or hot water placed on a cotton swab, but other options are available, such as

TABLE 42-5. **Clinical Neurosensory Testing**

Subjective assessment: visual analogue scale
Objective assessment
Level A: static two-point discrimination, brush-stroke directional discrimination
Level B: contact detection
Level C: pinprick nociception, thermal discrimination

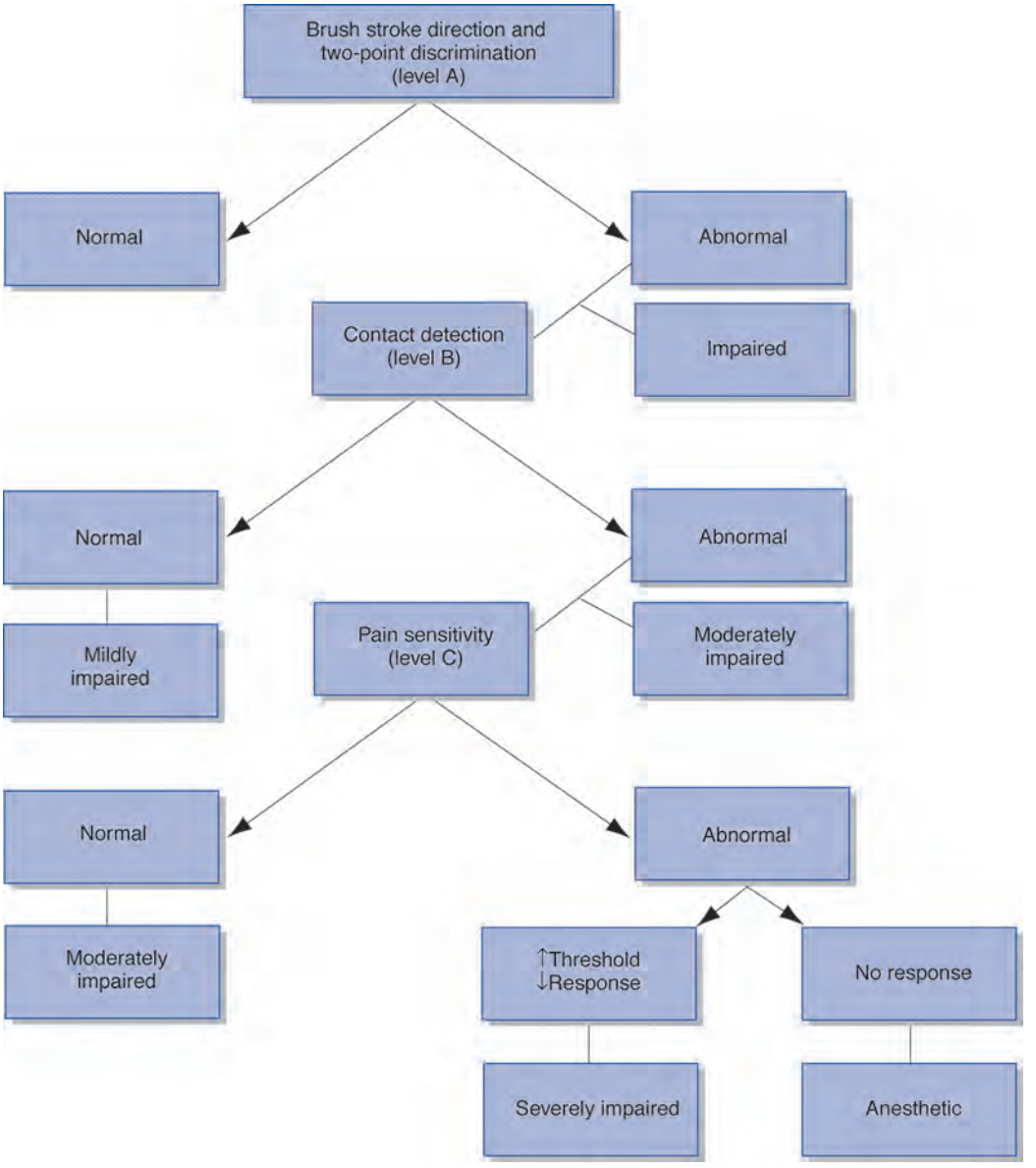


FIGURE 42-7. Algorithm for objective clinical neurosensory test.

Minnesota thermal disks made of copper, stainless steel, glass, and polyvinyl chloride.

Although these five tests employed in the CNT are considered "objective" tests, they are, in reality, "subjective" because they require a "subjective" patient response. Few purely objective tests of nerve function are available, and these include trigeminal somatosensory evoked potentials and magnetic source imaging.[49] Unfortunately, these tests are not readily available and are not considered a part of the routine assessment of a nerve-injured patient. Also, there are little data on the trigeminal nerve and the patterns of responses to these objective tests based on specific types of nerve injuries.

Finally, taste may be assessed by a variety of methods, but, it is performed as either whole -mouth or localized taste testing. Solutions such as 1 M sodium chloride (salt), 1 M sucrose (sweet), 0.4 M acetic acid (sour), and 0.1 M quinine (bitter) are used. There are many difficulties and limitations with gustatory assessment in the patient with an LN injury. The perception of taste alteration (cranial nerve VII) is extremely variable and has little correlation with the degree of LN (cranial nerve V) injury. For example, a patient with a fourth- or fifth-degree LN injury may not report any taste alteration subjectively but may test abnormally upon formal taste testing. The complex sense of taste is mediated not only by the chorda tympani branch of the facial nerve but also through feedback mechanisms in the nasopharynx, oropharynx, and hypopharynx as well as the nucleus tractus solitarius in the brainstem.[50] Taste buds are located throughout the oral cavity, not solely on the anterior two thirds of the

tongue surface, but the palate, buccal mucosa, and floor of mouth. Also, after microneurosurgical LN repair, the objective and subjective neurosensory recovery of tongue sensation and taste perception also are inconsistent.[51]

Diagnostic nerve blocks can be a useful component of the patient evaluation when dysesthesia or unpleasant sensations predominate the clinical scenario. The goals of the diagnostic nerve block is to localize the source of nerve injury, determine whether delivery of peripheral anethesia resolves the painful symptoms, and estimate the prognosis for recovery after either pharmacologic or surgical therapy. If surgical management includes consideration for a nerve transection procedure for pain relief, the nerve block may use a long-acting local anesthetic solution to allow the patient several hours to determine whether permanent anesthesia is tolerable. The preferred local anesthetic solution for a routine nerve block, however, is of a low concentration (e.g., 0.25% lidocaine) to selectively block the smaller A-delta and C fibers while not affecting the larger myelinated fibers. If the low concentration fails to relieve the pain, a higher concentration is used in the same location. Diagnostic blocks begin in the most central location and proceed with progressively more peripheral injections along the course of the nerve with constant reassessment of the area of involvement both objectively and subjectively. If the patient presents with symptoms consistent with sympathetically mediated pain or causalgia, a stellate ganglion block may be performed as well, but these symptoms usually indicate a problem not amenable to peripheral microneurosurgery. Other painful syndromes that generally are not relieved with diagnostic nerve blocks include anesthesia dolorosa and deafferentation pain, and similar to other neuropathies with predominating symptoms of dysesthesia, these syndromes are used managed with pharmacotherapy.

NONSURGICAL TREATMENT

Pharmacologic management of peripheral nerve injuries is reserved for patients who present with unpleasant abnormal sensations or dysesthesia. In the majority of these cases, pharmacologic treatment should be managed with a consultation from an experienced individual such as a neurologist or facial pain specialist. Many systemic (Table 42-6) and topical (Table 42-7) medications are available.[51] Whereas the systemic drugs may have significant side effects, topical agents offer the advantages of little systemic absorption, possibly only minor irritation (which can be relieved with a period of abstinence), and over-the-counter availability in many cases. There are also many combinations of topical agents that can be used, such as a eutectic mixture of local anesthetics (EMLA) that contains 2.5% lidocaine and 2.5% prilocaine. Many of the topical agents are prepared in a pleuronic lecithin organogel base. For most oral and maxillofacial surgeons, long-term pharmacologic management with drug dosage adjustments based upon clinical response is not part of their routine practice, so the prompt referral to a microneurosurgeon or neurologist may offer the best chance for long-term successful patient management. Pharmacotherapy for dysesthesia includes the use of membrane-stabilizing agents, such as antidepressants and anticonvulsants, and many neurologists and surgeons consider gabapentin (neurontin) as a first-line agent. There are also dorsal horn inhibitors or γ-aminobutyirc acid (GABA) agonists, including muscle relaxants and benzodiazepines. Consideration may be given to a trial of a topical agent such as capsaicin cream 0.025% or 0.075% three times a day and/or a systemic medication with few side effects, such as baclofen (Lioresal) 10 mg orally three times a day, pregabalin (Lyrica) 50 mg orally three times a day, or gabapentin (Neurontin) 100 mg orally three times a day. The most common side effect of these medications is sedation or drowsiness that may require frequent daily dose adjustments. Again, surgeons unfamiliar with the drugs, as well as the spectrum of pharmacologic options, should consider obtaining a neurology consultation.

It may be appropriate to manage perioperative paresthesia after third molar removal or implant placement with a short course of systemic corticosteroid therapy in an attempt to decrease perineural edema caused by the local response to the nerve injury. Although there is little evidence to suggest that systemic steroids actually provide any effect, the use of steroids when a nerve injury occurs indicates that the surgeon has recognized the problem and has taken some initiative to potentially improve outcome based upon a knowledge of neural anatomy and injury physiology, which is advantageous when considering medicolegal involvement issues.

TABLE 42-6. **Systemic Pharmacologic Agents**

Local anesthetics
Corticosteroids
Nonsteroidal anti-inflammatory agents
Antidepressants
Narcotic analgesics
Anticonvulsants
Muscle relaxants
Benzodiazepines
Antisympathetic agents

TABLE 42-7. **Topical Medications**

Category	Example
Topical anesthetics	5% viscous lidocaine gel; 20% benzocaine gel; 2.5% lidocaine with 2.5% prilocaine
Neuropeptides	Capsaicin cream (0.025% or 0.075%)
Nonsteroidal anti-inflammatory drugs	Ketoprofen 10–20% PLO base; diclofenac 10–20% PLO base
Sympathomimetics	Clonidine 0.01% PLO base or patch
N-methyl-D-aspartate blocking agents	Ketamine 0.5% PLO base
Anticonvulsants	Carbamazepine 2% PLO base
Tricyclic antidepressants	Amitriptyline 2% PLO base
Antispasmodics	Baclofen 2% PLO base

PLO = pleuronic lecithin organogel.

TABLE 42-8. **Microneurosurgeon Referral Indications**

Observed nerve transection
Complete postoperative anesthesia
Persistent paresthesia (lack of improvement in symptoms) at 4 wk
Presence or development of dysesthesia

Perhaps the most important initial consideration in patient management should be prompt referral, when indicated, to a specialist for pharmacologic or surgical management. The indications for referral include but are not limited to those listed in Table 42-8.

In the past, before considerating surgical management, a variety of neuroablative techniques had been used to ameliorate painful neuropathies. Some of these included radiofrequency thermal neurolysis, cryoneurolysis, and alcohol and glycerol injections at the site of injury as well as at the gasserian (trigeminal) ganglion. Based on the published complications of these types of procedures as well as the recurrence rates of the symptoms of dysesthesia, caution should be employed when considering these options.[53,54] The use of a low-level ("soft") laser (gallium-aluminum-arsenide, wavelength of approximately 820 nm) holds promise in the area of neural healing. Several studies have shown improvement in objective and subjective neurosensory function with the use of laser therapy in some of the more difficult clinical scenarios, such as longstanding nerve injuries, orthognathic-related IAN paresthesia, and prolonged dysesthesia unresponsive to pharmacologic or surgical therapy.[55–57] However, the current limited availability of the low-level laser and the lack of approval by the U.S. Food and Drug Administration (FDA) preclude its routine use for patients with nerve injuries, although clinical trials continue.

TRIGEMINAL NEURALGIA

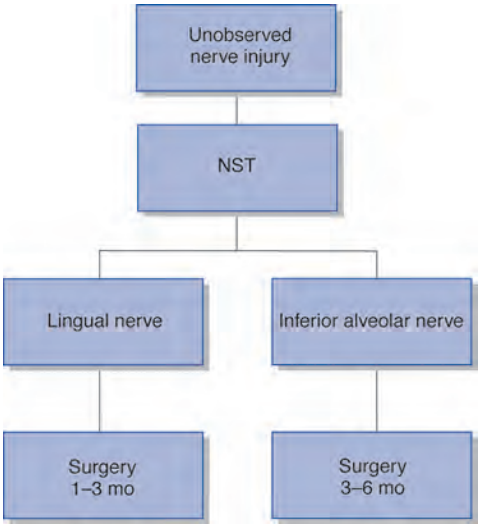
Whereas it is not the intention of this chapter to cover the various facial pain syndromes, it is appropriate to discuss briefly the subject of trigeminal neuralgia (TN), which is typically not amenable to microneurosuregry but should be ruled out in the assessment of patients who present with complaints related to the trigeminal nerve system. The reported incidence of TN is 4/100,000 as a spontaneous occurrence. Female patients older than 50 years are more commonly affected by TN symptoms, and the right side of the face is involved more often than the left. The typical symptoms are lancinating or stabbing ("electric shock"–like) paroxysms of facial pain involving the second and third divisions of the trigeminal nerve (V2 and V3). Although various theories have been proposed, including arterial compression of the trigeminal nerve in the pons region (Janetta), the etiology of TN is unknown in most cases and, therefore, has been designated idiopathic trigeminal neuralgia (ITN). The diagnosis of TN is made based upon the Sweet diagnostic criteria, 1969, that includes paroxysmal pain, pain provoked by light touch (with trigger zones), pain confined to the trigeminal distribution, unilateral pain, and a normal clinical neurosensory test. The International Classification of Headache Disorders (ICHD) has established criteria to differentiate two forms of TN, classic TN and symptomatic TN. *Classic TN* is the most common form with paroxysmal attacks lasting from 1 second to 2 minutes affecting the trigeminal nerve. The pain has at least one of the following features: intense, sharp, superficial or stabbing, or precipitated by a trigger point or area. The attacks in classic TN are stereotyped in each individual patient occurring at the same time and location each day. There is no clinical neurologic deficit on examination. Finally, the symptoms of classic TN cannot be attributed to another disorder. The second form, *symptomatic TN*, has all of the features of classic TN with the additional finding of a causative lesion, other than vascular compression, identified by special studies (magnetic resonance imaging [MRI], lumbar puncture, evoked potentials, hematologic studies) or exploration of the posterior cranial fossa. Another form of TN is known as *atypical TN* because it may meet most, but not all, of the Sweet diagnostic criteria of classic TN. TN is managed with pharmacologic therapy, and usually multidrug regimens are used. A variety of medications have been used, including antiepileptic drugs, gabapentin, baclofen, clonezepam, lamotrigine, oxcarbazepine, toprimate, and carbamezepine. For acute exacerbations of TN symptoms, several treatment options include peripheral local anesthetic nerve block, intravenous lidocaine (100 mg over 20 min), intravenous antiepilepric drugs (dilantin, fosphenytoin, or valproic acid), and analgesics (opioid and nonopioid) that may have little clinical effect. Surgical options for TN are limited to a few specialized procedures, with minimal morbidity, that may provide some form of symptomatic relief in 80% to 85% of patients. These include percutaneous stereotactic radiofrequency thermal lesioning of the trigeminal ganglion, posterior fossa exploration and microvascular decompression of the trigeminal root, and gamma knife radiation to the trigeminal nerve root entry zone, which has shown promising results.

TREATMENT ALGORITHMS

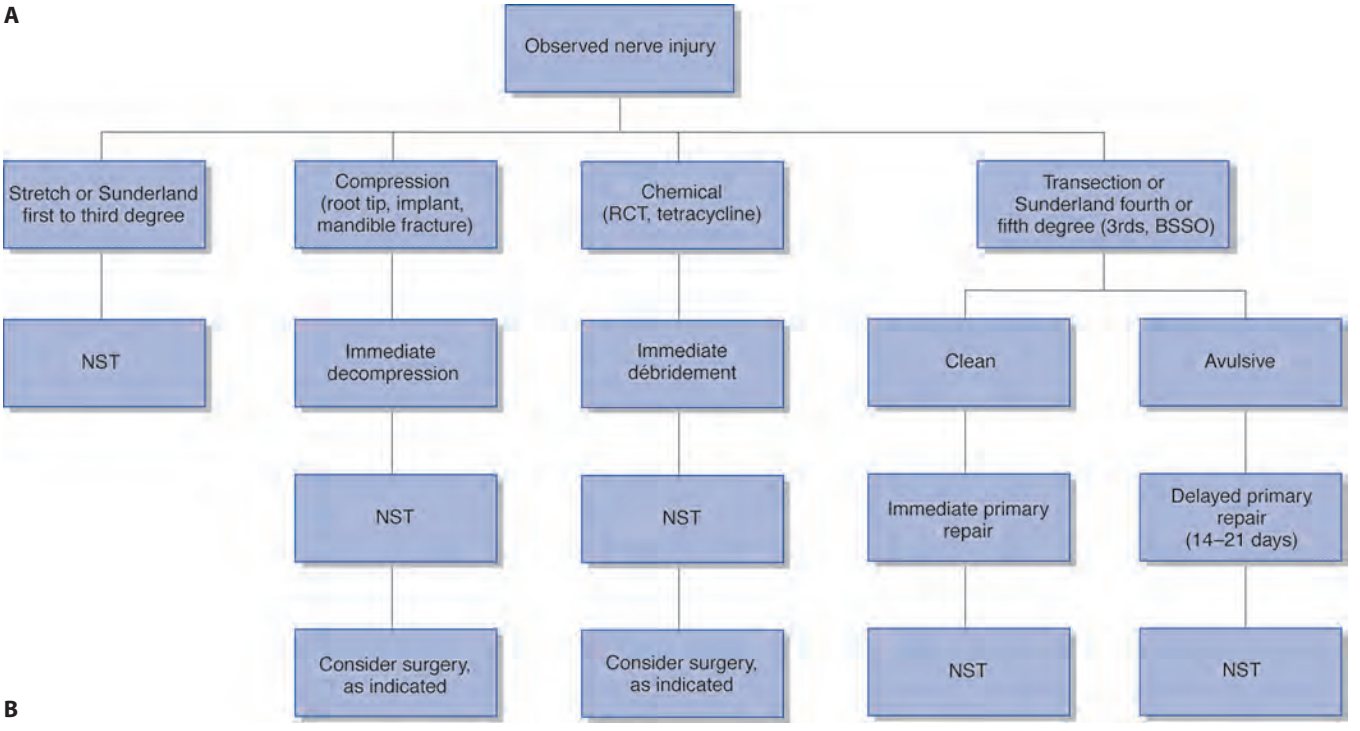
The decision to proceed with microneurosurgery must be made after a careful patient assessment over a defined period of time. The dilemma for clinicians is that sufficient time must be given to allow for spontaneous neurosensory recovery but that prompt surgical intervention may afford the best chance for recovery. Time is a critical issue in the management of nerve injuries for three main reasons. First, at the site of injury, distal nerve degeneration (wallerian degeneration—named for Augustus Waller, 1892) occurs owing to an interruption of anterograde and retrograde axonal transport across the site of injury. This results in a progressive loss of neural tissue due to lack of stimulation that may compromise future repair attempts by causing a greater length of neuromatous nerve segment requiring resection at the time of nerve repair. Second, at the nerve cell

bodies, there is ganglion cell death that occurs early after injury.[58] This may result in a large percentage (>50%) of cell body death in a short period of time after the injury (weeks to months), so that any "peripheral" attempt at microneurosurgical repair would have, at best, a 50% functional neurosensory recovery. Third, as the time from the nerve injury increases, there is a higher likelihood that central intracranial somatosensory cortical changes may occur, and these would make peripheral repair ineffective.[59] The central nervous system remains "plastic" in that longstanding peripheral painful neuropathies become "expected" in the area of the brain devoted to that region and, despite peripheral nerve transection as an attempt at painful symptom resolution, the brain will continue to interpret the information not as "anesthesia," but as "dysesthesia" (similar to "phantom-limb" pain syndromes).

Microneurosurgery is indicated for persistent paresthesia that fails to improve over successive examinations, including both subjective and objective interval assessments. Microneurosurgery is not indicated if there is continued improvement at each interval assessment. The currently accepted recommendations are to consider microneurosurgery, when indicated, for the LN within 1 to 3 months after the injury and for the IAN within 3 to 6 months after the injury (Figure 42-8). The rationale for the difference in time is that the IAN lies within a bony canal or "physiologic conduit" that can



A



B

FIGURE 42-8. Nerve treatment algorithms. **A,** Unobserved nerve injury. **B,** Observed nerve injury. BSSO = bilateral sagittal split osteotomy; NST = neurosensory testing; RCT = root canal therapy.

guide spontaneous regeneration, so more time is allotted for that process to occur, whereas an LN injured within soft tissue does not have a "conduit" to guide spontaneous regeneration. In general, the oral and maxillofacial surgeon should schedule weekly follow-up examinations with the patient over a period of approximately 4 weeks. If there is persistent paresthesia or a worsening of symptoms, referral should be made to a microneurosurgical specialist for prompt definitive evaluation and management.

The indications for microneurosurgery include the complete (100%) postoperative anesthesia, or less than 50% residual sensation in an injury classified as a Sunderland grade III, IV, or V. Some surgeons consider another indication to be the level of sensation, which is unacceptable to the patient, even if the objective tests show greater than 50% residual sensation of a Sunderland I or II injury. In these cases, the patient must be informed of the reasonable expectation for recovery after microneurosurgery, which, according to the literature, may be at or below their current level of sensibility. Also, whereas as a general rule unpleasant sensations are managed pharmacologically and decreased sensations are managed surgically, some cases of early dysesthesia or hyperpathic pain may indicate the early formation of a neuroma at the site of nerve injury, and consideration should be given toward early microneurosurgical intervention.

For an unobserved nerve injury, the plan should be to continue neurosensory testing for 1 month and then to refer for surgery in the 1- to 3-month (LN) or 3- to 6-month (IAN) time periods. For an observed nerve injury, treatment should focus on the specific etiology. For a suspected traction injury (Sunderland first-, second-, and third-degree injuries), the patient should be tested for 1 month for signs of expected spontaneous recovery. In the case of nerve compression, immediate decompression should be considered. This includes removal of a root displaced into the IAN canal, removal or replacement when there is evidence of implant impingement within the confines of the IAN canal, or reduction and alignment of a displaced posterior mandible fracture including the IAN canal. Neurosensory testing should be performed after decompression, and microneurosurgery should be considered as indicated. Chemical injuries should be débrided promptly. For observed transection injuries (Sunderland fourth- or fifth-degree injuries), an immediate primary repair may be performed for a clean transection injury (e.g., scalpel transection). For an avulsive injury (e.g., LN entangled in a bur), consideration is given to a delayed primary repair performed at 3 weeks after the injury. This allows time for the proximal and distal nerve stumps to define the extent of fascicular injury and to determine whether the immediate surrounding environment is conducive to nerve repair surgery, at a time when there are very high levels of neurotropic and neurotrophic factors (at 21 days after injury). After microneurosurgery, patients should be examined with repeat neurosensory testing following the first signs or symptoms of return of sensation. Sensory reeducation exercises should be considered early in the postoperative period to potentially augment neurosensory recovery.

TABLE 42-9. **Classification of Sensory Recovery (Medical Research Council System)**

Grade (Stage)	Recovery of Sensibility
S0	No recovery
S1	Recovery of deep cutaneous pain
S1+	Recovery of some superficial pain
S2	Return of some superficial pain and tactile sensation
S2+	S2 with overresponse
S3*	Return of some superficial pain and tactile sensation without overresponse; two-point discrimination > 15 mm
S3+	S3 with good stimulus localization; two-point discrimination = 7–15 mm
S4	Complete recovery, S3+; two-point discrimination = 2–6 mm

*S3 score indicates significant clinical recovery (from Wyrick JD, Stern PJ. Secondary nerve reconstruction. Hand Clin 1992;8:587).
Adapted from Mackinnon SE. Surgical management of the peripheral nerve gap. Clin Plast Surg 1989;16:587.

The reported success rates of microneurosurgical reconstruction after nerve injury are variable in the literature. This is due to many factors including the lack of standardization in the diagnosis and management process and also with wide variations of the following[60]: patient age, etiology of the nerve injury, time of delay from injury to repair, experience and training of the microneurosurgeon, specific surgical techniques used, the length of the nerve gap, the methods of clinical neurosensory testing, the use of normative values for control sites, the length of the follow-up period, and important, the criteria used by the surgeon to define "success" (Table 42-9). A global review of the literature might indicate a success rate of 30% to 50% after microneurosurgery, including direct and indirect (gap or graft) repair techniques. These studies generally use subjective patient assessements to quantify the results into a scale used by the patient to define the sensation after microneurosurgery as worse, no change, some improvement, or good improvement. In general, direct repair (neurorrhaphy) is preferred over gap repair (e.g., using an autogenous nerve graft or conduit) and has higher reported success rates.[61] Perhaps the largest study to date indicates an overall "success" rate of 76.2% in 521 patients.[62] The success criteria were defined as light touch detected more than 80% of the time and a 30% decrease in postoperative pain level. Although the success criteria are less than ideal, the study results suggested some important trends in outcome after microneurosurgery: hypoesthetic (decreased sensation) injuries improved more after microneurosurgery than did hyperesthetic (unpleasant sensation) injuries; the LN recovered more than did the IAN overall after microneurosurgery; and there was a decrease in success associated with a delay from injury to repair of longer than 6 months. Another report of 51 microneurosurgical reconstructions (direct and gap repairs) found that

10 patients subjectively reported good improvement, 18 patients some improvement, 22 patients no improvement, and 1 patient reported feeling worse after surgery.[63] This indicates that 55% of patients (28/51) showed some improvement, or "success." In another study of 53 surgical patients, with a mean follow-up of 13 months, light touch improved from 0% to 51% and pinprick nociception improved from 34% to 77%. Patients in this study also experienced improved taste and an increased number of fungiform papillae, and there was a decrease in incidence of accidental tongue biting. Interestingly, there was no correlation of success with delay from time of injury to repair. No patient became completely normal, and there was no reduction in dysesthesia; however, most patients considered the surgery worthwhile. In order to most appropriately interpret the literature and allow proper assessment and management of patients who sustain nerve injuries based upon a critial review of evidence-based medicine, there is certainly a need for standardization in all aspects of evaluation and management of the nerve-injured patient.

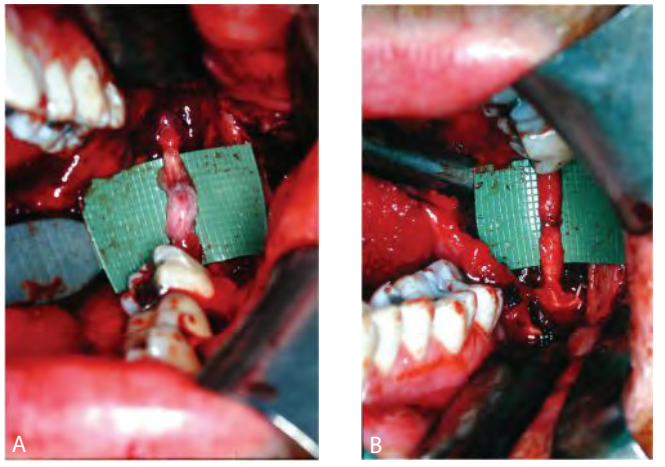


FIGURE 42-9. Lingual nerve (LN) direct repair. **A,** Left LN neuroma after exposure. **B,** Direct repair of left LN after neuroma resection.

SURGICAL TREATMENT

Microneurosurgical reconstruction surgery involves an orderly sequence of surgical procedures including nerve identification and exposure (either via access through soft tissues for the LN [Figure 42-9] or via an osteotomy for the IAN [Figure 42-10]), dissection of the nerve from the surrounding tissues, assessment of the degree of injury, manipulation of the nerve (neurolysis), which may include neuroma resection, débridement of the nerve stumps as required, and repair with a direct anastomosis or the use of a nerve graft or conduit. Many of the techniques of trigeminal nerve repair closely mimic those of hand surgery (although access is more limited) and use similar microneurosurgical instrumentation. In general, surgical loupe magnification (×3.5 magnification) is adequate for most microneurosurgeons. An operating microscope (×12 magnification) provides a higher magnification, but it is cumbersome and difficult to use effectively with a transoral exposure in the posterior oral cavity, although it may be more useful with a transfacial approach to the IAN. Most experienced microneurosurgeons have had some form of advanced training in the technical aspects of this specific type of surgery, including the use of microinstruments and an operating microscope for both vascular and

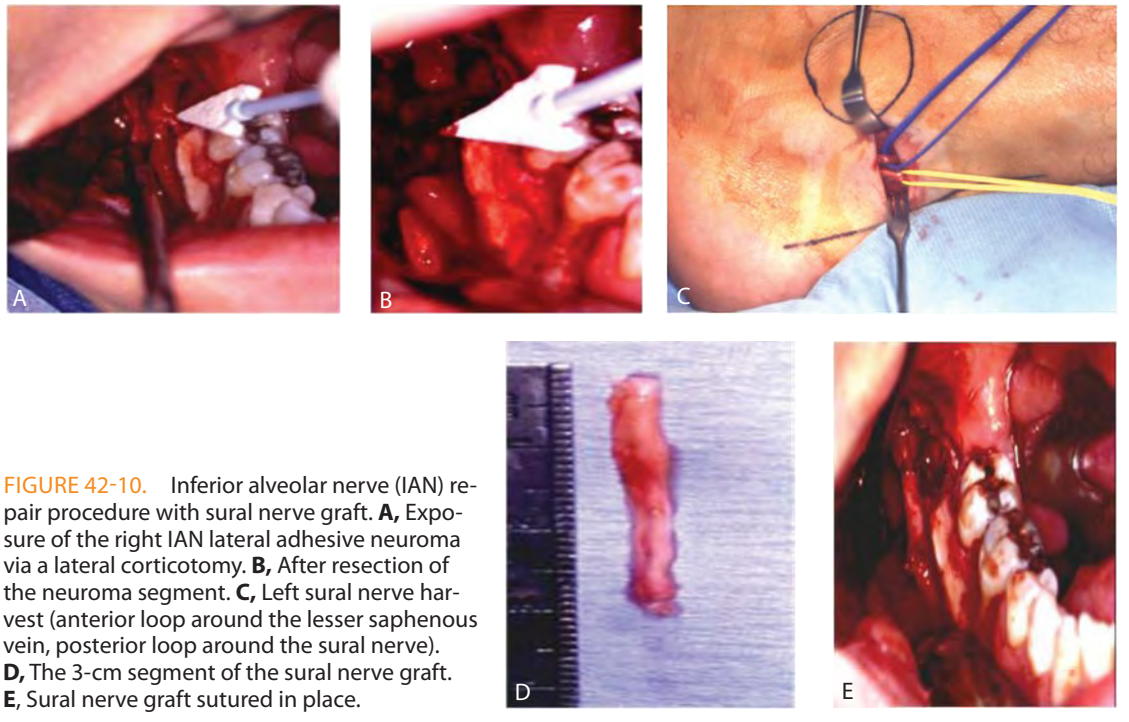


FIGURE 42-10. Inferior alveolar nerve (IAN) repair procedure with sural nerve graft. **A,** Exposure of the right IAN lateral adhesive neuroma via a lateral corticotomy. **B,** After resection of the neuroma segment. **C,** Left sural nerve harvest (anterior loop around the lesser saphenous vein, posterior loop around the sural nerve). **D,** The 3-cm segment of the sural nerve graft. **E**, Sural nerve graft sutured in place.

neural tissue handling and repair, although, as mentioned previously, these techniques are becoming more widespread with dissemination of the principles and practice of microneurosurgery, as well as microvascular surgery, in oral and maxillofacial surgery residency training programs in the United States and abroad.

Exposure

Surgical access to the LN or IAN may be accomplished transfacially or transorally. The transfacial approach to the IAN in the posterior mandible affords wide surgical exposure and access; however, it necessitates a facial incision with subsequent scar formation. The intraoral approach provides a more difficult surgical access and requires more diligence during the procedure involving the posterior regions of the oral cavity, but it avoids a facial scar. The decision regarding surgical access depends on an individual patient's anatomy; the site of nerve injury; planned surgical procedures; patient preference; and surgeon's preference, skill, and experience.

External Neurolysis

Microdissection of the nerve once exposed involves liberation of the nerve from the surrounding tissues to facilitate inspection. For the LN, this procedure may commonly involve the release of the nerve from a lateral adhesive neuroma in the area of the lingual plate in the third molar region or from within the healing third molar extraction site itself, whereas for the IAN, a lateral corticotomy is generally required in order to perform an external neurolysis and mobilization of the IAN from within the inferior alveolar canal. Several techniques have been described for lateral decortication in the area of the third molar for IAN exposure, ranging from a simple nerve transpositioning procedure to a modified buccal corticotomy or a unilateral sagittal split ramus osteotomy (Figure 42-11).[64] The specific location of the injury as well as the surgeon's preference frequently dictate the specific approach used. The LN is usually exposed via a modified incision used for third molar surgery with a gingival sulcular lingual extension (Figure 42-12). As another

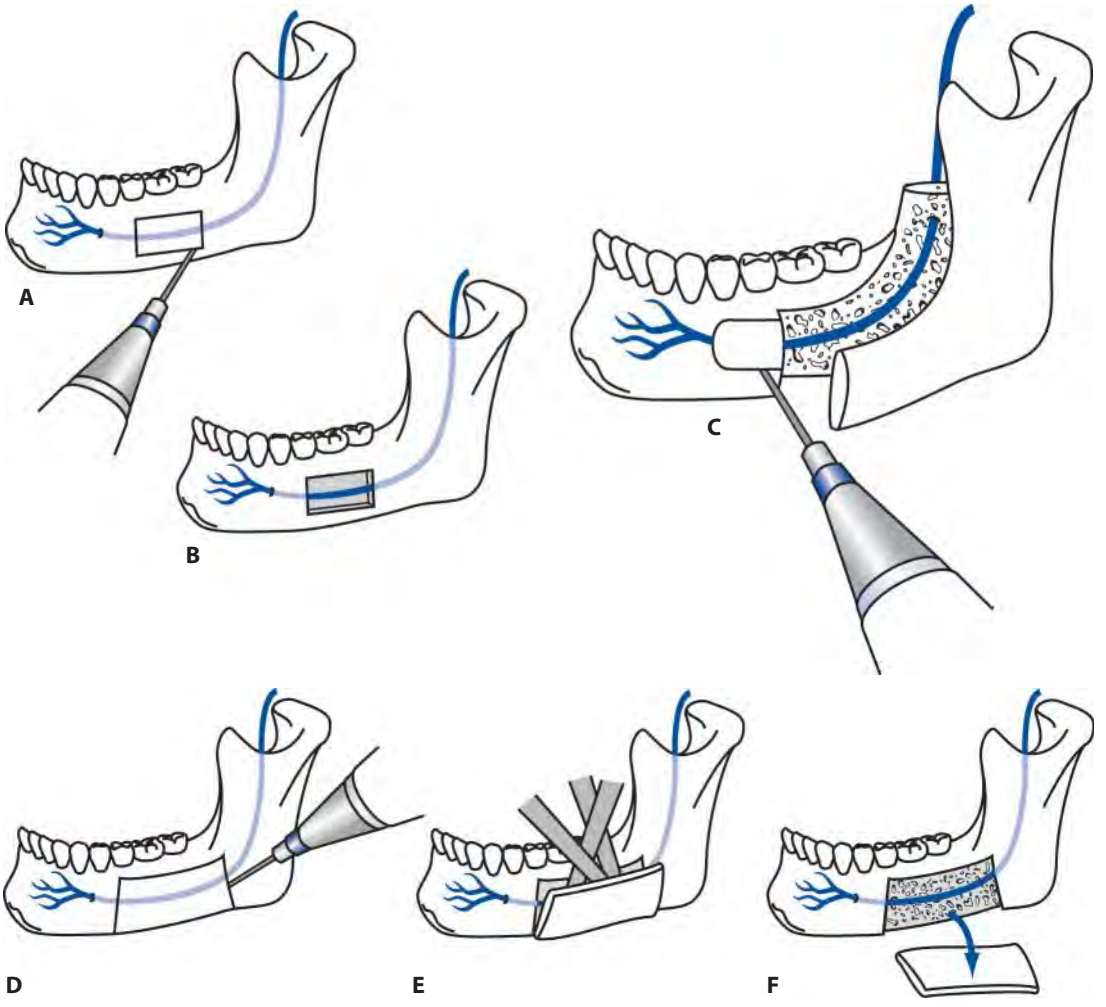


FIGURE 42-11. Exposure techniques for the IAN. **A,** Lateral decortication of the mandible. **B,** With exposure of the inferior alveolar neurovascular bundle. **C,** Sagittal ramus osteotomy with anterior extension via lateral decortication to the mental foramen. **D,** Lateral mandibular decortication. **E,** Bone removal with chisels. **F,** Wide exposure of the neurovascular bundle.

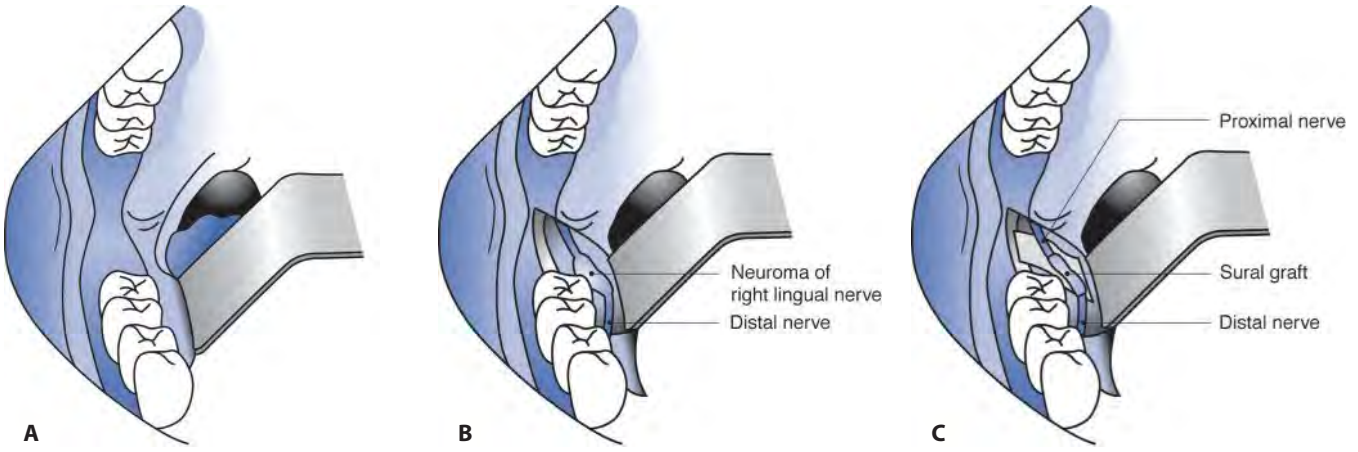


FIGURE 42-12. LN exposure. **A,** Incision design via a distobuccal extension and lingual gingival sulcus approach. **B,** Right LN exposure with neuroma. **C,** Right LN repair with an interpositional nerve graft.

example, external neurolysis of the infraorbital nerve (V2) may be performed via reduction and fixation of the displaced zygomaticomaxillary complex fracture impinging on the neurovascular bundle at its exit through the infraorbital foramen. It has been suggested that external neurolysis may provide definitive treatment for a nerve injury if the nerve compression is less than 25% of the normal diameter, if the paresthesia is of short duration (<6 mo), and if there is no evidence of neuroma formation.[65] Typically, external neurolysis is followed by techniques of "internal" neurolysis that will address the specific fascicular injury by internal nerve manipulation.

Internal Neurolysis

The term *internal neurolysis* refers to surgical manipulation within the confines of the epineurium that is done in order to prepare the nerve for subsequent repair. Sophisticated maneuvers that involve attempts to dissect the individual fascicles of the nerve (especially in a polyfascicular nerve) may compromise repair by unnecessary removal of tissue and/or induction of cicatrix formation owing to excessive manipulation in the interfascicular spaces. Several methods of internal neurolysis have been described, including epifascicular epineurotomy, epifascicular epineurectomy, and interfascicular epineurectomy (Figure 42-13). The first two techniques prepare the epineurium for repair, and the third technique involving interfascicular dissection may cause further fascicular disruption and tissue scarring. Extensive internal neurolysis procedures should be used with caution, with the goal of doing no further harm to the uninvolved segments of the proximal and distal nerve stumps.

Nerve Stump Preparation

Perhaps the most critical portion of the microneurosurgical procedure involves thorough inspection of the proximal and distal nerve stumps via magnification. It should be noted that there may already be an existing discontinuity from a transection injury to the nerve and identification of the proximal and distal stumps may be difficult owing to scar tissue formation. When a neuroma of any type is present,

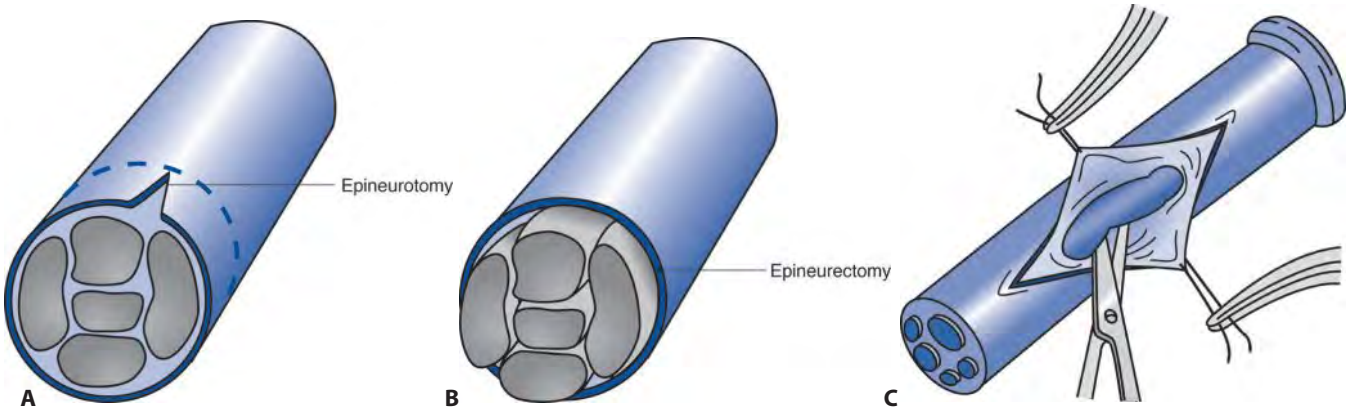


FIGURE 42-13. Internal neurolysis. **A,** Epifascicular epineurotomy. **B,** Epifascicular epineurectomy. **C,** Interfascicular epineurectomy. (**A** and **B,** Adapted from LaBanc JP. Reconstructive microneurosurgery of the trigeminal nerve. In Peterson **L J**, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 2. Philadelphia: JB Lippincott; 1992; p. 1067.)

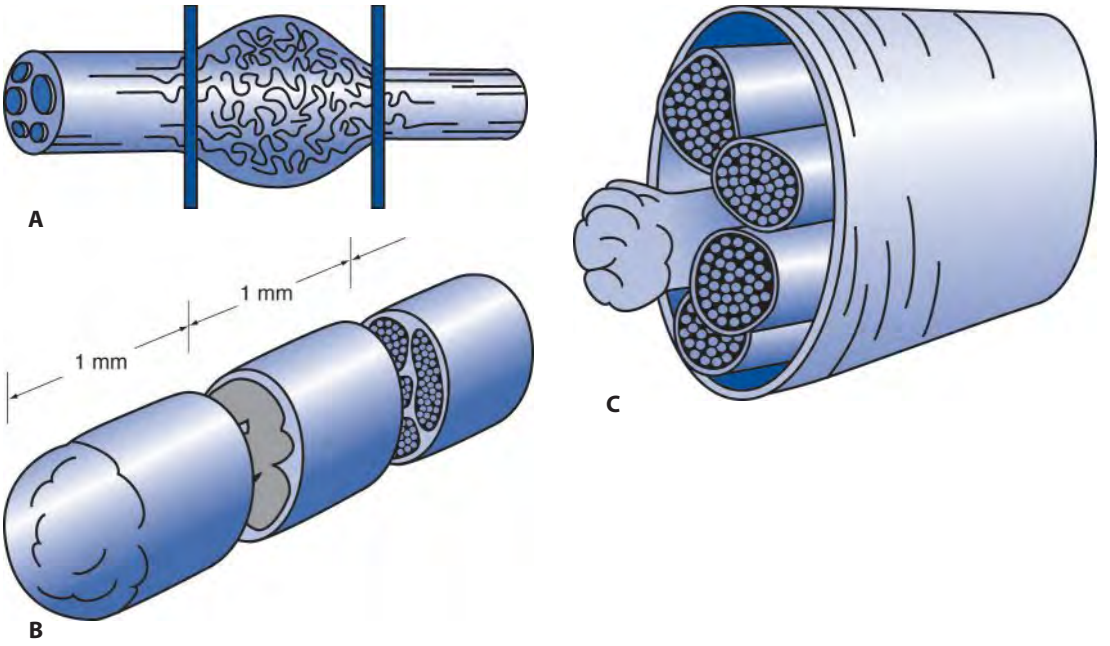


FIGURE 42-14. Nerve stump preparation. **A,** Neuroma; resection at the "clinical margin" of the neuroma fails to complete nerve preparation. **B,** Neuroma resection in 1-mm increments. **C,** Mushrooming fascicle.

meticulous attention to detail is required in order to remove all neuromatous tissue (Figure 42-14). It must be recognized that with any neuroma, the clinical appearance of neuronal edema or atrophy may be less apparent than the actual, more severe internal fascicular disruption (see Figure 42-14A). The failure by the microneurosurgeon to resect enough nerve tissue to encounter normal nerve fascicular tissue will inevitably result in a failure of neurosensory recovery. Once the nerve is divided, when necessary, into proximal and distal stumps, care must be taken to sequentially resect small (1-mm) portions of the nerve trunk in both directions (see Figure 42-14B) until healthy glistening white "mushrooming" fascicles are seen to herniate through the edges of the epineurium (see Figure 42-14C). The microneurosurgeon should be confident that the repair will be performed in order to anastomose healthy nerve tissues, rather than neuromatous tissue, and this requires an aggressive approach at this point and may result in a greater need for consideration of indirect (graft) repair over direct neurorrhaphy. The informed consent process for all microneurosurgical procedures should include a discussion of options for gap repair in the event that this becomes necessary during the surgical procedure.

Approximation

The trigeminal nerve is similar to other peripheral nerves in that it does not tolerate tension well; therefore, tension-free closure after microneurosurgical repair is mandatory.[66] The deleterious effects of tension on a repair site result from vascular compromise and subsequent fibrosis at the nerve repair site, essentially resulting in the formation of a surgically induced neuroma. Approximation is the act of bringing the nerve stumps into direct contact and assessing the degree of tension that may be present. At the time of approximation, a decision must be made regarding whether to use an interpositional graft or conduit. In general, mobilization with primary epineurial repair is usually possible when the LN gap is less than 10 mm and when the IAN gap is less than 5 mm. The difference in acceptable gap length is due to the relative immobility of the IAN within the confines of the inferior alveolar canal, whereas there is usually more mobility of the LN stumps with dissection into the pterygomandibular space in a posterior direction and the floor of the mouth in an anterior direction.

Coaptation

Coaptation is the process of aligning the proximal and distal nerve stumps into the correct premorbid cross-sectional fascicular orientation. This is a very difficult maneuver to accomplish with a polyfascicular nerve that has undergone any degree of proximal or distal nerve structural alteration in diameter or fascicular pattern. This surgical step of coaptation of the LN or IAN is usually not performed with diligence in because of the complex polyfascicular pattern of the nerve as well as the limited visualization in the posterior oral cavity.

Neurorrhaphy

Neurorrhaphy is the act of bringing the nerve ends together and maintaining this position with sutures. A *direct neurorrhaphy* is performed with the proximal and distal nerve stumps, and an *indirect neurorrhaphy* is performed when using an interpositional graft between the nerve stumps, such as a nerve graft or conduit. The polyfascicular trigeminal

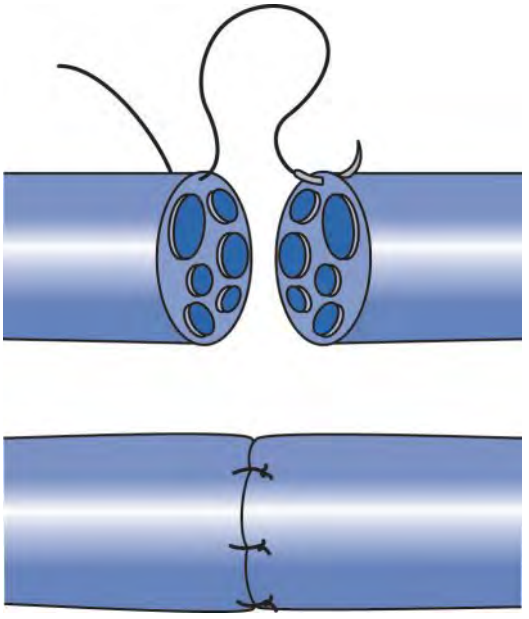


FIGURE 42-15. Direct epineurial neurorrhaphy.

nerve is repaired using epineurial sutures, not perineurial sutures (Figure 42-15), because any attempt to suture the individual fascicles would be technically difficult as well as prone to induce excessive scar tissue in the area of nerve repair. Generally, 8-0 to 10-0 monofilament nonresorbable nylon suture is preferred, because a resorbable suture material would tend to invoke an inflammatory response and disturb the area of anticipated neural healing. At least two sutures should be used per anastomosis site to prevent rotation of the nerve stumps, but no more than three or four sutures should be used per anastomosis. The first suture is usually placed on the medial side of the anastomosis because it is the more difficult site of repair to access. The epineurium is pierced with the needle approximately 0.5 to 1.0 mm from the edge of the nerve stump. The second suture is placed 180 degrees from the first suture row on the lateral aspect of the repair site, and then an assessment is made regarding the necessity for the placement of one or two additional sutures.

Nerve Grafts

When neurorrhaphy is not possible without tension and a nerve gap exists, an interpositional graft must be considered for indirect neurorrhaphy.[67] The options for autogenous nerve grafting include, but are not limited to, the sural nerve, the greater auricular nerve, and possibly the medial antebrachial cutaneous nerve.[68] The sural nerve is preferred for grafting because it most appropriately matches the nerve diameter and the fascicular number and pattern of the trigeminal nerve (Table 42-10).[69] The area of the nerve superior to the lateral malleolus exhibits less branching than at or below the lateral malleolus. The sural nerve, or medial sural cutaneous nerve, is a branch of the sacral plexus (S1, S2) and supplies sensory

TABLE 42-10. **Size of Donor Nerve Grafts Relative to Injured Nerve**

	Donor Nerve		
Injured Nerve	Sural (2.1 mm)	Greater Auricular (1.5 mm)	Greater Auricular Cable (3.0 mm)
Inferior alveolar (2.4 mm)	88%	63%	125%
Lingual (3.2 mm)	66%	47%	94%

Adapted from Brammar JP, Epker BN. Anatomic-histologic survey of the sural nerve: implications for inferior alveolar nerve grafting. J Oral Maxillofac Surg 1988;46:111.

information to the posterior lower extremity and the dorsolateral foot. Sural grafts up to 20 cm in length are possible, and patients tolerate the donor site deficit well.[70] The greater auricular nerve is a poor choice for trigeminal repair. As a branch of the cervical plexus (C1, C2), the greater auricular nerve supplies sensation to the pre- and postauricular regions, the lower third of the ear, and the skin overlying the posteroinferior border at the angle of the mandible. Patients are generally not amenable to sacrificing sensation of one facial region to regain sensation in another neighboring location. In addition, the small diameter of the greater auricular nerve makes it useful only when used as a cable graft (Figure 42-16). The sole advantage of a greater auricular graft over a sural graft is in situations when it can be harvested via the same incision for another procedure, such as the repair of an extraoral mandibular fracture or management of pathology. The basic premise with graft repair is that the nerve graft will supply the Schwann cell conduit sheaths and the growth factors necessary to support and encourage axonal sprouting through the graft toward the target site. In addition, there are currently available FDA-approved human allogeneic nerve grafts from cadaveric donors that may be used for repair of IAN and LN discontinuties; early studies show promising results. This allograft is avialble in various lengths and diameters that can be used for the trigeminal system. Although the allograft is decellularized, it mainatins certain neurotrophic and neurotropic factors such as basement membrane laminin.

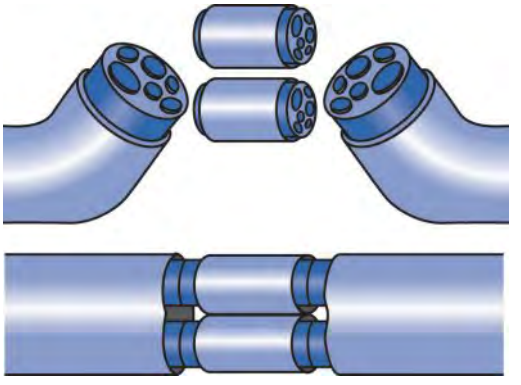


FIGURE 42-16. Greater auricular nerve cable graft.

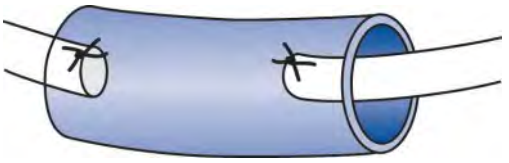


FIGURE 42-17. Entubulation (conduit) nerve repair.

Entubulation Techniques

In an attempt to avoid donor site morbidity, a variety of entubulation techniques have been proposed to create conduits to support nerve regeneration (Figure 42-17). These conduits include both autogenous and alloplastic materials (Table 42-11). The autogenous options include vein,[71–73] collagen,[74,75] and muscle grafts.[76] Alloplastic materials include polyglycolic acid,[77] polymeric silicone,[78] and expanded polytetrafluoroethylene.[79–82] It appears that the use of these alloplastic materials has a high success in the animal model, but poor human clinical outcomes. Perhaps this is due to the fact that these materials have no inherent strength to prevent compression during the postoperative period, although newer bioabsorbable synthetic materials for nerve conduits are under investigation.

POSTSURGICAL MANAGEMENT

In the majority of cases, patients experience a variable period of complete anesthesia after nerve repair. In general, the nerve regeneration process progresses at approximately 1 mm/day (~3 cm/mo) from the cell body to the target site. For example, with a direct IAN repair, the approximate distance from the trigeminal ganglion to the lower lip and chin is nearly 10 cm; therefore, complete nerve regeneration of an IAN repair would take about 100 days, or 12 weeks, after microneurosurgical repair. With graft or conduit indirect repair, the time frame is lengthened owing to slowed regeneration through the graft site, and recovery is variable. As mentioned, the use of sensory reeducation exercises ("biofeedback") in the postoperative period may improve or accelerate neurosensory recovery. For the LN or IAN, this may simply involve self-performance of a two-point discrimination or brushstroke directional discrimination test while looking in a mirror, using the uninvolved side of the tongue or lower lip and chin as the control site in order to quantify normalcy. It should be remembered that a poor outcome after attempted microneurosurgery may preclude future surgical options; therefore, the best chance for microneurosurgical success occurs at the first (and most likely, last) surgical intervention.

TABLE 42-11. **Materials for Entubulation (Conduit) Repair**

Autogenous Materials
Collagen
Muscle
Fascia
Vein
Alloplastic Materials
Polyglycolic acid
Polyester
PTFE
ePTFE
Silicone, polymeric silicone
Allogeneic Materials
Cadaveric nerve allograft

ePTFE = expanded polytetrafluoroethylene; PTFE = polytetrafluoroethylene.

MEDICOLEGAL ISSUES

Oral and maxillofacial surgeons recognize the potential medicolegal issues related to nerve injuries that occur secondary to third molar removal, and these adverse outcomes account for a large proportion of the malpractice claims in this country and abroad.[83] Based on the information contained in this chapter and recent trends in liability defense, all oral and maxillofacial surgeons should have a minimum understanding of the diagnosis and management of nerve injuries according to the so-called legal parameters of care, with which the legal profession is familiar.[84] These are summarized as follows:

- Spontaneous sensory recovery occurs in most, but not all patients. It is difficult to predict early, it may not be "complete," and it may not be to the patient's satisfaction. Nerves in soft tissue (LN) have a lower rate of spontaneous regeneration than do those in bony canals (IAN).
- All nerve injuries should be documented and evaluated with a history, examination, and neurosensory testing (objective and subjective). The injury should be classified (Seddon or Sunderland). In cases of observed or known nerve injury, prompt referral for microsurgery provides the best opportunity for sensory recovery.
- Repeat examinations at frequent intervals may be necessary. Patients should be followed up for at least 1 month. Complete recovery in 1 month indicates neurapraxia, and no further treatment is indicated. Neurosensory dysfunction that lasts longer than 1 month indicates a higher-grade injury with uncertain spontaneous neurosensory recovery. Microneurosurgical consultation should be considered.
- Nerve injuries that show improvement (objective and/or subjective) may be followed up expectantly. Once improvement stops for a period of time, it usually does not begin again.
- Most nerve injuries resolve within 3 to 9 months, but *only* if improvement begins before 3 months. Patients who are anesthetic at 3 months usually do not achieve significant neurosensory recovery. Prompt microsurgery is usually indicated.
- Patients with partial sensory loss and/or painful sensations *that they find unacceptable* should be considered for microsurgery if objective and subjective findings have not improved or returned to normal by 4 months. Microsurgical delay decreases the chance of success because

progressive distal nerve degeneration and/or the development of a central pain syndrome occur.

- Some painful neuropathies may be managed nonsurgically under the supervision of a microneurosurgeon or other experienced individual (e.g., neurologist).
- Angry uninformed patients with nerve injuries are less likely to improve with any treatment, surgical or nonsurgical. A discussion regarding options and the risk of nerve injury should be provided so that the patient can give informed consent. Local anesthetic injections carry a risk of nerve injury.
- Early surgical intervention (i.e., at 3–4 mo) is *more likely* to produce neurosensory improvement than is late intervention. Surgery delayed beyond 12 months is seriously compromised by distal nerve degeneration and the development of chronic pain syndromes.
- Surgery is *more likely* to improve responses to objective sensory testing and/or to reduce functional impairment than it is to reduce pain or subjective feelings of numbness.

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Appendix
Nerve Terminology Review*

allodynia: Pain due to a stimulus that does not normally provoke pain.

analgesia: Absence of pain in the presence of stimulation that would normally be painful.

anesthesia: Absence of any sensation in the presence of stimulation that would normally be painful or nonpainful.

anesthesia dolorosa: Pain in an area or a region that is anesthetic.

atypical neuralgia: A pain syndrome that is not typical of classic nontraumatic trigeminal neuralgia.

axonotmesis (Seddon) or second- through fourth-degree injuries (Sunderland): Nerve injury characterized by axonal injury with subsequent degeneration and regeneration. *Third-degree injury:* Characterized by axonal damage and a breach of the endoneurial sheath, resulting in intrafascicular disorganization. The perineurium and epineurium remain intact. The mechanism is typically traction or compression. *Fourth-degree injury:* Characterized by disruption of the axon, endoneurium, and perineurium, resulting in severe fascicular disorganization. The epineurium remains intact. Possible mechanisms include traction, compression, injection injury, and chemical injury.

causalgia: Burning pain, allodynia, and hyperpathia after a partial injury of a nerve.

central pain: Pain associated with a primary central nervous system lesion (spinal cord or brain trauma, vascular lesions, tumors).

chemoreceptor: A peripheral nerve receptor that is responsive to chemicals, including catecholamines.

deafferentation pain: Pain occurring in a region of partial or complete traumatic nerve injury in which there is interruption of afferent impulses by destruction of the afferent pathway or other mechanism.

dysesthesia: An abnormal sensation, either spontaneous or evoked, that is unpleasant. All dysesthesias are a type of paresthesia but not all paresthesias are dysesthesias.

endoneurium: A connective tissue sheath surrounding individual nerve fibers and their Schwann cells.

epineurium: A loose connective tissue sheath that encases the entire nerve trunk.

fascicle: A bundle of nerve fibers encased by the perineurium.

hyperalgesia: An increased response to a stimulus that is normally painful.

hyperesthesia: An increased sensitivity to stimulation, excluding the special senses (i.e., seeing, hearing, taste, touch, and smell).

hyperpathia: A painful syndrome characterized by increased reaction to a stimulus, especially a repetitive stimulus. The threshold is increased as well.

hypoalgesia: Diminished pain in response to a normally painful stimulus.

hypoesthesia: Decreased sensitivity to stimulation, excluding the special senses (i.e., seeing, hearing, taste, touch, and smell).

mechanoreceptor: A peripheral nerve receptor preferentially activated by physical deformation from pressure and associated with large sensory axons.

mesoneurium: A connective tissue sheath, analogous to the mesentery of the intestine, that suspends the nerve trunk within soft tissue.

*Adapted from LaBanc JP, Gregg JM. Glossary. Trigeminal nerve injury: diagnosis and management. Oral Maxillofac Surg Clin North Am 1992;4:563.

monofascicular pattern: Characteristic cross-section of a nerve containing one large fascicle.

neuralgia: Pain in the distribution of a nerve or nerves.

neurapraxia (Seddon) or first-degree injury (Sunderland): Nerve injury characterized by a conduction block, with rapid and virtually complete return of sensation or function and no axonal degeneration.

neuritis: A special case of neuropathy now reserved for inflammatory processes affecting nerves.

neurolysis: The surgical separation of adhesions from an injured peripheral nerve.

neuroma: An anatomically disorganized mass of collagen and nerve fascicles, and a functionally abnormal region of a peripheral nerve resulting from a failed regeneration after injury.

neuropathy: A disturbance of function or a pathologic change in a nerve.

neurotization: Axonal invasion of the distal nerve trunk.

neurotmesis (Seddon) or fifth-degree injury (Sunderland): Nerve injury characterized by severe disruption of the connective tissue components of the nerve trunk, with compromised sensory and functional recovery. *Fifth-degree injury:* Characterized by complete disruption of the nerve trunk with considerable tissue loss. Possible mechanisms include laceration, avulsion, and chemical injury.

nociceptor: A receptor preferentially sensitive to a noxious stimulus or to a stimulus that would become noxious if prolonged.

oligofascicular pattern: Characteristic cross-section of a nerve containing 2 to 10 rather large fascicles.

paresthesia: An abnormal sensation, either spontaneous or evoked, that is not unpleasant. A global term used to encompass all types of nerve injuries.

perineurium: A thick connective tissue sheath surrounding fascicles.

polyfascicular pattern: Characteristic cross-section of a nerve containing greater than 10 fascicles of different sizes, with a prevalence of small fascicles.

protopathia: The inability to distinguish between two different modes of sensation, such as a painful and a nonpainful pinprick.

sympathetically mediated pain: A general term that refers to a family of related disorders including causalgia, reflex sympathetic dystrophy, minor causalgia, Sudeck's atrophy, and postherpetic neuralgia, which may be sympathetically maintained.

synesthesia: A sensation felt in one part of the body when another part is stimulated.

wallerian degeneration: The distal degeneration of the axon and its myelin sheath following injury.

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CHAPTER

Cleft Lip and Palate

Bernard J. Costello, DMD, MD, and Ramon L. Ruiz, DMD, MD

The comprehensive treatment of cleft lip and palate deformities requires thoughtful consideration of the anatomic complexities of the deformity and the delicate balance between intervention and growth. Comprehensive and coordinated care from infancy through adolescence is essential in order to achieve an ideal outcome, and surgeons with formal training and experience in all of the phases of care must be actively involved in the planning and treatment.[1–3] Specific goals of surgical care for children born with cleft lip and palate include the following:

- Normalized esthetic appearance of the lip and nose
- Intact primary and secondary palate
- Normal speech, language, and hearing
- Nasal airway patency
- Class I occlusion with normal masticatory function
- Good dental and periodontal health
- Normal psychosocial development

Successful management of the child born with a cleft lip and palate requires coordinated care provided by a number of different specialties including oral/maxillofacial surgery, otolaryngology, genetics/dysmorphology, speech/language pathology, orthodontics, prosthodontics, and others.[4] In most cases care of patients with congenital clefts has become a subspecialty area of clinical practice within these different professions. In addition to surgery for cleft repair, treatment plans routinely involve multiple treatment interventions to achieve the above-stated goals. Because care is provided over the entire course of the child's development, long-term follow-up is critical under the care of these different health care providers. The formation of interdisciplinary cleft palate teams has served two key objectives of successful cleft care: (1) coordinated care provided by all of the necessary disciplines, and (2) continuity of care with close interval follow-up of the patient throughout periods of active growth and ongoing stages of reconstruction. The best outcomes are achieved when the team's care is centered on the patient, family, and community rather than a particular surgeon, specialty, or hospital. The idea of having an objective team that does not revolve around the desires of one particular individual or discipline is sometimes impeded by competitive interactions between surgical specialties. Historic battles over surgical domains between surgical specialties and economic factors contribute to these conflicts and negatively affect the work of the team. Healthy team dynamic and optimal patient care are achieved when all members are active participants, when team protocols and referral patterns are equitable and based on the surgeons' formal training and experience instead of specialty identity, and when the needs of the child are placed above the needs of the team.

This chapter presents an overview of the concepts for reconstruction of the cleft lip and palate deformity. The surgical reconstruction of clefts requires that the surgeon undertaking this important work maintain a cognitive understanding of the complex malformation itself, the varied operative techniques employed, facial growth considerations, and the psychosocial health of the patient and family. The objectives of this chapter will be to present the overall staged reconstructive approach for repair of cleft lip and palate from infancy through the time of skeletal maturity, as well as a focused discussion of the specific surgical procedures involved in primary cleft lip and palate repair. Secondary revision procedures, bone graft reconstruction of the cleft maxilla, and orthognathic surgery for cleft-related dysmorphology are discussed in Chapter 42, "Reconstruction of the Alveolar Cleft," Chapter 43, "Reconstruction of Cleft Lip and Palate: Secondary Procedures," and Chapter 60, "Orthognathic Surgery in the Patient with Cleft Palate."

HISTORY OF CLEFT LIP AND PALATE REPAIR

The history of cleft lip and palate care has always been closely linked to dentistry and oral and maxillofacial surgery. The birth and roots of what is now the American Cleft Palate-Craniofacial Association are strongly rooted in dentistry.

The first documented cleft lip repair was performed in ‘ c 390 on a patient who later became the Governor General of several regions in China, although nothing is known about the actual surgeon.[5,6] Jehan Yperman is believed to have been the first to describe unilateral and bilateral cleft lip repair.[6,7] The first diagrammatic representation of cleft lip repair and cleft palate obturator use is credited to Ambrose Pare in the fourteenth century.[6,8] Much later the first documented successful cleft palate repair was performed by a dentist, Le Monnier, in 1766 in Paris.[6,9] The concepts of cleft lip and palate repair have evolved from straight line repairs to a variety of techniques using various cutbacks, triangles, and Z-plasties.[6–14] During the 1950s, Asensio, an oral and maxillofacial surgeon from Guatemala, developed a novel technique for cleft lip repair, which involved the rotation of the philtral segment inferiorly and advancement of the lateral segment medially using a quadrangular flap. Although he used this approach in Guatemala throughout the 1950s, he did not report it until much later.[15,16] Ralph Millard of Miami described his classic rotational-advancement technique in the mid-1950s, and his concepts changed cleft repair forever.[17,18] Millard is credited with perhaps the most important technical development related to cleft lip repair, and today the majority of surgeons use his original technique or some close modification of it.

In the mid-nineteenth century, Hullihen, recognized as the father of American oral and maxillofacial surgery, published a treatise on comprehensive care of cleft lip and palate deformities.[19] Another pioneer, Truman Brophy, was the professor of oral surgery and dean of the Chicago College of Dentistry and contributed greatly to the care of many patients with clefts. Brophy published a text detailing his experiences with the management of various malformations of the mouth and their surgical repairs including the details of cleft repair.[20] One of his pupils was Chalmers Lyons who started a residency program in oral surgery at the University of Michigan in 1917.[20] Lyons developed the largest cleft practice in America and contributed extensively to the literature.[20]

Many of the concepts related to interdisciplinary care with a cleft palate team care were introduced by Robert Ivy, an oral and maxillofacial surgeon who later became dually qualified in plastic surgery.[20] Robert Ivy trained both in dentistry and medicine at the University of Pennsylvania. After his training in dentistry, Ivy further developed his interests in maxillofacial surgery as an assistant to his uncle, Matthew Cryer, who was a professor in oral surgery at the University of Pennsylvania. Robert Ivy became interested in clefts during his training as the first dental intern at Philadelphia General Hospital at the University of Pennsylvania. His interests in maxillofacial injury led him to serve in France in World War I as an assistant to Vilray Blair. After the war Ivy and Blair’s collaboration resulted in two landmark publications by Ivy, *Essentials of Oral Surgery* and *Fractures of the Jaws*. Through work with his state representatives in Harrisburg, Pennsylvania, he was able to start the very first cleft palate clinics in Lancaster, Pittsburgh, Philadelphia, Erie, and Scranton that provided interdisciplinary care to children for cleft lip and palate deformities. When Reed Dingman put forth a resolution of the American Society of Maxillofacial Surgeons condemning oral and maxillofacial surgeons practicing in the hospital setting, Ivy resigned his membership and sent a letter of protest to the organization that he helped build in support of his dental colleagues.[20]

In the 1950s the concept of primary or early bone grafting of the cleft maxillary defect was introduced by Schmid.[21] Although the concept was initially met with enthusiasm from a number of surgeons, primary bone grafting was eventually abandoned due to unfavorable outcomes. During the decades that followed, the negative skeletal, dental, and growth-related consequences of primary bone grafting became better understood.[22,23] During the early 1970s oral and maxillofacial surgeons Boyne and Sands were the first to publish their favorable outcomes using autogenous particulate bone grafts for reconstruction of the cleft maxilla/alveolus later in childhood during the mixed dentition rather than earlier in life.[24] Although their work and results represented a landmark discovery in the field of cleft reconstruction, cleft palate teams were slow to integrate his approach into their treatment protocols because of the negative associations that lingered following the days of primary bone grafting. Today their principles of secondary bone grafting represent the standard approach for almost all of the world’s cleft centers.[24,25]

Orthognathic reconstruction of the patient with cleft deformities has been discussed by many authors.[26–36] Early techniques limited some surgeons’ options to procedures centered on mandibular setback.[37] During the 1970s the use of total maxillary osteotomy was pioneered by Bell.[38] His novel ideas provided oral and maxillofacial surgeons with an understanding of the biologic basis for maxillary osteotomy, described the vascular supply that allowed the procedures to be performed safely, and as a result incorporated the Le Fort I osteotomy into modern-day practice.[38] Since that time a number of technical refinements have been described for use of the Le Fort I osteotomy specifically in the cleft patient. Much of this work has been done by two of Bell’s former pupils, Fonseca and Turvey, who went on to make substantial contributions to the skeletal reconstruction of patients with clefts.[39] Another dual qualified oral and maxillofacial surgeon, Posnick, has published the most complete descriptions of surgical technique modifications for patients undergoing midfacial advancement in the absence of prior bone graft reconstruction and his extensive experiences with the long-

term stability of midfacial advancement after correction of various types of cleft deformities with orthognathic techniques.[26–30] Distraction osteogenesis has gained recent popularity for correction of midfacial hypoplasia but has yet to show significant advantages over traditional techniques for the majority of patients.[32,33,40–42]

Comprehensive and coordinated care has become more prevalent across the world, involving many different types of specialty care for children with clefts. Posnick has provided the most comprehensive, succinct, and evidenced-based discussions on the topic of cleft lip and palate reconstruction from infancy through adolescence.[26] These efforts as well as craniofacial training programs associated with oral and maxillofacial surgery have helped to solidify the role of oral and maxillofacial surgery in the comprehensive care of patients with clefts.

EMBRYOLOGY

To understand the goals of lip and palate repair from an anatomic standpoint the cleft surgeon must have an appreciation for the failure of embryogenesis that results in clefting. There are critical points in the development of the fetus when the fusion of various prominences creates continuity and form to the lip, nose, and palate. Anomalies occur when the normal developmental process is disturbed between these components. Each of these prominences is made up of ectomesenchyme derived from neural crest tissue of the mesencephalon and rhombencephalon. Mesoderm is also present within these prominences as mesenchymal tissue. The prescribed destiny of each of these cells and tissues is controlled by various genes to alter the migration, development, and apoptosis and form the normal facial tissues of the fetus. At the molecular level there are many interdependent factors such as signal transduction, mechanical stress, and growth factor production that affect the development of these tissues. Currently only portions of this complex interplay of growth, development, and apoptosis are clear.

At approximately 6 weeks of human embryologic development the median nasal prominence fuses with the lateral nasal prominences and maxillary prominences to form the base of the nose, nostrils, and upper lip. The confluence of these anterior components becomes the primary palate. When this mechanism fails, clefts of the lips and/or maxilla occur. At approximately 8 weeks the palatal shelves elevate and fuse with the septum to form the intact secondary palate. When one palatal shelf fails to fuse with the other components, then a unilateral cleft of the secondary palate occurs. If both of the palatal shelves fail to fuse with each other and the midline septum, then a bilateral cleft of the palate occurs.

Fusion occurs when programmed cell death (apoptosis) occurs at the edges of the palatal shelves. The ectodermal component disintegrates and the mesenchyme fuses to form the intact palate. Soon after this the anterior primary palate fuses with the secondary palate and ossification occurs. At any point, if failure of fusion occurs with any of the above components, a cleft will occur of the primary and/or secondary palates. Clefts may be complete or incomplete based on the degree of this failure of fusion.

GENETICS AND ETIOLOGY

Clefts of the upper lip and palate are the most common major congenital craniofacial abnormality and are present in approximately 1 in 700 live births.[43] Although inheritance may play a role, cleft lip and palate is not considered a single-gene disease. Instead clefts are thought to be of a multifactorial etiology with a number of potential contributing factors. These factors may include chemical exposures, radiation, maternal hypoxia, teratogenic drugs, nutritional deficiencies, physical obstruction, or genetic influences. One prevailing theory relates the process of clefting as a threshold in which multiple factors come together to raise the individual above a threshold at which time the mechanism of fusion fails.[44,45] Recently multiple genes have been implicated in the etiology of clefting.[46–48] Some of these genes include the *MSX*, *LHX*, *goosecoid*, and *DLX* genes. Additional disturbances in growth factors or their receptors that may be involved in the failure of fusion include fibroblast growth factor, transforming growth factor-, platelet-derived growth factor, and epidermal growth factor.

Clefts of the lip occur more commonly in males than in females.[49] In addition left-sided cleft lips are more common than right-sided cleft lips, and unilateral cleft lips are more common than the bilateral cleft of the lip.[50] Bilateral clefts of the lip are most often associated with clefting of both the primary and secondary palates. Cleft palate alone is seen in approximately 1 in 2,000 live births and this incidence is similar in all racial groups.[51] Significant differences in the prevalence of clefts exist when specific ethnic/racial populations are examined. For example, African Americans have a birth prevalence that is less common than the total population, but Asians tend to have a higher prevalence.

In the majority of cases unilateral cleft lip and palate is an isolated nonsyndromic birth defect that is not associated with any other major anomalies.[43,52,53] By comparison a much greater proportion of patients with an isolated cleft palate will have an associated syndrome or sequence.[43,53] Some of the more common syndromes seen in this group include Stickler's, Van der Woude's, or DiGeorge syndromes. It is important to identify the diagnosis early, as functional issues may arise early in life and go unnoticed. For example, patients with an isolated cleft palate should be evaluated early by an experienced pediatric ophthalmologist to evaluate the possibility of Stickler's syndrome. Patients with Stickler's syndrome may have ocular abnormalities that lead to retinal detachment. In an otherwise healthy-appearing child these findings may be difficult to diagnose and so early visual loss



FIGURE 43-1. Cleft lips come in a variety of configurations, such that each repair must be customized to establish the most normal morphology. **A,** Microform left unilateral cleft lip only, not requiring primary repair. **B,** Minor left incomplete unilateral cleft lip only. **C,** Left incomplete unilateral cleft lip and palate with a Simonart's band. **D,** Wide left complete unilateral cleft lip and palate.

may go unnoticed. In many cases long-term genetics follow-up is necessary to make a definitive diagnosis and to provide genetic counseling.

The chances of a recurrence of clefting within a family are dependent on many factors, including family history, severity, gender, degree of relationship to the affected individual, and the expression of a syndrome. Predicting the inheritance patterns of families who have a history of cleft lip and/or palate can be complicated. A skilled geneticist/dysmorphologist is best equipped to make these determinations based on pedigree analysis and genetic testing. Since most clefts are sporadic the chances of a family having another child with a cleft after having a child with a unilateral cleft lip and palate in which there was no family history of clefting is approximately 2 to 4%. The chances are higher if additional family history is present or if the cleft is bilateral.[54,55] The nature of any genetic influence will have an effect on the presence of a cleft. Such is the case in patients with autosomal dominant syndromes such as Stickler syndrome where 50% of the children may express the syndrome if one of the parents carries the altered gene.

CLASSIFICATION

The typical classification system used clinically to describe standard clefts of the lip and palate is based on careful anatomic description. Clefts can be unilateral or bilateral; microform, incomplete, or complete; and may involve the lip, nose, primary palate, and/or secondary palates (Figure 43-1). The presentation of clefts is extremely variable, and the individual repairs are custom-tailored to achieve the best symmetry and balance. More severe facial clefting is most commonly described using Tessier's orbitocentric system of numbering (Figure 43-2).[56] Other systems exist that are based on embryologic fusion planes, but these are cumbersome to use in routine clinical practice.[57]

PRENATAL COUNSELING

Recent advances in ultrasound imaging have revolutionized prenatal care and maternal-fetal medicine. Currently ultrasound images of clefts of the lip can be visualized as early as 16 weeks.[58–60] Diagnostic images of the palate are more difficult to acquire, making the correct prenatal diagnosis of a cleft palate less predictable. Palatal structures may be visualized using sagittal and coronal views, but this currently requires the very latest technology and a skilled ultrasonographer with experience performing this type of study.

When the diagnosis of cleft lip is made during pregnancy the family can then be referred to an experienced surgeon for a prenatal discussion. A prenatal consultation provides an excellent opportunity to explain the diagnosis, review the different stages of cleft lip and palate reconstruction that may be necessary, and prepare the parents for practical considerations such as feeding of a child with a cleft palate. This gives the family the opportunity to ask questions, calm fears, and learn about feeding techniques that will be important during the first week of life for their baby. Parents are empowered with this new knowledge, and the preparations made during a prenatal consultation allow them to anticipate the delivery of their baby with a greater comfort level regarding the necessary care of the child during the early postnatal period. The family is then referred to a cleft and craniofacial team in order to undergo a more thorough interdisciplinary approach.

Critical to this process is consultation with a geneticist/dysmorphologist to further discuss the issues associated with the birth and the possibility of other associated deformities. Additional testing may be warranted to evaluate the possibility of associated deformities, syndromes, or sequences that could affect the birthing process. Exceptionally skilled ultrasonographers can visualize airway development and other abnormalities that may require early intervention with fetal

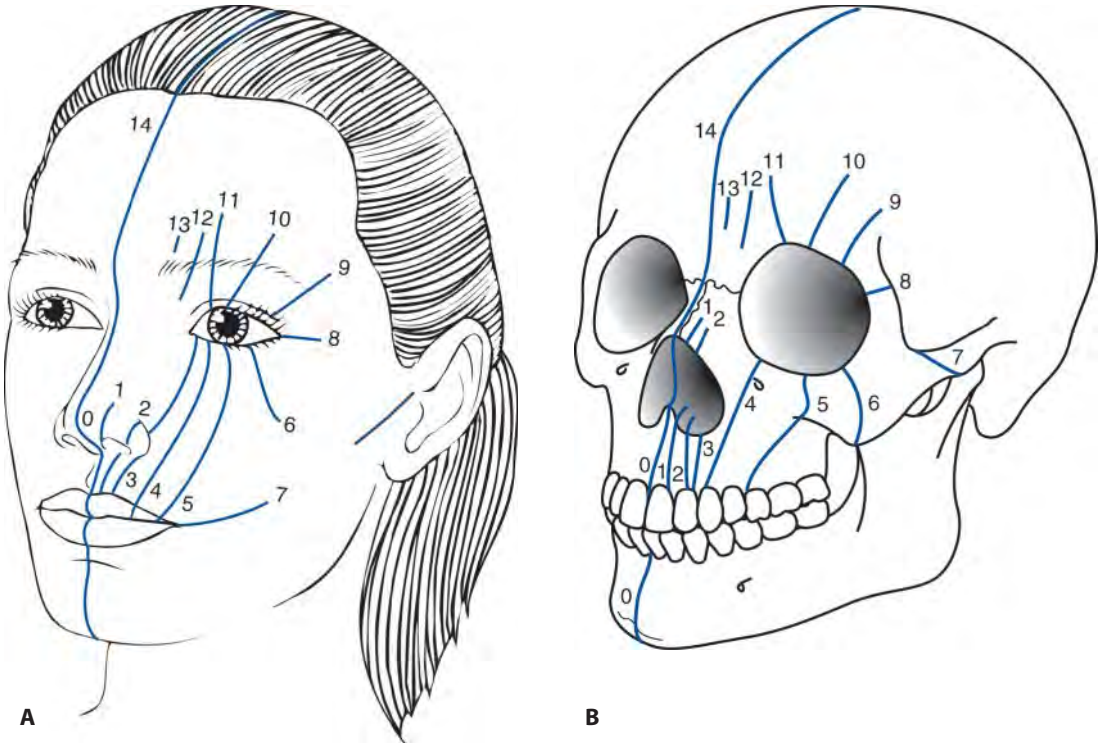


FIGURE 43-2. **A** and **B,** Complex facial clefts can be classified based on Tessier's original orbitocentric system of numbering. Clefts may involve all tissue planes including skin, mucosa, bone, teeth, muscle, brain, peripheral nerve, and other specialized tissues.

surgery, exit procedures, extracorporeal membrane oxygenation, or surgical airway management (tracheotomy) at the time of delivery.

In some medical centers fetal diagnosis and treatment teams are in place to deal with issues associated with various deformities diagnosed in the prenatal period. These teams foster a cohesive environment where information is exchanged through consultation. Much like in the environment of a cleft and craniofacial team, families can get the best information available to consider their child's treatment decisions using an interdisciplinary care model that is patient (mother and fetus), family, and community oriented.

FEEDING CONCERNS

Children born with isolated cleft lip can feed quite well and even have the opportunity to breastfeed in most instances. However, infants with cleft palate can have difficulty feeding due to the inability to form an adequate seal between the tongue and palate for creation of sufficient negative pressure to suck fluid from a bottle. Nasal regurgitation and inefficient handling of secretions and foodstuffs may also be observed during early development. Specialized nipples and bottles are necessary to improve feeding immediately after birth. The most useful devices combine oversized nipples with reservoir spaces and large openings, a squeezable bottle to push fluid into the nipple assembly, and a one-way valve that allows the bolus of fluid to pass from the bottle to the nipple only in order to minimize the amount of work the child must perform to feed. These include a variety of nipples with reservoirs that collect a variable volume of liquid that can be expressed more easily when sucking is inefficient or not possible. Bottles that can be squeezed to allow for manual flow of liquid to the infant are helpful for improving feeding. No single bottle and nipple combination tends to work better than another, but trials with a variety of types using different techniques are helpful in optimizing feeding early in life. Close attention to weight gain is necessary for these children. Generally, in 24 hours each infant should have approximately 2 to 3 ounces of milk for each pound of weight. Feeding sessions should last no longer than 35 minutes as longer sessions are fatiguing and burn more calories than the baby can consume. Infants should be weighed at least weekly using the same scale, preferably at their pediatrician's office.

The subject of breast-feeding an infant with a cleft palate is controversial, with some practitioners encouraging the practice and others strongly opposed to it. There are clear advantages to breast-feeding a newborn, including passive immunologic contribution of the mother to the child in the form of secretory immunoglobulin A and an experience that enhances bonding between the mother and child during such a critical period.[61,62] At the same time the infant's inability to create negative oral pressure will often make successful nursing difficult, if not impossible. It is relatively common to

encounter an exclusively breast-fed infant with severe dehydration and failure to thrive secondary to these difficulties. This is especially a concern in infants that have a wide cleft of the secondary palate, where breast-feeding may not be possible. The authors' approach with regard to breast-feeding in the presence of a cleft palate is to use a combined protocol that includes intermittent feeding with the use of a specialized bottle (as described above) and attempts at nursing. Breast milk may be pumped for use with the specialized nipple and bottle that will provide the nutritional and immunologic benefits desired. This also allows the parents to keep a more quantitative record of how many ounces have been ingested over the course of the day since this is normally difficult with breast-feeding alone. At the same time the mother and baby are not deprived of an opportunity to incorporate breast-feeding into the daily regimen. This approach obviously requires rigorous documentation of the child's weight, consultation with a lactation consultant and infant feeding specialist, and frequent follow-up evaluations through the surgeon and/or pediatrician.

TREATMENT PLANNING AND TIMING: OVERVIEW

The timing of cleft lip and palate repair is controversial. Despite a number of meaningful advancements in the care of patients with cleft lip and palate, a lack of consensus exists regarding the timing and specific techniques used during each stage of cleft reconstruction. Surgeons must continue to carefully balance the functional needs, esthetic concerns, and the issue of ongoing growth when deciding how and when to intervene. In no other type of surgical problem is the issue of early surgery's effect on growth more apparent than in the treatment of cleft lip and palate deformities. The decision to surgically manipulate the tissues of the growing child should not be made lightly and should take into account the possible growth restriction that can occur with early surgery. Nevertheless many patients with congenital deformities will benefit from surgical intervention based on functional or psychosocial reasons. Understanding the growth and development of the craniofacial skeleton is critical to the treatment planning process.[33] In many cases waiting for a greater degree of growth to occur is advantageous unless compelling functional or esthetic issues are present that can not or should not wait.

Due to many different treatment philosophies the timing of treatment interventions is considerably variable amongst cleft centers. Therefore, it is difficult to produce a timing regimen that everyone agrees on. Each stage of surgical reconstruction and the suggested timing based on the patient's age are presented in Table 43-1. Special considerations may alter the sequencing or timing of the various procedures based on individual functional or esthetic needs.

Cleft lip repair is generally undertaken at some point after 10 weeks of age. One advantage of waiting until the child is 10 to 12 weeks of age is that it allows a complete medical evaluation of the patient so that any associated congenital defects affecting other organ systems (eg, cardiac or renal anomalies) may be uncovered. The surgical procedure itself may be easier when the child is slightly larger and the anatomic landmarks more prominent and well defined. Historically the anesthetic risk-related data suggested that the safest time period for surgery in this population of infants could be outlined simply by using the "rule of 10's." This referred to the idea of delaying lip repair until the child was at least 10 weeks old, 10 pounds in weight, and with a minimum hemoglobin value of 10 dL/mg.[63,64] Today more sophisticated pediatric techniques, advances in intraoperative monitoring, and improved anesthetic agents have all resulted in the ability to provide safe general anesthesia much earlier in life.[65] Despite the ability to provide safe anesthesia earlier in life, there is no measurable benefit to performing lip repair prior to 3 months of age.[64,66,67] Some surgeons have advocated that lip repair be carried out in the first days of infancy based on the idea of capitalizing on early "fetal-like" healing. Unfortunately these hoped-for benefits have not been observed, and problems with excessive scarring and less favorable outcomes have been encountered instead.[68–70] Children may have more scarring at this early age, and their tissues are smaller and more difficult to manipulate. Consequently the esthetic outcomes may be worse if surgery is performed at an earlier age, and since there are no clear benefits to earlier repair the recommendations for repair stand at approximately 3 months of age.

Cleft palate repair is usually performed at approximately 9 to 18 months of age. In deciding the timing of repair the surgeon must consider the delicate balance between facial growth restriction after early surgery and speech development that requires an intact palate. Most children will require an intact palate to produce certain speech sounds by 18 months of age. If developmental delay is present and speech will not likely develop until later, then the repair can be delayed further. There is little evidence to suggest any benefit to palate repair prior to 9 months of age.[71–73] Repairs prior

TABLE 43-1. **Staged Reconstruction of Cleft Lip and Palate Deformities**

Procedure	Timing
Cleft lip repair	After 10 weeks
Cleft palate repair	9–18 months
Pharyngeal flap or pharyngoplasty	3–5 years or later based on speech development
Maxillary/alveolar reconstruction with bone grafting	6–9 years based on dental development
Cleft orthognathic surgery	14–16 years in girls, 16–18 years in boys
Cleft rhinoplasty	After age 5 years but preferably at skeletal maturity; after orthognathic surgery when possible
Cleft lip revision	Anytime once initial remodeling and scar maturation is complete but best performed after age 5 years

to this time are associated with a much higher incidence of maxillary hypoplasia later in life and show no improvements in speech. For these reasons most surgeons will perform primary palate repair at approximately 9 to 12 months of age.

As the child continues to develop, approximately 20% of children will have inadequate closure of the velopharyngeal mechanism (velopharyngeal insufficiency or VPI), and this may produce hypernasal speech.[74] These children are usually diagnosed at 3 to 5 years of age when a detailed speech examination can be obtained by a skilled speech pathologist familiar with clefts. When VPI is shown to be consistent and due to a definable anatomic defect, surgery is often helpful in correcting this problem. A pharyngeal flap or sphincter pharyngoplasty may be used to treat VPI, with the goal of improving closure between the oral and nasal cavities and reducing nasal air escape during the production of certain sounds. The details of assessment, diagnosis, and treatment of VPI associated with cleft palate are discussed in Chapter 43, "Reconstruction of Cleft Lip and Palate: Secondary Procedures." Approximately 75% of patients with any type of cleft will present with clefting of the maxilla and alveolus.[24–26] Bone graft reconstruction of this site is performed during the mixed dentition prior to the eruption of the permanent canine and/or the permanent lateral incisor. The timing of this procedure is based on dental development and not chronologic age. Based on work by Boyne and Sands, most surgeons reconstruct this area during the mixed dentition prior to eruption of the permanent canine. Earlier reconstruction of this area has been associated with a high degree of maxillary growth restriction requiring orthognathic correction later in life in a much higher percentage of patients.[22,24] The gold standard for reconstruction in this area is autogenous bone from the anterior iliac crest. Cranial bone, rib, tibia, symphysis of the mandible, zygoma, and allogeneic bone have all been studied, but none have been shown to be appreciably better than the iliac crest.[26,75,76]

Orthognathic reconstruction of maxillary and mandibular discrepancies is performed at 14 to 18 years of age based on individual growth characteristics.[26–36,38] This is done in conjunction with orthodontics prior to and after surgery. However, in some cases of severe maxillary hypoplasia, early Le Fort I osteotomy may be performed to optimize facial esthetics and occlusion with the supposition that revision osteotomies will likely be necessary. These early osteotomies may complicate later treatment. Early orthognathic correction is reserved for the most severe dysmorphology, and in most cases the authors prefer standard orthognathic techniques.[31–33] Attempts at using distraction osteogenesis have been associated with a higher complication rate than with standard orthognathic techniques.[32,42,77] Orthognathic correction of the deformities associated with cleft lip and palate defects is discussed in Chapter 60, "Orthognathic Surgery in the Patient with Cleft Palate."

As with the timing of other interventions, lip and nasal revision is best reserved until after the majority of growth is complete. Most of the lip and nasal growth is complete after age 5 years. Lip revision can be considered prior to school age at about 5 years of age. However, this may be performed earlier if the deformity is severe. Nasal revision is performed after age 5 years as most of the nasal growth is also complete by this time. If orthognathic reconstruction is likely, then rhinoplasty is usually best performed after orthognathic surgery as maxillary advancement improves many characteristics of nasal support. However, when nasal deformity is particularly severe, rhinoplasty can be considered earlier even if orthognathic surgery is expected. Multiple early revisions of the lip or nose should be avoided so that excess scarring does not potentially impair ongoing growth. Secondary revisions of cleft lip and palate deformities are discussed in Chapter 43, "Reconstruction of Cleft Lip and Palate: Secondary Procedures."

CLEFT LIP AND PALATE REPAIR

Presurgical Taping and Presurgical Orthopedics

Facial taping with elastic devices is used for application of selective external pressure and may allow for improvement of lip and nasal position prior to the lip repair procedure. In the authors' opinions these techniques often have greater impact in cases of wide bilateral cleft lip and palate where manipulation of the premaxillary segment may make primary repair technically easier. Although one of the basic surgical tenets of wound repair is to close wounds under minimal tension, attempts at improving the arrangement of the segments using taping methods have not shown a measurable improvement.[78–80]

Some surgeons prefer presurgical orthopedic (PSO) appliances rather than lip taping to achieve the same goals.[81,82] PSO appliances are composed of a custom-made acrylic base plate that provides improved anchorage in the molding of lip, nasal, and alveolar structures during the presurgical phase of treatment (Figure 43-3). Although the use of appliances probably makes for an easier surgical repair, there

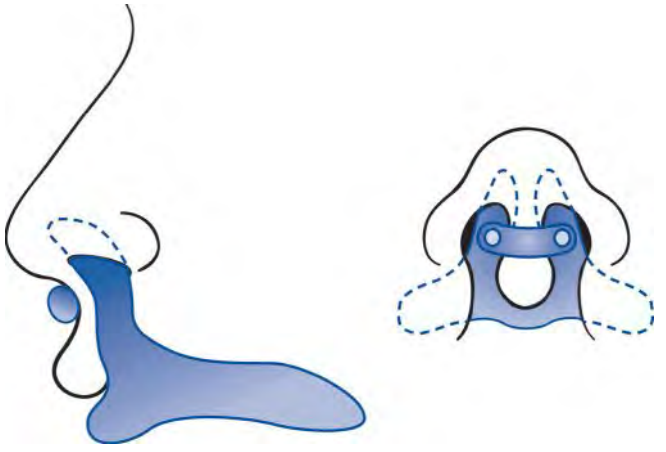


FIGURE 43-3. Frontal and lateral views of the Grayson nasoalveolar molding appliance showing the nasal projections that help to theoretically mold the nasal cartilages and maxillary segments into a more appropriate configuration prior to repair.

has been a lack of clinical evidence to demonstrate that there is any measurable improvement in esthetics of the nose or lip, dental arch relationship, tooth survival, or occlusion. Studies have looked at the dental arch relationship outcomes in patients who have infant presurgical orthopedic devices, and no improvement in dental arch relationship was seen.[83,84] Additionally no long-term improvement in speech outcome has be demonstrated in patients who had PSOs.[85] Furthermore concerns regarding potential negative consequences with these types of appliances have been raised.[86] PSOs also add significant cost and time to treatment early in the child's life. Many appliances require a general anesthetic for the initial impression used to fabricate the device. Frequent appointments are necessary for monitoring of the anatomic changes and periodic appliance adjustment.

The Latham appliance was popular for expanding and aligning the maxillary segments of the patient with a cleft palate.[87] It is a pin-retained device that is inserted into the palate with acrylic extensions onto the alveolar ridges. A screw mechanism is then used to manipulate the segments as desired. The Latham appliance has been shown to be associated with significant growth restriction of the midface when used in infancy to approximate the segments prior to definitive repair.[86] Children who have had Latham appliances have been shown to have significant midfacial growth restriction in adolescence 100% of the time whereas children who have not had the Latham appliance have midface hypoplasia 25 to 35% of the time.[42,80,86]

The nasoalveolar molding appliance has become popular with some surgeons in attempts to manipulate the segments without pin retention prior to lip and nose repair (see Figure 43-3). The appliance popularized by Grayson is adjustable by removing or adding acrylic and manipulating protrusive elements that attempt to mold the nasal cartilages. This device attempts to align the alveolar segments, lip structures, and nasal cartilages to optimize repair. Unfortunately the hoped-for advantages of this appliance have not been realized. Additionally no long-term data are available regarding growth in the craniofacial skeleton after using this protocol. The limited short-term data that are available cannot be extrapolated to determine the ultimate outcome on growth, function, or esthetics. Some surgeons use gingivoperiosteoplasty in conjunction with the PSO, using limited flaps to close the alveolus cleft during the primary repair of the lip or palate. Many surgeons who use this appliance in conjunction with their primary lip repairs will perform a gingivoperiosteoplasty in attempts to have bone form at the alveolus. This is more easily performed with the segments aligned in close proximity as the flaps are small.[82] Experiences with similar techniques in the 1960s involving primary bone grafting were poor with respect to growth.[22,23] Additionally there has been no convincing long-term objective data showing improvement in either lip or nose esthetics.

In their current state of technical refinement there is no evidence that any of the PSOs offer an improved outcome with respect to esthetics, function, or growth in patients with cleft lip and palate. Coupled with the fact that appliances are time-consuming and have a high cost of fabrication and utilization, it is difficult to advocate their uniform use. As with other interventions considered for patients with clefts, costly and unproven interventions should be avoided, although they may prove to be helpful in some select cases.[88] Hopefully, long-term data will be forthcoming and positive to help determine which patients may benefit from PSO appliance treatment.

Lip Adhesion

Some surgeons attempt to surgically approximate the segments of the cleft lip prior to definitive lip repair in an attempt to achieve a better relationship of both the lip structures and the dental arches.[89–91] This is achieved by advancing small flaps of tissue across the cleft site. While some surgeons advocate the use of this technique in wide bilateral clefts, it is rarely performed in unilateral cases. When used, the lip adhesion is usually completed at 3 months of age. In most cases this will convert a wide complete cleft into a wide incomplete cleft as the scar will eventually be excised from the cleft site recreating a similar wide deformity. The definitive lip repair is then completed 3 to 9 months later by excising the scar and reapproximating the remaining lip structures. Furthermore at the second procedure there is usually less supple tissue to work with when performing the definitive repair due to scarring. As with most endeavors in cleft surgery, repeated early interventions tend to complicate later refinements due to excessive scarring. In general adequate mobilization of the flaps in one stage will make tension-free skin closure possible in almost every case without the need for taping, presurgical orthopedic appliances, and/or lip adhesion.

Unilateral Cleft Lip Repair

Clefts of the lip and nose that are unilateral present with a high degree of variability, and thus each repair design is unique (see Figure 43-1).[26,92] The repair technique preferred by the authors for cleft lip and nasal deformities is shown in Figures 43-4 and 43-5 and is usually performed after 10 weeks of age.[17,18,26,63] The basic premise of the repair is to create a three-layered closure of skin, muscle, and mucosa that approximates normal tissue and excises hypoplastic tissue at the cleft margins. Critical in the process is the reconstruction of the orbicularis oris musculature into a continuous sphincter. The Millard rotation-advancement technique has the advantage of allowing for each of the incision lines to fall within the natural contours of the lip and nose. This is an advantage because it is difficult to achieve "mirror image" symmetry in the unilateral cleft lip and nose with the normal side immediately adjacent to the surgical site. A Z-plasty technique such as the Randall-Tennison repair may not achieve this level of symmetry because the

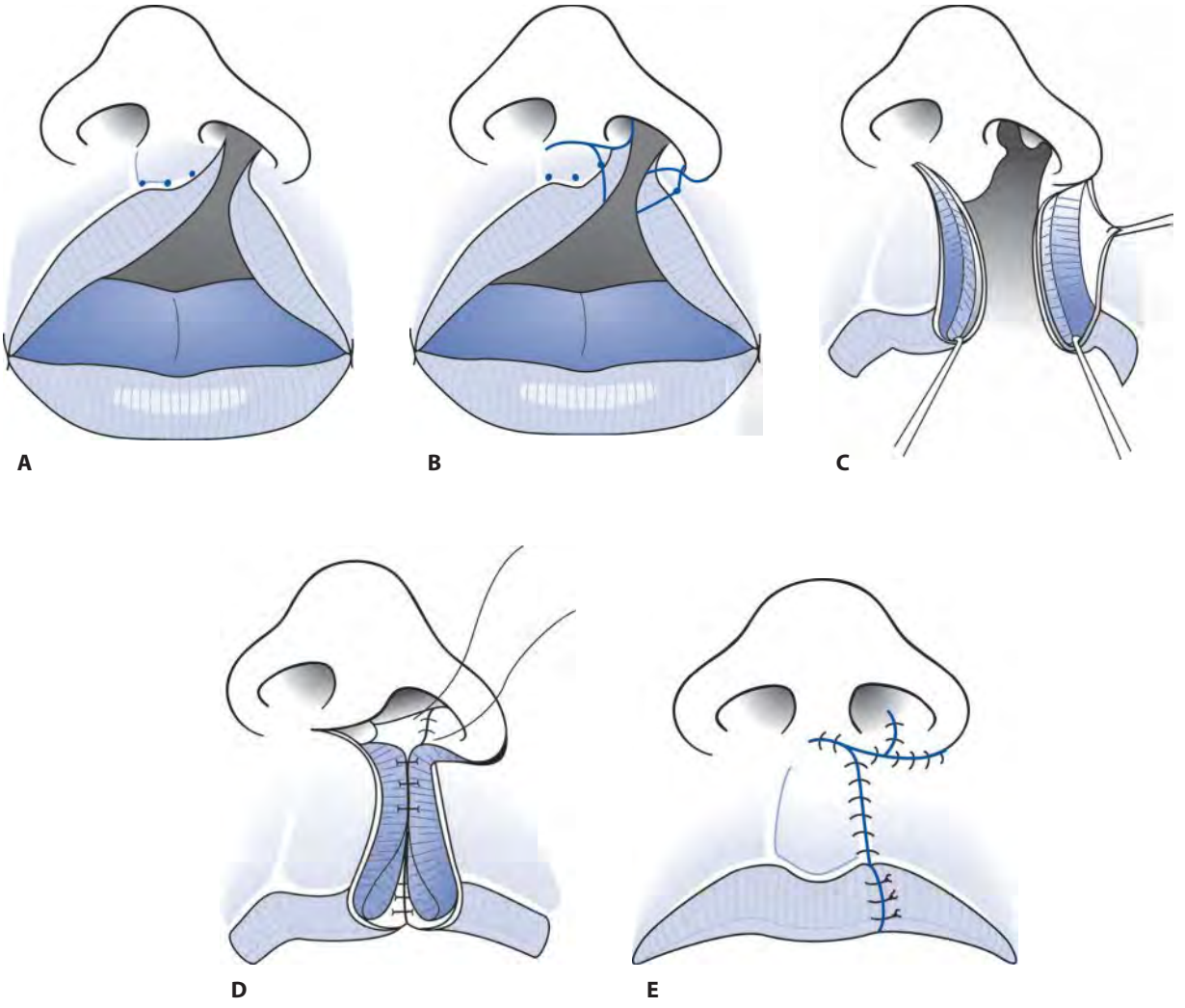


FIGURE 43-4. **A,** A complete unilateral cleft of the lip is shown highlighting the hypoplastic tissue in the cleft site that is not used in the reconstruction. Note the nasal deformities that are typical in the unilateral cleft, including displaced lower lateral nasal cartilages, deviated anterior septum, and nasal floor clefting. **B,** The typical markings for the authors' preferred repair are shown highlighting the need to excise the hypoplastic tissue and approximate good vermilion and white roll tissue for the repair. **C,** Once the hypoplastic tissue has been excised, the three layers of tissue are dissected (skin, muscle, and mucosa). It is important to completely free the orbicularis oris from its abnormal insertions on the anterior nasal spine area and lateral alar base. Nasal flaps are also incorporated into the dissection to repair the nasal floor (not shown). **D,** The orbicularis oris muscle is approximated with multiple interrupted sutures, and the vermilion border/white roll complex is reconstructed. The nasal floor and mucosal flaps are approximated. **E,** The lateral flap is advanced and the medial segment is rotated downward to create a healing scarline that will resemble the natural philtral column on the opposite side. The incision lines are hidden in natural contours and folds of the nose and lip.

Z-shaped scar is directly adjacent to the linear nonclefted philtrum (Figure 43-6). Achieving symmetry is more difficult when the rotation portion of the cleft is short in comparison to the advancement segment.

Primary nasal reconstruction may be considered at the time of lip repair to reposition the displaced lower lateral cartilages and alar tissues. Several techniques are advocated, and considerable variation exists with respect to the exact nasal reconstruction performed by each surgeon.[93,94] The primary nasal repair may be achieved by releasing the alar base, augmenting the area with allogeneic subdermal grafts, or even a formal open rhinoplasty. Since lip repair is done at such an early point in growth and development, the authors prefer minimal surgical dissection due to the effects of scarring on the subsequent growth of these tissues. McComb described a technique that has become popular, consisting of dissecting the lower lateral cartilages free from the alar base and the surrounding attachments through an alar crease incision.[93,95–97] This allows the nose to be bolstered and/or stented from within the nostril to improve symmetry.

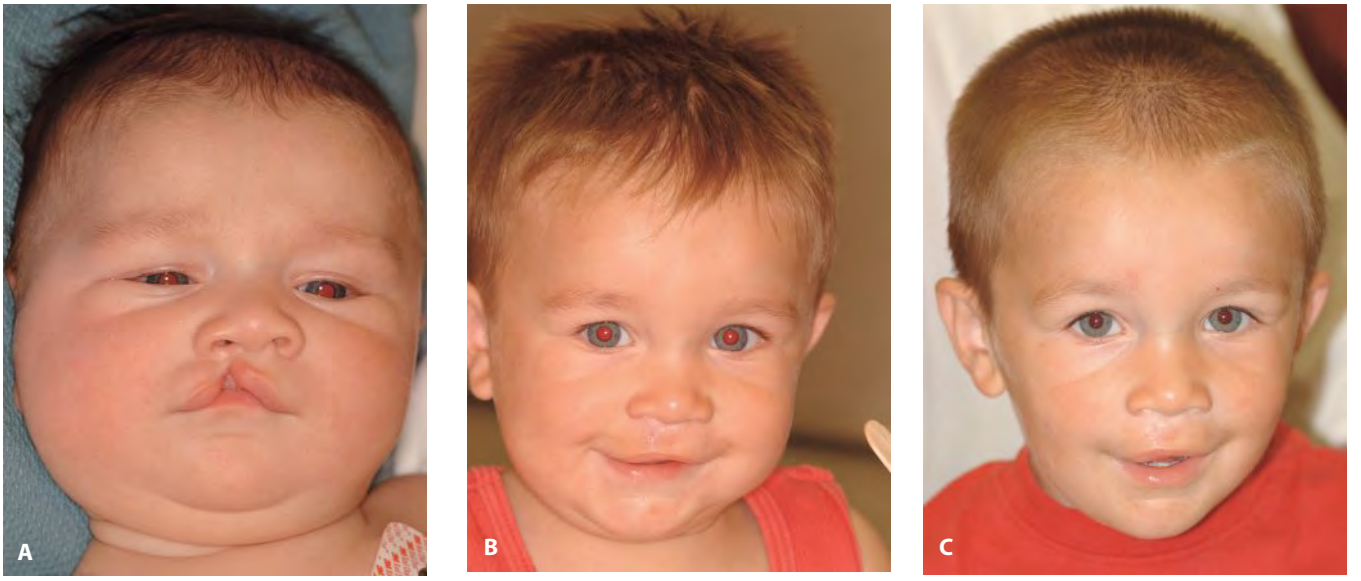


FIGURE 43-5. **A,** Three-month-old child with a right-sided incomplete unilateral cleft lip. Note the short philtrum near the midline that must be rotated downward to avoid notching and to improve symmetry. **B,** Nine-month-old boy after the rotation-advancement repair of his cleft lip and nasal deformities. **C,** The same child in B 2½ years after his cleft lip and nasal repairs.

Bilateral Lip Repair

Bilateral cleft lip repair can be one of the most challenging technical procedures performed in children with clefts. The lack of quality tissue present and the widely displaced segments are major challenges to achieving exceptional results, but superior technique and adequate mobilization of the tissue flaps usually yields excellent esthetic results (Figures 43-7–43-10). Additionally the columella may be quite short in length, and the premaxillary segment may be significantly rotated. Adequate mobilization of the segments and attention to the details of only using appropriately developed tissue will yield excellent results even in the face of significant asymmetry.



FIGURE 43-6. A typical scar that may result from a Z-type lengthening repair. Although the length and symmetry of the lip is good, an unnatural contour can occur due to the Z shape of the closure.

Some surgeons have used aggressive techniques to surgically lengthen the columella and preserve hypoplastic tissue using banked fork flaps.[98,99] Early and aggressive tissue flaps in the nostril and columella areas do not look natural after significant growth has occurred and result in abnormal tissue contours. While surgical attempts at lengthening the columella may look good initially, they frequently look abnormally long and excessively angular later in life (Figure 43-11). Revision of these iatrogenic deformities is difficult and some of the contour irregularities will not be able to be revised adequately. Usually if the hypoplastic tissue is excised and incisions within the medial nasal base and columella are avoided, the long-termes thetic results are excellent.

The authors prefer a primary nasal reconstruction that can be performed in a similar fashion to the unilateral technique described by McComb.[100] This allows for release and repositioning of the lower lateral cartilages and alar base on both sides without aggressive degloving of the entire nasal complex. Other open rhinoplasty techniques have been suggested using either direct incision on the nasal tip or through prolabial unwinding techniques.[100–103] As with most early maneuvers aggressive rhinoplasty at this time may incur early scarring that affects the growth potential of the surrounding

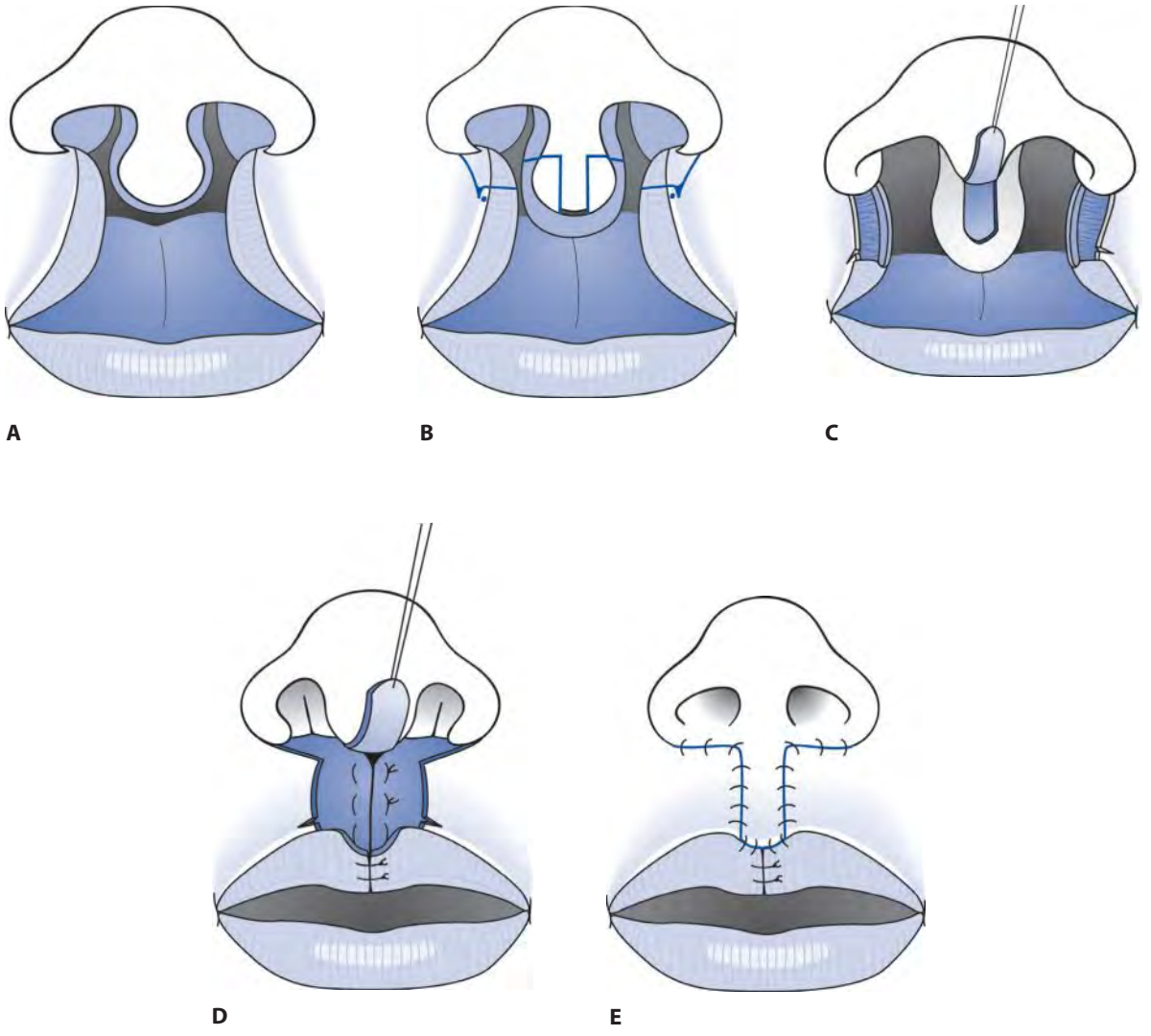


FIGURE 43-7. **A,** The bilateral cleft of the lip and maxilla shown here is complete and highlights the hypoplastic tissue along the cleft edges. The importance of the nasal deformity is evident in the shorter columella and disrupted nasal complexes. **B,** Markings of the authors' preferred repair are shown with an emphasis on excision of hypoplastic tissue and approximating more normal tissue with the advancement flaps. **C,** A new philtrum is created by excising the lateral hypoplastic tissue and elevating the philtrum superiorly. Additionally the lateral advancement flaps are dissected into three distinct layers (skin, muscle, and mucosa). Nasal floor reconstruction is also performed. **D,** The orbicularis oris musculature is approximated in the midline with multiple interrupted and/or mattress sutures. This is a critical step in the total reconstruction of the functional lip. There is no musculature present in the premaxillary segment, and this must be brought to the midline from each lateral advancement flap. The nasal floor flaps are sutured at this time as well. The new vermillion border is reconstructed in the midline with good white-roll tissue advanced from the lateral flaps. **E,** The final approximation of the skin and mucosal tissues is performed leaving the healing incision lines in natural contours of the lip and nose.

tissues, making revision more difficult and long-term esthetics less than ideal.

Cleft Palate Repair

The term *primary palate* is used to describe the anatomic structures anterior to the incisive foramen (eg, the alveolar ridge, maxilla, piriform rim). The term *secondary palate* refers to those structures posterior to the incisive foramen. Therefore, when surgeons refer to the initial or "primary" cleft palate repair, they are actually describing the closure of the secondary palate structures that include the hard palate, soft palate, and uvula. The structures of the embryologic primary palate are reconstructed later in childhood during the cleft maxillary/ alveolar bone graft procedure.

There are two main goals of cleft palate repair during infancy: (1) the water-tight closure of the entire oronasal communication involving the hard and soft palate; and (2) the

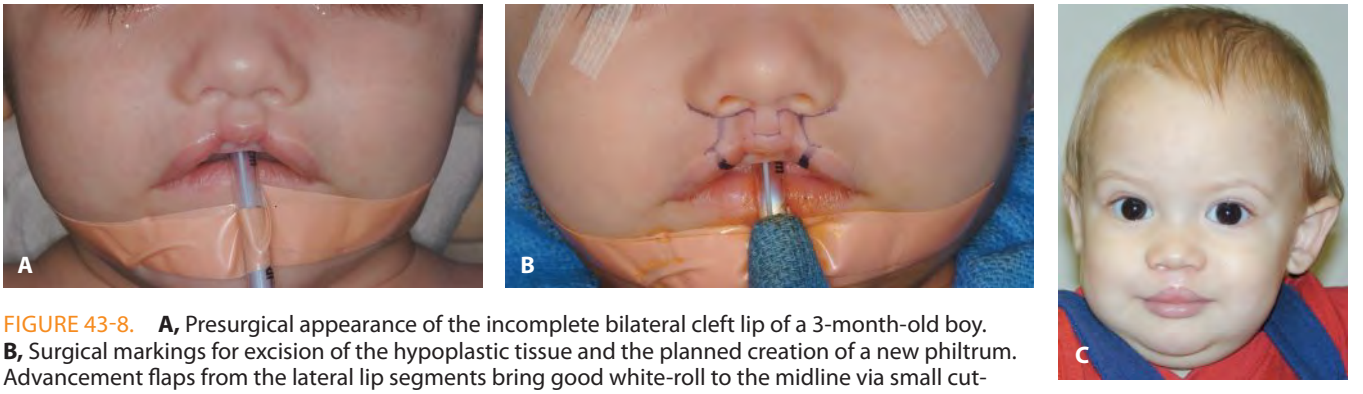


FIGURE 43-8. **A,** Presurgical appearance of the incomplete bilateral cleft lip of a 3-month-old boy. **B,** Surgical markings for excision of the hypoplastic tissue and the planned creation of a new philtrum. Advancement flaps from the lateral lip segments bring good white-roll to the midline via small cut-backs. **C,** The same child at 1 year of age after the repair of his bilateral cleft lip.



FIGURE 43-9. **A,** Presurgical appearance of a bilateral cleft lip and palate with impressive asymmetry and rotation of the premaxillary segment. Note the significant nasal asymmetry and bunching of the orbicularis oris laterally. **B,** The same child at 14 months of age.



FIGURE 43-10. **A,** Presurgical frontal view of a wide bilateral cleft lip and palate with significant asymmetry and lack of columella length. **B,** Presurgical left lateral view of a wide bilateral cleft lip and palate with a protrusive premaxillary segment. Note the short columella length. **C,** The same child at 10 months of age after repair of her bilateral cleft lip and palate. No presurgical taping or orthopedic appliances were used.

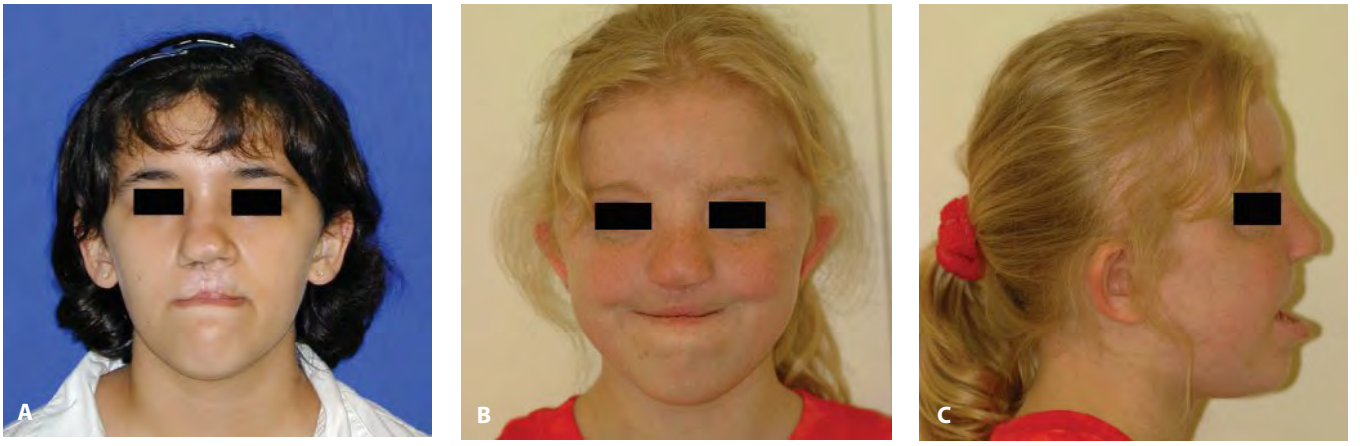


FIGURE 43-11. **A,** Frontal view of a teenage girl who had undergone columella lengthening and banked fork flaps during her initial repair and multiple attempts at secondary rhinoplasty by another surgeon prior to orthognathic surgery. **B,** Frontal view of a patient who underwent columella lengthening and banked fork flaps during her initial repair. **C,** Lateral view of the patient from B with a columella that is curved upwards and abnormally angular.

anatomic repair of the musculature within the soft palate that is critical for normal creation of speech. The soft palate, or velum, is part of the complex coupling and decoupling of the oral and nasal cavities involved in the production of speech. When a cleft of the soft palate is present there are abnormal muscle insertions located at the posterior edge of the hard palate. Surgery must not simply be aimed at closing the palatal defect but rather at the release of abnormal muscle insertions. Muscle continuity with correct orientation should be established so that the velum may serve as a dynamic structure.

The exact timing of repair of a palate cleft is controversial. Generally the velum must be closed prior to the development of speech sounds that require an intact palate. On average this level of speech production is observed by about 18 months of age in the normally developing child. If the repair is completed after this time, compensatory speech articulations may result. Repair completed prior to this time allows for the intact velum to close effectively, appropriately separating the nasopharynx from the orophayrynx during certain speech sounds.[104–107]

In patients with cleft palate, concerns for normal speech development are frequently balanced with the known biologic consequences of surgery during infancy; namely, the problem of surgery during the growth phase resulting in maxillary growth restriction.[33,72,73,108] When repair of the palate is performed between 9 and 18 months of age, the incidence of associated growth restriction affecting the maxillary development is approximately 25%.[31,33,109–111] If repair is carried out earlier than 9 months of age, then severe growth restriction requiring future orthognathic surgery is seen with greater frequency.[22,26,31,33,109,112–114] At the same time proceeding with palatoplasty prior to 9 months of age is not associated with any increased benefit in terms of speech development so the result is an increase in growth-related problems with an absence of any functional benefit.[115,116] Using only the chronologic age it seems that carrying out the operation during the 9 to 18 months timeline best balances the need to address functional concerns such as speech development with the potential negative impact on growth. To date no case-controlled rigorous clinical trial has examined what is likely the most critical factor in dictating the exact timing of cleft repair—the individual child's true language age. In cases where significant developmental delay is present surgery should be delayed since speech formation is not yet an issue and there is a likely benefit in terms of growth of the maxilla. Delaying palatal closure is relevant in situations where the cleft palate is associated with other complex medical conditions, neurodevelopmental delay, complex craniofacial anomalies, and/or the presence of a tracheotomy.

Another approach used to balance speech issues with growth-related concerns is to stage the closure of the secondary palate with two operations. Generally this involves the repair of the soft palate early in life as an initial step, followed by closure of the hard palate later in infancy. The idea is that timely repair of the soft palate, which is critical for speech, is accomplished while hard palate repair with mucoperiosteal stripping is delayed until growth is further along.[117,118] Although this technique is not advocated by the majority of surgeons, some surgeons may feel that repairing the hard palate portion later may offer the advantages of less growth restriction, easier repair of larger clefts, and less chance for fistula formation. No convincing data exist to favor this approach over a single-stage repair, but the practice is continued by some centers where anecdotal evidence suggests that there may be some benefit. In contrast most North American speech and language pathologists prefer closure of the palate as a single operation.[117]

Cleft palate reconstruction requires the mobilization of multilayered flaps to reconstruct the defect due to the failure of fusion of the palatal shelves. Generally when the initial palate closure is performed, this refers to closure of the tissues posterior to the incisive foramen. This is done in a

layered fashion by first closing the nasal mucosa and then the oral mucosa. Since the main function of the palate is to close the space between the nasopharynx and oropharynx during certain speech sounds, the surgeon must also reconstruct the musculature of the velopharyngeal mechanism. The musculature of the levator palatini is abnormally inserted on the posterior aspect of the hard palate and therefore must be disinserted and reconstructed in the midline.[26,119] Therefore, the soft palate is closed in three layers by approximating the nasal mucosa, levator musculature, and the oral mucosa. The hard palate portion is closed in two layers using nasal mucosa flaps and then oral mucosa flaps. Both the hard and soft palate repairs must be done in a tension-free manner to avoid wound breakdown and fistula formation. Adequate mobilization of the flaps during the dissection is essential to achieve tension-free closure. At times some surgeons may elect to incorporate vomer flaps into the repair if there is difficulty in mobilizing the lateral flaps to the midline.

Many techniques have been described for repair of the palate.[120–127] The Bardach two-flap palatoplasty uses two large full-thickness flaps that are mobilized with layered dissection and brought to the midline for closure (Figure 43-12).[26,120] This technique preserves the palatal neurovascular bundle as well as a lateral pedicle for adequate blood supply. The von Langenbeck technique is similar to the Bardach palatoplasty but preserves an anterior pedicle for increased blood supply to the flaps.[26,121] This technique is also successful in achieving a layered closure but may be more difficult when suturing the nasal mucosa near the anteriorly based pedicle attachments. The authors do not favor push-back techniques as they may incur more palatal scarring, restrict growth, and do not show a measurable benefit in speech.

Another common technique is the Furlow double-opposing Z plasty, which attempts to lengthen the palate by taking advantage of a Z-plasty technique on both the nasal mucosa and the oral mucosa (Figure 43-13).[26,124–127] This technique can be effective at closing the palate but has been reported by some to have a higher rate of fistula formation at the junction of the soft and hard palates where theoretical lengthening of the soft palate may compromise the closure.[26,128–133] No benefit has been convincingly demonstrated with any particular repair technique when one looks at dental arch form, speech outcome, feeding, or any other functional variable. At this point in our understanding surgeons often consider their own experiences and training when repairing clefts, since definitive data suggesting that one repair is preferable over another are lacking.

In very wide clefts some surgeons will advocate the consideration of a pharyngeal flap at the primary palatoplasty procedure to assist in closure since revision palatoplasty is sometimes unsuccessful in eradicating fistulas. Those who use this technique usually perform it in extremely wide clefts

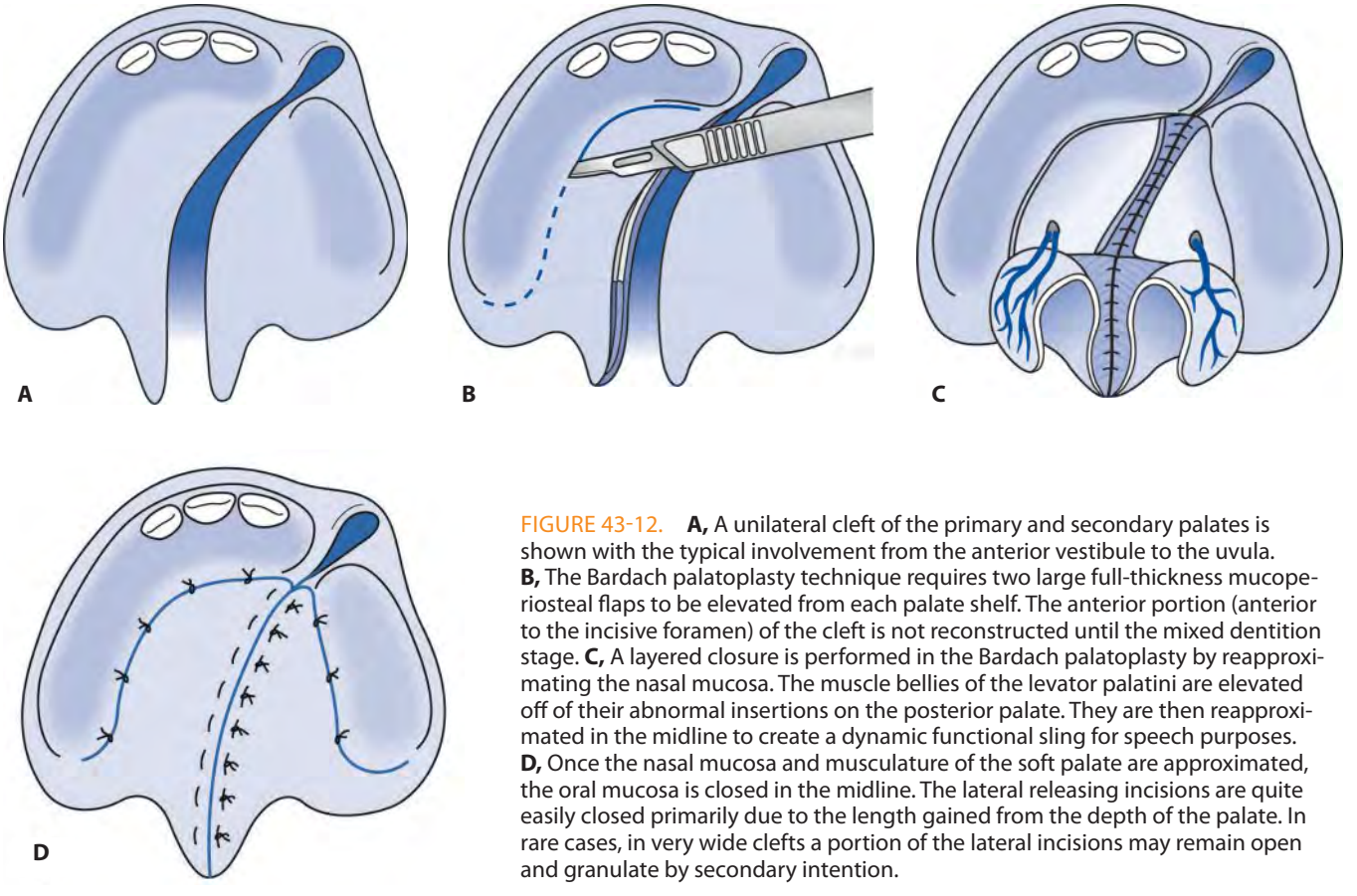


FIGURE 43-12. **A,** A unilateral cleft of the primary and secondary palates is shown with the typical involvement from the anterior vestibule to the uvula. **B,** The Bardach palatoplasty technique requires two large full-thickness mucoperiosteal flaps to be elevated from each palate shelf. The anterior portion (anterior to the incisive foramen) of the cleft is not reconstructed until the mixed dentition stage. **C,** A layered closure is performed in the Bardach palatoplasty by reapproximating the nasal mucosa. The muscle bellies of the levator palatini are elevated off of their abnormal insertions on the posterior palate. They are then reapproximated in the midline to create a dynamic functional sling for speech purposes. **D,** Once the nasal mucosa and musculature of the soft palate are approximated, the oral mucosa is closed in the midline. The lateral releasing incisions are quite easily closed primarily due to the length gained from the depth of the palate. In rare cases, in very wide clefts a portion of the lateral incisions may remain open and granulate by secondary intention.

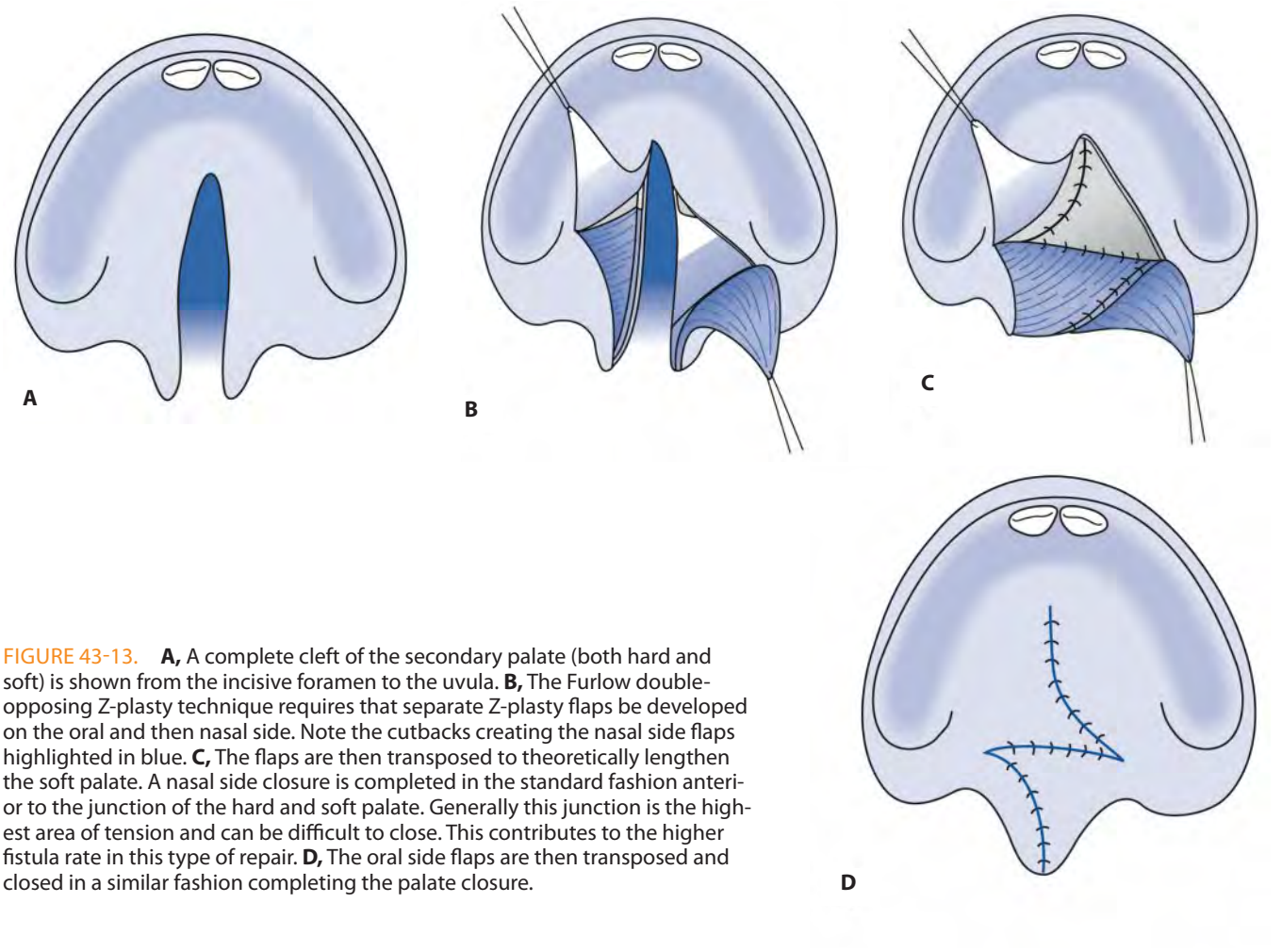


FIGURE 43-13. **A,** A complete cleft of the secondary palate (both hard and soft) is shown from the incisive foramen to the uvula. **B,** The Furlow double-opposing Z-plasty technique requires that separate Z-plasty flaps be developed on the oral and then nasal side. Note the cutbacks creating the nasal side flaps highlighted in blue. **C,** The flaps are then transposed to theoretically lengthen the soft palate. A nasal side closure is completed in the standard fashion anterior to the junction of the hard and soft palate. Generally this junction is the highest area of tension and can be difficult to close. This contributes to the higher fistula rate in this type of repair. **D,** The oral side flaps are then transposed and closed in a similar fashion completing the palate closure.

and do so very selectively. This allows the central portion of the closure to be filled with posterior pharyngeal wall tissue making the closure of the nasal and palatal mucosa easier. Patients with Pierre Robin syndrome or Treacher Collins syndrome may have exceptionally wide clefts that are difficult to close with no tension, and this technique may be considered. The drawbacks of using a pharyngeal flap during the repair of the palate include a significantly increased risk for complications such as bleeding, snoring, obstructive sleep apnea, or hyponasality. The details of pharyngeal flap surgery and revision palatoplasty techniques are discussed in Chapter 43, "Reconstruction of Cleft Lip and Palate: Secondary Procedures."

COMPLEX FACIAL CLEFTING

Clefting of the facial structures other than the typical nasolabial region is rare and often presents difficult challenges to the reconstructive cleft surgeon. Therefore it is important to consider referring patients with complex facial clefting to surgeons with experience in this particular area. Comprehensive interdisciplinary care is mandatory to achieve the best results including involvement of neurosurgery, ophthalmology, orthodontics, speech pathology, and other members of the craniofacial team. Some interventions such as eye lubrication may be necessary within hours after birth, and accurate prenatal diagnosis of severe facial clefting is helpful in planning for early care.

The etiology of the various facial clefts may be related to failure of embryologic fusion, physical obstruction in fetal life, association with an encephalocele or tumor, amniotic bands, or other anatomic disruptions during fetal life.[55] The vast majority of complex facial clefts are sporadic events and not related to a single gene disease. Many complex facial clefts involve the orbit, and the classification system most often used is orbitocentric in design (see Figure 43-2). Paul Tessier described a numbering system for facial clefting phenomena to make description and surgical planning more easily discussed.[56] Other systems exist but have a more cumbersome nomenclature.[57]

Primary repair of severe facial clefts is often more difficult than even the most difficult standard bilateral clefts.[134–136] While mobilization of the lip and nose structures is rather straightforward, the closure of clefts in the orbital region can be challenging due to the lack of eyelid and adjacent tissue for advancement and/or rotation. Revision surgery is the norm in this group and should include a skilled ophthalmologic surgeon early in the process for the best results.



FIGURE 43-14. **A,** A 2-month-old boy with a Tessier no. 7 left facial cleft associated with craniofacial microsomia (Kaban type IIb), congenital ear anomalies, a lateral tongue cleft, and a left-sided epibulbar dermoid. **B,** The same child at 8 months after the primary repair.

The staged reconstruction of these types of severe facial clefts is similar to the more common cleft lip and palate protocols. However, several functional issues are present in patients with complex facial clefting that require more immediate attention. For example, patients with large Tessier no. 7 clefts may have problems with retaining foodstuffs in their oral cavities due to the discontinuity of the orbicularis oris (Figure 43-14). This may prompt early repair and reestablishment of the orbicularis oris musculature for functional concerns.

For those patients with orbital clefts a skilled pediatric ophthalmologist should evaluate the child early to avoid severe corneal abrasion and desiccation. Immediate lubrication of the globes is necessary to prevent severe irreversible corneal damage until eyelid structures can be mobilized to cover the globe adequately. Early after birth tarsorrhaphy stitches can be used to gain adequate closure of the lids for corneal protection. Ignoring the need for eye protection may result in severe corneal scarring that may cause blindness and prompt consideration for corneal transplantation. Corneal transplants in infants are often not successful but are possible in patients with severe orbitofacial clefts. Another concern is the support of the globes at the orbit floor that may be involved in some facial clefts. The timing of orbital reconstruction is dependent on the functional needs of the cleft area in each patient. These are just some of the concerns present in complex facial clefting, and a customized treatment plan must be formulated for each patient.

OUTCOME ASSESSMENT

Decision-making in cleft care should be based on evidenced-based research and a critical look at outcomes. Unfortunately there is little evidenced-based research available to guide clinicians through the many treatment protocols for cleft care.[113] Although the clinical experience of the surgeon certainly has value, this must be integrated with a constant review of evidence-based research. Typically enthusiasm by a surgeon or a particular group of surgeons regarding a specific intervention because of personal experiences may help popularize that intervention but with little outcome data to support its use. Too frequently the long-term results are not forthcoming, and the treatment regimen may still persist. Unfortunately some of the treatment regimens used today are based on the poor outcomes and mishaps of previous surgeons rather than regimens chosen as a consequence of published evidence of the actual success of a particular treatment.

Additionally the pressures of a costly health care system have made treatment decision questions even harder to investigate.[88] A need to understand the outcome differences between treatment philosophies will be critical to help determine which protocols will be most beneficial to the patient without extending valuable health care resources on unproven or ineffective methods. For this reason among many others, the need to discard unproven and unnecessary interventions has never been greater. Outcomes studies based on functional results such as appearance, facial growth, occlusion, patient satisfaction, and psychosocial development are all critical in this process. Surgeons involved in the care of patients with clefts must critically review the literature on a regular basis and not be tempted by poorly evaluated techniques popularized by clinical reports.

CONCLUSIONS

The comprehensive care of patients with clefts requires an interdisciplinary approach that demands precise surgical

execution of the various procedures necessary to correct cleft deformities, as well as frequent long-term follow-up. Clinicians experienced in the comprehensive interdisciplinary care of patients with clefts are best equipped to deal with these concerns. The treatment of patients with cleft and craniofacial deformities should be free of bias and should demand team care that is patient, family, and community oriented. Only in this fashion can the overall treatment be optimally successful. This type of care maximizes the patient's ability to grow into adulthood and succeed in life without focusing on their deformity.

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44

CHAPTER

Reconstruction of the Alveolar Cleft

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In the management of patients with cleft lip and cleft palate, the decision regarding alveolar cleft grafting is one of the most controversial. Is grafting of the residual alveolar defect indicated? If so, at what age is it most appropriate, what material is most ideal, and should adjunctive procedures such as orthodontic expansion be used before or after grafting? Lastly, what are appropriate measures of success? This chapter reviews what is known, discusses these controversies, and provides a rationale for the approach to the residual alveolar cleft defect.

RATIONALE FOR GRAFTING

Although some authors have advocated nongrafting techniques[1] or prosthodontic approaches, the general consensus is that achieving continuity between the cleft alveolar segments has significant advantages, regardless of how and when this is accomplished. Potential advantages include[2]

1. Grafting achieves stability of the arch and prevents collapse of the alveolar segments. This provides improved orthodontic stability.
2. Grafting preserves the health of the dentition. Grafting provides room for the canine and lateral incisors to erupt into the arch into stable alveolar bone and maintains bony support of teeth adjacent to the cleft.[3,4]
3. Grafting restores continuity not only of the alveolus but also of the maxilla at the piriform rim. This supports the ala and provides improved stability and support for the nose. This may have a direct aesthetic benefit and may also prove to be of long-term benefit when formal rhinoplasty procedures are performed.[3]
4. Palatal and nasolabial fistulae are often present even after palatoplasty. Grafting of the alveolar defect provides an opportunity for the surgeon to address the residual oronasal fistula. This may have potential benefit for both hygiene and speech. Many cleft patients present with chronic upper respiratory and sinus disease, which may be related to reflux into the nasal cavity and sinus. There is some evidence that the residual fistula, whether labial or palatal, can have an effect on speech articulation and nasality. There is evidence that closure of the fistula and grafting the cleft defect can improve nasal emission and nasality.[5]

MEASURING OUTCOMES

Before discussing the controversies associated with reconstruction of the residual alveolar cleft, it is important to accept some consistent measure of successful outcome. Most reports rely on descriptive data. This makes comparison of different approaches difficult. To evaluate bone graft success, Bergland and colleagues[6] described a semiquantitative approach that divided grafts into four types based on alveolar crest height. Although this is effective, it has been suggested that occlusal alveolar bone height does not adequately measure success.[7] Support of the ala and opportunity for successful tooth movement into the site or placement of an endosseous implant also require apical bone formation. A modification of the Bergland scale that measures both occlusal and basal bone height may be a better tool for evaluating graft success. Although the Bergland scale and modifications of it rely on a two-dimensional radiograph to evaluate bone fill within a three-dimensional cleft, studies show good correlation between bone volume as predicted by these two-dimensional radiographs and that shown on three-dimensional computed tomography (CT) scans.[8] Recently, investigators

have used three-dimensional imaging almost exclusively to quantify the results of alveolar cleft bone grafting techniques.[9–11] To date, no research has been conducted to show that a critical volume of bone is required at an alveolar cleft site in order for the graft to meet success criteria. Therefore, it appears at this time that three-dimensional imaging certainly has its place in research but two-dimensional imaging continues to be sufficient for routine monitoring of clinical outcomes. An exception to this might include evaluation of the grafted site for the treatment planning of dental implants after skeletal maturity has been achieved.

TIMING OF THE GRAFT

Perhaps the most controversial topic in managing the alveolar cleft is when grafting should be performed. In the traditional literature, terminology is not consistent. Outcome measures for various approaches are also defined inconsistently, which makes comparison difficult. Here, alveolar grafting is grouped according to timing as defined later (Table 44-1).

Primary Grafting

Some define primary alveolar bone grafting as that which is performed simultaneously with lip repair.[12] Others have stated that any grafting that is performed at younger than 2 years of age is considered primary grafting. Still others have defined primary grafting as grafting performed before the palate is repaired.[13,14]

Primary grafting performed at the time of lip repair has failed to result in acceptable outcome. Long-term studies show abnormal maxillary development with maxillary retrognathia, concave profile, and increased frequency of crossbite compared with patients without grafts.[15,16]

Primary grafting performed after the closure of the lip and before the closure of the palate has proved successful in a limited number of centers when a very specific protocol is followed.[13,14] A prosthesis is placed before the lip is closed to mold the alveolar segments into close proximity. The lip is then closed, which further aids in molding the segments. The segments must be in close proximity with good arch form before an onlay rib graft is placed across the labial surface of the cleft in a subperiosteal tunnel that is developed by limited dissection.

Advocates of this approach have not experienced problems with altered facial growth and malocclusion, most likely the result of the limited dissection used in these cases. They have reported improved occlusion and graft success in these patients compared with patients grafted at other ages.[17] It is still difficult to wholeheartedly endorse this approach. Several additional anesthetics and surgeries are needed at a young age. This technique may not be possible in all patients, such as those with isolated alveolar clefts without palatal clefting or those in whom segments cannot be orthopedically aligned. In one center, because of these limitations, nearly one half of patients could not be treated with primary grafting.[13] Outcomes may also not be as good as with other approaches. In one study, there was an increased incidence of malformation of permanent lateral incisors in the primary graft group and decreased success of the graft, with only 41% of primary grafts (54% if pregrafting orthopedics was included) resulting in adequate bone height when measured with a Bergland scale.[18] This was compared with 73% success of those sites grafted in the mixed-dentition stage (after eruption of the permanent central incisors and before eruption of the maxillary canines).

TABLE 44-1. **Timing of Alveolar Bone Grafting**

<2 Years of Age: Primary Grafting
After lip repair
Before palate repair
≥2 Years of Age: Secondary Grafting
2–5 yr: Early secondary
6–12 yr: Mixed dentition secondary (after central incisor eruption and before the canine erupts)
6–8 yr: Early mixed dentition
9–12 yr: Late mixed dentition
>12 yr: Late secondary grafting

Early Secondary Grafting

Grafting after the child reaches 2 years of age and before 6 years is considered early secondary grafting. The literature does not support early secondary grafting.

Secondary Grafting during the Mixed Dentition (After Eruption of the Maxillary Central Incisors and before Eruption of the Canines)

Alveolar reconstruction with grafting during the eruption of the permanent dentition may be best for various reasons. Rationale for grafting and for timing of grafting during this time period include

1. There is minimal maxillary growth after age 6 to 7 years, and the effect of grafting at this time will result in minimal to no alteration of facial growth.[19,20]
2. Cooperation with orthodontic and perioperative care is predictable. General anesthesia is not required for routine orthodontic procedures such as expansion.
3. The donor site for graft harvest is of acceptable volume for predictable grafting with autogenous bone.
4. Bone volume may be improved by eruption of the tooth into the newly grafted bone.[21]
5. Grafting during this phase allows placement of the graft before eruption of permanent teeth into the cleft site, which achieves one of the primary goals of grafting—to enhance the health of teeth in and adjacent to the alveolar cleft

The landmark papers by Boyne and Sands[22,23] established that grafting in the mixed dentition achieves many of the goals of

TABLE 44-2. **Factors Contributing to Timing of Grafting during the Mixed Dentition**

Dental age vs. chronologic age
Presence of the lateral incisor
Position of the lateral incisor
Degree of rotation/angulation of the central incisor
Trauma/mobility of premaxillary segment (bilateral clefts)
Social issues
Size of the patient and of the cleft
Occlusion
Need for adjunctive procedures
Dynamic of the team

reconstruction of the cleft alveolus. The ideal patient is between the ages of 8 and 12 years with a maxillary canine root that is one half to two thirds developed. This timing is supported by several well-documented studies.[6,24–28] However, some authors have suggested that earlier grafting should be considered as a means of preserving the lateral incisor as well.[15,29,30] These authors have suggested that grafting be considered as early as 6 years of age. There is some evidence that grafting between the ages of 6 and 8 years, in addition to achieving the expected goal of preserving the canine, can preserve the lateral incisor as well, but this remains controversial. Despite clear indications that grafting in the mixed dentition is preferable to either primary, early secondary, or late secondary grafting, it is not entirely clear whether this grafting should be performed early (age 6–8 yr) or late (age 8–12 yr). Various individual factors should be evaluated when determining the ideal time for grafting during the mixed dentition (Table 44-2).

Dental versus Chronologic Age

Many outcomes of grafting are related to preserving health of the dentition adjacent to and erupting into the cleft site. It makes sense that the timing of the graft be determined on the basis of dental rather than chronologic age. When the maxillary central incisors begin to erupt, regardless of chronologic age, the patient should be evaluated for grafting, taking into consideration the other factors discussed later. In some patients, this may be much earlier than the traditionally recommended age for evaluation.

Presence of the Lateral Incisor

Many proponents of earlier mixed-dentition grafting advocate this timing because of the opportunity to salvage the lateral incisor.[15,27,29] During the evaluation, attention should be directed to the presence of the lateral incisor and to whether this tooth appears to be normally formed. The incidence of congenitally missing permanent lateral incisors within the alveolar cleft is between 35% and 60%.[18,31] If a lateral incisor is present and appears to be well formed, earlier grafting may be beneficial. Even if the tooth is not perfectly formed, it may still be beneficial to attempt to preserve it. The grafted alveolus will often thin to the point that alveolar width is not adequate for definitive reconstruction with an endosseous implant without additional grafting.[32] Retaining the lateral incisor will maintain bone width and perhaps eliminate the need for yet another graft at the time of implant placement.

Position of the Lateral Incisor

If the lateral incisor is mesial to the cleft, it often has adequate space for eruption. However, if the lateral incisor is located in the posterior segment, earlier grafting may be necessary to preserve the lateral incisor.[12] In one review, 36% of patients with cleft lip and alveolus had missing lateral incisors.[31] Of the 64% who had lateral incisors, 90% of the lateral incisors were located distal to the cleft. In the same series of patients, 57% of those with cleft lip and palate had missing lateral incisors, and of the remaining 43%, 86% of the lateral incisors were located distal to the cleft.[31] Therefore, a significant number of patients may benefit from earlier grafting to preserve the lateral incisor (Figure 44-1).

Rotation of the Central Incisor

The maxillary permanent central incisor will often erupt in a rotated and angled position (Figure 44-2). This reflects the

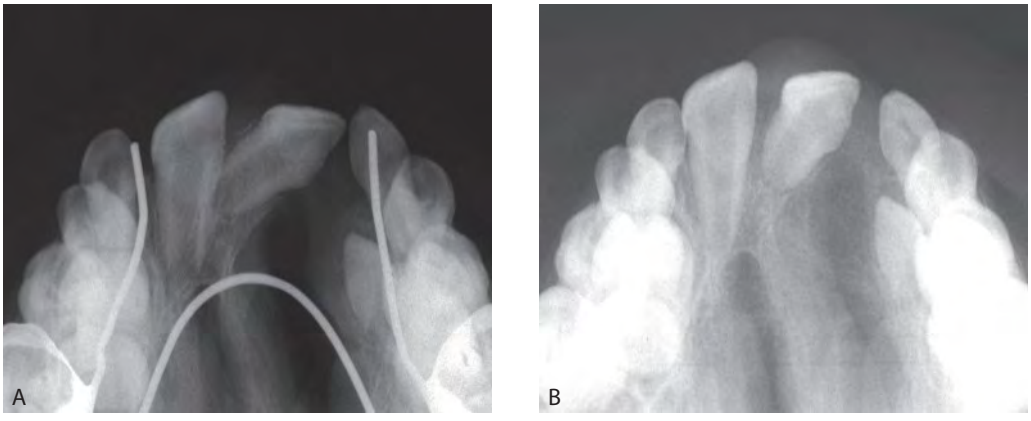


FIGURE 44-1. **A,** Occlusal radiograph shows lateral incisor distal to the cleft. **B,** Grafting which was performed at age 7 years to facilitate eruption of the lateral incisor.

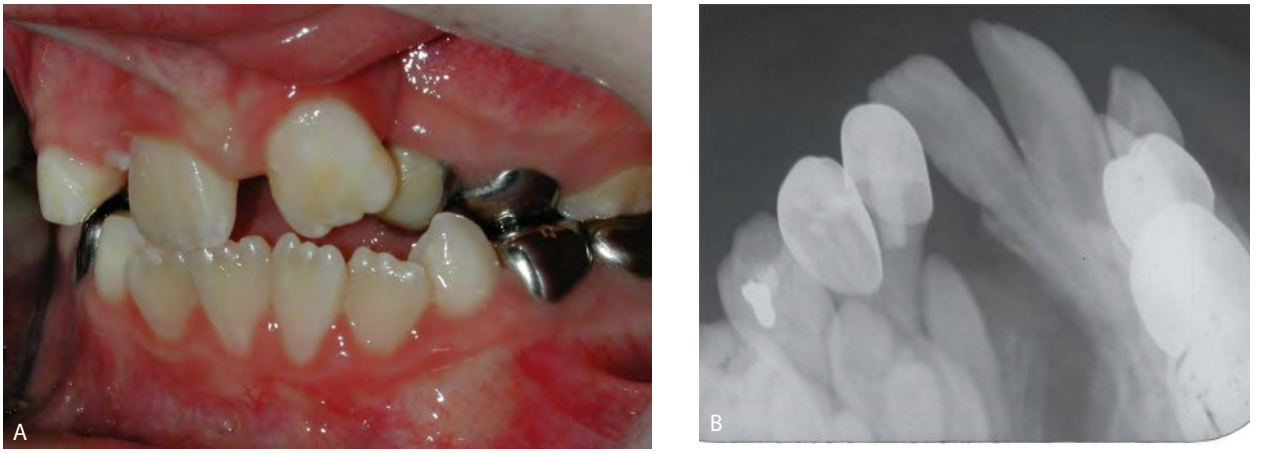


FIGURE 44-2. **A,** Photograph of a typical unilateral cleft. There is rotation of the central incisor and angulation of the crown toward the cleft. This maintains bone support for the root of the tooth. **B,** Occlusal radiograph shows that the cleft defect is larger than it appears clinically and support for the incisor root is provided by only a thin margin of bone.

morphology of the underlying bone. In extreme cases, the crowding of the two incisors can preclude normal oral hygiene methods, and this can result in decay of the central incisor. The patient or parent may also be concerned with the position of the incisors for aesthetic reasons. If a decision is made to rotate these teeth into alignment, it may be necessary to graft the alveolar defect before this orthodontic tooth movement.[33] Failure to consider the morphology of the bone on the distal surface of the erupted central incisor can result in bone loss and periodontal defects as a result of orthodontic tooth movement. Because the incisor teeth erupt at approximately age 6 years, the surgeon may choose to graft at an earlier age so that orthodontic movement of the incisors can be accomplished.

Social Issues

The window for mixed-dentition grafting is large (age 6–12 yr). This is also during a period of tremendous social development for the patient. If a graft is necessary, the timing of surgery should respect the social and educational development of the child. Slightly earlier grafting, when it may cause less interference with education or other important opportunities for social development, may be preferable to grafting at an exact stage of dental development.

Size of the Patient and of the Cleft

Petite patients with large cleft defects are challenging. Adequate closure of the defect may be difficult, and harvesting an adequate amount of graft material may be challenging as well. This is particularly true for large bilateral cleft defects. In these patients, the lateral incisor is often absent, the oronasal communication is often quite large, and the premaxilla is frequently in less than ideal position. In these large defects, later grafting is often better, to wait for growth of the patient and orthodontic alignment of the cleft segments.

Need for Other Procedures

Patients are often evaluated for velopharyngeal incompetence, minor aesthetic revision of the nose or the lip, and pressure-equalizing tubes for otitis media. It is reasonable to coordinate the timing of surgery for the alveolar cleft with other procedures that may be necessary. If velopharyngeal flap surgery is planned during the mixed-dentition phase, it should take precedence over the alveolar graft. Improved speech is more important to the child's development than achieving continuity of the alveolus. Alveolar grafting would be compromised if performed simultaneously with velopharyngeal flap surgery, and in these patients, it is appropriate to delay the graft until the velopharyngeal flap surgery is accomplished and speech therapy re-instituted. Minor soft tissue, nasal, and lip revision are often desired by the patient and parents. These can be accomplished with alveolar grafting. The grafting process can distort the nose and soft tissue; these soft tissue procedures should be performed first with alveolar grafting undertaken in the same setting and with care not to disrupt the aesthetic procedures already performed.

Dynamic of the Team

Cleft management should always involve a multidisciplinary team, with the wide expertise to develop a proper treatment plan. Difficulties may arise when the priorities of one specialty compete with those of another. If the surgical team is faced with an orthodontic provider who feels strongly that it is appropriate to align the maxillary central incisors as soon as they erupt, it will be necessary for the alveolar defect to be grafted earlier to prevent compromise of osseous support for the central incisors. Some orthodontists and surgeons believe that palatal expansion is necessary before grafting. These teams may find that it is more appropriate to graft patients at a later age, because it may take months to achieve the desired expansion before the graft.

Secondary Grafting after Eruption of the Permanent Canine (Late Secondary Grafting)

Late secondary grafting has received some support; however, data show that when all the goals of alveolar reconstruction are considered, it has a less than acceptable outcome. Patients older than 12 years of age who undergo grafting have been reported to have decreased success when evaluated using the Bergland scale,[6,18,28,30,34,35] loss of osseous support of teeth adjacent to the cleft,[21] and increased morbidity.[30] There is less opportunity to salvage the lateral incisor, and there is a delay in correction of the orthodontic condition. This delayed grafting does allow for increased options with regard to donor site for graft material because harvest of the mandibular symphysis becomes possible. Such grafts are difficult in the mixed-dentition stage, where it is difficult to obtain adequate bone without damaging unerupted teeth.

SOURCE OF BONE GRAFT

The selection of the ideal grafting material is somewhat dependent on the timing of the graft. In primary bone grafting, the rib is the only site for adequate quantity of bone with acceptable morbidity. In the mixed-dentition stage, the rib is not as appropriate as other sites such as the calvaria or iliac crest. These options would also be possible sources for bone for late secondary grafting, as well as grafts from the mandibular symphysis and possibly the tibia.

Because the data suggest that grafting during the mixed dentition is ideal, discussion focuses on comparing various sources of graft material for this group of patients. The advantages and disadvantages of various potential sources of bone are outlined in Table 44-3.

Iliac Crest

Potential advantages of the iliac crest bone graft include low morbidity and high volume of viable osteoblastic cells (cancellous bone); two teams may work simultaneously, and this procedure is well accepted by the patient.

Bone can be harvested from the iliac crest through various approaches. Some have suggested that a lateral approach is appropriate in the growing patient.[36] This procedure disrupts the iliotibial tract and has a higher incidence of gait disturbance and postoperative pain.[37] In theory, it may be appropriate to avoid the anterior crest, which does not complete its growth until after age 20 years.[37] However, the cartilaginous cap overlying the crest is reduced in thickness to about 1 cm by age 9 years. Damage to the crest at this time could lead to disturbance in growth and cosmetic deformity of the crest; however, splitting the crest longitudinally, which allows access to the underlying cancellous marrow, has been used for harvest of bone in this age group with no reported growth alteration and less postoperative gait disturbance than with the lateral subcrestal approach.[38,39]

Calvarial Bone

Calvarial bone has been recommended by some as an alternative to iliac crest grafting.[40,41] Some authors have concerns about the potential for success when the calvarium is used as a graft source.[42,43] This may be related to the technique of harvest. Bone grafts consisting of diploic bone have been shown to be more successful than those grafts harvested using a high-speed rotary device to shave off primarily cortical bone from the surface of the calvarium.[43] However, even when harvesting calvarial bone in such a way as to maximize diploic bone, results may not be as good as with iliac crest bone. In one study in which primarily diploic bone was carefully harvested from the calvarium, the results were still less successful (80% graft success) than with traditional iliac crest bone (93% graft success).[44] It is likely that either source is effective as long as primarily diploic bone is used. This limitation may render calvaria as a less useful source for large clefts and bilateral clefts.

Calvarial grafts may have decreased morbidity compared with iliac crest harvest. There is less postoperative pain and no gait disturbance. Other potential advantages include decreased surgery time. Cranial bone grafts can be harvested more quickly than iliac crest grafts. If a single team is

TABLE 44-3. **Comparison of Graft Sources**

Site	Advantage	Disadvantage	Considerations
Ilium	Large quantity cancellous bone, two teams	Mild transient gait disturbance	All clefts, particularly large and bilateral clefts
Calvaria	Minimal postoperative discomfort, incision hidden, low morbidity	Limited cancellous/diploic bone, increased operative time	Unilateral clefts, lower success
Mandibular symphysis	Same operative field, rapid procurement, minimal pain	Limited bone	Older children with small defects
Rib	Two teams	Poor source cancellous bone, postoperative pain, visible scar, risk of pneumothorax	Not recommend except for primary grafting
Proximal tibia	Abundant cancellous bone, easy procedure, mild postoperative pain, two teams		Not recommend in patients who have not completed growth

Adapted from Ochs MW. Alveolar cleft bone grafting (part 2): secondary bone grafting. J Oral Maxillofac Surg 1996;54:83–88.

performing surgery, this may be significant. However, it is not possible to harvest the cranial graft simultaneously with the alveolar cleft repair. Grafting from the iliac crest if two teams are used can decrease overall operating time compared with calvarial grafting. Lastly, the incision for graft harvest is hidden in the hairline, which may have a cosmetic advantage.

Grafting from the calvarium has potential disadvantages. There is a perceived increased risk by patients and their families, although several studies show that the morbidity of bone harvest from the calvaria is minimal.[37] As mentioned previously, the volume of diploic bone is limited, making this less predictable for large or bilateral clefts.

Allogeneic Bone

In an effort to eliminate the morbidity and time necessary to harvest bone from any autogenous site, some authors have evaluated allogeneic bone as a potential source of graft material. Studies have shown that allogeneic bone can be used successfully to graft secondary alveolar cleft defects and that results can be compared favorably with those achieved with autogenous bone.[45] However, the demands of bone healing in the alveolar defect where there is potential communication between the graft and the nasal and oral cavity may make this less predictable in large cleft defects or bilateral clefts. In general, bone healing with autogenous bone is biologically different than with allogeneic bone. Autogenous bone grafts initiate an angioblastic response early in the healing process, and some of the transplanted cells remain viable, resulting in a more rapid formation of new bone. In contrast, allogeneic bone grafts demonstrate slower revascularization, because no viable cells are transferred with the graft.[45,46] There is also a theoretical risk of disease transmission from allogeneic sources of bone. Mathematically, the risk is quite small but may be of concern to patients and families.

Bone Morphogenic Protein

Bone morphogenic protein (BMP) and its use in maxillofacial reconstruction have recently become topics of interest in oral and maxillofacial surgery. The potential benefits of avoiding a secondary surgical site and obtaining the same results as those with autogenous grafting have prompted surgeons to use the material in a variety of maxillofacial bony defects. The U.S. Food and Drug Administration (FDA) has approved the use of BMP for specific maxillofacial applications in adults but has conducted no research for its use in skeletally immature patients. (INFUSE Bone Graft package insert, Medtronic).

The off-label use of BMP for alveolar cleft grafting in skeletally immature patients with cleft lip and palate has been investigated in several clinical trials.[9,11] In these trials, the investigators compared the results of cleft sites grafted with an autogeneous iliac crest bone graft (AICBG) and sites grafted with BMP on an absorbable collagen sponge (BMP/ACS) in the same sample population. The mature bone volume obtained in both groups was measured by comparing pre- and postoperative CT scans. The cleft sites grafted with BMP on an ACS resulted in bone volume that was comparable with that in group grafted with autogenous bone in both the horizontal and vertical dimensions. In addition, teeth erupted normally through these BMP-grafted sites. As expected, both the length of hospital stay and the amount of postoperative pain were less for those patients grafted with BMP than for those grafted with an AICBG.[9,11] As has been previously seen with the use of BMP in the maxillofacial region, a few patients in each study who received a BMP graft developed severe swelling in the postoperative period that was not consistent with cellulitis and that resolved without intervention.[9,11]

The preparation of the cleft site for BMP/ACS mirrors the technique used to prepare the site for a conventional bone graft. The buccal advancement flaps, palatal flaps, and nasal floor are developed followed by repair of the nasal floor and palatal tissue. The absorbable collagen sponge is cut into small squares and soaked according to manufacturer's guidelines (INFUSE Bone Graft, Medtronic). After the sponge has soaked for the recommended time period, it is placed into the defect and the buccal flaps are advanced and the four-cornered flaps are closed.

The potential advantages of using BMP are all related to eliminating a second surgical site. The patient will likely have a shorter hospital stay due to less postoperative pain. Gait and sensory disturbances can be completely avoided. Complications at a secondary surgical site are eliminated and overall surgical time may be shortened. There are several potential disadvantages of using BMP in the reconstruction of alveolar clefts. Although it resolves spontaneously, severe postoperative swelling that is unique to this material can be both worrisome to the patient as well as place tension across suture lines. In addition, the lack of structural integrity of the absorbable collagen sponge can lead to an unsupported ala.[9] Also, at this point, the refining process to manufacture BMP is extremely expensive, which makes its routine use somewhat unlikely.

The results of using an AICBG or BMP/ACS are similar in the amount of bone obtained as well as the ability to achieve dental eruption through the graft. Unfortunately, the ACS does not have the structural integrity to support the ala and the use of BMP makes this technique quite costly. At this point in time, it seems that even with the advantages that come with avoiding a secondary surgical site, autogenous bone grafts for the reconstruction of alveolar clefts continues to be the gold standard.

Pre-versus Postsurgical Orthodontics

Controversy exists regarding the use of orthopedic expansion of the cleft segments and the relationship between expansion and grafting. This issue has not been entirely settled. Most authors prefer presurgical expansion, citing easier expansion because of less resistance, improved access to the cleft for closure of the nasal floor, better postoperative hygiene, and less chance of reopening the oronasal fistula.[26,27] Presurgical expansion may also allow orthopedic movement of the



FIGURE 44-3. **A,** Occlusal photograph of a typical unilateral cleft. There is rotation of the central incisors and collapse of the lesser segment. Expansion of the lesser segment will bring the arch into better form and facilitate grafting without widening the cleft. **B,** Similar cleft that has been expanded. **C,** This cleft is already wide without any expansion. It would not be appropriate to expand this cleft before grafting even if a cross-bite is present.

premaxillary segment in the bilateral cleft patient, which can eliminate traumatic occlusion that can negatively affect graft success. Proponents of expansion after grafting cite advantages of improved bone consolidation when the graft is placed under a dynamic load during healing, a smaller soft tissue defect to close, less difficulty in procuring an adequate volume of bone, and a narrower defect, which will regenerate bone more quickly.[29] Both approaches have been used in conjunction with autogenous grafting in the mixed-dentition stage with success.[12]

In practice, both approaches are valid, and the decision should be based on individual clinical presentation.[34] Small unilateral clefts with collapse of the arch may be easier to graft with some presurgical expansion. In these cases, such expansion may not increase the size of the defect appreciably, and better alignment of the segments can improve hygiene of the teeth adjacent to the cleft and improve access (Figure 44-3). In these cases, the end point of presurgical expansion is improved arch form, not necessarily resolution of cross-bite. Bilateral clefts with collapse of the lateral segments may also benefit from presurgical expansion. Expanding the lateral segments may allow the premaxilla, which is often anteriorly positioned, to be brought back into better relation with the arch, improving arch form, and in some cases, eliminating traumatic occlusion (Figure 44-4). If this is not done before grafting, it may be difficult to obtain ideal arch

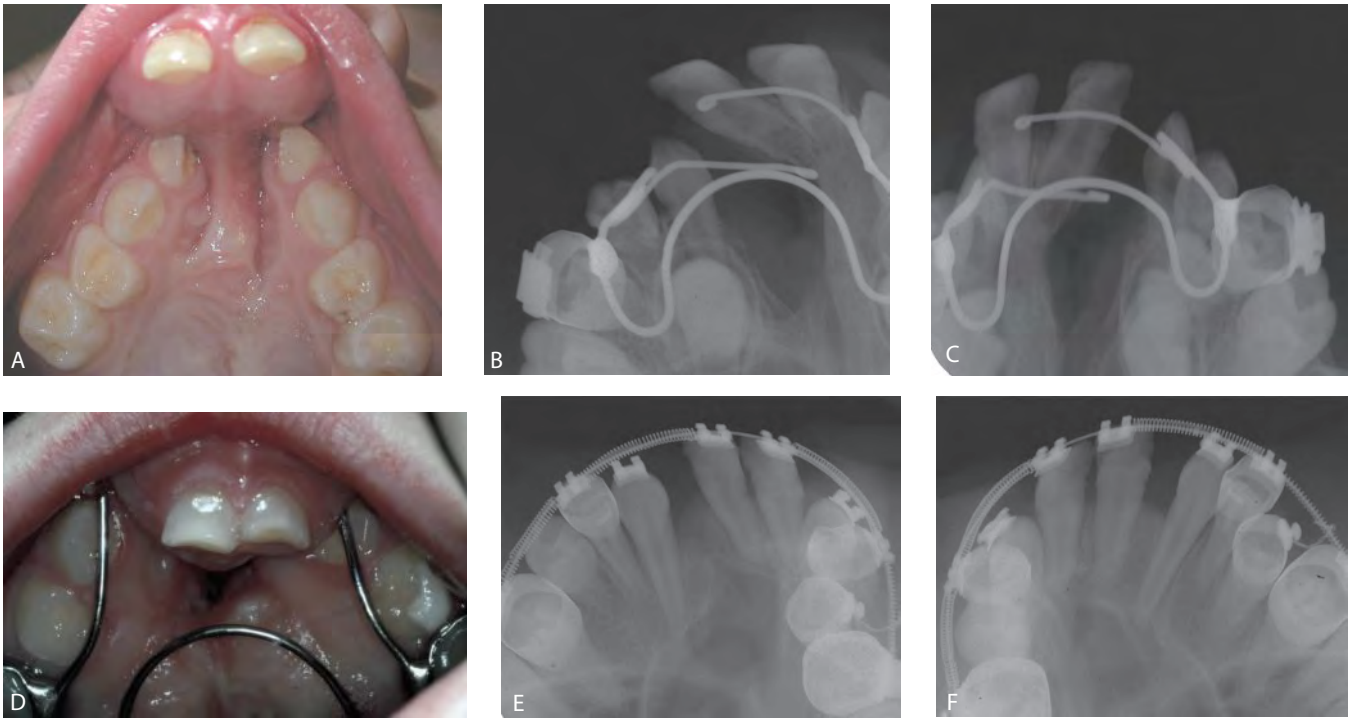


FIGURE 44-4. **A,** Occlusal photograph of bilateral cleft with collapse of the lateral segments and protrusion of the premaxilla. **B** and **C,** Right and left oblique occlusal radiographs show the cleft defect. **D,** Expansion of the bilateral cleft allows the premaxilla to move posteriorly and improves arch form and alignment of the segments. **E** and **F,** Occlusal radiographs of the same patient after grafting with eruption of the canine.

continuity and positioning of the segments after grafting may be difficult. In patients with reasonable arch form, good alignment of the segments, and dental development corresponding to ideal timing for grafting, it makes little sense to delay grafting in order to expand preoperatively, even in the presence of a buccal cross-bite. These cross-bites may be related only to the anterior-posterior discrepancy, and even if they are truly representative of transverse deficiency, they can be treated easily with expansion after the graft. These clefts can be expanded without opening the oronasal fistula or having a negative effect on the graft.

Not only is there controversy regarding pre-versus postsurgical expansion, there are also two schools of thought regarding orthodontic movement of the erupted teeth adjacent to the cleft. Some authors suggest that aligning the teeth adjacent to the cleft produces better hygiene and an improved result.[26] However, orthodontic movement of teeth adjacent to the cleft is not typically desired.[47] Orthodontic movement of teeth adjacent to the cleft before grafting increases the risk of moving these teeth into the cleft site, compromising osseous support. Studies have directly correlated the success of grafting with the presence of adequate bone on the distal surface of the central incisor preoperatively.[48] These defects cannot subsequently be grafted because the bone graft will not adhere to the tooth surface. The central incisor adjacent to the cleft site is usually rotated and angled with the crown tipped toward the cleft. This rotation and angulation decreases the mesial-distal dimension of the tooth and allows for the best bony support of the tooth (see Figure 44-2). Orthodontic forces of rotation and tipping will have the undesirable effect of increasing the mesial-distal dimension, encroaching on the bony support at the cementoenamel junction of the tooth. Orthodontic root torque to correct the angulation of the tooth will have the undesired effect of pushing the apical portion of the root toward the cleft site. The underlying osseous cleft is frequently much larger than the overlying soft tissue defect may indicate, giving a false sense of security to the orthodontist who may want to move these teeth in the absence of a graft (Figure 44-5).



FIGURE 44-5. Occlusal radiograph of a patient who had orthodontic rotation of the maxillary central incisor adjacent to the cleft before grafting the defect. There is loss of bone on the distal surface of the root to the apex. The tooth required removal.

SURGICAL TECHNIQUE FOR GRAFTING THE CLEFT ALVEOLUS

The ideal technique will meet the following criteria:

1. Predictable closure of the nasal floor produces a watertight barrier between the graft and the nasal cavity.
2. There is access to closure of residual palatal and labial fistula.
3. Keratinized attached tissue is maintained around the teeth adjacent to the cleft and in the site where the yet unerupted lateral incisor and canine will erupt.
4. Mobilization of tissue is adequate to close large defects without tension, when such defects are present.
5. The vestibule is not shortened, and scarring is not excessive.

Given these requirements, the technique most often used employs advancing buccal gingival and palatal flaps. This approach has some disadvantages including

1. Difficulty obtaining closure in large bilateral clefts.
2. Defects at the site of the releasing incision created by advancing the flap heal by secondary intention.
3. A four-corner suture line that approximates the flaps directly overlying the graft, which may lead to dehiscence.
4. The possibility that elevating large full-thickness mucoperiosteal flaps leads to growth alteration in young patients. However, when compared with finger flaps and trapezoidal flaps, which can shorten the vestibule and place nonkeratinized tissue around the dentition, this approach remains the best.

The procedure can be broken down as follows. The first step requires development of full-thickness mucoperiosteal buccal flaps (Figure 44-6). Consideration may be given to a papilla preserving incision, but this is not required in the mixed dentition. Palatal flaps are then developed, incorporating whatever residual palatal defect may be present to allow for closure of the residual palatal fistula. Some diagrams show this incision being made from a palatal approach. This may be possible in wide clefts but, in practice, is more easily accomplished by starting reflection of the palatal flaps from a sulcular incision placed on the palatal side of the dentition followed by reflection of full-thickness palatal flaps toward the palatal defect. The palatal flaps can then be separated from the nasal tissue along the cleft margin by sharp dissection with scissors from the anterior extending posteriorly as the flaps are elevated (Figure 44-7A). In this manner, the maximum palatal soft tissue is preserved for closure, while assuring adequate nasal mucosa to obtain a watertight nasal closure. Once the buccal and palatal flaps have been developed, access is readily obtained to the nasal mucosa, which is then reflected and sutured, burying the knots to obtain a watertight nasal closure (see Figure 44-7B and C). Most schematic diagrams of cleft closure show this portion of the procedure being performed from the palatal aspect. However, it is generally most readily accomplished in narrow clefts from the anterior through the cleft defect. Once the nasal

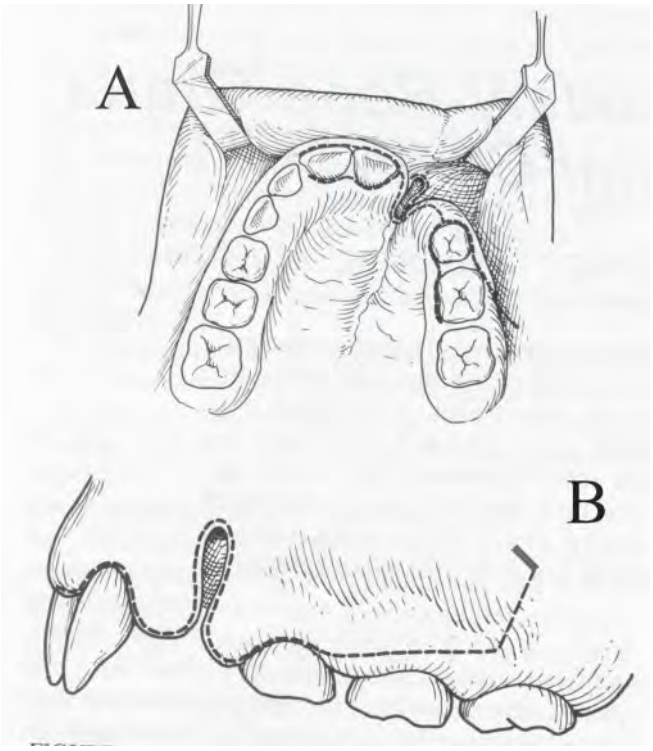


FIGURE 44-6. **A** and **B,** Sulcular incision used to develop sliding flaps for closure over the graft. (**A** and **B,** Adapted from Hall HD, Posnick JC. Early results of secondary bone grafts in 106 alveolar clefts. J Oral Maxillofac Surg 1983;41:289–294.)

mucosa is closed, the palatal defect is closed by first closing the palatal flaps, converting the cleft palate into a single flap (see Figure 44-7D). The graft material is then placed into the cleft from the anterior, making certain to fill all voids completely to the piriform rim. Graft material can be condensed using an orthodontic band pusher or periosteal elevator (see Figure 44-7E). It is helpful to place a malleable retractor to protect the nasal floor as the bone is packed into place. Finally, the labial flaps can be advanced, and they are sutured to each other and then to the palatal flap, producing the classic four-corner closure over the crest of the ridge (see Figure 44-7F and G). In most cases, the sliding flaps will be advanced one papilla on either side of the cleft, or in some cases, only a single papilla advancement from the posterior segment is necessary. It may be necessary to perform a small backcut or to release or score the periosteum to obtain a tension-free closure. It is best to use a resorbable monofilament suture.

A palatal stent can be used to stabilize the cleft and protect the soft tissue closure. This may compromise hygiene and blood supply to the palatal flaps and, in most cases, is not required for success. In the bilateral cleft, if there is a traumatic occlusion to the anterior maxillary dentition, a mandibular bite plane is helpful to open the bite and prevent mobility of the premaxilla.

It is appropriate to use intraoperative antibiotics. Previous studies show that graft success and incidence of infection are not improved by the use of postoperative antibiotics.[49] Some surgeons feel more comfortable with a 1-week course of antibiotics, particularly when the soft tissue closure is questionable.

The postoperative diet should be limited to full liquids for approximately 5 days. This can be advanced to a soft mechanical diet. However, it is critical that the patient refrain from incising food with the anterior dentition; rather the patient should cut food into small pieces and masticate primarily on the posterior teeth. In bilateral cases, this is particularly important because any trauma to the premaxilla will cause mobility of the segment, leading to graft failure. Radiographic evidence of graft consolidation should be visible within 8 weeks. The surgeon should confirm successful consolidation of the graft before any orthodontic manipulation of the teeth adjacent to the cleft.

OVERVIEW

This chapter has outlined historic benefits of grafting, discussed many of the controversies, and provided data on the benefits and disadvantages of several approaches.

The following is a stepwise approach to managing the alveolar cleft from one perspective:

1. At age 5 to 6 years, an orthodontic evaluation is performed. The ability of the patient to cooperate with orthodontic treatment is assessed, the arch is evaluated for collapse, and erupted supernumerary teeth in the area of the cleft are identified. Radiographic examination should include a panoramic film as well as an intraoral view that allows detailed evaluation of the cleft site. Periapical films can be used for this, but a lateral oblique occlusal film is best. An occlusal film is placed in the standard position while directing the beam obliquely to the midline along the long axis of the cleft (Figure 44-8).
2. If erupted supernumerary teeth are identified in the area of the cleft, these are extracted now or, at a minimum, 8 weeks before the graft (Figure 44-9A).
3. Orthodontic expansion is performed if there are specific goals that can be met before grafting. These would include decreasing traumatic occlusion to the premaxillary segment in bilateral cleft patients and correcting arch collapse that will compromise grafting. No attempt is made to correct the cross-bite at this stage, and there is no attempt to orthodontically correct rotation of the permanent central incisor (see Figure 44-9A).
4. The alveolar cleft is grafted when the patient is between 6 and 8 years of age. Two teams perform the surgery with graft harvest from the iliac crest simultaneous with the cleft closure.
5. The graft is evaluated with a lateral oblique occlusal radiograph 3 months after surgery (see Figure 44-9B).
6. Final orthodontic expansion is performed if indicated, and permanent incisor teeth are then rotated into proper alignment.

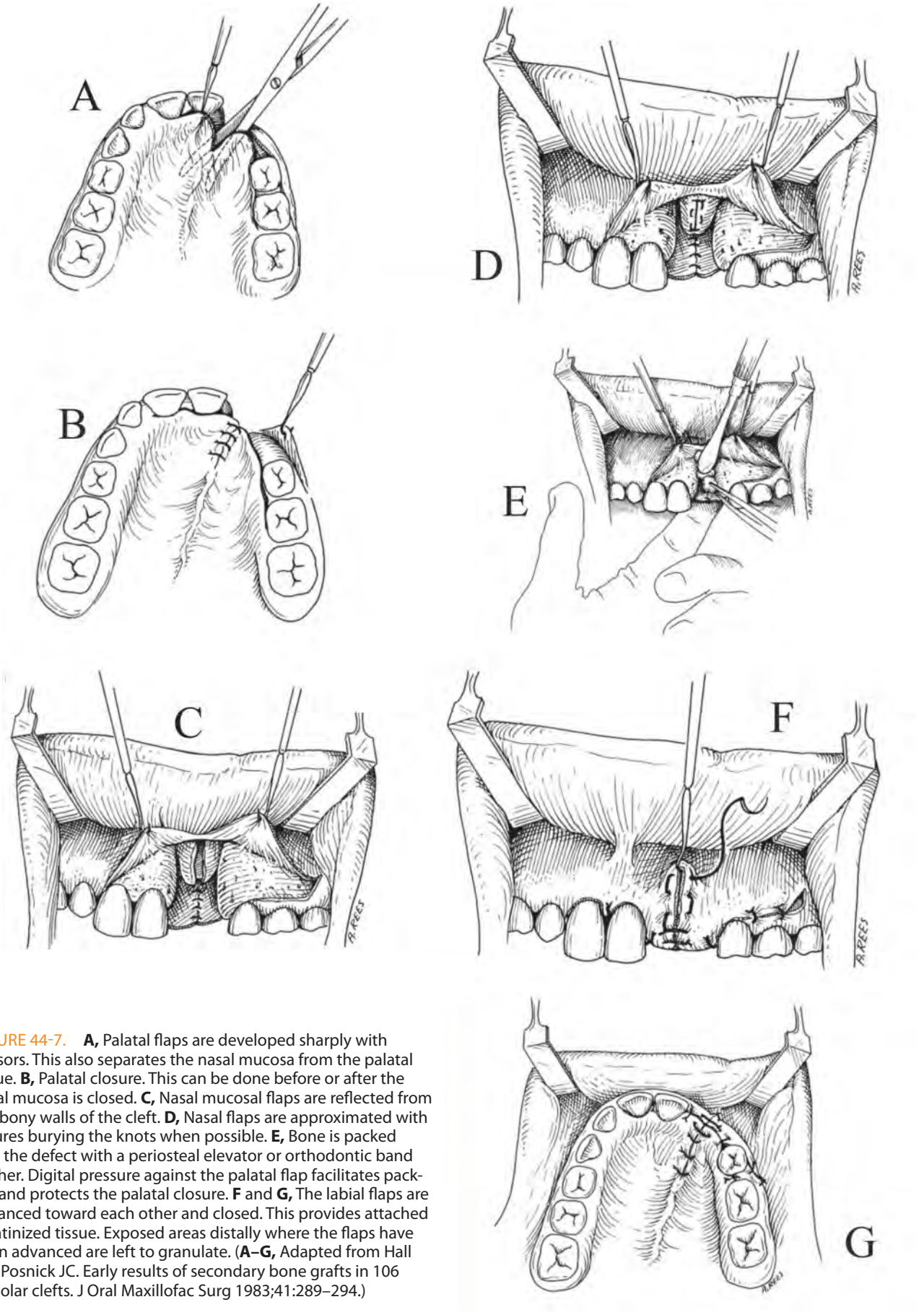


FIGURE 44-7. **A,** Palatal flaps are developed sharply with scissors. This also separates the nasal mucosa from the palatal tissue. **B,** Palatal closure. This can be done before or after the nasal mucosa is closed. **C,** Nasal mucosal flaps are reflected from the bony walls of the cleft. **D,** Nasal flaps are approximated with sutures burying the knots when possible. **E,** Bone is packed into the defect with a periosteal elevator or orthodontic band pusher. Digital pressure against the palatal flap facilitates packing and protects the palatal closure. **F** and **G,** The labial flaps are advanced toward each other and closed. This provides attached keratinized tissue. Exposed areas distally where the flaps have been advanced are left to granulate. (**A–G,** Adapted from Hall HD, Posnick JC. Early results of secondary bone grafts in 106 alveolar clefts. J Oral Maxillofac Surg 1983;41:289–294.)

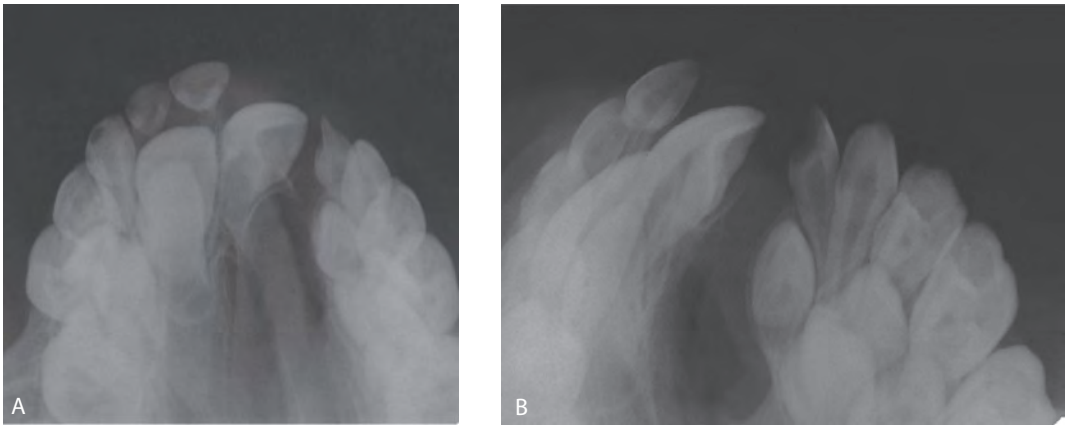


FIGURE 44-8. **A,** Traditional maxillary occlusal radiograph. The cleft is identifiable, but overlap of the bone makes it difficult to determine the size of the defect and relationship of the teeth to the defect. **B,** Oblique occlusal radiograph is exposed by directing the beam obliquely to the midline, along the long axis of the cleft. Note that the morphology of the cleft is better identified, as is the relationship of unerupted teeth to the defect.

7. Conventional orthodontic treatment is performed at a more traditional age, after eruption of the remaining permanent dentition. Patients are periodically monitored for eruption of the canine in the cleft. Some authors have indicated that in 30% to 73% of patients, eruption of the canine into the alveolar graft requires surgical uncovering of the tooth or uncovering and orthodontic assistance.[15,50,51] Others have reported that nearly all of these teeth can be expected to erupt without surgical intervention[27] (see Figure 44-9C). If uncovering is necessary, techniques to preserve attached tissue are used as would be appropriate for impacted canines in noncleft patients.
8. Missing lateral incisors are managed with space development and implant placement as opposed to canine substitution. This is accomplished after definitive orthodontic treatment and orthognathic surgery, if indicated, after maxillary growth is complete. Even when bone height is adequate and teeth adjacent to the graft have good support, the graft undergoes resorption, resulting in a narrow ridge. This is not unlike the bone resorption found with congenitally absent lateral incisors in noncleft patients. Successful implant restoration is possible, but further grafting is likely needed before adequate labial-palatal width is available for implant placement[33] (Figure 44-10). Attention to soft and hard tissue is critical in these patients to achieve aesthetic results.

CONCLUSION

Restoration of the cleft alveolus and maxilla by grafting is a critical part of the overall management of the patient with cleft palate. A systematic approach can improve predictability. This is best accomplished during the mixed-dentition stage. Adjunctive expansion may be accomplished before or after grafting, depending on the needs of the patient.

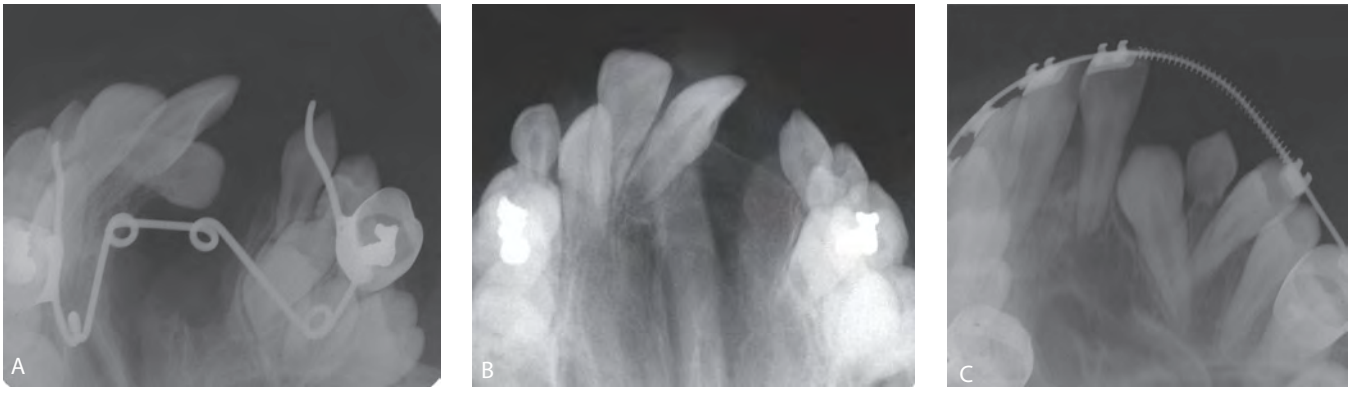


FIGURE 44-9. **A,** The cleft has been expanded. The canine is in position to begin eruption. There is a supernumerary/malformed lateral incisor erupting horizontally into the cleft. **B,** The supernumerary tooth has been removed. The defect was grafted 2 months after extraction and the film shows good bone consolidation. **C,** The maxillary canine can be seen erupting into the graft.

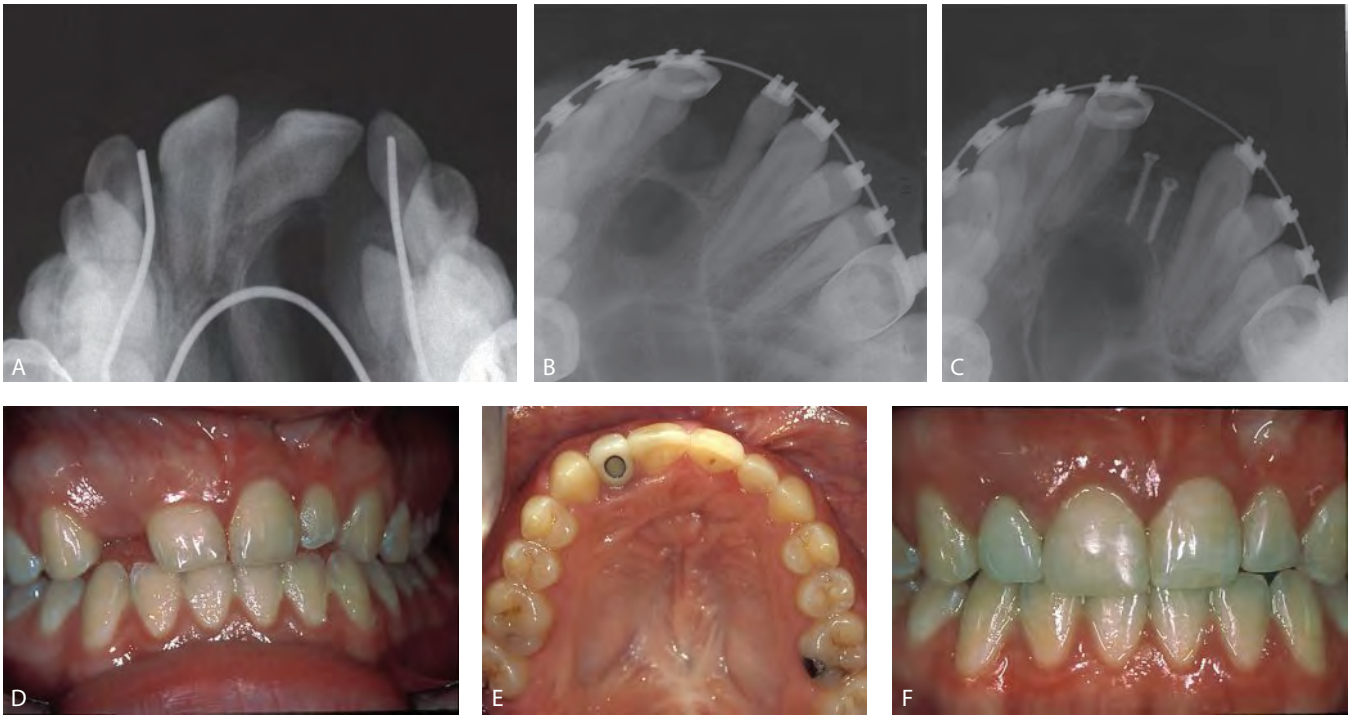


FIGURE 44-10. **A,** Occlusal radiograph shows thin bridge of bone with inadequate height. A malformed lateral incisor was maintained to preserve as much width as possible. **B,** The tooth was removed. **C,** After 2 months of healing, an onlay bone graft was placed. **D,** Clinical view after grafting. **E** and **F,** View after placement of the implant and reconstruction.

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CHAPTER

Nonsyndromic Craniosynostosis

G. E. Ghali, DDS, MD, and Andrew R. Banker, DDS, MD

In its basic form, craniosynostosis represents premature suture fusion. It occurs in approximately 1 per 1000 live births in the United States. Craniosynostosis may be classified as *nonsyndromic* or *syndromic*. Most forms of craniosynostosis are isolated and not associated with any other conditions and are, therefore, nonsyndromic. Syndromic craniosynostosis is covered in Chapter 46. The pathogenesis of craniosynostosis is complex and probably multifactorial.[1] Moss theorized that craniosynostosis such as seen in Apert's and Crouzon's syndromes results from abnormal tensile forces transmitted to the dura from an anomalous cranial base through key ligamentous attachments.[2] This hypothesis fails to explain craniosynostosis in patients with a normal cranial base configuration. The cause of craniosynostosis may be postulated to be the result of either primary suture abnormalities, sufficient extremes of forces that overcome the underlying expansive forces of the brain, inadequate intrinsic growth forces of the brain, or various genetic and environmental factors.[3] Cranial vault growth achieves approximately 80% of the adult size at birth and definitive size by 2.5 to 3 years of age.[4] The existence of the six major sutural regions allows for head expansion as well as transvaginal head deformation.[5] Recall that posterior fontanelle closure (3–6 mo) generally precedes anterior fontanelle closure (9–12 mo).

FUNCTIONAL CONSIDERATIONS

The major functional problems associated with craniosynostosis are intracranial hypertension, visual impairment, limitation of brain growth, and neuropsychiatric disorders.[6] In general, the functional problems increase as the number of sutures involved increases.[6] These functional abnormalities are gradual in their development, difficult to detect, and often irreversible in nature.

Intracranial Hypertension

Intracranial hypertension is defined as a pressure of greater than 15 mmHg. Studies by Marchac and Renier[7] have demonstrated a 13% incidence of intracranial hypertension with single-suture stenosis and up to a 42% incidence in multisuture-stenosed children. The clinical symptoms of intracranial hypertension include headaches, irritability, and difficulty sleeping. The radiographic signs may include cortical thinning or a lückenschädel (hammered metal) appearance of the inner table of the skull; these clinical and radiographic signs are relatively late developments. If intracranial hypertension goes untreated, it affects brain function; if persistent, this may necessitate early operative intervention during the first few months of life. Intracranial hypertension most likely affects those with the greatest disparity between brain growth and intracranial capacity and may occur in as many as 42% of untreated children with more than one suture affected. Currently, intracranial volume is measured using computed tomography (CT) scans, a noninvasive method appropriate for use in children with craniosynostosis.[8] It might be possible to identify individuals who are at a greater risk for developing intracranial hypertension and would benefit the most from early surgery.

Visual Impairment

Untreated intracranial hypertension in the acute phase may lead to papilledema, and chronically, to some degree of optic atrophy causing a partial to complete blindness.[9] Some forms of craniosynostosis may involve orbital hypertelorism and may lead to compromised visual acuity and restricted binocular vision.

Limitation of Brain Growth

Brain volume in the normal child almost triples during the first year of life. By 2 years of age, the cranial capacity is four times that at birth. If brain growth is to proceed unhindered, open sutures at the level of the cranial vault and base must spread during phases of rapid growth for marginal ossification.

In craniosynostosis, premature suture fusion is combined with continuing brain growth. Depending on the number and location of prematurely fused sutures and the timing of closure, the growth potential of the brain may be limited. Surgical intervention can provide suture release and reshaping to restore a more normal intracranial volume. In general, this does not completely reverse craniosynostosis and diminished volume is often the end result.

Neuropsychiatric Disorders

Neuropsychiatric disorders are believed to be secondary to cerebral compression. Disorders range from mild behavioral disturbances to overt mental retardation. It has been shown that children with craniosynostosis and associated neuropsychiatric disorders often improve after cranial vault reconstruction.[10]

DIAGNOSIS

One should suspect craniosynostosis in any infant with an abnormal head shape. Definitive diagnosis is based on clinical and radiographic evaluations. The clinical evaluation involves the palpation of the skull for any movement, ridging, and presence of the anterior and posterior fontanelles. Quantitative measurements of the superior orbital rims, relative to the most anterior aspect of the cornea, also may help in planning treatment for superior orbital rim advancements.

The radiographic evaluation of craniosynostosis is used to define quantitatively aberrant anatomy, plan surgical procedures, and most important, provide a means to demonstrate to the parents the difference between stenosed and nonstenosed sutures. Conventional skull radiographs, such as plain skull films and lateral cephalograms, are inexpensive and widely available. The preoperative assessment of patients with suspected or known craniosynostosis is based on these conventional radiographs. Most cases of synostosis can be demonstrated on plain skull films. Normal or patent cranial sutures manifest as a line. The absence of a radiolucent line in the normal anatomic position of a suture may suggest craniosynostosis.

Currently, CT scans provide improved hard tissue imaging. The definition of these elements of the bony facial structures on high-resolution CT images with or without three-dimensional reconstruction is unmatched by other imaging techniques (Figure 45-1). The development of CT scanning, particularly three-dimensional reformatting, and the maturation of readily available means of craniofacial surgery have led to a close dependence on CT scanning for preoperative surgical planning. CT scanning also has been used to document surgical changes in vivo and to follow developments longitudinally.[11–22]

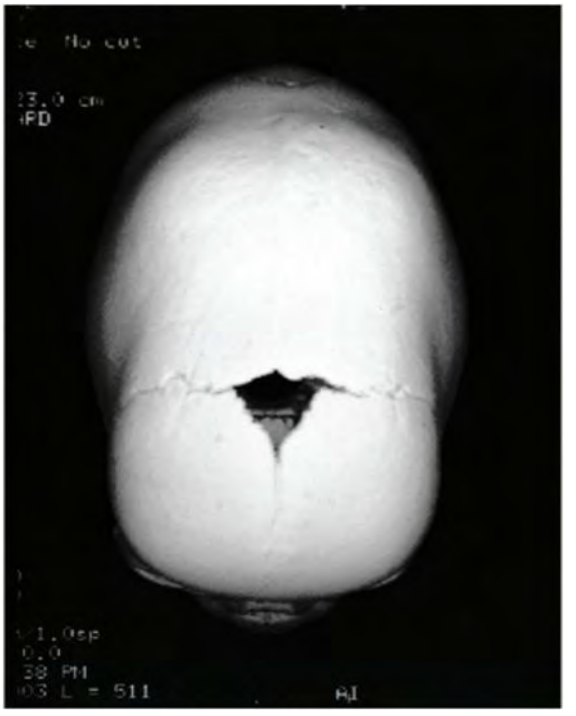


FIGURE 45-1. Computed tomography scan with three-dimensional reconstruction. Patency of metopic and coronal sutures as well as anterior fontanelle; premature fusion of sagittal suture. (Reproduced with permission from Ghali GE, Sinn DP, Tantipasawasin S. Management of nonsyndromic craniosynostosis. Atlas Oral Maxillofac Surg Clin North Am 2002;10:3.)

CLASSIFICATION

The classification of craniosynostosis is based on the shape of the skull, which usually reflects the underlying prematurely fused suture or sutures.[23,24] The major cranial vault sutures that may be involved include the left and right coronal, metopic, sagittal, and lambdoid.

Unilateral Coronal Synostosis

Unilateral coronal synostosis results in flatness on the ipsilateral side of the forehead and supraorbital ridge region. The head is inherently asymmetrical in shape with a flattened or retropositioned forehead on the ipsilateral side, especially when viewed from the top (Figure 45-2). The term for this deformity is *anterior plagiocephaly.* One should rule out infant molding or positional plagiocephaly and congenital torticollis as other possible diagnoses. Premature fusion of the unilateral coronal suture represents 20% of the isolated or nonsyndromic cases of synostosis in the United States. Characteristic morphologic features occur on the ipsilateral side. The frontal bone is flat, and the supraorbital ridge and lateral orbital rim are recessed (Figure 45-3). The orbit is shallow,

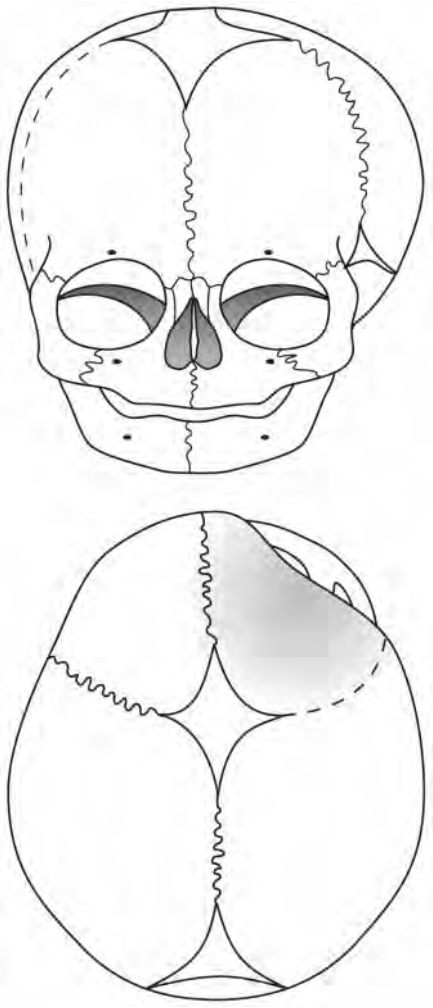


FIGURE 45-2. Frontal and superior views. Characteristic right anterior plagiocephaly. (Adapted from Ghali GE, Sinn DP, Tantipasawasin S. Management of nonsyndromic craniosynostosis. Atlas Oral Maxillofac Surg Clin North Am 2002;10:4.)



FIGURE 45-3. Superior view. Recessed frontal bone, supraorbital ridge, and lateral orbital rim on patient's right side. (Reproduced with permission from Ghali GE, Sinn DP, Tantipasawasin S. Management of nonsyndromic craniosynostosis. Atlas Oral Maxillofac Surg Clin North Am 2002;10:5.)

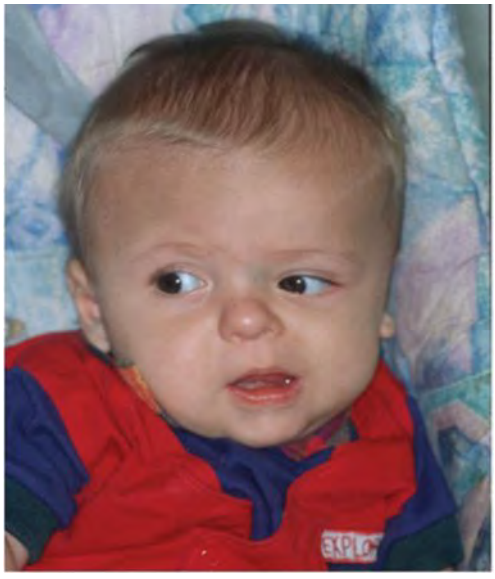


FIGURE 45-4. Frontal view. Nasal bridge and root deviation to the affected (right) side characteristic in plagiocephaly. (Reproduced with permission from Ghali GE, Sinn DP, Tantipasawasin S. Management of nonsyndromic craniosynostosis. Atlas Oral Maxillofac Surg Clin North Am 2002;10:5.)

and the anterior cranial base is short in the anteroposterior dimension. The root of the nose may be constricted and deviated to the affected side (Figure 45-4). The ipsilateral zygoma and infraorbital rim also may be flat and recessed.

Bilateral Coronal Synostosis

Bilateral coronal synostosis is the most common cranial vault suture synostosis pattern associated with Apert's and Crouzon's syndromes. Bilateral coronal synostosis results in recession of the supraorbital ridges, which causes the overlying eyebrows to sit posterior to the corneas. In addition to the recessed supraorbital bone, the forehead appears to be lower and there is sagittal shortening of the skull (Figure 45-5). The term for this cranial vault deformity is *brachycephaly*. The anterior cranial base is short in the anteroposterior dimension and wide transversely. The overlying cranial vault is high in the superoinferior dimension, with anterior bulging of the upper forehead that results from compensatory growth of the patent metopic suture (Figure 45-6). The orbits are also shallow (exorbitism), with the eyes bulging (exophthalmus) and abnormally separated (orbital hypertelorism). Brachycephaly represents 20% of the isolated craniosynostosis cases in the United States and is the most common syndrome-associated synostosis.

Metopic Synostosis

Metopic synostosis usually occurs in isolation and results in a triangular shape to the skull (Figure 45-7). The term for this cranial vault deformity is *trigonocephaly*. The associated cranial vault deformity consists of relative hypotelorism, an elevated supraorbital ridge medially, and posteroinferior

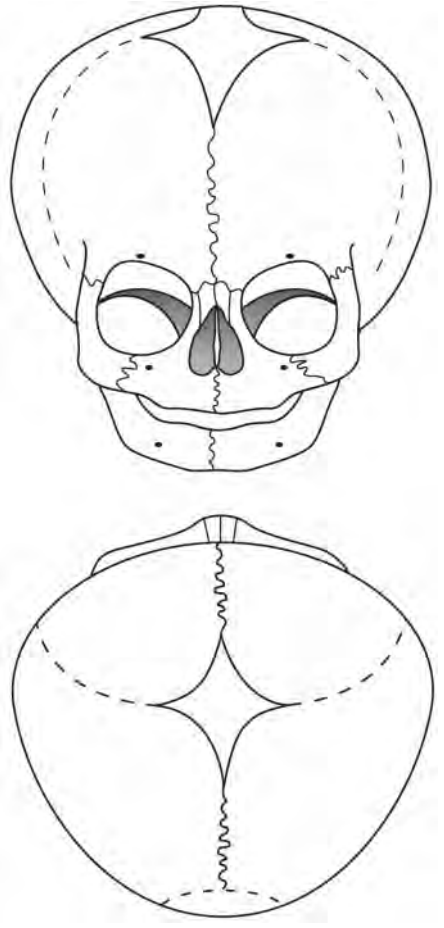


FIGURE 45-5. Frontal and superior views. Characteristic brachycephaly. (Adapted from Ghali GE, Sinn DP, Tantipasawasin S. Management of nonsyndromic craniosynostosis. Atlas Oral Maxillofac Surg Clin North Am 2002;10:4.)



FIGURE 45-6. Frontal view. High cranial vault and transverse widening characteristic of brachycephaly. Note visible ridging in the bicoronal suture region. (Reproduced with permission from Ghali GE, Sinn DP, Tantipasawasin S. Management of nonsyndromic craniosynostosis. Atlas Oral Maxillofac Surg Clin North Am 2002;10:7.)

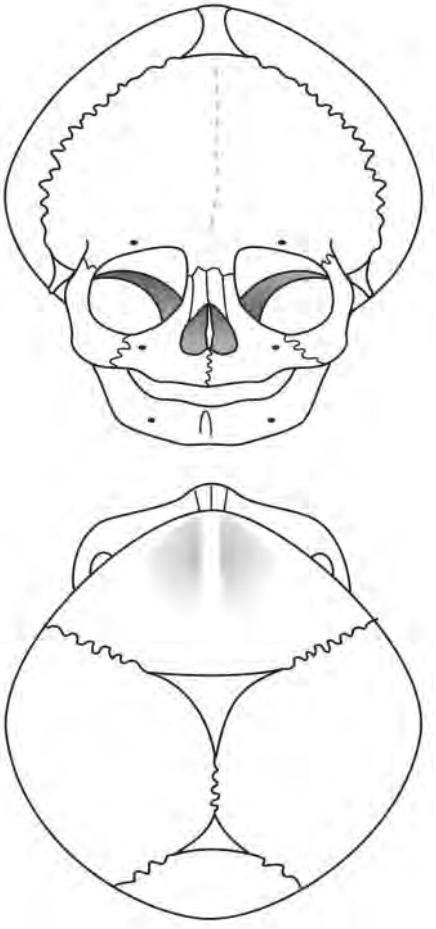


FIGURE 45-7. Frontal and superior views. Characteristic trigonocephaly. (Adapted from Ghali GE, Sinn DP, Tantipasawasin S. Management of nonsyndromic craniosynostosis. Atlas Oral Maxillofac Surg Clin North Am 2002;10:8.)

recession of the lateral orbital rims and lateral aspect of the supraorbital ridges. Palpation often reveals a prominent midline keel in the region of the metopic suture (Figure 45-8). The bitemporal width is decreased, which results in an abnormal anterior cranial vault shape and decreased anterior cranial vault volume. The overlying forehead is sloped posteriorly to approximately the level of the coronal sutures. Trigonocephaly represents 10% of the nonsyndromic craniosynostosis cases in the United States.

Sagittal Synostosis

Sagittal synostosis, the most common form, is rarely associated with increased intracranial pressure. The skull typically has anteroposterior elongation with a compensatory transverse narrowing (Figure 45-9). The term for this cranial vault deformity is *scaphocephaly.* The deformity consists of an elongated anteroposterior dimension and a narrow transverse dimension to the cranial vault (Figure 45-10). Usually, the midface and anterior cranial vault sutures are not affected. Scaphocephaly represents 50% of all single-suture craniosynostosis cases in the United States.



FIGURE 45-8. Intraoperative view after shaving of the head demonstrates prominent midline ridging associated with trigonocephaly. (Reproduced with permission from Ghali GE, Sinn DP, Tantipasawasin S. Management of nonsyndromic craniosynostosis. Atlas Oral Maxillofac Surg Clin North Am 2002;10:9.)



FIGURE 45-10. Intraoperative view after shaving of the head demonstrates prominent sagittal suture ridging associated with scaphocephaly. (Reproduced with permission from Ghali GE, Sinn DP, Tantipasawasin S. Management of nonsyndromic craniosynostosis. Atlas Oral Maxillofac Surg Clin North Am 2002;10:11.)

Unilateral Lambdoid Synostosis

Unilateral lambdoid synostosis results in flatness of the affected ipsilateral parietooccipital region. The location of the ear canal and external ear is more posterior and inferior on the ipsilateral side than on the contralateral side. This configuration is more noticeable when the patient is examined from the superior view and is relatively inconspicuous from the frontal or profile views. The term for this cranial vault deformity is *posterior plagiocephaly.* One should rule out infant molding and congenital torticollis (Figure 45-11). With positional (or deformational) plagiocephaly, the ipsilateral ear and forehead are positioned anteriorly, and the ear is not inferiorly displaced as it is with true unilateral lambdoid fusion. The use of head-molding helmet therapy has received renewed interest in the past decade as the preferred treatment of children with positional head shape abnormalities.[25,26] The overall incidence of true unilateral lambdoid synostosis is less than 3% of all isolated synostosis cases in the United States.

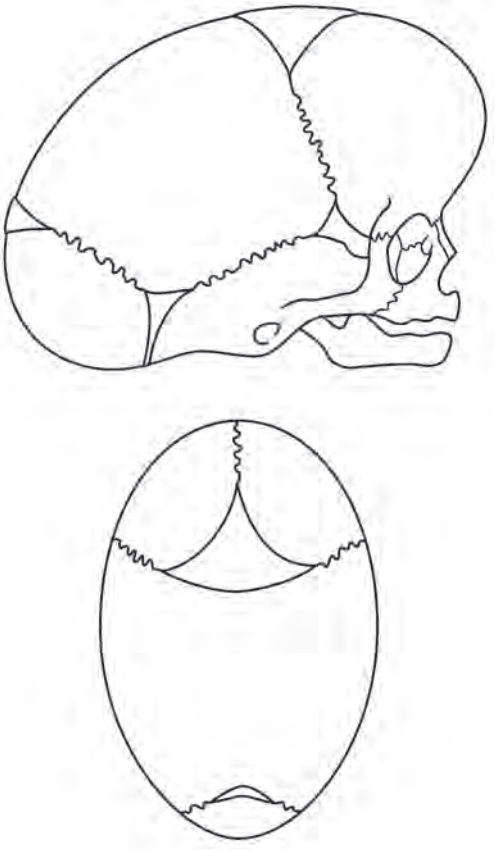


FIGURE 45-9. Lateral and superior views. Characteristic scaphocephaly. (Adapted from Ghali GE, Sinn DP, Tantipasawasin S. Management of nonsyndromic craniosynostosis. Atlas Oral Maxillofac Surg Clin North Am 2002;10:10.)

PRINCIPLES OF MANAGEMENT

Multidisciplinary Team Approach

The multidisciplinary team approach was developed in response to the failures that commonly occurred when various aspects of care were not coordinated and when the relationships among coexisting problems were not known.[4,27] The objectives of this approach are diagnosis, formulation, and execution of treatment plans and longitudinal follow-up for patients with craniofacial deformities; the team should meet at least monthly for regular outpatient evaluations. Transcripts of these evaluations are forwarded with recommendations to primary care providers and appropriate agencies. Children younger than 5 years are usually evaluated annually, whereas children older than 5 years are seen every other year.



FIGURE 45-11. Frontal **(A)** and superior **(B)** views of a child with positional plagiocephaly. Radio-graphic and clinical examination demonstrated no evidence of craniosynostosis; note asymmetry of external ear position.

The frequency of evaluation varies with the stability of the deformity and its consequences.

The craniofacial team should consist of a pediatric anesthesiologist, a pediatric ophthalmologist, a surgeon, an audiologist, a maxillofacial prosthodontist, an orthodontist, a psychologist, a geneticist, an otolaryngologist, a pediatrician, social workers, a speech pathologist, and a nurse.[28–32] All these team members have integral roles at various times in the child's development.[33,34]

Current Surgical Approach

The goals of craniosynostosis suture release are twofold. The first goal is to allow the brain to grow and expand without restriction. The second goal is to establish a more normal contour to the forehead, supraorbital ridges, and skull.[35–37] In most cases, an intracranial approach is used for cranial vault and orbital osteotomies, with reshaping and advancement of bony segments for ideal age-appropriate bony morphology. When planning the time and type of surgical intervention, one must consider the functions, future growth and development of the craniofacial skeleton, and the maintenance of normal body image. Simple craniosynostosis can be managed successfully with frontocranial remodeling.

Although the timing of craniosynostosis repair remains controversial and individualized, we prefer early surgical repair between the ages of 4 and 8 months.[38,39] Early surgical repair allows for rapid frontal lobe growth, which supports the forehead and supraorbital ridge advancement. At this age, the cranium is highly malleable and, therefore, easier to contour; a positive effect on facial growth may be achieved and future deformities may be lessened. Also, during this period of rapid growth, residual bony defects heal more rapidly. In severe forms of craniosynostosis, additional revision of the cranial vault and orbit is necessary during infancy or early childhood to increase intracranial volume further, which allows for continued brain growth and avoids or reduces the likelihood of intracranial hypertension.

A craniotomy is performed by a pediatric neurosurgeon to remove the deformed section of cranium and provide access for the additional craniofacial osteotomies. The skeletal segments are reshaped, replaced into position, and stabilized with the use of resorbable plates and screws. These plates, which are composed of polylactic and polyglycolic acid, are completely resorbed by hydrolysis within 9 to 14 months while maintaining tensile strength for initial stabilization.[40–43] As a result, growth restrictions are minimized as is the potential for transcranial migration.

SURGICAL CONSIDERATIONS

Unilateral Coronal Synostosis

Multiple surgical approaches for the correction of unilateral coronal synostosis (Figures 45-12 and 45-13) have been described.[44–46] Good long-term results are obtained when treatment of coronal synostosis includes suture release along with cranial vault and orbital osteotomies for reshaping and advancement in infancy. At the Louisiana State University Health Sciences Center in Shreveport, unilateral orbital

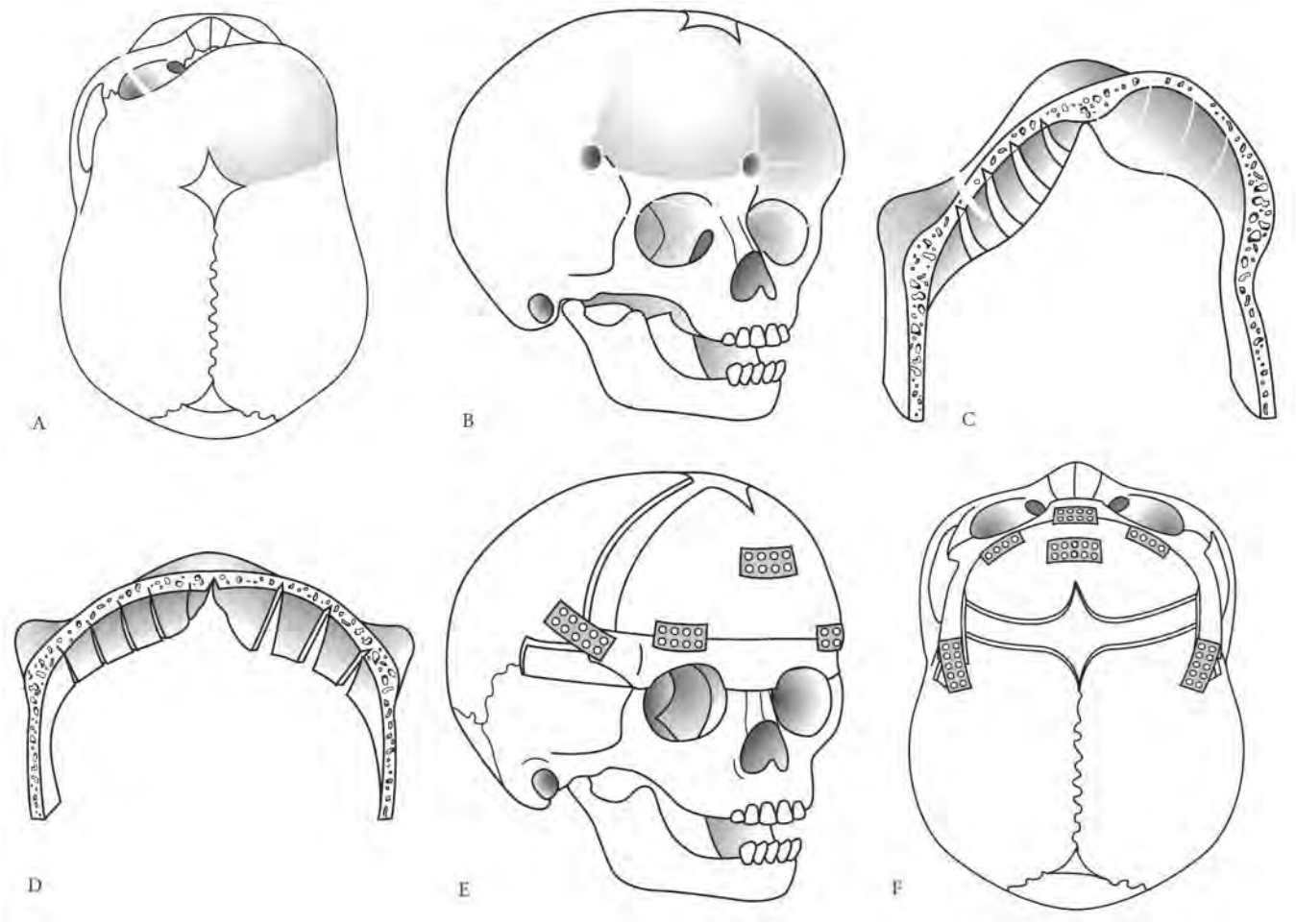


FIGURE 45-12. A child with unilateral coronal synostosis resulting in left-sided anterior plagiocephaly. **A,** Asymmetrical forehead and orbit viewed from above. Note marked left supraorbital retrusion and right forehead and cranial vault bulging. **B,** Bur holes prepared for bifrontal craniotomy at the level of the supraorbital region, allowing a 1 cm fronto-orbital unit (bandeau), which extends into the temporal fossa via tongue-in-groove (tenon) extensions. Note that the degree of extension into the lateral and inferior orbital rims is variable based on aesthetics. **C,** The removed bandeau is contoured bilaterally via removal of wedges from the left orbital roof and scoring the right orbital roof. **D,** The bandeau is reshaped to achieve symmetry by bending the left side and straightening the right side. **E** and **F,** Stabilization of forehead and bandeau achieved via resorbable plates and screws. (A–F, Adapted from Ghali GE, Sinn DP, Tantipasawasin S. Management of nonsyndromic craniosynostosis. Atlas Oral Maxillofac Surg Clin North Am 2002;10:14–15.)

rim advancement and frontal bone reshaping are ideally performed at 6 to 8 months of age. Other centers have reported good results when treatment is provided between the ages of 2.5 and 3 years. To achieve optimal symmetry, we prefer to use a bilateral surgical approach. Symmetry of the cranial vault and orbit must be achieved during surgery because results generally do not improve over time. Stabilization is achieved by using direct intraosseous wires or resorbable plates and screws.

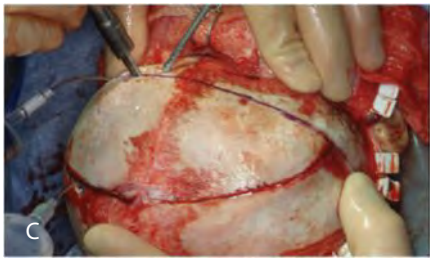
Bilateral Coronal Synostosis

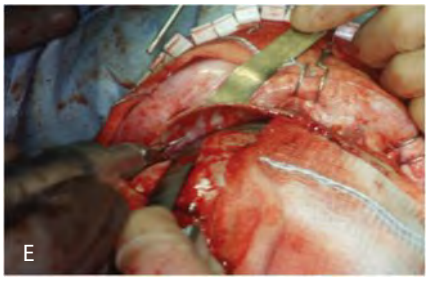
The treatment of bilateral coronal synostosis (Figures 45-14 and 45-15) requires suture release and simultaneous bilateral orbital rim and frontal bone advancements.[47,48] Surgery is performed when the patient is between 6 and 8 months of age. Other centers have reported good results with children treated between the ages of 2.5 and 3 years of age. The osteotomies for the bilateral orbital rim advancement are made superior to the nasofrontal and frontozygomatic sutures and extend to the squamous portion of the temporal bone. Stabilization is achieved with direct transosseous wires or resorbable plates and screws. The more normalized shape provides the needed increase in intracranial volume within the anterior cranial vault.

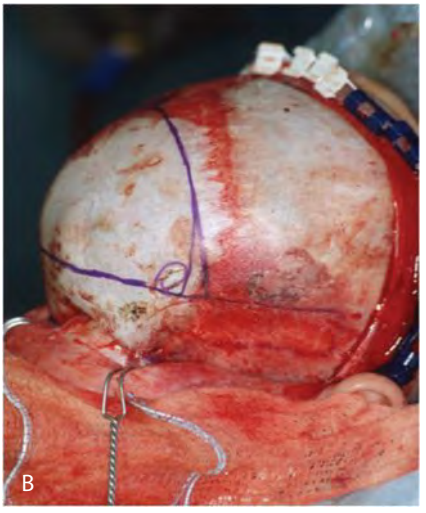
Metopic Synostosis

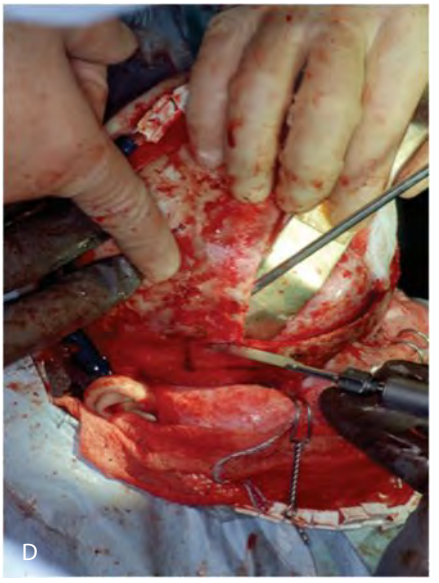
Surgical treatment of metopic synostosis (Figures 45-16 and 45-17) involves metopic suture release, simultaneous bilateral orbital rim advancements, and lateral widening via frontal bone advancement. These procedures are usually performed at 6 to 8 months of age.[49] Orbital hypotelorism is corrected by splitting the supraorbital ridge unit vertically in















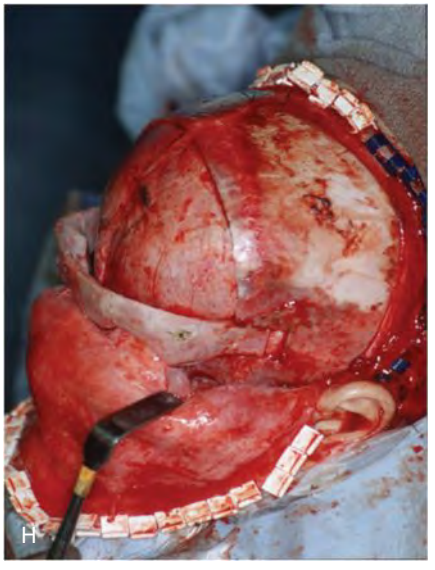


FIGURE 45-13. **A,** A 6-month-old patient with right anterior plagiocephaly placed in the supine position and the head secured in a Mayfield headrest. A coronal incision is used and the anterior scalp flap is elevated subperiosteally along with the temporalis muscle. Extension may be carried pre- or postauricular as needed. **B,** Subperiosteal dissection is achieved bilaterally circumferentially in the periorbital, lateral canthal, lateral orbital, and zygomatic buttresses. Care is taken to maintain the integrity of the medial canthal ligaments. Posterior scalp flap is dissected subperiosteally to between the coronal and the lambdoid sutures. Area of proposed bifrontal craniotomy and bur holes are marked. **C,** Neurosurgeon performs bifrontal craniotomy using a Midas Rex drill. **D,** Frontal and temporal lobes of the brain are gently repositioned to perform upper orbital and temporal osteotomies through the skull base. Reciprocating saw is used to perform bilateral tongue-in-groove extensions from external approach to the level of pterion. **E,** Attention is turned to the anterior skull base osteotomy and the saw is directed internally across the skull base anterior to the olfactory bulbs while retracting the frontal lobe. **F,** In addition to frontal lobe retraction, the orbital contents must be protected via retraction at this time. The level of the osteotomy at the lateral orbital rim is customized as needed from as high as the frontozygomatic suture to as low as the lateral aspect of the orbital floor into the inferior orbital fissure. **G,** Bandeau has been removed and asymmetry noted before reshaping. **H,** Left oblique view after remodeling and recontouring of the bandeau but before frontal bone placement. Resorbable plates and screws are used for fixation. *(Continued)*



FIGURE 45-13. *(Continued)* **I,** Retraction of bifrontal lobes demonstrates differential degree of advancement on the right side at anterior skull base. **J,** Superior view of anterior cranial vault before reshaping. **K,** Superior view of the anterior cranial vault after osteotomies, reshaping, and resorbable plate and screw fixation of the bone segments. Barrel-staving cuts may be made in the temporal and parietal bones as needed for reshaping purposes. **L,** A 6-month-old boy with right unilateral plagiocephaly. He underwent anterior cranial vault and bilateral superior orbital rim osteotomies with reshaping and advancement by the procedure described. Preoperative frontal view is shown. **M,** Frontal view 6 weeks after reconstruction. **N,** Preoperative superior view. **O,** Superior view 6 weeks after reconstruction. **P,** Frontal view 2 years after reconstruction. **Q,** Superior view 2 years after reconstruction. *(Continued)*



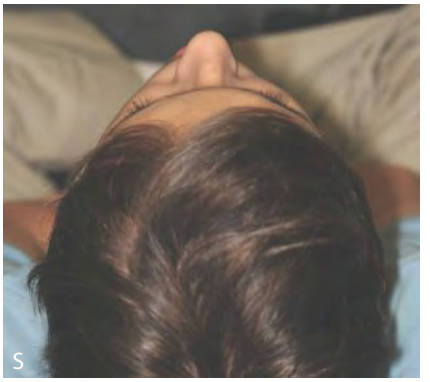


FIGURE 45-13. *(Continued)* **R,** Frontal view 12 years after reconstruction. **S,** Superior view 12 years after reconstruction. (**A–S,** Reproduced with permission from Ghali GE, Sinn DP, Tantipasawasin S. Management of nonsyndromic craniosynostosis. Atlas Oral Maxillofac Surg Clin North Am 2002;10:16–24.)

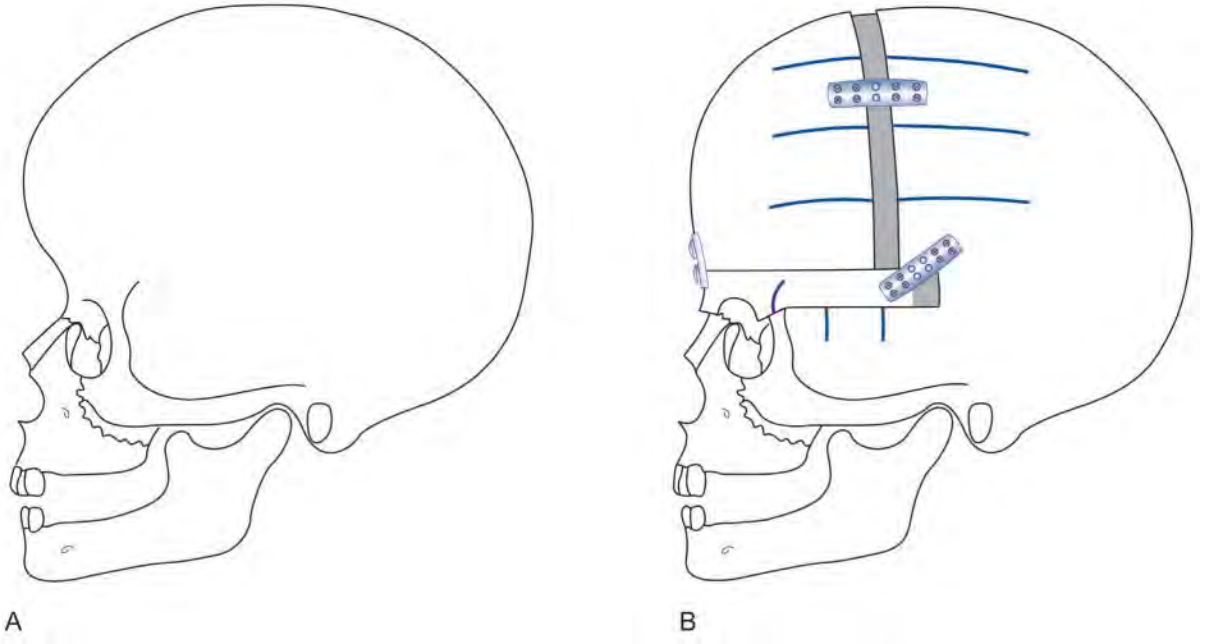


FIGURE 45-14. Brachycephaly before and after anterior cranial vault and bilateral superior orbital rim osteotomies, reshaping, and advancements. **A,** Site of osteotomies as indicated. Dissection and osteotomies are similar to those previously described for plagiocephaly repair. **B,** After osteotomies, reshaping, and fixation of bandeau and frontal plates. (**A** and **B,** Adapted from Ghali GE, Sinn DP, Tantipasawasin S. Management of nonsyndromic craniosynostosis. Atlas Oral Maxillofac Surg Clin North Am 2002;10:25.)

the midline and placing autogenous cranial bone grafts to increase the intraorbital distance. Stabilization is achieved with direct transosseous wires or resorbable microplate fixation. The microplate fixation is usually placed at the inner surface of the cranial bone. The abnormally shaped bone that has been removed is cut into sections of appropriate shape for the new forehead configuration. The anterior cranial base, anterior cranial vault, and orbit are given a more aesthetic shape, and the volume of the anterior cranial vault is increased, which allows the brain adequate space. Autogenous bone may be taken from the posterior cranium, when required, to enhance frontal reconstruction.

Sagittal Synostosis

Historically, when premature closure of a sagittal suture (Figures 45-18 and 45-19) was recognized in early infancy, most neurosurgeons believed that simple release of the

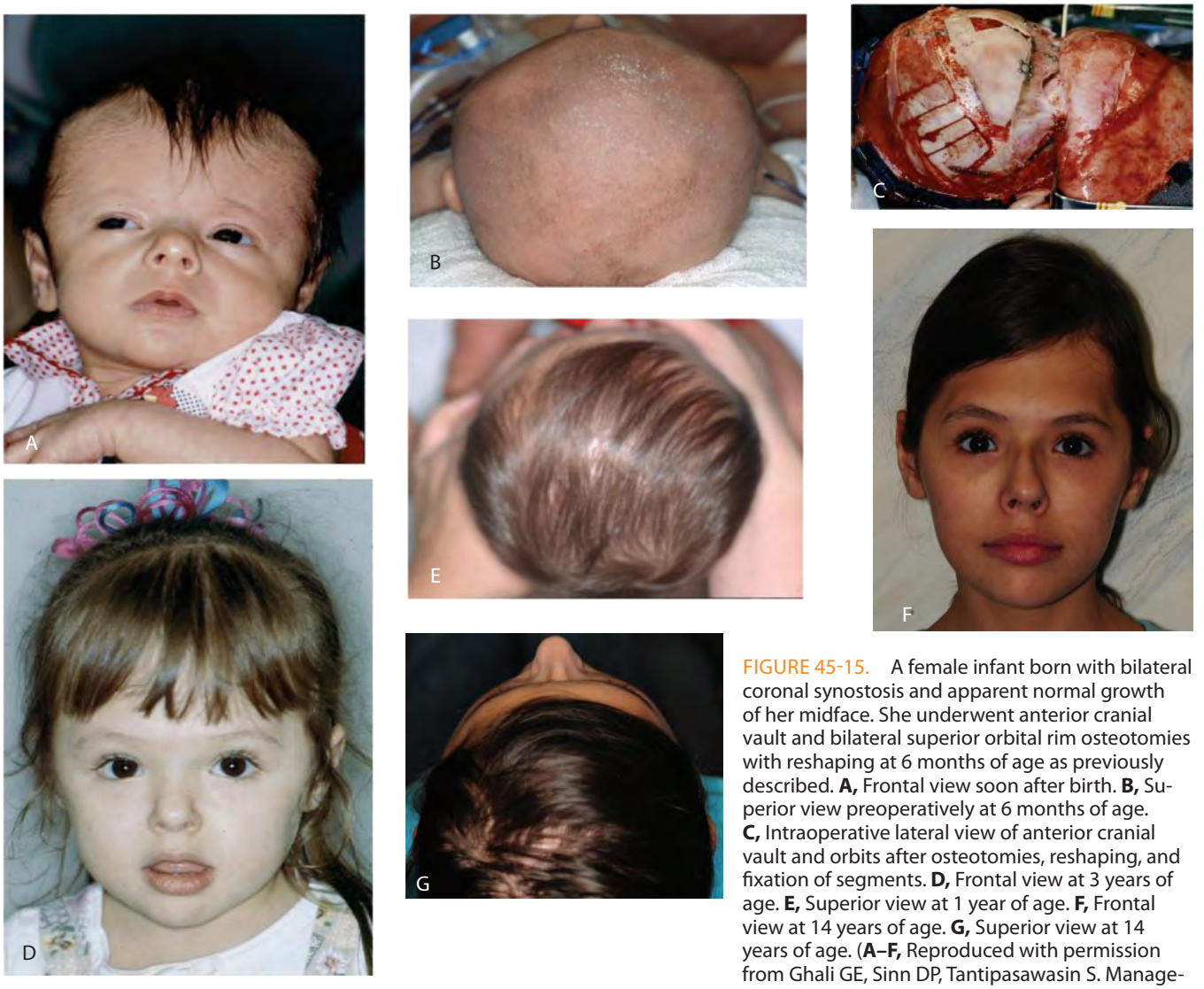


FIGURE 45-15. A female infant born with bilateral coronal synostosis and apparent normal growth of her midface. She underwent anterior cranial vault and bilateral superior orbital rim osteotomies with reshaping at 6 months of age as previously described. **A,** Frontal view soon after birth. **B,** Superior view preoperatively at 6 months of age. **C,** Intraoperative lateral view of anterior cranial vault and orbits after osteotomies, reshaping, and fixation of segments. **D,** Frontal view at 3 years of age. **E,** Superior view at 1 year of age. **F,** Frontal view at 14 years of age. **G,** Superior view at 14 years of age. (**A–F,** Reproduced with permission from Ghali GE, Sinn DP, Tantipasawasin S. Management of nonsyndromic craniosynostosis. Atlas Oral Maxillofac Surg Clin North Am 2002;10:25–27.)

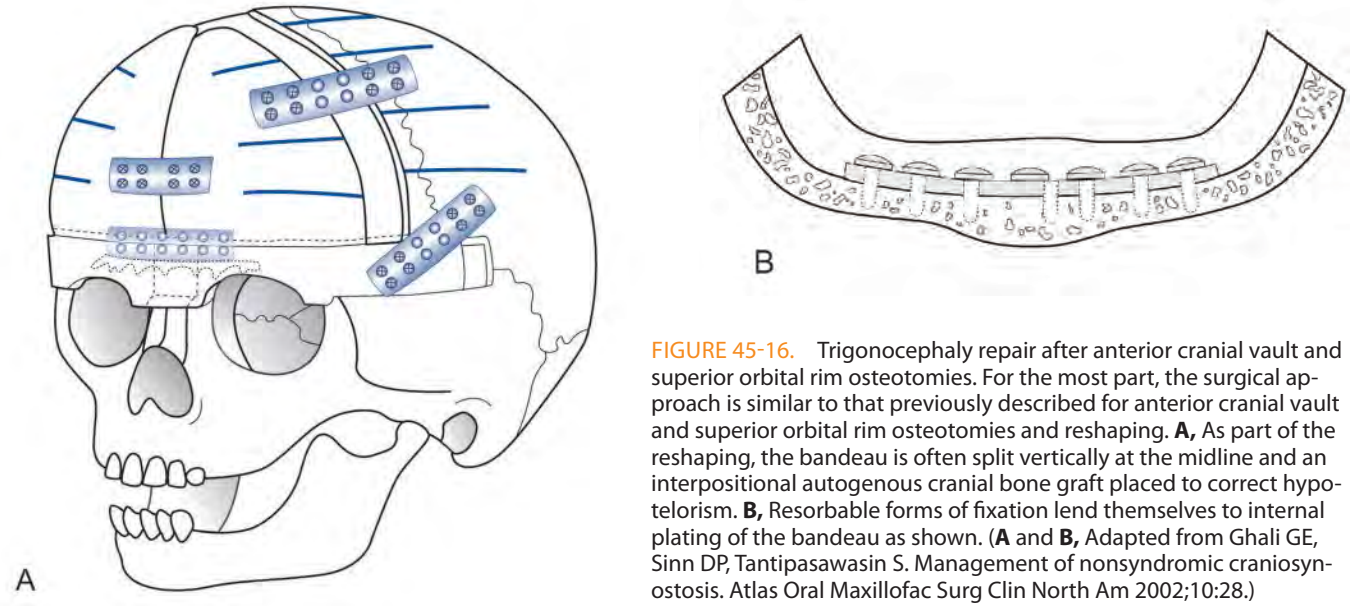


FIGURE 45-16. Trigonocephaly repair after anterior cranial vault and superior orbital rim osteotomies. For the most part, the surgical approach is similar to that previously described for anterior cranial vault and superior orbital rim osteotomies and reshaping. **A,** As part of the reshaping, the bandeau is often split vertically at the midline and an interpositional autogenous cranial bone graft placed to correct hypotelorism. **B,** Resorbable forms of fixation lend themselves to internal plating of the bandeau as shown. (**A** and **B,** Adapted from Ghali GE, Sinn DP, Tantipasawasin S. Management of nonsyndromic craniosynostosis. Atlas Oral Maxillofac Surg Clin North Am 2002;10:28.)



FIGURE 45-17. A 10-month-old girl with metopic synostosis resulting in trigonocephaly. She underwent anterior cranial vault reshaping, bilateral superior orbital rim advancements, and bitemporal widening via barrel-staving osteotomies. **A,** Frontal view before surgery. **B,** Frontal view after reconstruction. **C,** Superior view before surgery. **D,** Superior view after reconstruction. **E,** Frontal view at 2 years of age. **F,** Superior view at 2 years of age. **G,** Frontal view at 7 years of age. **H,** Inferior view at 7 years of age. **I,** Intraoperative frontal view outlining proposed osteotomy and bifrontal craniotomy sites. **J,** Superior view of bandeau before reshaping. **K,** Superior view of bandeau after reshaping and resorbable plate and screw stabilization. **L,** Frontal view of bandeau after reshaping and resorbable plate fixation. **M,** View of same bandeau from cranial aspect. **N,** Superior view after fixation of bandeau. Pivoting point of rotation is about the glabellar region. Observation of the gap between the bandeau and the anterior cranial base assists in assessing ideal placement and bitemporal expansion. **O,** Superior oblique view of anterior cranial vault prior to reshaping. **P,** Superior oblique view of anterior cranial vault after osteotomies, reshaping, and fixation. (**A–P,** Reproduced with permission from Ghali GE, Sinn DP, Tantipasawasin S. Management of nonsyndromic craniosynostosis. Atlas Oral Maxillofac Surg Clin North Am 2002;10:28–34.)

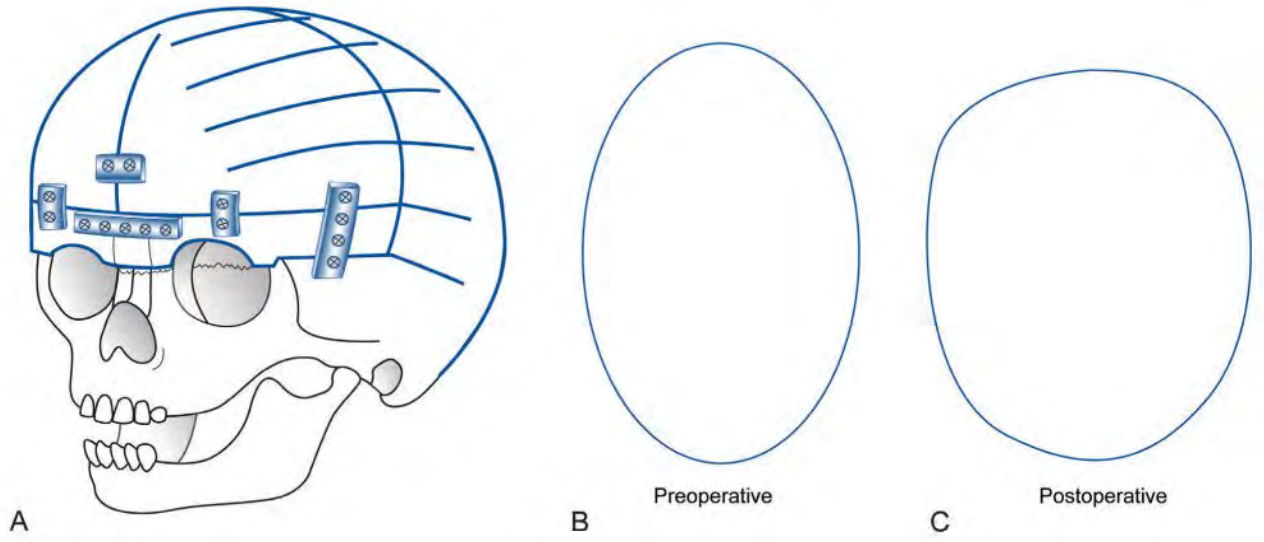


FIGURE 45-18. A child after total cranial vault and upper orbital osteotomies for the treatment of scaphocephaly. **A,** Forehead is symmetrically tilted back. The occiput is symmetrically tilted forward. The anteroposterior dimension is thereby shortened and secured via resorbable plates and screws. Barrel-stave cuts are made laterally to widen the transverse dimension or the squamous portion of the temporal plates as osteotomized, interchanged, and stabilized with resorbable plates and screws. Superior view preoperatively **(B)** and postoperatively **(C).** Total cranial vault reshaping as well as orbital rim alteration is accomplished to increase the biparietal width and decrease the frontal and occipital prominences. (**A–C,** Adapted from Ghali GE, Sinn DP, Tantipasawasin S. Management of nonsyndromic craniosynostosis. Atlas Oral Maxillofac Surg Clin North Am 2002;10:35.)

sagittal suture through a strip craniectomy without simultaneous skull reshaping was adequate treatment. [4,5,40,50] Our results using this technique alone have been less than favorable, and a residual cranial vault deformity usually results. If improvements in cranial vault shape are to be achieved, most cases require a formal total cranial vault reshaping at the age of 4 to 8 months. In our center, a recent trend toward earlier surgery (≤4 mo) involving removal of the stenosed suture along with extensive barrel-staving and postoperative helmet-molding is gaining popularity. Early results using this protocol have shown promising outcomes with less blood loss and shorter operative times. Variations in the degree of the scaphocephalic deformity are common, depending on the extent of sagittal suture stenosis. When the posterior half is fused, the patient is treated in the prone position with the posterior two thirds of the cranial vault reshaped. When the anterior half is fused, the patient is treated in the supine position with the anterior two thirds of the cranial vault reshaped, with or without superior orbital rim reshaping. When the entire suture is fused, a combination of both approaches may be necessary. Unless a significant concomitant supraorbital deformity exists, we prefer to treat full sagittal suture stenosis (anterior and posterior) at one operative setting in the prone position via a total cranial vault reshaping. For older children (>1 yr) or children with a need for upper orbital reconstruction, we prefer the supine position at one operative setting or, rarely, in two stages, with posterior reconstruction preceding anterior and orbital reconstruction by 4 to 6 months. Other centers have reported good results when routinely staging full sagittal synostosis.

Unilateral Lambdoid Synostosis

Many surgeons consider simple strip craniectomy of the involved suture or partial craniectomy of the region to be adequate treatment. More extensive vault craniectomy and reshaping are generally necessary. If improvements in cranial vault shape are required after 10 to 12 months of age, formal posterior cranial vault reshaping is performed.

SUMMARY

In approximately 1 in 1000 live births in the United States, an infant has some variant of a craniofacial deformity. If cleft lip and palate deformities are included, the incidence is even greater. Surgical management of these patients has been advocated to occur from the first few weeks after birth until well into the teens. Many of these patients require multiple, staged procedures that involve movements of the bone and soft tissue from both the intracranial and the extracranial approaches. The surgical approach to most of these congenital deformities was radically changed by techniques introduced to the United States by Paul Tessier of France in 1967. From his imaginative intracranial and extracranial approaches, numerous advances have been made that have improved the management of these complex pediatric craniofacial deformities.



FIGURE 45-19. A 6-month-old girl with anterior and posterior sagittal suture synostosis resulting in scaphocephaly. She underwent total cranial vault reshaping without the need for any orbital osteotomies. A prone position was used throughout the procedure. **A,** Lateral view before surgery. **B,** Lateral view after reconstruction. **C,** Superior view before surgery. **D,** Superior view after reconstruction. **E,** Frontal view 6 years after reconstruction. **F,** Lateral view 6 years after reconstruction. **G,** Prone positioning is necessary and requires careful protection of both the airway and the globes. **H,** Intraoperative superior view of proposed osteotomy sites for total cranial vault reshaping. **I,** Intraoperative superior view of the osteotomies, reshaping, and resorbable plate fixation. **J,** Intraoperative left lateral view before reshaping. **K,** Intraoperative left lateral view after osteotomies, reshaping, and resorbable plate fixation. (**A–K,** Reproduced with permission from Ghali GE, Sinn DP, Tantipasawasin S. Management of nonsyndromic craniosynostosis. Atlas Oral Maxillofac Surg Clin North Am 2002;10:36–40.)

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CHAPTER

Craniofacial Dysostosis Syndromes: Evaluation and Treatment of the Skeletal Deformities

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INTRODUCTION

Cranial sutures are a form of bone articulation in which the margins of the bones are connected by a thin layer of fibrous tissue. The cranial vault is composed of six major sutural areas and several minor sutures, which serve two critical functions during the postnatal period. Initially, the sutures allow head deformation during vaginal delivery. Later, during an infant's postnatal development, cranial vault sutures facilitate head expansion to accommodate propulsive brain growth.[1] Only small amounts of pressure (5 mmHg) from the growing brain are required to stimulate bone deposition at the margins of a cranial bone.[2,3] Under normal conditions, the brain volume will triple within the first year of life, and by age 2 years, the cranial capacity is four times that at birth.[4] Normally, closure of the cranial vault sutures occurs earlier than closure of the membranous facial bone sutures, which often remain patent until adulthood.

The term *craniosynostosis* is defined as a premature fusion of a cranial vault suture. With rare exception, this is an intrauterine event. A more accurate description of craniosynostosis may be a *congenital absence* of the cranial vault sutures. The result is fusion of the bones adjacent to the suture and arrested sutural growth of the adjacent bones. The classic theory known as *Virchow's law* states that premature fusion of a cranial vault suture results in limited development of the skull perpendicular to the fused suture and a compensatory "overgrowth" through the sutures that remain open.[5] The result is a dysmorphology with characteristics depending on the sutures affected and potential neurologic consequences related to underlying brain compression. Most forms of craniosynostosis represent nonsyndromic malformations limited to the cranial vault and orbital regions. Management typically requires a combined neurosurgical and craniofacial approach for release of the involved suture and reshaping of the dysmorphic skeletal components. Discussion of the management of nonsyndromic craniosynostosis is beyond the scope of this chapter. *Craniofacial dysostosis (CFD)* is the term applied to syndromal forms of craniosynostosis. These disorders are characterized by sutural involvement that not only includes the cranial vault but also extends into the skull base and midfacial skeletal structures. CFD syndromes have been described by Carpenter, Apert, Crouzon, Saethre-Chotzen, and Pfeiffer.[6] Although the cranial vault and cranial base are thought to be the regions of primary involvement, there is also significant impact on midfacial growth and development.[7,8] In addition to cranial vault dysmorphology, patients with these inherited conditions exhibit a characteristic "total midface" deficiency that is syndrome-specific and must be addressed as part of the staged reconstructive approach.

GENETIC ASPECTS

FGFR-related CFD syndrome includes: *FGFR1*-related craniosynostosis (Pfeiffer's syndrome types 1, 2, and 3); *FGFR2*-related craniosynostosis (Apert's syndrome; Beare-Stevenson syndrome; Crouzon's syndrome; *FGFR2*-related isolated coronal synostosis; Jackson-Weiss syndrome; Pfeiffer's syndrome types 1, 2, and 3); and *FGFR3*-related craniosynostosis, (Crouzon's syndrome with acanthosis nigricans, *FGFR3*-related isolated coronal synostosis, Muenke's syndrome).

The eight disorders considered as part of the *FGFR*-related craniosynostosis spectrum are *Pfeiffer's syndrome, Apert's syndrome, Crouzon's syndrome, Beare-Stevenson syndrome, FGFR2-related isolated coronal synostosis, Jackson-Weiss syndrome, Crouzon's syndrome with acanthosis nigricans,* and *Muenke's syndrome.* All but Muenke's syndrome and *FGFR2*-related isolated coronal synostosis generally present with bicoronal synostosis or cloverleaf skull anomaly.

The diagnosis of Muenke's syndrome (*FGFR3*-related coronal synostosis) is based on identification of a disease-causing mutation in the *FGFR3* gene. The diagnosis of *FGFR2*-related isolated coronal synostosis is based on identification of a disease-causing mutation in the *FGFR2* gene. The diagnosis of the other six *FGFR*-related craniosynostoses is based on clinical findings; however, molecular genetic testing of the *FGFR1*, *FGFR2*, and *FGFR3* genes may be helpful in establishing the diagnosis of these syndromes in questionable cases.

FGFR-related craniosynostosis is inherited in an autosomal dominant manner. Affected individuals have a 50% chance of passing the mutant gene to each child. Prenatal testing is available; however, its use is limited by poor predictive value. Molecular testing is necessary to establish the diagnosis for two of the disorders, Muenke's syndrome and *FGFR2*-related isolated coronal synostosis. Individuals with Muenke's syndrome may have unilateral coronal synostosis or megalencephaly without craniosynostosis: The accurate diagnosis depends upon identification of a disease-causing mutation in the *FGFR3* gene. *FGFR2*-related isolated coronal synostosis is characterized only by uni- or bicoronal craniosynostosis: The accurate diagnosis depends upon identification of a disease-causing mutation in the *FGFR2* gene.

FUNCTIONAL CONSIDERATIONS

Brain Growth and Intracranial Pressure

If the rapid brain growth that normally occurs during infancy is to proceed unhindered, the cranial vault and base sutures must remain open and expand during phases of rapid growth, resulting in marginal ossification. In craniosynostosis, premature fusion of sutures causes limited and abnormal skeletal expansion in the presence of continued brain growth. Depending on the number and location of prematurely fused sutures, the growth of the brain may be restricted.[2,9–11] In addition, abnormal cranial vault and midfacial morphology occurs as determined by Virchow's law. If surgical release of the affected sutures and reshaping to restore a more normal intracranial volume and configuration are not performed, decreased cognitive and behavioral function is likely to be the end result.

Elevated intracranial pressure (ICP) is the most serious functional problem associated with premature suture fusion. A "beaten-copper" appearance along the inner table of the cranial vault seen on a plain radiograph or the loss of brain cisternae as observed on a computed tomography (CT) scan may suggest elevated ICP,[12] but these are considered soft radiographic findings.

Intracranial hypertension can be established invasively by means of a burhole craniotomy used to place either an epidural or an intraparenchymal pressure sensor. Increased ICP is most likely to affect individuals with great disparity between brain growth and intracranial capacity and may occur in as many as 42% of untreated children in whom more than one suture is affected.[2,10,13] Unfortunately, there is no absolute agreement on what levels of ICP are normal at any given age in infancy and early childhood.

The clinical signs and symptoms related to elevated ICP may have a slow onset and be difficult to recognize in the pediatric population. Although standardized CT scans allow for indirect measurement of intracranial volume, it is not yet possible to use these studies to make judgments as to who requires craniotomy for decompression.[14,15] Careful neurosurgical and pediatric ophthalmologic evaluation are critical components of the data gathering required to formulate a definitive treatment plan in a patient with craniosynostosis.

Vision

Untreated craniosynostosis with elevated ICP will cause papilledema and eventual optic nerve atrophy, resulting in partial or complete blindness. If the orbits are shallow (exorbitism) and the eyes are proptotic (exophthalmos), as occurs in the CFD syndromes, the cornea may be exposed and abrasions or ulcerations may occur. An eyeball extending outside of a shallow orbit is also at risk of trauma. If the orbits are extremely shallow, herniation of the globe itself may occur, necessitating emergency reduction followed by tarsorrhaphies or urgent orbital decompression.

Some forms of CFD result in a marked degree of orbital hypertelorism, which may compromise visual acuity and restrict binocular vision. Divergent or convergent nonparalytic strabismus or exotropia occurs frequently and should be considered during the diagnostic evaluation. This may be the result of congenital anomalies of the extraocular muscles themselves. Paralytic or nonparalytic unilateral or bilateral upper eyelid ptosis also occurs with greater frequency with CFD than in the general population.

Hydrocephalus

Hydrocephalus affects as many as 10% of patients with a CFD syndrome.[16–19] Although the etiology is often not clear, hydrocephalus may be secondary to a generalized cranial base stenosis with constriction of all the cranial base foramina, which affects the patient's cerebral venous drainage and cerebrospinal fluid (CSF) flow dynamics. Hydrocephalus may be identified with the help of CT or magnetic resonance imaging (MRI) to document progressively enlarging ventricles. Difficulty exists in interpreting ventricular findings as seen on a CT scan especially when the skull and cranial base are brachycephalic. The skeletal dysmorphology seen in a child with severe cranial dysmorphology related to craniosynostosis may translate into an abnormal ventricular shape that is not necessarily related to abnormal CSF flow. Serial imaging and clinical correlation is indicated, and a great deal of clinical judgment is often required in making these assessments.

Effects of Midface Deficiency on Airway

All newborn infants are obligate nasal breathers. Many infants born with a CFD syndrome have moderate to severe hypoplasia of the midface as a component of their malformation. They will have diminished nasal and nasopharyngeal spaces with resulting increased nasal airway resistance (obstruction). The affected child is thus forced to breathe through the mouth. For a newborn infant to ingest food through the mouth requires sucking from a nipple to achieve negative pressure as well as an intact swallowing mechanism. The neonate with severe midface hypoplasia will experience diminished nasal airflow and be unable to accomplish this task and breathe through the nose at the same time.[20–23] Complicating this clinical picture may be an elongated and ptotic palate and enlarged tonsils and adenoids. The compromised infant expends significant energy respiring, and this may push the child into a catabolic state (negative nitrogen balance). Failure to thrive results unless either nasogastric tube feeding is instituted or a feeding gastrostomy is placed. Evaluation by a pediatrician, pediatric otolaryngologist, and feeding specialist with craniofacial experience can help distinguish minor feeding difficulties from those requiring more aggressive treatment.

Sleep apnea of either central or obstructive origin may also be present. If the apnea is found to be secondary to upper airway obstruction based on a formal sleep study, a tracheostomy may be indicated. In rare situations, "early" midface advancement is useful to improve the airway and allow for tracheostomy decannulation. Central apnea may occur from poorly treated intracranial hypertension and other contributing factors. If this is the case, the condition may improve by reducing the ICP to a normal range through cranio-orbital or posterior cranial vault decompression or expansion.

The literature confirms that as many as 50% of children with Apert's, Crouzon's, or Pfeiffer's syndrome develop obstructive sleep apnea (OSA) during the first 6 years of life.[24,25] Documented OSA in a child with CFD is frequently treated with tracheostomy. It may also be treated by adenoidectomy, tonsillectomy, midface advancement, or continuous positive airway pressure (CPAP). If left untreated or ineffectively treated, OSA will result in disabilities ranging from failure to thrive; recurrent upper respiratory infection; cognitive dysfunction; developmental delay; cor pulmonale; or sudden death.[26] Overwhelmingly, the documented midface hypoplasia in many CFD children is a primary cause of OSA. If feasible, effective surgical midface advancement is the preferred biologic treatment approach.

Clinical symptoms of OSA will include heavy snoring, difficulty in awake breathing, observed apnea during sleep, excessive perspiration during sleep, and daytime somnolence. Polysomnography remains the gold standard for diagnosis. In the pediatric population, *apnea*, defined as absence of air flow for more than two breadths, and *hypopnea*, defined as a reduction of greater than or equal to 50% of nasal flow signal amplitude of more than two breadths, defines OSA syndrome. The *apnea-hypopnea index (AHI)* is the number of obstructive apneas in combination with hypopneas (followed by desaturation) per hour. The *oxygenation-desaturation index (ODI)* is the number of desaturations (≥4% decrease with respect to the baseline) per hour. A score of less than 1.0 is considered normal. Definition of the severity of the condition in children includes between 1 and 5 events as mild OSA; 6 to 25 as moderate OSA; and greater than 25 events as severe OSA. In the child with CFD and moderate to severe midface deficiency who is documented to have OSA, neither tonsillectomy nor adenoidectomy generally provides sufficient opening of the upper airway.

Ishii and coworkers[27] used lateral cephalometric radiographs to evaluate the nasopharyngeal airway after Le Fort III advancement in Apert's and Crouzon's syndrome individuals with OSA. They were able to document improvement in the nasopharyngeal airway in the study patients after successful midface advancement surgery.

Arnaud and colleagues[28,29] completed a studies to review respiratory improvement in CFD patients suffering with OSA that underwent monobloc advancement (DO approach). Fourteen of the 16 study patients (88%) showed improvement as measured by oxygen level during sleep. Tracheostomy removal was possible in 4 of 6 study patients. In one of the patients, a tracheostomy was required 6 months after midface advancement due to recurrent severe OSA.

Witherow and colleagues[30] found a significant improvement in OSA in all patients diagnosed with CFD (Apert's, Crouzon's, Pfeiffer's) as documented by polysomnography at 24 months after undergoing monobloc advancement (distraction osteogenesis approach). Before monobloc advancement, 14 of their patients had severe OSA with either tracheostomy or the use of CPAP in place. For the study patients undergoing monobloc advancement, only 43% (6/14) had resolution of their OSA. Unfortunately, 8 of the

14 patients (57%) remained dependent on tracheostomy or CPAP despite the monobloc advancement procedure.

Nelson and associates[31] studied 18 CFD individuals, all presenting with bilateral coronal synostosis and midface deficiency. In 15/18 patients (83%), either tracheostomy or CPAP was required for treatment of OSA before midface advancement. After the midface advancement procedures, 5 of the tracheostomy patients were decannulated, and in 6, CPAP was no longer required (11/15 = 73%). The mean post-surgical follow-up was 3 years.

In the CFD patient undergoing midface advancement, the lack of universal success in the relief of OSA could be for a combination of reasons including inadequate midface advancement, midface relapse after initial advancement, other anatomic factors not addressed by the midface advancement (septum, turbinates, soft palate, tonsils, adenoids, mandible, tracheomalacia), a central sleep apnea component, or inadequate (abnormal) contraction forces of the pharyngeal dilator muscles during sleep. In CFD, not only is the anatomy of the upper airway different for each syndrome, but a wide variation of deformity is also seen within each syndrome. Differences in the soft tissues and the quality of the pharyngeal muscles compared with normal may be attributed to a mutation of the fibroblast growth factor receptor (FGFR).[32] CFD patients with OSA who are nonresponders to an effective midface advancement may have pharyngeal wall collapse due to poor pharyngeal dilator muscle function during sleeping. Whereas a tendency for airway collapse can be overcome through midface advancement, the individual must also have sufficient ability to control/contract (maintain tone) of the pharyngeal muscles while asleep.

It would appear that in some CFD individuals (Apert's, Crouzon's, Pfeiffer's syndromes) despite effective midface advancement (Le Fort III/monobloc/facial bipartition), the long-term dependence on tracheostomy or CPAP for resistant OSA should be anticipated. In patients with persistent OSA despite optimal maxillofacial skeletal morphology, airway collapse due to inadequate pharyngeal muscle tone appears to play a role.

Dentition and Occlusion

The incidence of dental and oral anomalies is higher among children with CFD syndromes than in the general population. In Apert's syndrome in particular, the palate is high and constricted in width. The incidence of isolated cleft palate in patients with Apert's syndrome approaches 30%.[13] Clefting of the secondary palate may be submucous, incomplete, or complete. Confusion has arisen over whether the oral malformations and absence of teeth that are often characteristic of these conditions are a result of congenital or iatrogenic factors (e.g., injury to dental follicles associated with early midface surgery). The midfacial hypoplasia seen in the CFD syndromes often results in limited maxillary alveolar bone to house a full complement of teeth. The result is severe crowding, which often requires serial extractions in order to address the problem. An Angle class III skeletal relationship in combination with anterior open bite deformity is typical.

Hearing

Hearing deficits are more common among patients with the CFD syndromes than in the general population.[24] In Crouzon's syndrome, conductive hearing deficits are common, and atresia of the external auditory canals may also occur. Otitis media is more common in Apert's syndrome, although the exact incidence is unknown. Middle ear disease may be related to the presence of a cleft palate that results in eustachian tube dysfunction. Congenital fixation of the stapedial footplate is also believed to be frequent. The possibility of significant hearing loss is paramount in importance and should not be overlooked because of preoccupation with other, more easily appreciated craniofacial findings.

Extremity Anomalies

Apert's syndrome results in joint fusion and bony and soft tissue syndactyly of the digits of all four limbs.[24] These Apert-associated extremity deformities are often symmetrical. Partial or complete fusion of the shoulder, elbow, or other joints is common. Broad thumbs, broad great toes, and partial soft tissue syndactyly of the hands may be seen in Pfeiffer's syndrome, but these are variable features. Preaxial polysyndactyly of the feet may also be seen in Carpenter's syndrome.

MORPHOLOGIC CONSIDERATIONS

Examination of the patient's entire craniofacial region should be meticulous and systematic. The skeleton and soft tissues are assessed in a standard way to identify all normal and abnormal anatomy. Specific findings tend to occur in particular malformations, but each patient is unique. The achievement of symmetry and normal proportions and the reconstruction of specific aesthetic units are essential to forming an unobtrusive face in a child born with a CFD syndrome.

Frontoforehead Aesthetic Unit

The frontoforehead region is dysmorphic in an infant with CFD.[25–30] Establishing normal position of the forehead is critical to overall facial symmetry and balance. The forehead may be considered as two separate aesthetic components: the supraorbital ridge–lateral orbital rim region and the superior forehead (Figure 46-1).[31,32] The supraorbital ridge–lateral orbital rim region includes the glabella and supraorbital rim extending inferiorly down each frontozygomatic suture toward the infraorbital rim and posteriorly along each temporoparietal region. The morphology and position of the supraorbital ridge–lateral orbital rim region is a key element of upper facial aesthetics. In a normal forehead, at the level of the frontonasal suture, an angle ranging from 90 to 110 degrees, is formed by the supraorbital ridge and the nasal

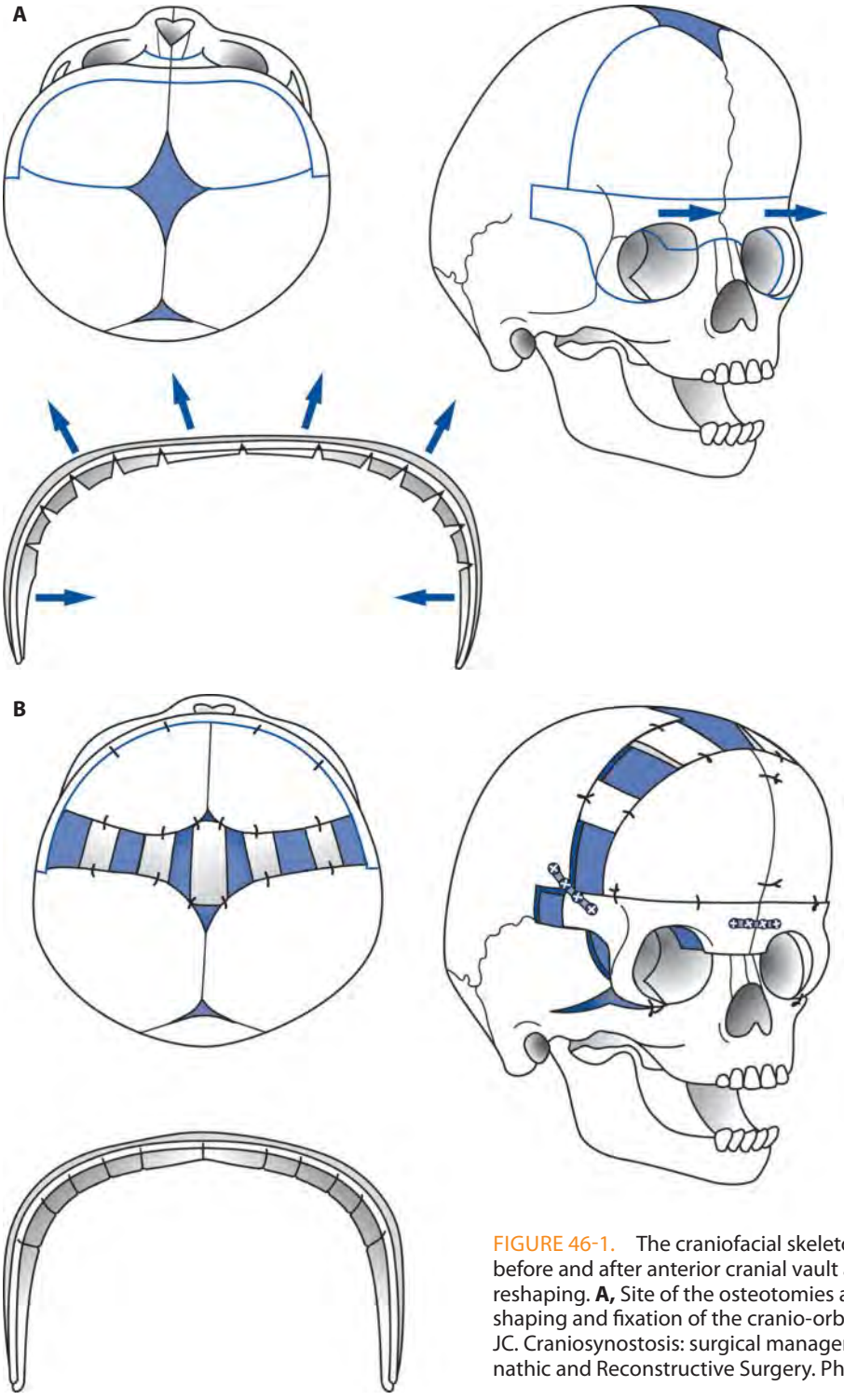


FIGURE 46-1. The craniofacial skeleton of a child with bilateral coronal synostosis before and after anterior cranial vault and three-quarter orbital osteotomies with reshaping. **A,** Site of the osteotomies as indicated. **B,** After osteotomies and reshaping and fixation of the cranio-orbital regions. (**A** and **B,** Adapted from Posnick JC. Craniosynostosis: surgical management in infancy. In Bell WH, editor. Orthognathic and Reconstructive Surgery. Philadelphia: WB Saunders; 1992; p. 1839.)

bones when viewed in profile. In addition, the eyebrows, overlying the supraorbital ridge, should be anterior to the cornea. When the supraorbital ridge is viewed from above, the rim should arc posteriorly to achieve a gentle 90-degree angle at the temporal fossa with a center point of the arc at the level of each frontozygomatic suture. The superior forehead component, approximately 1.0 to 1.5 cm up from the supraorbital rim, should have a gentle posterior curve of about 60 degrees, leveling out at the coronal suture region when seen in profile.

Posterior Cranial Vault Aesthetic Unit

Symmetry, form, and the appropriate intracranial volume of the posterior cranial vault are closely linked. Posterior cranial vault flattening may result from either a unilateral or

a bilateral lambdoidal synostosis, which is rare; previous craniectomy with reossification in a dysmorphic flat shape, which is frequent; or postural molding because of repetitive sleep positioning.[33] A short anteriorposterior cephalic length may be misinterpreted as an anterior cranial vault (forehead) problem when the occipitoparietal (posterior) skull represents the primary region of the deformity. Careful examination of the entire cranial vault is essential to defining the dysmorphic region so that appropriate therapy may be carried out.

Orbitonasozygomatic Aesthetic Unit

In CFD syndromes, the orbitonasozygomatic regional deformity is a reflection of the cranial base malformation. For example, in Crouzon's syndrome when bilateral coronal suture synostosis is combined with skull base and midfacial deficiency, the orbitonasozygomatic region will be dysmorphic and consistent with a short (anteroposterior) and wide (transverse) anterior cranial base.[34] In Apert's syndrome, the nasal bones, orbits, and zygomas, like the anterior cranial base, are transversely wide and horizontally short (retruded), resulting in a shallow hyperteloric upper midface (zygomas, orbits, and nose).[34] Advancing the midface without simultaneously addressing the increased transverse width will not adequately correct the dysmorphology.[35]

Maxillary–Nasal Base Aesthetic Unit

In the CFD patient with midface deficiency, the upper anterior face (nasion to maxillary incisor) is vertically short, and there is a lack of horizontal anteroposterior projection of the midface.[36,37] These findings may be confirmed with cephalometric analysis that indicates a sella-nasion angle (SNA) below the mean value and a short upper anterior facial height (nasion to anterior nasal spine). The width of the maxilla in the dentoalveolar region is generally constricted with a high-arched palate. In order to normalize the maxillonasal base region, multidirectional surgical expansion and reshaping are generally required. The maxillary lip-to-tooth relationship and occlusion are normalized through Le Fort I osteotomy and orthodontic treatment as part of the staged reconstruction.

Quantitative Assessment

A quantitative analysis of measurements taken from CT scans, surface anthropometry, cephalometric analysis, and dental casts is critical to data gathering for evaluation of craniofacial deformities.[38–43] This analysis will confirm or refute clinical impressions, aid in the treatment planning of intraoperative skeletal movements and reshaping, and provide a framework for objective assessment of immediate and long-term results. These methods of assessment rely on the measurement of linear distances, angles, and proportions based on accurate, reliable, and reproducible anatomic landmarks found to be useful for patient evaluation.

CT Scan Analysis

The use of CT scans has clarified our appreciation of the dysmorphology of a child born with a craniofacial malformation.[44,45] Accurate standardized points of reference have been identified in the cranio-orbitozygomatic skeleton based on axial CT images.[42,43] Knowledge of differential facial bone growth patterns and normal measurement values can now be used to improve diagnostic accuracy, assist in the staging of reconstruction by understanding growth vectors, and offer the option of making intraoperative measurements that correlate with the preoperative CT scan measurements and ideal dimensions. This information can effectively guide the surgeon in the reconstruction of an individual with a craniofacial malformation and also allows for accurate postoperative reassessment.

Anthropometric Surface Measurements

Cross-sectional studies of the patterns of postnatal facial growth based on anthropometric surface measurements have been carried out in growing white children.[38–41,46] This published material has proved useful in the quantitative evaluation and recognition of discrepancies in postnatal development in the head and face of patients with specific craniofacial syndromes. This is particularly useful when evaluating basic distances, angles, and proportions of the head, face, and orbits in patients affected with CFD syndromes.

Cephalometric Analysis

Cephalometric radiography, first introduced by Broadbent in 1931, has been traditionally used to study the morphology and patterns of growth of the maxillofacial skeleton.[47] The large collection of normative data developed allows clinicians to monitor an individual's facial growth. The interpretation of cephalometric radiographs remains useful in the analysis of facial heights and maxillary, mandibular, and chin positions and their relationships to one another, the cranial base, and the dentition.[30,36,48,49] The lateral cephalometric radiograph offers an accurate view from the midsagittal plane if the facial skeleton being analyzed is relatively symmetrical. Unfortunately, the number of anatomic landmarks that can be identified accurately in the cranio-orbitozygomatic region is limited because of the overlap of structures, which makes predictably locating these anatomic landmarks more difficult.

SURGICAL MANAGEMENT

Historical Perspectives

As early as 1890, Lannelongue[50] and then Lane[51] reported a surgical approach to the treatment of craniosynostosis. Lannelongue's aim was to remove the fused suture (strip craniectomy) in the hopes of controlling the problem of brain compression within a congenitally small cranial vault. By the

turn of the century, Harvey Cushing, the most prominent neurosurgeon of his day, suggested that surgical intervention for the problem of craniosynostosis was misdirected and that more attention should instead be given to the schooling of these children. Schillito disagreed with Cushing and enthusiastically supported the concept of surgical intervention to improve the outlook for these children. He believed that the linear "strip" craniectomy of the fused sutures would "release" the skull and allow the cranium to reshape itself and continue to grow in a normal and symmetrical fashion.[52] The strip craniectomy procedures were supposed to allow for a new suture line at the site of the previous synostosis. With the realization that this goal was not biologically feasible, attempts were made to remove portions of the cranial vault surgically and to either leave large open areas or use the removed segments as free grafts to refashion the cranial vault shape. Problems with these methods included uncontrolled postoperative skull molding, re-ossification into dysmorphic configurations, and the occurrence of large residual skull defects.

The concept of simultaneous release of the fused suture combined with more meticulous cranial vault reshaping carried out in infants was initially suggested by Rougerie and coworkers[53] and then refined by Hoffman and Mohr in 1976[54] for children born with unilateral coronal synostosis. Whitaker and colleagues[55] proposed a more formal anterior cranial vault and orbital reshaping procedure for unilateral coronal synostosis in 1977. Marchac and associates[41] published their experience with a "floating forehead" technique for simultaneous unilateral coronal and bilateral coronal suture release and forehead and upper orbital osteotomies with advancement to manage craniosynostosis in infancy. Unfortunately, the "floating forehead" technique resulted in unpredictable re-ossification of the open cranial vault areas. In addition, bitemporal constrictions or bulging and vertex of the skull concavities or bulging were frequent developments as re-ossification occurred. In any case, the hoped for midface growth did not materialize.

In the 1950s, Gillies and Harrison[56] reported experience with an extracranial Le Fort III "type" osteotomy to improve the anterior projection of the midface in an adult with Crouzon's syndrome. The initial Gillies procedure was actually carried out in 1942. He mobilized the midface by a variety of osteotomies preformed through skin excisions directly over each osteotomy site. The midface was mobilized and advanced and intermaxillary fixation (IMF) applied. After removal of the IMF (2 wk after the operation) the metal cast cap (dental) splint was attached to a plaster head cap, which was maintained for 3 weeks. There is no evidence that Gillies and Harrison used any bone grafts in the surgical gaps created. The early enthusiasm for this technique later turned to discouragement when the patient's facial skeleton relapsed to its preoperative status. There was relapse—both at the maxillary incisor level and of the eye proptosis.

Dr. Tessier was aware of Gillies' previous work and the accompanying difficulties. In 1967, Tessier[57] described a new (intracranial–cranial base) approach to the management of Crouzon's syndrome. This work was first presented in France at a meeting in Montpelier in 1966 and the following year at the International Plastic Surgery Meeting in Rome. Tessier's landmark presentations and publications were the beginning of modern craniofacial surgery. To overcome the earlier problems encountered by Gillies and Harrison, Tessier developed an innovative basic surgical approach that included new locations for the Le Fort III osteotomy, a combined intracranial-extracranial (cranial base) approach, use of a coronal (skin) incision to expose the upper facial bones, and use of fresh autogenous bone graft. He also applied an external fixation device to help maintain bony stability until healing had occurred.

In 1971, Tessier[58] described a single-stage frontofacial advancement in which the fronto-orbital bandeau was advanced as a separate element in conjunction with the Le Fort III complex below and the frontal bones above.[57–66] Seven years later, Ortiz-Monasterio and coworkers[67,68] refined the "monobloc" osteotomy to advance the orbits and midface as one unit, combined with frontal bone (anterior cranial vault) repositioning to correct the upper and midface deformity of Crouzon's syndrome. In 1979, Van der Meulen[69] described the "medial fasciotomy" for the correction of midline facial clefting. Van der Meulen split the monobloc osteotomy vertically in the midline, removed central nasal and ethmoid bone, and then moved the two halves of the facial skeleton together for correction of the orbital hypertelorism. To correct the midface dysplasia and associated orbital hypertelorism in Apert's syndrome, Tessier refined the vertical splitting and reshaping of the midline split monobloc segments, thus correcting the midface deformity in three dimensions in a procedure now known as *facial bipartition*. More recently, Posnick and colleagues[70,71] have independently documented the advantages of Tessier's facial bipartition technique for the correction of the Apert upper and midface deformity.

The widespread use of autogenous cranial bone grafting has virtually eliminated the need for rib and hip grafts when bone replacement is required in cranio-orbitozygomatic procedures. This represents another of Tessier's contributions to craniofacial surgery.

In 1968, Luhr[72] introduced the use of small metal (vitalium) plates and screws to stabilize maxillofacial fractures and then osteotomies. In current practice, the use of internal miniplate and microplate and screw (titanium) fixation (of various sizes) is the preferred form of fixation when stability and three-dimensional craniomaxillofacial reconstruction of multiple osteotomized bone segments and grafts are required. In infants and young children, for the stabilization of non–load-bearing osteotomy segments such as the cranial vault stabilization with resorbable materials is now generally preferred. This avoids issues or uncertainty concerning either growth restriction or brain trauma from retained hardware.

More recently, the intraoperative placement of a distraction device as a method of achieving advancement of the midface in patients with severe forms of craniofacial hypoplasia has been added to the surgeon's armamentarium.[47]

If used, distraction osteogenesis is not applied until after successful completion of standard osteotomies and disimpaction in the operating room. The distraction apparatus is anchored to the "stable" skeleton either internally or externally (through a "halo" head frame) and then to the palatal (intraoral) and infraorbital rims or zygomatic buttresses. Advancement of the "total midface" can then proceed. Once adequate (midface) advancement has been accomplished (on an outpatient basis) over a period of several weeks, the patient is generally returned to the operating room for stabilization and final reconstruction. The final reconstruction may require additional segmental osteotomies, bone grafting, or placement of plate and screw fixation. The "distraction approach" to the midface deformity is a labor-intensive, technique-specific, and relatively crude method of accomplishing horizontal advancement with difficulty in controlling the vertical dimension of the midface and without the ability to alter the transverse deformity or deficiency. In our opinion, the current level of distraction technology leaves it an adjunctive rather than a primary technique. It is most useful when the midfacial hypoplasia is severe to the extent that conventional techniques cannot reliably allow the immediate (in the operating room) desired advancement and when complex vertical and transverse reconstruction is not required.

Philosophy Regarding Timing of Intervention

In considering the timing and type of intervention the experienced surgeon will take several biologic realities into account: the natural course of the malformation (i.e., is the dysmorphology associated with Crouzon's syndrome progressively worsening or is it a nonprogressive craniofacial deformity?), the tendency toward growth restriction of an operated bone (aesthetic unit) that has not yet reached maturity (i.e., we know that operating on a palate of a child born with a cleft in infancy will cause scarring and later result in maxillary hypoplasia in a significant percentage of individuals), and the uncertain relationship between the underlying growing viscera (i.e., brain or eyes) and the congenially affected and surgically altered skeleton (i.e., if the cranial vault is not surgically expanded by 1 yr of life in a patient with multiple suture synostosis, will brain compression occur?).

In attempting to limit functional impairment and also achieve long-term ideal facial aesthetics, an essential question the surgeon must ask is, "During the course of craniofacial development, does the operated-on facial skeletal of the child with CFD tend to grow abnormally, resulting in further distortions and dysmorphology or are the initial positive skeletal changes (achieved at operation) maintained during ongoing growth?" Unfortunately, the theory that craniofacial procedures carried out early in infancy will "unlock growth" has not been documented through the scientific method.[57,73,74]

Incision Placement

For exposure of the craniofacial skeleton above the Le Fort I level, the approach used is the coronal (skin) incision. This allows for a relatively camouflaged access to the anterior and posterior cranial vault, orbits, nasal dorsum, zygomas, upper maxilla, pterygoid fossa, and temporomandibular joints. For added cosmetic advantage, placement of the coronal incision more posteriorly on the scalp and with postauricular rather than preauricular extensions is useful. When exposure of the maxilla at the Le Fort I level is required, a circumvestibular maxillary intraoral incision is used. Unless complications occur that warrant unusual exposure, no other incisions are required for managing any aspect of the CFD patient's reconstruction. These incisions (coronal [scalp] and maxillary [circumvestibular]) may be reopened as needed to further complete the patient's staged reconstruction.

Management of Cranial Vault Dead Space

Cranial reshaping in the CFD patient provides space for the compressed brain to expand into. Unfortunately, after anterior cranial vault expansion and monobloc advancement, an immediate extradural (retrofrontal) dead space is combined with the osteotomy-created gap across the skull base (connecting the anterior cranial fossa and the nasal cavity). This combination of factors may complicate the postoperative recovery (e.g., CSF leakage, infection, bone loss, fistula formation). After frontofacial advancement, the nasal cavity–cranial fossa communication is managed by being gentle to the tissues; good hemostasis; effective repair of any dural tears (dural grafting as needed); complete separation of dural and nasal mucosal tissue planes by interposing a combination of bone grafts, tissue sealants, and flaps; avoidance of pressure gradients across the opening while the nasal mucosa is healing; and prevention of "over-" or "under-" shunting (when a shunt is in place). The preferred way to manage the retrofrontal (lobes of the brain) dead space and the gap across the skull base osteotomy site (separating the cranial fossa and nasal cavities) after frontofacial advancement remains controversial but all agree that it is a critical aspect of the reconstruction.

In the CFD patient, relatively rapid (6–8 wk) filling of the surgically expanded intracranial volume by the previously compressed frontal lobes of the brain has been documented after cranio-orbital expansion in infants. It has also been shown to occur after frontofacial advancement in children and young adults when the volume increase remains in a physiologic range. These observations support the conservative management of the retrofrontal dead space in younger patients. More gradual and less complete filling of the space is thought to occur in older children and adults. If so, this may be particularly troublesome when the anterior cranial fossa dead space communicates directly with the nasal cavity (i.e., monobloc advancement, facial bipartition, intracranial Le Fort III) across the (open-gap) skull base interface. When feasible, closing off (sealing) the nasal cavity from the cranial fossa across the skull base osteotomy at the time of operation is preferred. Insertion of a pericranial flap or other fillers can help to separate the cavities. The use of fibrin glue to seal the anterior cranial base provides a temporary separation between the cavities, allowing time for the reepithelialization (healing)

of the torn nasal mucosa. To reconstruct the defect across the skull base, (gap) bone grafts of various types have also been used. Until the torn nasal mucosa heals, potential communication between the nasal cavity and the cranial fossa may result in the transfer of air, fluid, bacteria and nasocranial fistula formation. To facilitate nasal mucosa healing and limit a pressure gradient across the communication, postoperative endotracheal intubation may be extended for 3 to 5 days and/or bilateral nasopharyngeal airways may be placed after extubation. The avoidance of positive-pressure ventilation, enforcement of sinus precautions, and restriction of nose blowing further limit reflux of air, fluid, and bacteria (nose to cranial fossa) during the early postoperative period. When anterior cranial vault procedures are performed and aerated frontal sinuses are present, management is by either cranialization or obliteration.

Aside from a learning curve in mastering the technical skills of completing the monobloc osteotomies and disimpaction, the surgical morbidity from these procedures primarily results from a combination of the anticipated retrofrontal dead space, unavoidable tears in the nasal mucosa, and management of nasocranial communication across the skull base gap with the potential for fluid, air, and bacterial contamination. Two benchmark studies clarify these issues.

Posnick and associates[75] studied the issue of retrofrontal dead space, the communication across the skull base, nasal mucosal lacerations, and associated morbidity in a consecutive series of patients (N = 23) undergoing either monobloc or facial bipartition osteotomies combined with cranial expansion by a single surgeon (JCP) during a 4-year time frame (1987–1991). The extradural (retrofrontal) dead space was measured from consistent CT scan images at specific postoperative intervals (immediate, 6–8 wk, and 1 yr). The study confirmed the presence of an immediate retrofrontal dead space that generally filled in with the expanding brain/dura by 6 to 8 weeks after surgery. Specific intraoperative measures were taken by the surgeon to close (seal) the nasofrontal communication using flaps, fibrin glue, and Gelfoam. Precautions to prevent a pressure gradient across the communication (repair of dural tears, sinus precautions, and nasal stinting) were meticulously adhered to with the objective of providing time for nasal mucosal healing. The infection rate in this study group was limited to 2 of 23 patients or 9%. In both patients who developed infection, a retrofrontal (extradural) fluid collection with drainage across the residual nasofrontal communication occurred. Both patients healed without major sequela but did require further reconstruction of resorbed portions of the cranial vault.

Wolfe[76] completed a critical analysis of 81 monobloc advancements carried out over a 27-year period. This was a retrospective chart analysis of a series of patients undergoing either monobloc (frontofacial) advancement (MFFA) or facial bipartition (MFFA plus FB). The procedures were carried out at seven different craniofacial centers and included 49 MFFAs and 32 MFFAs plus FBs. This included the techniques of osteotomy followed by placing the osteotomized units in their preferred location in the operating room (classic approach) and osteotomies carried out followed by distraction (buried versus external). There were significant complications in the distracted (DO) group and fewer in the nondistracted (classic osteotomy) group. Complications included 2 deaths (cardiac arrest in 1 patient and complications arising from hypovolemia in the other). One case was aborted owing to large volume blood loss; there were 3 infections/sequestrations; and 1 CSF leak (no meningitis). The authors concluded that the MMFA or MMFA plus FB provided a superior morphologic result to the Le Fort III advancement. All patients were believed to also require orthognathic surgery. Blood loss and operative time were equivalent for both classic and distraction procedures. The authors concluded that for the majority of patients, the classic approach offered improved morphologic results. The incidence of infection and CSF leaks was not diminished through the use of the distraction approach.

Bradley and coworkers[77] recently completed a single-center retrospective chart review study comparing differences in morbidity in a series of CFD patients undergoing combined cranial vault expansion and monobloc advancement for correction of the upper and middle face deformities/hypoplasia. They describe three different sequential treatment groups over a period of 23 years. Group I patients (1979–1989: N = 12) underwent monobloc advancement without any special attention to the retrofrontal dead space or the communication through the skull base between the anterior cranial fossa and the nasal cavity. Group II (1989–1995: N = 11) patients underwent varied attempts at closure of the skull base gap with pericranial flaps and fibrin glue. Group III (1995–2002: N = 24) patients underwent monobloc osteotomies and disimpaction but without any immediate advancement. An internal distraction device was placed across the osteotomized zygoma on each side. After 7 days, the monobloc and forehead advancement was initiated at 1 mm/day for approximately 2 to 4 weeks. The infection rate for group III patients was significantly lower (2 of 24 patients or 8%) than for those in groups I and II. Neither of the two infections in group III resulted in bone loss. Not surprisingly, the group I patients had the greatest morbidity.

Advancement of the monobloc osteotomy in the CFD patient using the DO technique described by Bradley and coworkers[77] is likely to facilitate early nasal mucosa healing and thereby limit communication of fluid, air, and bacteria across the surgically created skull base gap. By allowing a 1-week delay in the actual advancement, the nasal mucosa may achieve sufficient healing to explain a drop in the infection rate from the 9% described by Posnick and associates[75] to the 8% described by Bradley and coworker's group III patients.

Bradley and coworkers[77] also describe a greater advancement in their group III (DO treatment) patients compared with their groups I and II patients. Confounding variables in their study may explain these differences including greater experience and improved surgical technique by the surgeons in the later years of their study (group III patients) and effect of co-morbidities (e.g., high rate of infection in group I and

group II patients that likely increased relapse and limited long-term advancement). More important, no correlation is shown by Bradley and coworkers[77] between a number of millimeters of advancement of the frontofacial osteotomies in the CFD patient and either greater functional gains or enhanced facial aesthetics. In fact, with a monobloc (frontofacial) osteotomy as much aesthetic damage is done by overadvancement (enophthalmos) as by underadvancement (residual eye proptosis). In addition, the achievement of a "normal" occlusion is rarely a treatment objective at the time of monobloc advancement. Accomplishing an ideal occlusion without creating enophthalmos requires a separate Le Fort I osteotomy to differentially advance the maxilla often combined with maxillary segmentation and mandibular (sagittal split) osteotomies. To achieve the most favorable facial balance for the CFD patient, an experienced clinician's aesthetic sense of the preferred morphology and focused technical expertise to alter the skeleton intraoperatively is essential. Several key technical aspects include

1. The ability to remove, segment, and then reshape and stabilize (plates and screws) the anterior cranial vault.
2. The ability to separate the orbits and midface as a unit (monobloc) from the skull base.
3. The ability to further segment the monobloc (at the upper orbits) and reconstruct (with cranial grafts) as needed.
4. The ability to separate the monobloc into halves (facial bipartition) and then alter the two facial halves to achieve the most favorable morphology. This often requires simultaneously increasing the maxillary transverse width and decreasing the upper face width to correct hypertelorism of the orbits, zygomas, nose, and bitemporal regions (e.g., Apert's syndrome). Facial bipartition also provides the ability to correct transverse facial arc of rotation deformities. For example, changing the Apert's syndrome patients' concave facial arc of rotation toward a normal convexity is an essential aspect of the reconstruction.

Any potential advantage of limiting morbidity due to infection across the skull base with the DO technique should be considered in light of limitations to achieve the previously mentioned key technical and aesthetic aspects (points 1–4). Added morbidity with the DO technique occurs: due to pin tract infection/scarring/loosening requiring reapplication; the need for device removal; and dependence on a patient's, family's, and clinician's continued commitment to staying the course for necessary outpatient DO procedures/adjustments to achieve an acceptable result must also be factored into the decision-making process.

When a CFD patient is to undergo intracranial volume expansion as part of the craniofacial procedure and they also require hydrocephalus management, the potential for morbidity increases. Complications may arise from excessive CSF drainage (overshunting). With "overshunting," there is decreased brain volume to fill any surgically created retrofrontal dead space. Frontofacial advancement and/or cranial vault expansion procedures should be carefully staged with ventriculoperitoneal (VP) shunting procedures. We believe that the presence or absence of a VP shunt is not in itself a major risk factor in the success of a frontofacial advancement procedure. An important aspect is satisfactory physiologic function of the ventricular system. Ultimately, the decision regarding the need for and sequencing of shunting is based on the patient's neurologic findings and the neurosurgeon's judgment. In a patient with a VP shunt in place before the surgery, experienced neurosurgical evaluation including CT scanning of the ventricular system is carried out to confirm physiologic shunt function.

Soft Tissue Management

A layered closure of the coronal incision (galea and skin) optimizes healing and limits scar widening. Resuspension of the midface periosteum to the temporalis fascia in a superior and posterior direction facilitates redraping of the soft tissues. Each lateral canthus should be adequately suspended or reattached in a superoposterior direction to the lateral orbital rim. The use of chromic gut for closure of the scalp skin in children may be used to obviate the need for postoperative suture or staple removal.

CROUZON'S SYNDROME

Crouzon's syndrome is a frequent form of CFD. It is characterized by multiple anomalies of the craniofacial skeleton with an autosomal dominant inheritance pattern. Its manifestations are generally less severe than those of Apert's syndrome and there are no malformations of the extremities. Typically, the cranial vault presentation is premature synostosis of both coronal sutures with a resultant brachycephalic shape to the skull. Cranial vault suture involvement other than coronal may include sagittal, metopic, or lambdoidal, either in isolation or in any combination. The cranial base and upper face sutures are variably involved, resulting in a degree of midface hypoplasia with an Angle class III malocclusion. The orbits are hypoplastic, resulting in a degree of proptosis with additional orbital dystopia that may produce a mild orbital hypertelorism and flatness to the (transverse) arc of rotation of the midface

By clinical examination alone, it may be difficult to accurately separate nonsyndromic bilateral coronal synostosis from Crouzon's syndrome, with its expected midface deficiency. It is now known that Crouzon's syndrome is caused by multiple mutations in the fibroblast growth factor receptor-2 *(FGFR2)* gene, some of which are identical to those seen in Pfeiffer's and Jackson-Weiss syndromes. It is likely that in the past many of these individuals have been misdiagnosed as having Crouzon's syndrome. The 5% of individuals with Crouzon's syndrome who have acanthosis nigricans (pigmentary changes in the skin fold regions) are said to have Crouzon's syndrome with acanthosis nigricans. It can be present in the neonatal period or appear later. Crouzon's syndrome with acanthosis nigricans

has been described with a specific Ala391Glu mutation in *FGFR3*.

Primary Cranio-orbital Decompression: Reshaping in Infancy

The initial treatment for Crouzon's syndrome generally requires bilateral coronal suture release and simultaneous anterior cranial vault and upper orbital osteotomies with reshaping and advancement (see Figure 46-1).[63–66] Our preference is to carry this out when the child is 9 to 11 months of age unless clear signs of increased ICP are identified earlier in life (Figure 46-2). Reshaping of the upper three quarters of the orbital rims and supraorbital ridges is geared toward decreasing the bitemporal and anterior cranial base width, with simultaneous horizontal advancement to increase the anteroposterior dimension. This also increases the depth of the upper orbits, with some improvement of eye proptosis. The overlying forehead is then reconstructed according to morphologic needs. A degree of overcorrection is preferred at the level of the supraorbital ridge when the procedure is carried out in infancy. In our opinion, by allowing additional growth to occur (waiting until the child is 9–11 mo old), the reconstructed cranial vault and upper orbital shape is better maintained with less need for repeat craniotomy procedures but without risking compression of the underlying brain.

The goals at this stage are to provide increased intracranial space in the anterior cranial vault for the brain; to increase the orbital volume, which allows the eyes to be positioned more



FIGURE 46-2. An 18-month-old girl with brachycephaly and midface deficiency with a mild degree of papilledema was referred for evaluation. She was found to have bilateral coronal synostosis and midface hypoplasia without extremity anomalies. The diagnosis of Crouzon's syndrome was made. She underwent cranio-orbital reshaping (see Figure 46-1). Several months later, a ventriculoperitoneal shunt was placed for management of hydrocephalus. Further staged reconstruction will include a total midface advancement procedure later in childhood followed by orthodontic treatment and orthognathic surgery in the early teenage years. **A,** Frontal view before surgery. **B,** Frontal view at 3 years of age, 1.5 years after undergoing cranio-orbital decompression and reshaping. **C,** Profile view before surgery. **D,** Profile view at 3 years of age, 1.5 years after undergoing first-stage cranio-orbital decompression and reshaping. **E** and **F,** Three-dimensional computed tomography (CT) scan views of craniofacial skeleton, just 1 week after cranio-orbital reshaping with advancement. (**A–F,** Reproduced with permission from Posnick JC. Crouzon syndrome: evaluation and staging of reconstruction. In Posnick JC, editor. Craniofacial and Maxillofacial Surgery in Children and Young Adults. Philadelphia: WB Saunders; 2000; p. 275.)

normally for better protection from exposure; and to improve the morphology of the forehead and upper orbits.

A postauricular coronal (scalp) incision is made, and the anterior scalp flap is elevated along with the temporalis muscle in the subperiosteal plane. Bilateral circumferential periorbital dissection follows, with detachment of the lateral canthi, but with preservation of the medial canthi and nasolacrimal apparatus to the medial orbital walls. The subperiosteal dissection is continued down the lateral and infraorbital rims to include the anterior aspect of the maxilla and zygomatic buttress. The neurosurgeon then completes the craniotomy to remove the dysmorphic anterior cranial vault. With protection of the frontal and temporal lobes of the brain (remaining anterior to each olfactory bulb), safe direct visualization of the anterior cranial base and orbits is possible at the time of orbital osteotomies.

The orbital osteotomies are then completed across the orbital roof and superior aspect of the medial orbital walls, laterally through the lateral orbital walls and inferiorly just into the inferior orbital fissures. The three-quarter orbital osteotomy units, with their tenon extensions, are removed from the field. The orbital units are reshaped and reinset into a preferred position. Orbital depth is thereby increased, and global proptosis is reduced. Fixation is generally achieved with 28-gauge interosseous wires or suture at each infraorbital rim and with plates and titanium or resorbable screws at the tenon extensions and frontonasal regions.

The removed calvaria is cut into segments, which are placed individually to achieve a more normally configured anterior cranial vault. The goal of reshaping is to narrow the anterior cranial base and orbital width slightly and provide more forward projection and overall normal morphology.

Repeat Craniotomy for Additional Cranial Vault Expansion and Reshaping in Young Children

After the initial suture release, decompression, and reshaping are carried out during infancy, the child is observed clinically at intervals by the craniofacial surgeon, pediatric neurosurgeon, pediatric ophthalmologist, and developmental specialist and undergoes interval CT scanning. Should signs of increased ICP develop, urgent brain decompression with cranial vault expansion and reshaping is performed.[47] When increased ICP is suspected, the location of brain compression influences for which region of the skull further expansion and reshaping is planned.

If the brain compression is judged to be anterior, then further anterior cranial vault and upper orbital osteotomies with reshaping and advancement are carried out. The technique is similar to that described previously.[47,65] If the problem is posterior compression, expansion of the posterior cranial vault, with the patient in the prone position, is required (Figure 46-3).

The "repeat" craniotomy carried out for further decompression and reshaping in the child with Crouzon's syndrome is often complicated by brittle cortical bone (which lacks a diploic space and contains sharp spicules piercing the dura), the presence of previously placed fixation devices in the operative field (e.g., Silastic sheeting, metal clips, stainless steel wires, plates, and screws), and convoluted thin dura compressed against (or herniated into) the inner table of the skull. All of these issues result in a greater potential for dural tears during the calvarectomy than would normally occur during the primary procedure. A greater potential for morbidity should be anticipated when reelevating the scalp flap, dissecting the dura free of the inner table of the skull and cranial base, and then removing the cranial vault bone.

Management of "Total Midface" Deformity in Childhood

The type of osteotomies selected to manage the "total midface" deficiency or deformity and residual cranial vault dysplasia should depend on the extent and location of the presenting dysmorphology rather than on a fixed approach to the midface malformation.[47,53–56,65,66,72,78–82] The selection of a monobloc (with or without additional orbital segmentation), facial bipartition (with or without additional orbital segmental osteotomies), or Le Fort III osteotomy to manage the basic horizontal, transverse, vertical orbital, and upper midface deficiencies or deformities in a patient with Crouzon's syndrome depends on the patient's presenting midface and anterior cranial vault morphology. The observed dysmorphology is dependent on the original malformation, the previous procedures carried out, and the effects of ongoing growth (Figures 46-4 and 46-5).

When evaluating the upper and midface in a child born with Crouzon's syndrome, if the supraorbital ridge is in good position when viewed from the sagittal plane (the depth of the upper orbits is adequate), the midface and forehead have a normal arc of rotation in the transverse plane (not concave), and the root of the nose is of normal width (minimal orbital hypertelorism), there is little need to reconstruct this region (the forehead and upper orbits) any further. In such patients, the basic residual midface deformity is in the lower half of the orbits, zygomatic buttress, and maxilla. If so, the deformity may be effectively managed using an extracranial Le Fort III osteotomy.

If the supraorbital ridges, anterior cranial base, zygomas, nose, lower orbits, and maxilla all remain deficient in the sagittal plane (horizontal retrusion), a monobloc osteotomy is indicated (see Figures 46-4 and 46-5). In these patients, the forehead is generally flat and retruded and will also require reshaping and advancement. If upper midface hypertelorism (increased transverse width) and midface flattening (horizontal retrusion) with loss of the normal facial curvature (concave arc) are also present, the monobloc unit is split vertically in the midline (facial bipartition), a wedge of interorbital (nasal and ethmoidal) bone is removed, and the orbits and zygomas are repositioned medially while the maxilla at the palatal level is widened. The facial bipartition is rarely

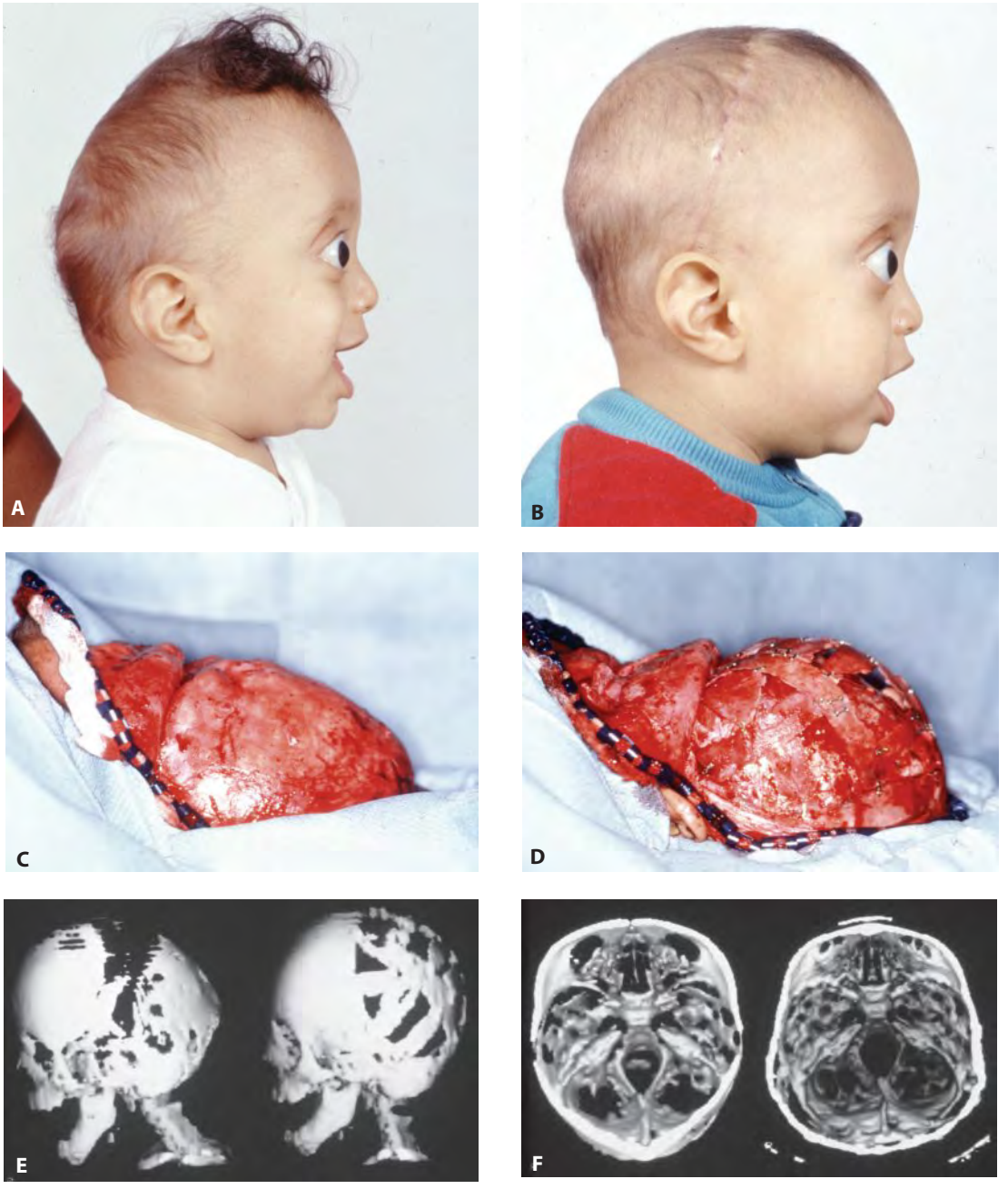


FIGURE 46-3. A child with Crouzon's syndrome is shown at 10 months of age. His deformities are characterized by mild bilateral coronal and marked bilateral lambdoid synostosis in combination with midface deficiency. He has diminished intracranial volume, resulting in brain compression. The orbits are shallow with resulting eye proptosis, and the midface is deficient with malocclusion. He is shown before and after undergoing posterior cranial vault decompression and reshaping to expand the intracranial volume. He later underwent placement of a ventriculoperitoneal shunt for management of hydrocephalus. He will require a total midface advancement (monobloc) with further anterior cranial vault reshaping after 5 years of age. This will be followed by orthognathic surgery in combination with orthodontic treatment in the teenage years. **A,** Profile view before surgery. **B,** Profile view after posterior cranial vault reconstruction. **C,** Intraoperative lateral view of the cranial vault (patient in prone position) as seen with the posterior scalp flap elevated. **D,** Same intraoperative view after posterior cranial vault decompression, reshaping, and fixation of bone segments with microplates and screws. **E,** Comparison of three-dimensional CT scan views before and after reconstruction. **F,** Comparison of three-dimensional CT scan views of the cranial base before and after reconstruction. (**A–F,** Reproduced with permission from Posnick JC. The craniofacial dysostosis syndromes: secondary management of craniofacial disorders. Clin Plast Surg 1997;24:429.)

required in Crouzon's syndrome, but the monobloc is. When a monobloc or facial bipartition osteotomy is carried out as the "total midface" procedure, additional segmentation of the upper and lateral orbits for reconstruction may also be required to normalize the morphology of the orbital aesthetic units.[83–88]

For most patients, a surgeon's attempt to simultaneously adjust the orbits and idealize the occlusion using the Le

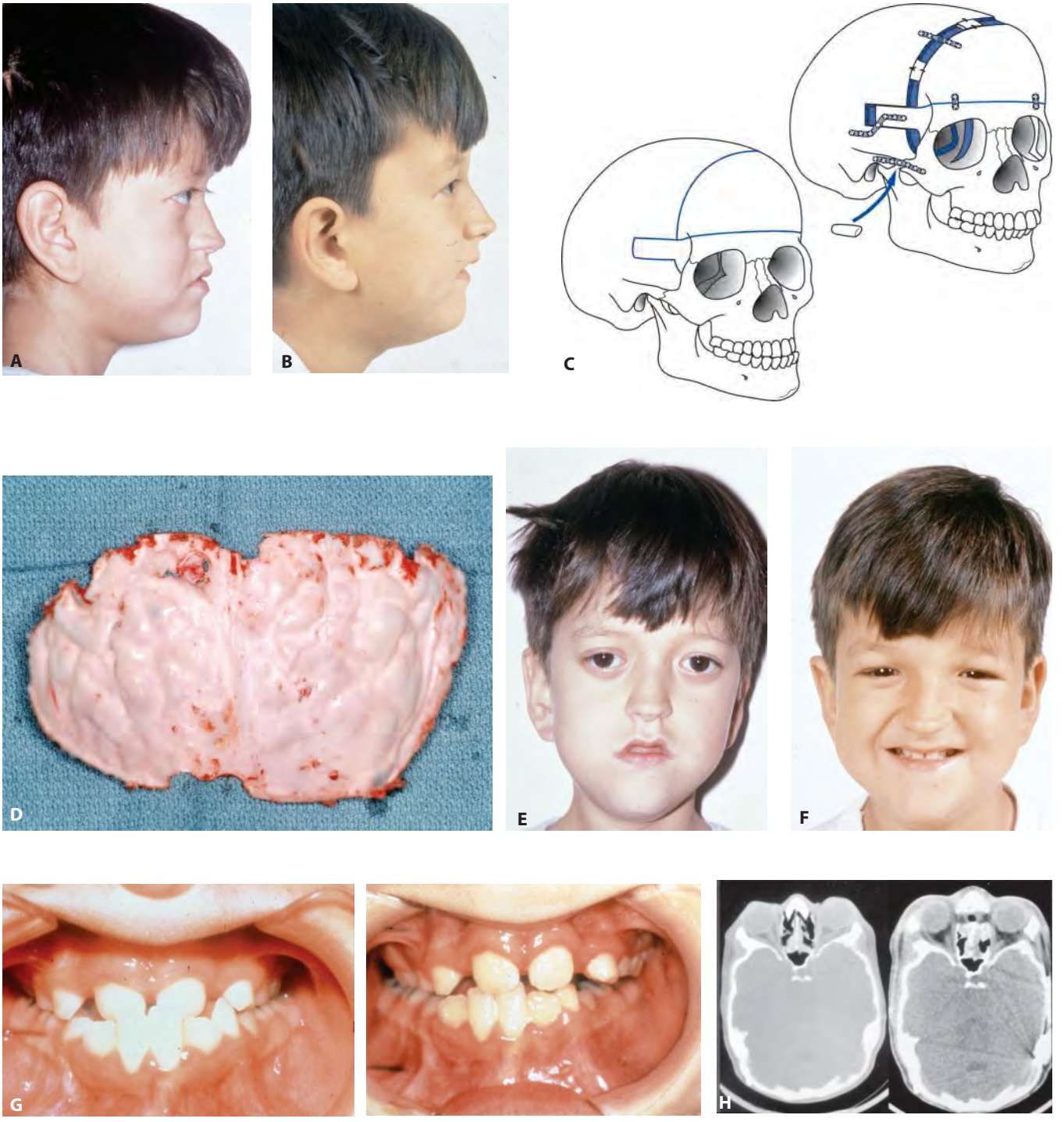


FIGURE 46-4. A child at 8 years of age with Crouzon's syndrome who underwent a limited first-stage cranio-orbital procedure at 6 weeks of age. He then underwent anterior cranial vault and monobloc (orbits and midface) osteotomies with advancement. **A,** Profile view before the monobloc procedure. **B,** Profile view after reconstruction. **C,** Craniofacial morphology before and after anterior the cranial vault and monobloc osteotomies with advancement as carried out. Osteotomy locations indicated. Stabilization with cranial bone grafts and miniplates and screws. **D,** View of the inner surface of the frontal bones after bifrontal craniotomy. Compression of the brain against the inner table has resulted in resorption of the inner skull. This is an indication of longstanding increased intracranial pressure (ICP). **E,** Frontal view before surgery. **F,** Frontal view after reconstruction. **G,** Occlusal views before and after reconstruction. **H,** Comparison of axial CT slices through the midorbits before and after reconstruction indicates resulting increased intraorbital depth and decreased proptosis achieved. *(Continued)*

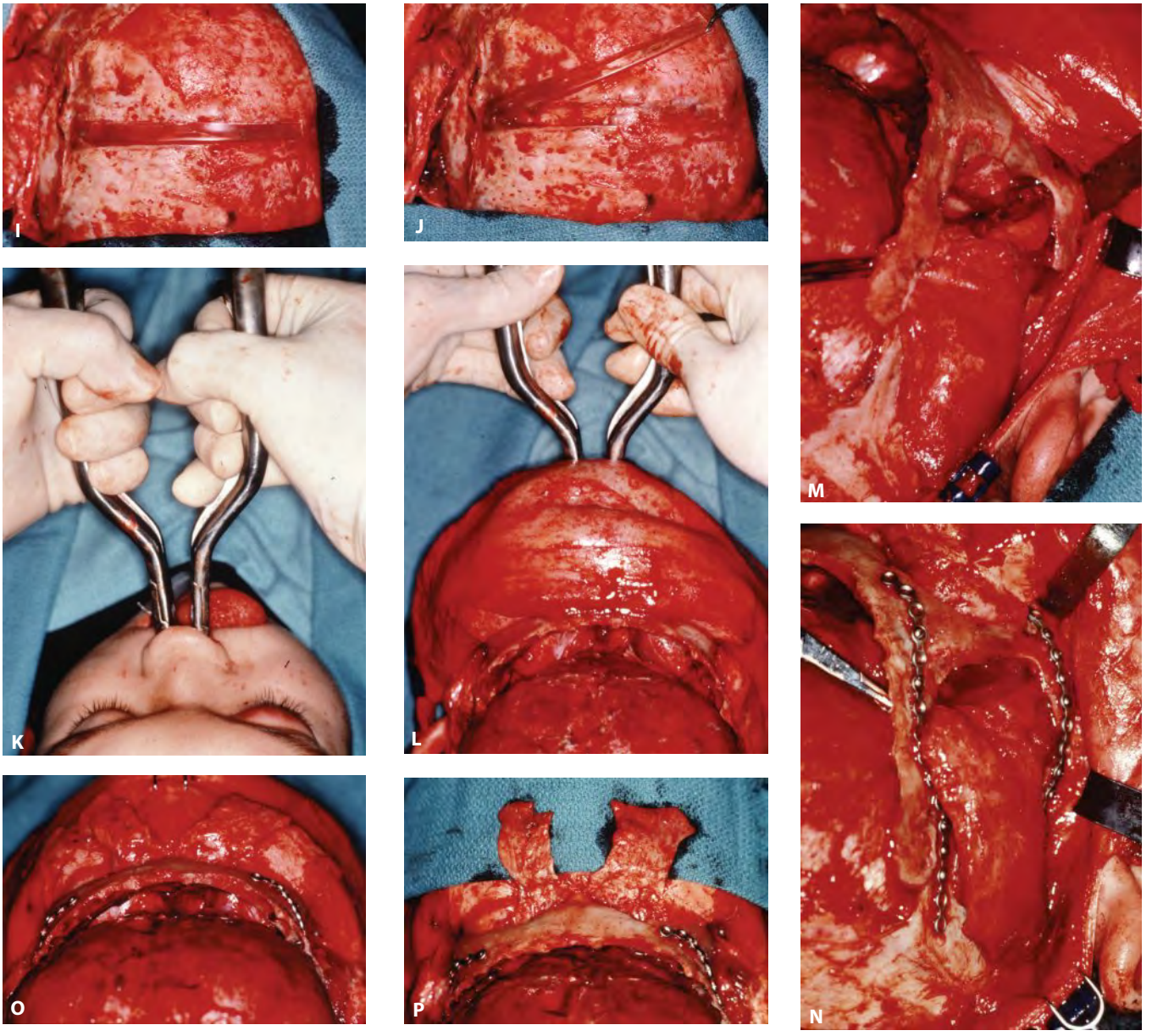
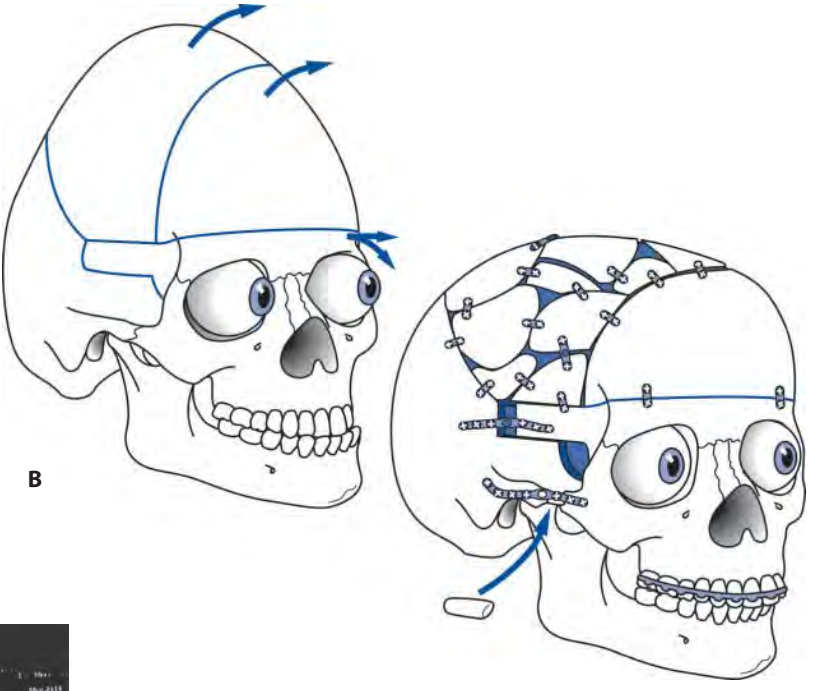
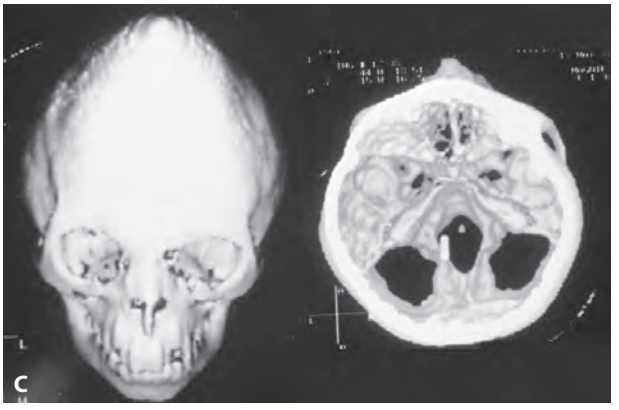


FIGURE 46-4. *(Continued)* **I,** Intraoperative bird's-eye lateral view of the cranial vault demonstrates Silastic strip that had been placed by the neurosurgeon when the patient was 2 months of age (8 yr earlier). **J,** Removing the Silastic strip along the sphenoid wing region is difficult owing to bone overgrowth. **K,** Intraoperative view of (Rowe) forceps in the nose and mouth after monobloc osteotomy but before disimpaction. **L,** Same view but with the coronal incision turned down, indicating the degree of advancement at the supraorbital ridge level after disimpaction. **M,** Lateral view of the zygomatic arch and tenon extension of the supraorbital rim after monobloc advancement just before miniplate fixation. **N,** Same view after miniplate fixation of the zygomatic arch and tenon extension. **O,** Bird's-eye view of the stabilized monobloc unit after advancement. There is increased intracranial volume (dead space) in the anterior cranial vault for brain expansion. **P,** Same view with elevated pericranial flaps, which will be turned in to close the opening between the nose and the anterior cranial base. (**A, B, D–P,** Reproduced with permission from Posnick JC. Craniofacial dysostosis: management of the midface deformity. In Bell WH, editor. Orthognathic and Reconstructive Surgery. Philadelphia: WB Saunders; 1992; p. 1888; **C,** adapted from Posnick JC. Craniofacial dysostosis: management of the midface deformity. In Bell WH, editor. Orthognathic and Reconstructive Surgery. Philadelphia: WB Saunders; 1992; p. 1888.)

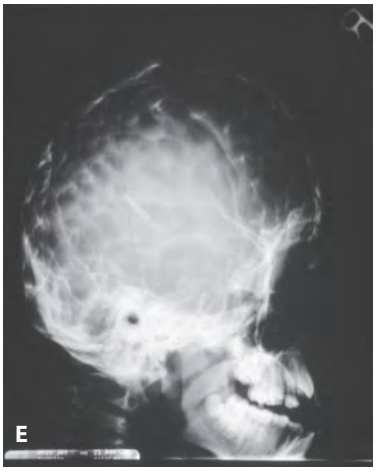
Fort III, monobloc, or facial bipartition osteotomy in isolation, without completing a separate Le Fort I osteotomy, is an error in judgment. The degree of horizontal deficiency observed at the orbits and maxillary dentition is rarely uniform. This further segmentation of the midface complex at the Le Fort I level is required to establish normal proportions. If a Le Fort I separation of the total midface complex is not carried out and the surgeon attempts to achieve a positive overbite and overjet at the incisor teeth, overadvancement of the orbits with enophthalmos will occur. The Le Fort I osteotomy is generally not performed at the time of the total midface procedure. This will await skeletal maturity and then













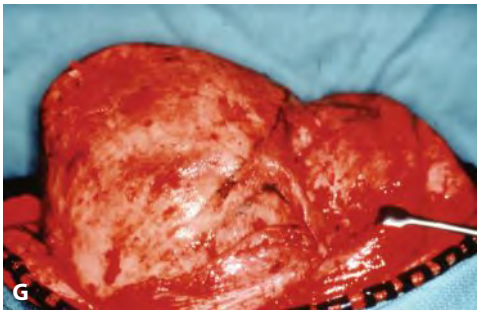




FIGURE 46-5. A 12-year-old boy with unrepaired Crouzon's syndrome who underwent total cranial vault and monobloc osteotomies with reshaping and advancement. **A,** Oblique view before surgery. **B,** The patient's craniofacial morphology before surgery. Osteotomy locations indicated. A second illustration after the osteotomies were completed with advancement, reshaping, and fixation. **C,** Three-dimensional CT scan views of the cranial vault and cranial base before surgery. **D,** Intraoperative view with forceps placed at the orbital rims indicates the extent of proptosis. **E,** Lateral skull radiograph with "fingerprinting" indicates longstanding increased ICP. **F,** Inner table internal side of the frontal bone indicates compression of the brain against the inner table of the skull. **G,** Intraoperative lateral view of the cranial vault and orbits through the coronal incision before osteotomies. **H,** Same view after osteotomies, reshaping, and stabilization of bone segments with miniplates and screws. *(Continued)*

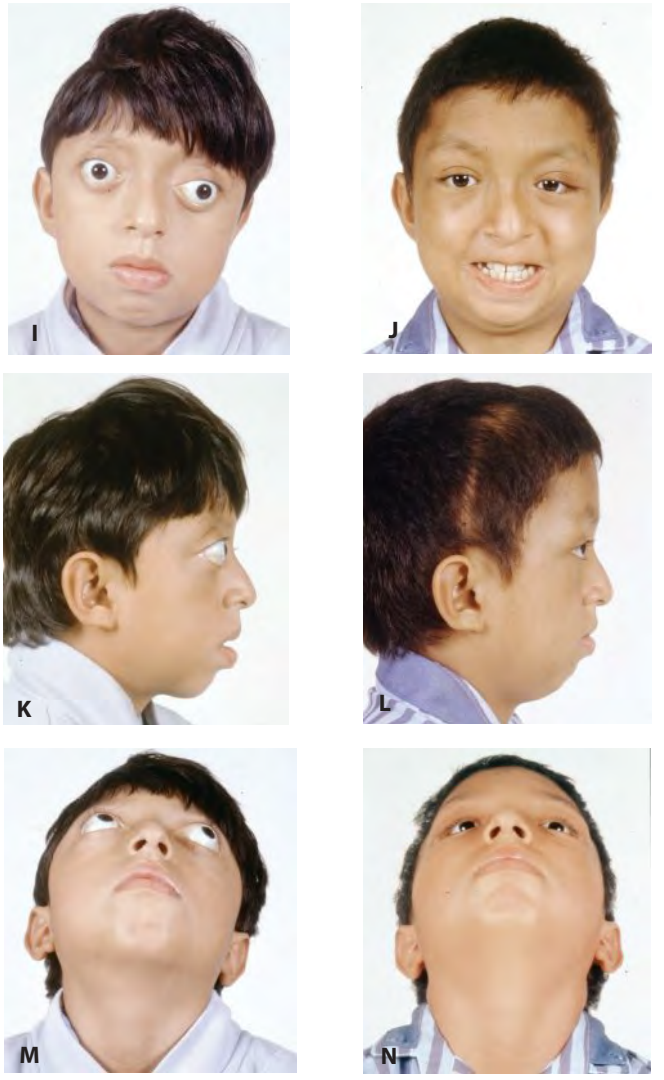


FIGURE 46-5. *(Continued)* **I,** Frontal view before surgery. **J,** Frontal view after reconstruction. **K,** Profile view before surgery. **L,** Profile view after reconstruction. **M,** Worm's-eye view before surgery. **N,** Worm's-eye view after reconstruction. **O,** Comparison of lateral cephalometric radiographs before and after reconstruction. **P,** Comparison of three-dimensional CT scan views before and after reconstruction. (**A, C–P,** reproduced with permission from Posnick JC. Craniofacial dysostosis: management of the midface deformity. In Bell WH, editor. Orthognathic and Reconstructive Surgery. Philadelphia: WB Saunders; 1992; p. 1888; **B,** adapted from Posnick JC. Craniofacial dysostosis: management of the midface deformity. In Bell WH, editor. Orthognathic and Reconstructive Surgery. Philadelphia: WB Saunders; 1992; p. 1888.)

be combined with orthodontic treatment. Until then, an Angle class III malocclusion will remain.

A major aesthetic problem specific to the Le Fort III osteotomy when its indications are less than ideal is the creation of irregular step-offs in the lateral orbital rims. This will occur when even a moderate (Le Fort III) advancement is carried out. These lateral orbital step-offs are unattractive and are visible to the casual observer at conversational distance. Surgical modification performed later is difficult, often with less than ideal aesthetic results. Another problem with the Le Fort III osteotomy is the difficulty in judging an ideal orbital depth. A frequent result is either residual proptosis or enophthalmos. Simultaneous correction of orbital hypertelorism or correction of a midface arc-of-rotation problem is not possible with the Le Fort III procedure. Excessive lengthening of the nose, accompanied by flattening of the nasofrontal angle, will also occur if the Le Fort III osteotomy is selected when the skeletal morphology favors a monobloc or facial bipartition

procedure. It is not possible to later correct the surgically created vertical elongation of the nose.

Final reconstruction, as discussed previously, of the cranial vault deformities and orbital dystopia in Crouzon's syndrome can be managed in patients as young as 5 to 7 years of age.[47] By this age, the cranial vault and orbits normally attain approximately 85% to 90% of their adult size.[43] When the upper midface and final cranial vault procedure is carried out at or after this age, the reconstructive objectives are to approximate adult dimensions in the cranio-orbitozygomatic region, with the expectation of a stable result (no longer influenced by growth) once healing has occurred (see Figure 46-4). Psychosocial considerations also support the upper midface and final cranial vault procedure taking place in patients 5 to 7 years of age. When the procedure is carried out at this age, the child may enter the first grade with an opportunity for satisfactory self-esteem. Routine orthognathic surgery will be necessary at the time of skeletal maturity to achieve an ideal occlusion, facial profile, and smile.

Orthognathic Procedures for Definitive Occlusal and Lower Facial Aesthetic Reconstruction

Although the mandible has a normal basic growth potential in Crouzon's syndrome, the maxilla does not. An Angle class III malocclusion, resulting from maxillary retrusion, with anterior open bite often results. A Le Fort I osteotomy to allow for horizontal advancement, transverse widening, and vertical adjustment is generally required in combination with an osteoplastic genioplasty (vertical reduction and horizontal advancement) to further correct the lower face deformity. Secondary deformities of the mandible should be simultaneously corrected through sagittal split ramus osteotomies. The elective orthognathic surgery is carried out in conjunction with orthodontic treatment planned for completion at the time of early skeletal maturity (~13–15 yr in girls and 15–17 yr in boys) (Figure 46-6).

Assessment of Results in the Crouzon Patient

The purpose of a quantitative assessment of the craniofacial complex, whether by CT scan analysis, anthropometric measurement, cephalometric analysis, or dental model analysis, is to help predict growth patterns, confirm or refute clinical impressions, aid in treatment planning, and provide a framework for objective assessment of the immediate and long-term reconstructive results.

Quantitative Assessment of Presenting Crouzon's Deformity and Surgical Results Based on CT Scan Analysis after First-Stage Cranio-orbital Reconstruction

Waitzman and colleagues[89,90] developed a method of analysis based on CT scan measurements that allows for a more quantitative assessment of the upper and midface skeleton in both the horizontal and the transverse planes than was previously available. This method of quantitative CT scan analysis was utilized to document the differences in the cranio-orbitozygomatic region between children with Crouzon's syndrome who had not yet undergone reconstruction and age-matched controls. Morphologic results achieved in those children 1 year after undergoing a classic suture release and anterior cranial vault and upper orbital procedure, designed to decompress and reshape these regions, were also evaluated.

The preoperative CT scan measurements of the children with untreated Crouzon's syndrome confirmed a widened anterior cranial vault at 108% of normal and a cranial length averaging only 92% of normal. In comparison with age-matched controls, orbital measurements revealed a widened anterior interorbital width at 122% of normal, an increased intertemporal width at 121% of normal, globe protrusion at 119% of normal, and a short medial orbital wall length at only 86% of normal. The distance between the zygomatic buttresses and the interarch widths were found to be increased at 106% and 103% of normal, respectively. The zygomatic arch lengths were substantially shortened at only 87% of the values of age-matched controls.

These findings confirmed clinical observations of brachycephalic anterior cranial vaults with shallow, frequently hyperteloric orbits and globe proptosis. Generally, the midface in Crouzon's syndrome is horizontally retrusive and transversely wide, which is reflected in wide and shortened zygomas. The same quantitative CT scan assessment was carried out in the children with Crouzon's syndrome more than 1 year after undergoing anterior cranial vault and upper orbital osteotomies with reshaping, to compare their values with the new age-matched control values; we were not able to demonstrate any significant improvement in the cranio-orbitozygomatic measurements.

Quantitative Intracranial Volume Measurements before and after Cranio-orbital Reshaping in Children with Crouzon's Syndrome

In a previous study, we documented the intracranial volumes in children with Crouzon's syndrome before and after cranio-orbital reshaping procedures. The intracranial volumes also were compared with those of an age- and gender-matched cohort, and we also reviewed the rate of cranial expansion with growth.[89,90] The study included 13 children who presented sequentially with Crouzon's syndrome and who subsequently underwent a classic first-stage cranio-orbital reconstruction by the senior author (JCP) in conjunction with a pediatric neurosurgeon. The primary method of osteotomy and bone graft fixation varied (e.g., wires, microplates, miniplates, and screws). The average age at the time of operation was 13 months (range, 6–46 mo). Postoperative clinical follow-up ranged from 12 to 60 months at the time of the study's completion. Of the children with Crouzon's syndrome who were evaluated preoperatively, 12 of 13 had intracranial volume values greater than the mean. When comparing postoperative volumes with the normative data, all 13 maintained volumes at or greater than the mean. Ten of the 13 achieved



FIGURE 46-6. A 19-year-old boy born with Crouzon's syndrome. When he was 11 years of age, the patient was seen by another surgeon and underwent a Le Fort osteotomy with advancement through an extracranial approach. He presented in his late teenage years with asymmetrical and dystopic orbits, zygomatic hypoplasia, a retrusive upper jaw, an asymmetrical lower jaw, and a long chin. He underwent a combined orthodontic and orthognathic approach, including a Le Fort I osteotomy (horizontal advancement), bilateral sagittal split osteotomies of the mandible (correction of asymmetry), and an osteoplastic genioplasty (vertical reduction and horizontal advancement). Stabilization was accomplished with miniplates and screws. During the same general anesthesia procedure, he underwent a reopening of his coronal (scalp) incision with harvesting of split cranial grafts to recontour and augment the orbits and zygomas. **A,** Frontal view before surgery. **B,** Frontal view after reconstruction. **C,** Profile view before surgery. **D,** Profile view after reconstruction. **E,** Oblique view before surgery. **F,** Oblique view after reconstruction. **G,** Occlusal view before surgery. **H,** Occlusal view after reconstruction. **I,** Oblique occlusal view before surgery. **J,** Oblique occlusal view after reconstruction. **K,** Articulated dental casts before surgery. **L,** Articulated dental casts after model reconstruction. (**A–L,** Reproduced with permission from Posnick JC. Crouzon syndrome: evaluation and staging of reconstruction. In Posnick JC, editor. Craniofacial and Maxillofacial Surgery in Children and Young Adults. Philadelphia: WB Saunders; 2000; pp. 299–300.)

intracranial volumes equal to or greater than 2 standard deviations (SD) above the mean. When reviewing each Crouzon's patient's cranial capacity over time, 5 of the 13 approximated the normal growth curve, whereas 6 of the 13 exceeded it. According to the findings of our study, for the majority of children born with Crouzon's syndrome, the cranial capacity will exceed the mean early in life and expand at a rapid rate in conjunction with cranio-orbital decompression and reconstruction. The biologic explanation for these unexpected findings remains unclear. It should be noted that other researchers have studied the intracranial volume of children born with craniosynostosis. Conflicting data have been reported, and further investigation is needed.

Quantitative Assessment of Presenting Deformity in Children with Crouzon's Syndrome and Surgical Results after Monobloc Osteotomy Based on CT Scan Analysis

In the midchildhood years, a group of children with Crouzon's syndrome were assessed using quantitative CT scan measurements. We found them to have cranial vault lengths averaging only 87% of the age-matched norms. The medial orbital walls were (horizontally) short at 87% of normal, whereas the extent of globe protrusion was excessive at 134% of age-matched norms. The zygomatic arch lengths averaged only 84% of normal. These findings confirmed horizontal (anteroposterior) deficiency of the upper and midface.

After undergoing a monobloc osteotomy (orbits and midface) combined with anterior cranial vault reshaping and advancement carried out through an intracranial approach, the children's' cranio-orbitozygomatic measurements were taken again. The mean cranial length initially achieved (after monobloc osteotomy) was 98%, and at 1 year, it was 92% of the control value. When compared with age-matched controls, the orbital measurements reflected improvement in the midorbital hypertelorism (midinterorbital width was 97% initially after operation and 102% at 1 yr) and orbital proptosis (early after surgery, 86% of values for age-matched normal children; 92% at 1 yr). The medial orbital wall length initially normalized at 101% and later at 97% of normal values. The zygomatic arch length initially corrected at 106% and later to 101% of normal.

APERT'S SYNDROME

Apert's syndrome has previously been classified on the basis of its clinical findings.[91] Postmortem histologic and radiographic studies have confirmed that skeletal deficiencies in the patient with Apert's syndrome result from a cartilage dysplasia at the cranial base, leading to premature fusion of the midline sutures from the occiput to the anterior nasal septum. Molecular genetic studies now clarify that two common mutations account for 98% of Apert's syndrome: P253R and S252W.[92] These mutations occur in the identical location as the *FGFR1* mutation in Pfeiffer's syndrome and the *FGFR3* mutation in Muenke's syndrome. In addition, a component of the syndrome is four-limb complex syndactly of the hands and feet. Fusion and malformation of other joints, including the elbows and shoulders, often occur. In Apert's syndrome, fused cervical vertebrae (68%), usually C5–6 can occur. Hydrocephalus is less frequent than in Crouzon's syndrome (2% vs. 10%). Occasional internal organ anomalies can occur. The integument (soft tissue envelope) also varies from that in Crouzon's syndrome, with a greater downward slant to the lateral canthi and a distinctive, S-shaped upper eyelid ptosis. The quality of the skin often varies from normal with acne and hyperhidrosis being prominent features.

Apert's syndrome has previously been classified on the basis of its clinical findings.[93,94] Postmortem histologic and radiographic studies suggest that skeletal deficiencies in the patient with Apert's syndrome result from a cartilage dysplasia at the cranial base, leading to premature fusion of the midline sutures from the occiput to the anterior nasal septum.[75,95–98] In addition, a component of the syndrome is four-limb symmetry complex syndactylies of the hands and feet (Figure 46-7). Fusion and malformation of other joints, including the elbows and shoulders, often occur. The soft tissue envelope also varies from that in Crouzon's syndrome, with a greater downward slant to the canthi lateral and a distinctive, S-shaped upper eyelid ptosis. The quality of the skin often varies from normal, with acne and hyperhidrosis being prominent features. Molecular genetic studies now clarify that two common mutations account for 98% of Apert's syndrome: P253R and S252W.[92] These mutations occur in the identical location as the *FGFR1* mutation in Pfeiffer's syndrome and the *FGFR3* mutation in Muenke's syndrome. At the molecular level, one of two *FGFR2* mutations involving amino acids (Ser252Trp and Pro253Arg) have been found to cause Apert's syndrome in nearly all patients studied.[99–104]

Primary Cranio-orbital Decompression: Reshaping in Infancy

The initial craniofacial procedure for Apert's syndrome generally requires bilateral coronal suture release and anterior cranial vault and upper three-quarter orbital osteotomies to expand the anterior cranial vault and reshape the upper orbits and forehead (see Figure 46-1).[105,106] Our preference is to carry this out when the child is 9 to 11 months of age, unless signs of increased ICP are identified earlier in life. The main goals at this stage are to decompress the brain and provide increased space for it in the anterior cranial vault and to increase the orbital volume to decrease globe protrusion. The fronto-orbital surgical technique is similar to that described for Crouzon's syndrome (Figure 46-8).

Further Craniotomy for Additional Cranial Vault Expansion and Reshaping in Young Children

As described for Crouzon's syndrome, after the initial suture release, decompression, and reshaping carried out during infancy, the child is observed clinically at intervals by the

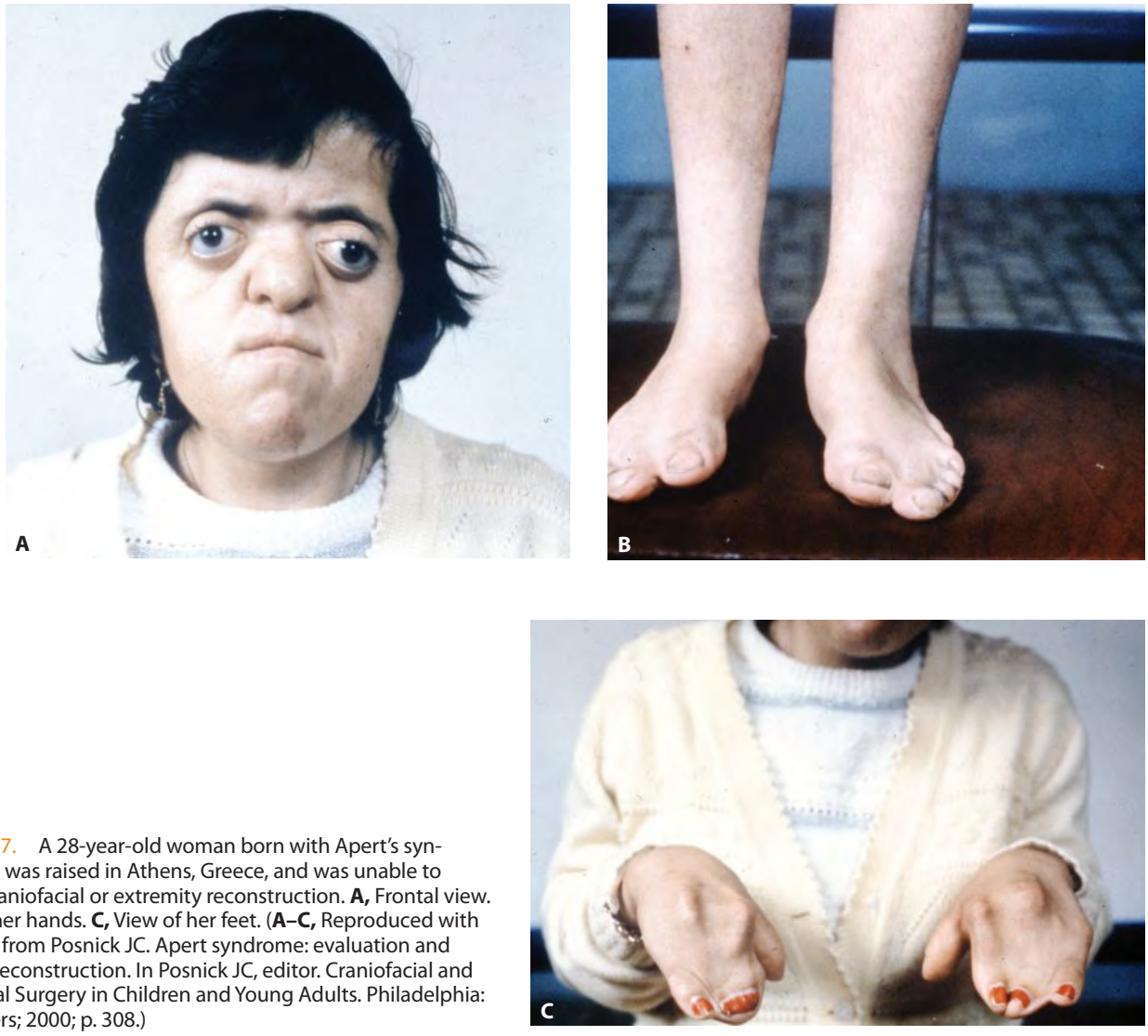


FIGURE 46-7. A 28-year-old woman born with Apert's syndrome. She was raised in Athens, Greece, and was unable to undergo craniofacial or extremity reconstruction. **A,** Frontal view. **B,** View of her hands. **C,** View of her feet. (**A–C,** Reproduced with permission from Posnick JC. Apert syndrome: evaluation and staging of reconstruction. In Posnick JC, editor. Craniofacial and Maxillofacial Surgery in Children and Young Adults. Philadelphia: WB Saunders; 2000; p. 308.)

craniofacial surgeon, pediatric neurosurgeon, pediatric ophthalmologist, and developmental pediatrician and undergoes interval CT scanning.[63,67,68] Should signs of increased ICP develop, further decompression with reshaping of the cranial vault to expand the intracranial volume is performed (Figure 46-9). In Apert's syndrome, the posterior cranial vault more commonly requires expansion. The technique is similar to that described for Crouzon's syndrome.

Management of the "Total Midface" Deformity in Childhood

In Apert's syndrome, for almost all patients, facial bipartition osteotomies combined with further cranial vault reshaping permit a more complete correction of the abnormal craniofacial skeleton than can be achieved through other midface procedure options (i.e., monobloc or Le Fort III osteotomies). When using the facial bipartition approach, a more normal arc of rotation of the midface complex is achieved with the midline split. This further reduces the stigmata of the preoperative "flat, wide, and retrusive" facial appearance. The facial bipartition also allows the orbits and zygomatic buttresses as units to shift to the midline (correction of hypertelorism) while the maxillary arch is simultaneously widened. Horizontal advancement of the reassembled midface complex is then achieved to normalize the orbital depth and zygomatic length. The forehead is generally flat, tall, and retruded, with a constricting band just above the supraorbital ridge, giving the impression of bitemporal narrowing. Reshaping of the anterior cranial vault is simultaneously carried out (Figure 46-10; see also Figures 46-8 and 46-9). See also Figure 46-9H for preoperative craniofacial morphology and planned and completed osteotomies and reshaping. Note that stabilization was achieved with cranial bone grafts and plate and screw fixation. A Le Fort III osteotomy is virtually never adequate for an ideal correction of the residual upper and midface deformity of Apert's syndrome.[107,108]

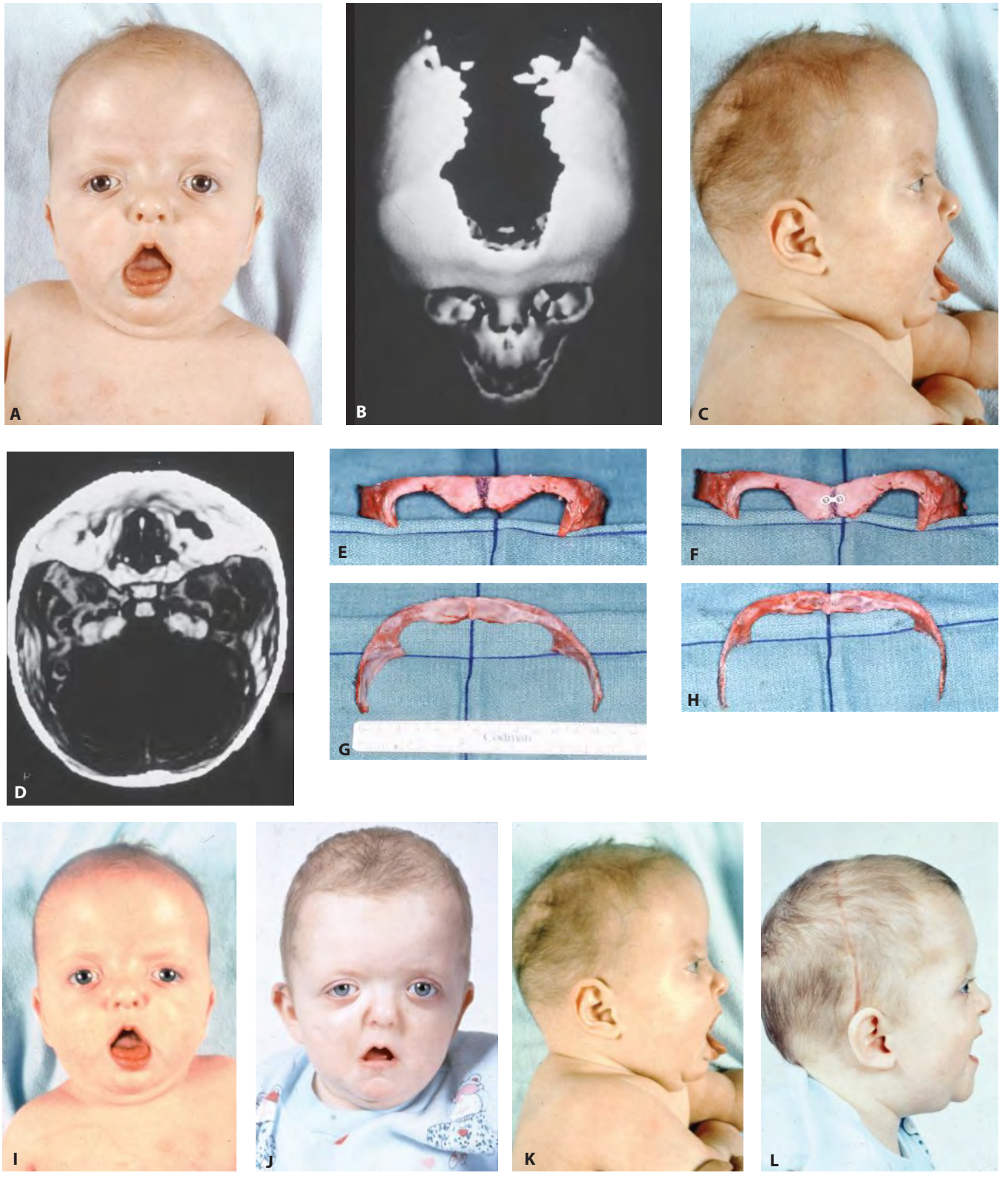


FIGURE 46-8. A 6-month-old girl with Apert's syndrome underwent anterior cranial vault and three-quarter orbital osteotomies with reshaping as described (see Figure 46-1). **A,** Frontal view before surgery. **B,** Three-dimensional CT scan view of cranial vault before surgery. **C,** Profile view before surgery. **D,** Three-dimensional CT scan view of the cranial base before surgery. **E,** Frontal view of the orbital osteotomy unit before reshaping. **F,** Frontal view of the orbital osteotomy unit after reshaping. **G,** Bird's-eye view of the orbital osteotomy unit before reshaping. **H,** Bird's-eye view of the orbital osteotomy unit after reshaping. **I,** Frontal view before surgery. **J,** Frontal view 1 year later. **K,** Profile view before surgery. **L,** Profile view 1 year later. *(Continued)*

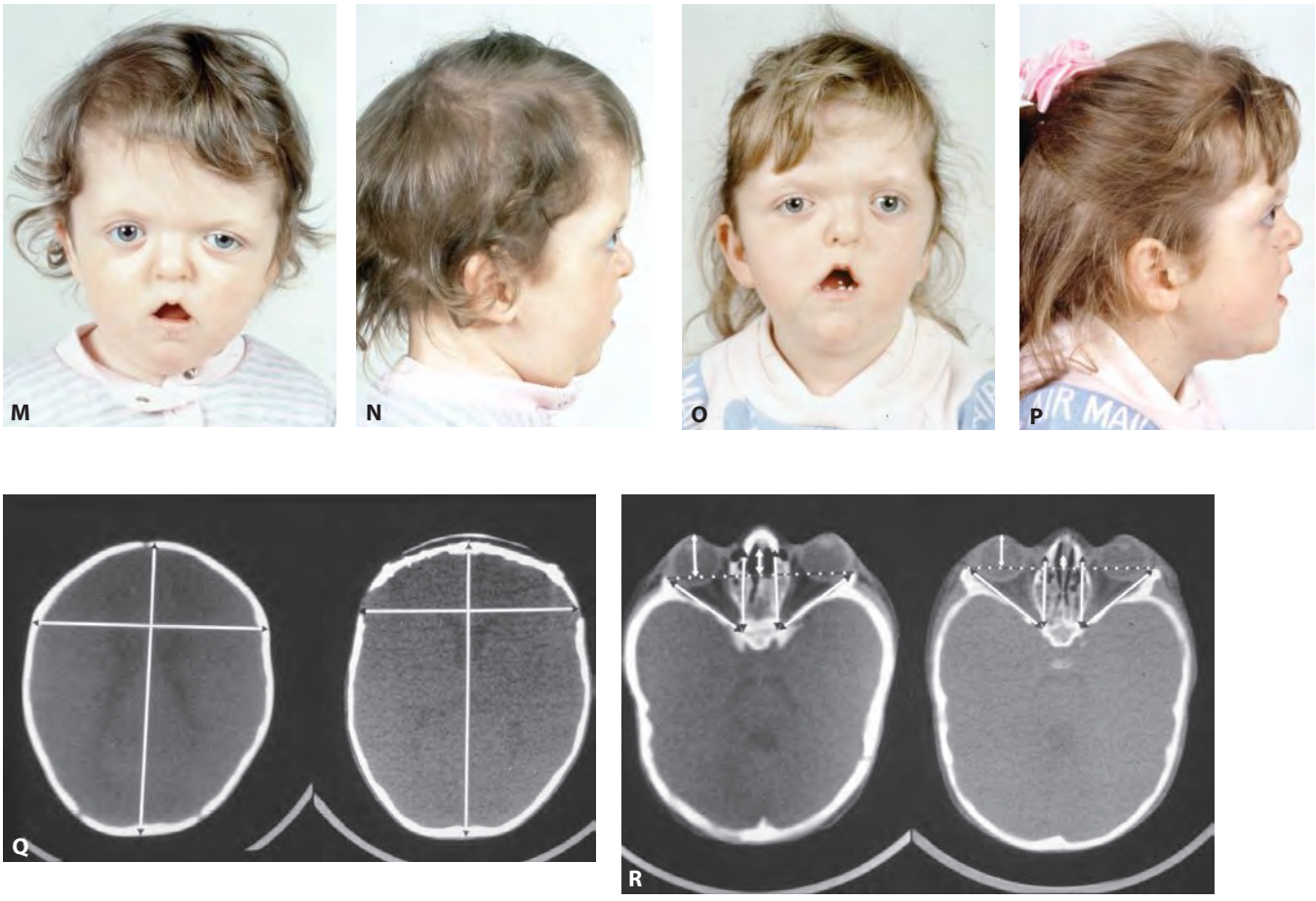


FIGURE 46-8. *(Continued)* **M** and **N,** Frontal and profile views 2 years after reconstruction. **O** and **P,** Frontal and profile views 3 years after first stage cranio-orbital reshaping; further staged reconstruction is required. **Q,** Comparison of standard axial CT slices through the cranial vault before and 1 year after cranio-orbital reshaping. The cranial vault length (cephalic length) has increased from 115 to 158 mm. The anterior cranial vault width (intercoronal distance) has remained stable at 115 mm. **R,** Comparison of standard axial CT slices through the midorbits before and 1 year after reconstruction. Marked globe protrusion of 16 mm has increased to 17 mm 1 year later. The anterior interorbital distance diminished from 29 to 25 mm, which still represented 137% of the age-matched control value. (Magnification of the individual CT scan images was not controlled for.) (**A–R,** Reproduced with permission from Posnick JC, Lin KY, Jhawar BJ, Armstrong D. Apert syndrome: quantitative assessment in presenting deformity and surgical results after first-stage reconstruction by CT scan. Plast Reconstr Surg 1994;93:489–497.)

Orthognathic Procedures for Definitive Occlusal and Lower Facial Aesthetic Reconstruction

The mandible has normal basic growth potential in Apert's syndrome. The extent of maxillary hypoplasia will result in an Angle class III malocclusion with severe anterior open-bite deformity. A Le Fort I osteotomy is required to allow for horizontal advancement, transverse widening, and vertical adjustment in combination with an osteoplastic genioplasty to vertically reduce and horizontally advance the chin, often combined with bilateral sagittal split osteotomies of the mandible. The elective orthognathic surgery is carried out in conjunction with detailed orthodontic treatment planned for completion at the time of early skeletal maturity (~13–15 yr in girls and 15–17 yr in boys).

Assessment of Results in the Apert Patient

Quantitative Assessment of Presenting Apert's Deformity and Surgical Results Based on CT Scan Analysis after First-Stage Cranio-orbital Reconstruction

In a previously published study, we applied a method of quantitative CT scan analysis to document the differences in the cranio-orbitozygomatic region between children with Apert's syndrome who had not been operated on and age-matched controls.[90] We also evaluated the morphologic results achieved in those children 1 year after a classic anterior cranial vault and upper orbital procedure was undertaken, which was designed to decompress the brain and reshape these regions. Eight consecutive infants and young children with Apert's syndrome who underwent a classic cranio-orbital procedure by a craniofacial surgeon (JCP) in

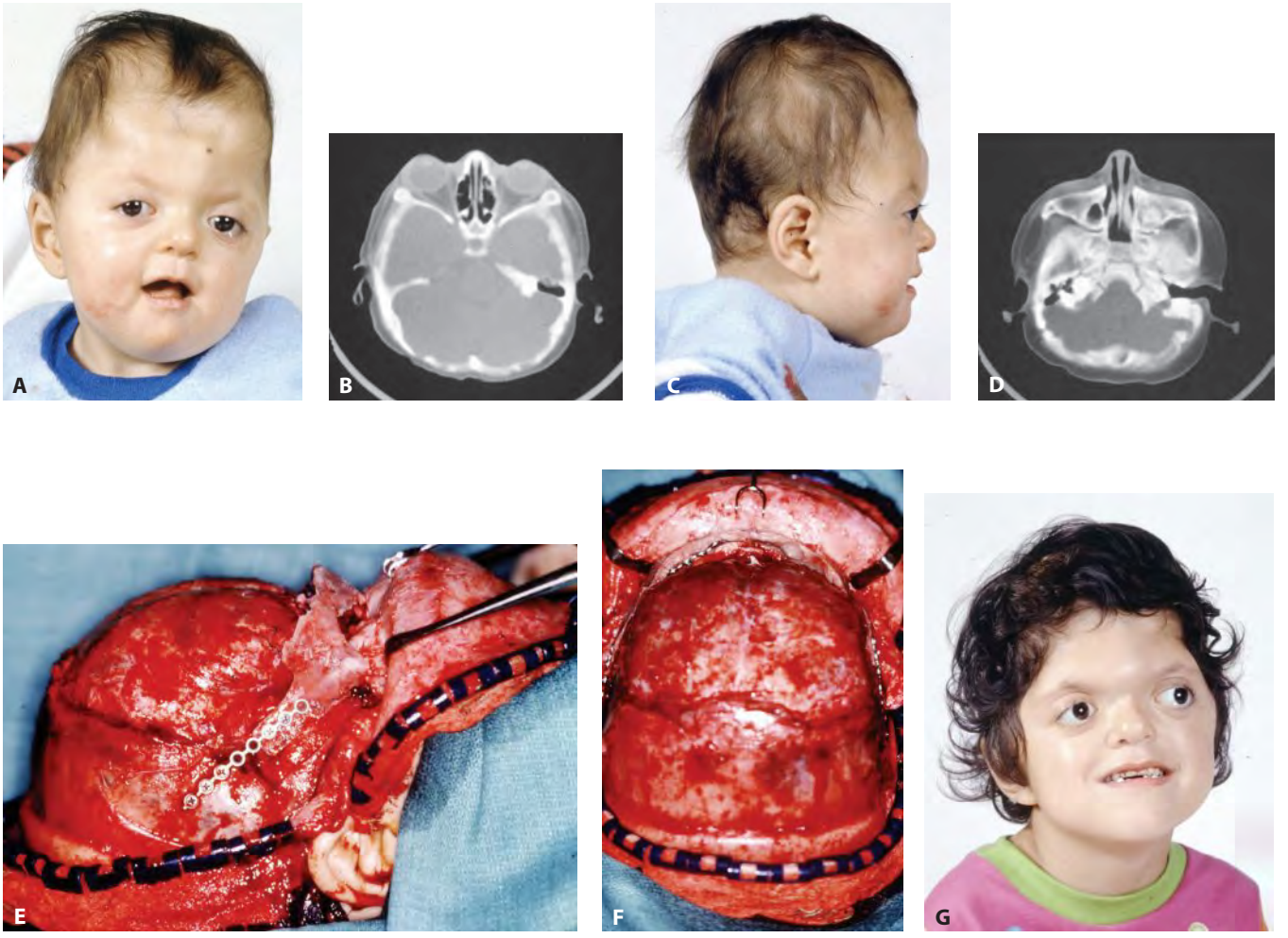
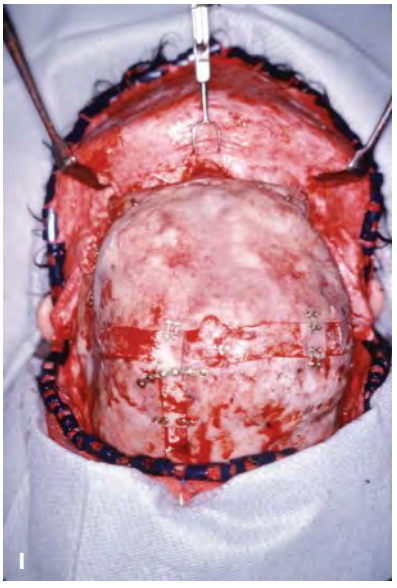


FIGURE 46-9. A child born with Apert's syndrome underwent bilateral "lateral canthal advancement" procedures when she was 6 weeks of age, carried out by the neurosurgeon working independently. At 18 months of age, she returned with turricephaly and a constricted anterior cranial vault requiring further cranio-orbital decompression and reshaping. At 5 years of age, she underwent anterior cranial vault and facial bipartition osteotomies with reshaping. As part of her staged reconstruction, she will require orthognathic surgery and orthodontic treatment planned for the teenage years. **A,** Frontal view at 8 months of age after a lateral canthal advancement procedure with residual deformity. **B,** Axial-sliced CT scan through the midorbits indicates dystopia, hypertelorism, and proptosis. **C,** Lateral view at 8 months of age. **D,** Axial-sliced CT scan through the zygomatic arches indicate midface deficiency.**E** and **F,** Lateral and bird's-eye views of the cranio-orbital region after three-quarter orbital osteotomies and reshaping and anterior advancement. **G,** Frontal view at 5 years of age just before further anterior cranial vault and facial bipartition osteotomies. **H,** Craniofacial morphology with planned and completed osteotomies and reconstruction. *(Continued)*



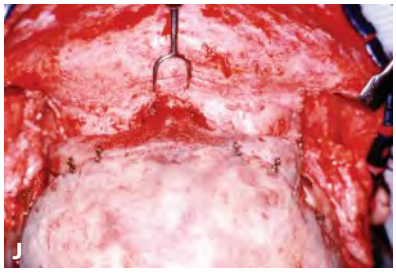


















FIGURE 46-9. *(Continued)* **I** and **J,** Bird's-eye view of the cranial vault and close-up view of the upper orbits after osteotomies with reshaping. **K,** Frontal view before surgery. **L,** Frontal view after facial bipartition reconstruction. **M,** Oblique view before surgery. **N,** Oblique view after reconstruction. **O,** Profile view before surgery. **P,** Profile view after reconstruction. **Q,** Worm's-eye view before surgery. **R,** Worm's-eye view after reconstruction. *(Continued)*

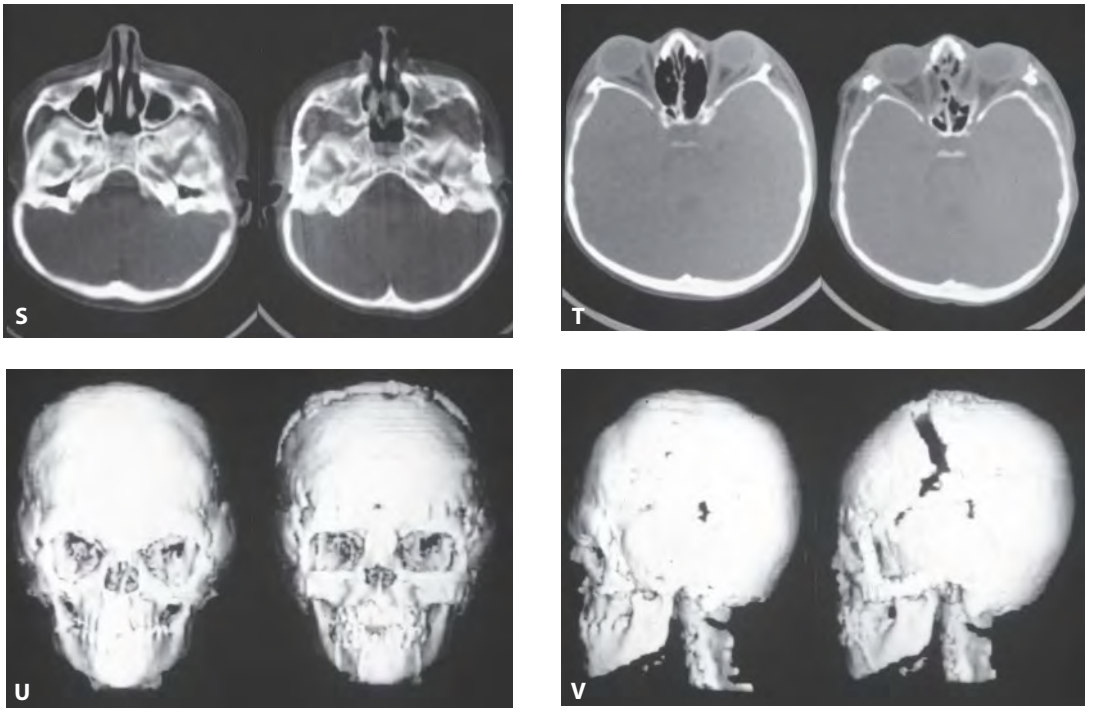


FIGURE 46-9. *(Continued)* **S,** Comparison of axial-sliced CT scans through the zygomas before and after reconstruction indicates a normalization of zygomatic arch length. **T,** Axial-sliced CT scan views through the midorbits before and after reconstruction indicate correction of orbital hyperteleorism and proptosis. **U** and **V,** Comparison of three-dimensional CT scan views of the craniofacial region before and after reconstruction, including improved morphology of orbits. **A–G** and **I–V,** Reproduced with permission from Posnick JC. Apert syndrome: evaluation and staging of reconstruction. In Posnick JC, editor. Craniofacial and Maxillofacial Surgery in Children and Young Adults. Philadelphia: WB Saunders; 2000; p. 316; **H,** adapted from Posnick JC. Apert syndrome: evaluation and staging of reconstruction. In Posnick JC, editor. Craniofacial and Maxillofacial Surgery in Children and Young Adults. Philadelphia: WB Saunders; 2000; p. 316.)

conjunction with a pediatric neurosurgeon over a 4-year period were reviewed. The series included seven girls and one boy, with an average age at surgery of 12 months (range, 9–23 mo). The average postoperative follow-up period was 34 months (range, 12–48 mo) at the close of the study. Preoperative and postoperative (>1 yr) CT scans were compared with those of age-matched controls. Percentages of normal were then compared for significant differences. All CT scans were reviewed by a consistent neuroradiologist experienced in CT scan measurements. Two of the children had clear evidence of hydrocephalus and required VP shunting before the craniofacial reconstruction procedures (at 3 and 5 mo of age). A third child had mildly increased ventricular size, but clinical correlations did not suggest the need for shunting.

Significant preoperative morphologic findings included a wide anterior cranial vault at 110% of normal, a maximum cranial length that averaged only 90% of normal, a substantially widened anterior interorbital width at 117% of normal, an increased lateral interorbital distance at 112% of normal, and a widened bitemporal width at 122% of normal. Globe protrusion was significant at 121% of normal, and the medial orbital wall length was less than normal at 92%. In the upper midface (zygomatic) region, both the width between the zygomatic buttresses and the interarch width were found to be increased at 109% of normal, whereas the zygomatic arch lengths were substantially shortened at 79% of normal. The measurements confirmed the clinical observations of brachycephalic, hyperteloric anterior cranial vaults, orbits, and zygomas, accompanied by eye proptosis and midface deficiency. Results of surgical reconstruction, as documented by CT scan measurements (analysis), showed that more than a year after surgery none of the craniofacial measurements had significantly improved ($P < .05$) in comparison with those of the new age-matched controls.

We also reviewed the medical records of this consecutive group of eight children with Apert's syndrome to document any morbidity associated with the cranio-orbital surgery carried out in infancy. There were no infections, wound difficulties, or central nervous system or ophthalmologic sequelae after any of the operations performed. One infant suffered intraoperative cardiac arrest due to intravascular volume depletion; the arrest responded to closed-chest cardiac massage and blood transfusion. The patient was discharged after an unremarkable further recovery. Several years later, she returned and underwent a facial bipartition procedure.

Detailed ophthalmologic examinations were also performed in all infants in the study. Two patients showed fun-

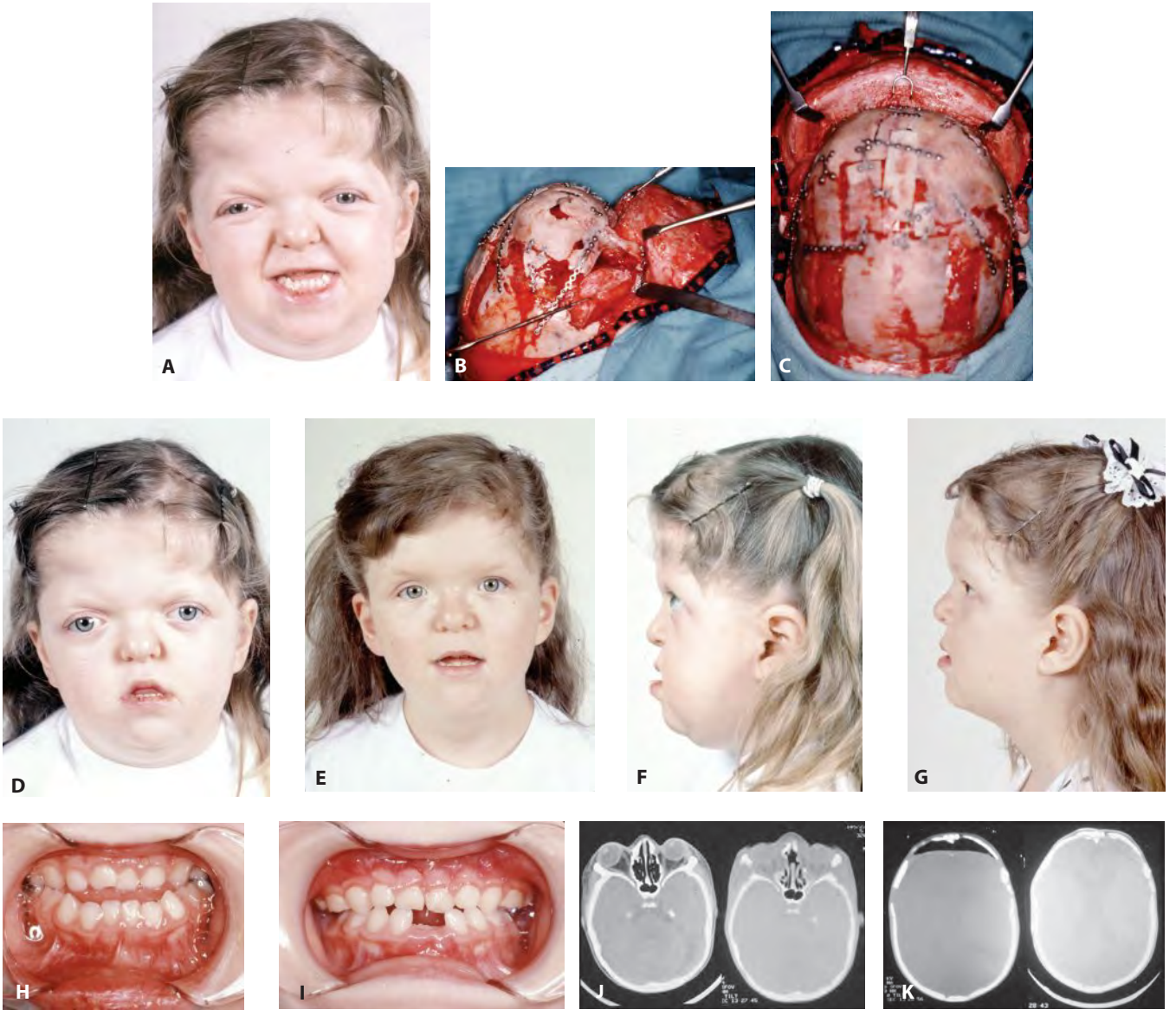


FIGURE 46-10. A 5-year-old girl with Apert's syndrome who underwent decompression and forehead and upper orbital reshaping at 6 months of age. She then presented to us with residual deformity requiring cranial vault and facial bipartition osteotomies with reshaping. She will require orthognathic surgery and orthodontic treatment later in the teenage years to complete her reconstruction. **A,** Frontal view before facial bipartition surgery. **B,** Intraoperative lateral view of the cranial vault and orbits through the coronal incision after reshaping. **C,** Bird's-eye view of the cranial vault after osteotomies and reshaping and fixation of the bone segments. **D,** Frontal view before surgery. **E,** Frontal view 2 years after reconstruction. **F,** Profile view before surgery. **G,** Profile view 2 years after facial bipartition reconstruction. **H,** Occlusal view before surgery. **I,** Occlusal view 6 months after reconstruction. **J,** Comparison of axial-sliced CT scan views through the midorbits before and after reconstruction demonstrates improvement in orbital hypertelorism and orbital depth with diminished eye proptosis. **K,** Standard axial CT scan slices through the cranial vault 1 week after facial bipartition (note dead space in the retrofrontal region), and at 1 year (notice that the initial retrofrontal dead space has been resolved by brain expansion). (**A–K,** Reproduced with permission from Posnick JC. Craniofacial dysostosis: staging of reconstruction and management of the midface deformity. Neurosurg Clin North Am 1991;2:683–702.)

duscopic evidence of increased ICP with papilledema before surgery. Both of these patients had previously undergone limited linear craniectomies. In both patients, the funduscopic examination results normalized by 6 months after the brain decompression and cranio-orbital expansion and reconstruction.

Quantitative measurement of CT scans of the cranio-orbitozygomatic region confirm clinically observed findings in these patients before cranio-orbital surgery in infancy and early childhood to be brachycephalic anterior cranial vaults and upper face hypertelorism (of orbits and zygomas) with eye proptosis and a flat midface. We found that early

brain decompression with cranial vault and upper orbital reshaping does not maintain a corrected shape in the craniofacial skeleton in children with Apert's syndrome. Although the cranio-orbitozygomatic dysmorphology did not seem to worsen when analyzed at least 1 year after surgery, values remained far from those of the new age-matched controls, thus confirming the need for a staged reconstructive approach.

Quantitative Intracranial Volume Measurements before and after Cranio-orbital Reshaping in Children with Apert's Syndrome

In published studies, we applied a proven method for obtaining intracranial volume measurements using CT scans to measure the intracranial volume of a consecutive series of children with Apert's syndrome before any craniofacial procedures.[70] We performed a classic cranio-orbital operation in each child (described previously for Crouzon's syndrome), followed by longitudinal follow-up, and remeasured the intracranial volume at least 1 year later. We also compared our patients' intracranial volumes with those of an age-matched cohort (according to norms established by Lichtenberg[109]) and reviewed each patient's cranial growth velocity.

The study included six girls and two boys with an average age at operation of 12 months (range, 9–23 mo). The average postoperative follow-up at the close of the study was 34 months (range, 12–48 mo).

Preoperative intracranial volume in the patients with Apert's syndrome ranged from 393 mL in a 2-month-old girl to 1715 mL in a 28-month-old girl. Comparison of the preoperative intracranial volume in patients with Apert's syndrome to those of the age- and-gender-controlled cohort group of Lichtenberg showed that six of the eight patients had values at least 2 SD above the mean. Interestingly, the other two were infants who underwent VP shunting for hydrocephalus earlier in life. Their measured preoperative intracranial volumes were 2 SD below the mean.

When our patients' postoperative intracranial volumes were compared with those of the Lichtenberg cohort group, all eight achieved values at least 2 SD above the mean. The majority of the measured preoperative and postoperative intracranial volume values of our patients with Apert syndrome followed a growth curve that greatly exceeded the rate expected for normal children. In three of the patients, cranial vault growth velocity seemed to match closely that expected for a normal child, but with a starting point determined by their preoperative values.

Our findings confirmed that untreated patients with Apert's syndrome are generally macrocephalic early in life, that classic cranio-orbital procedures carried out in childhood do not alter this trend, and that continued cranial volume expansion often exceeds the mean. The ability to develop "normal" intracranial volume standards and to identify variations from normal in specific syndromes and in individual patients before and after surgery continues to elude us.

Quantitative Assessment of Presenting Deformity in Children with Apert's Syndrome and Surgical Results after Facial Bipartition Osteotomy Based on CT Scan Analysis

In published studies, we assessed children with Apert's syndrome, in the midchildhood years, using quantitative CT scan measurements.[70] In the children with Apert's syndrome at this age, many of the measurements varied from normal when compared with those of age-matched controls. The orbital measurements showed a substantially increased anterior interorbital width (123% of normal), an increased midinterorbital width (122% of normal), and an increased intertemporal width (126% of normal). The globe protrusion beyond the sagittal plane of the lateral orbital walls was excessive (142% of normal). There was also a short medial orbital wall length (85% of normal). The width between the lateral orbital walls was excessive at 111% of normal. Zygomatic arch lengths were substantially shortened at 83% of normal value.

After undergoing facial bipartition osteotomies with three-dimensional repositioning, combined with cranial vault reshaping carried out through an intracranial approach, assessments of CT scan measurements were taken early after the operation and 1 year later. Analysis of the measurements showed an improvement toward normal range. When compared with those of age-matched controls, the orbital measurements reflected correction of the hypertelorism; the anterior interorbital width early after operation was 106% and later was 105% of normal. The midinterorbital width initially improved to 106% and later to 100% of normal. The width between the lateral orbital walls stabilized at 108% and the intertemporal width at 115% of normal, an improvement over the preoperative value of 126% of normal. The zygomatic arch length was initially overcorrected at 110%, then stabilized at 103% of normal.

Further study was carried out to evaluate the presence of extradural (retrofrontal) dead space after the facial bipartition osteotomy to reconstruct the Apert's syndrome upper and midface deformity. Seven patients with Apert's syndrome (mean age, 8 yr) underwent facial bipartition osteotomies with advancement. Extradural (retrofrontal) dead space was measured from a reproducible axial CT scan slice for each patient at postoperative intervals (1–2 wk, 6–8 wk, and 1 yr). An initial extradural (retrofrontal) dead space was identified early after surgery in each patient, with resolution occurring by the 6- to 8-week postoperative interval through expansion of the dura and frontal lobes of the brain. The dead space was confirmed to be closed in all patients at the 1-year postoperative interval.

We then reviewed the morbidity of the same consecutive series of patients with Apert's syndrome who underwent facial bipartition and cranial vault reshaping through an intracranial approach.[44] Of the seven children with Apert's syndrome, there were no deaths, cardiopulmonary sequelae, or injuries to the brain or eyes. New seizure activity or central nervous system problems did not occur, nor did infection develop in any of the patients after surgery.

PFEIFFER'S SYNDROME

In 1964, Pfeiffer described a syndrome consisting of craniosynostosis, broad thumbs, broad great toes, and occasionally, partial soft tissue syndactyly of the hands.[110] This syndrome is known to have an autosomal dominant inheritance pattern with complete penetrance documented in all recorded two- and three-generation pedigrees.[69] Variable expressivity of the craniofacial and extremity findings is common (Figures 46-11 and 46-12). Although some authors have found clinical

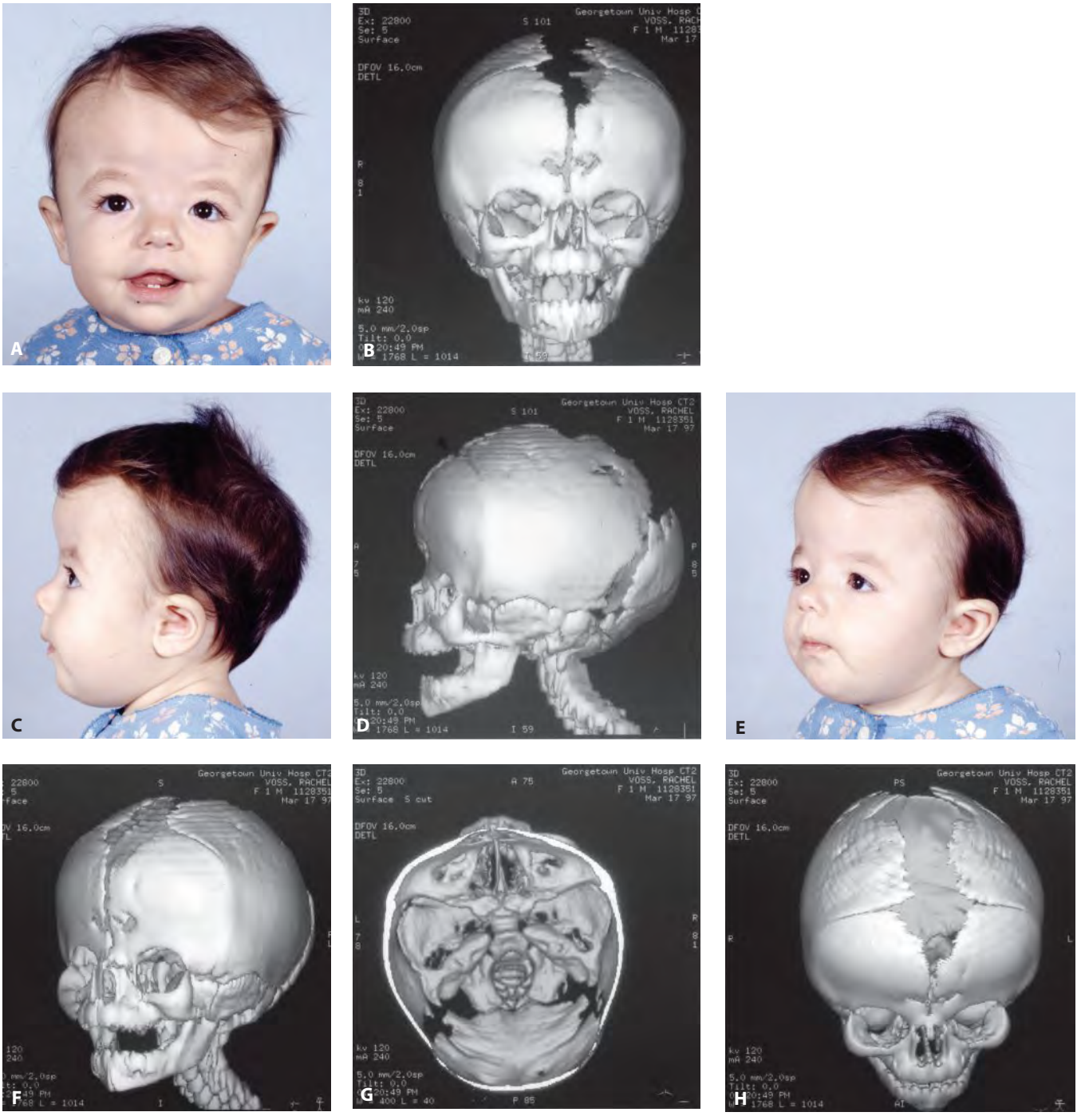


FIGURE 46-11. A 2-month-old child born with Pfeiffer's syndrome (type 1). She has bilateral coronal synostosis resulting in brachycephaly without suggestion of midface deficiency **A,** Frontal view. **B,** Frontal view of CT scan. **C,** Profile view. **D,** Profile view of CT scan. **E,** Oblique view. **F,** Oblique view of CT scan. **G,** Cranial base view of CT scan. **H,** Craniofacial view of CT scan. (**A–H,** Reproduced with permission from Posnick JC. Pfeiffer syndrome: evaluation and staging of reconstruction. In Posnick JC, editor. Craniofacial and Maxillofacial Surgery in Children and Young Adults. Philadelphia: WB Saunders; 2000; p. 344.)

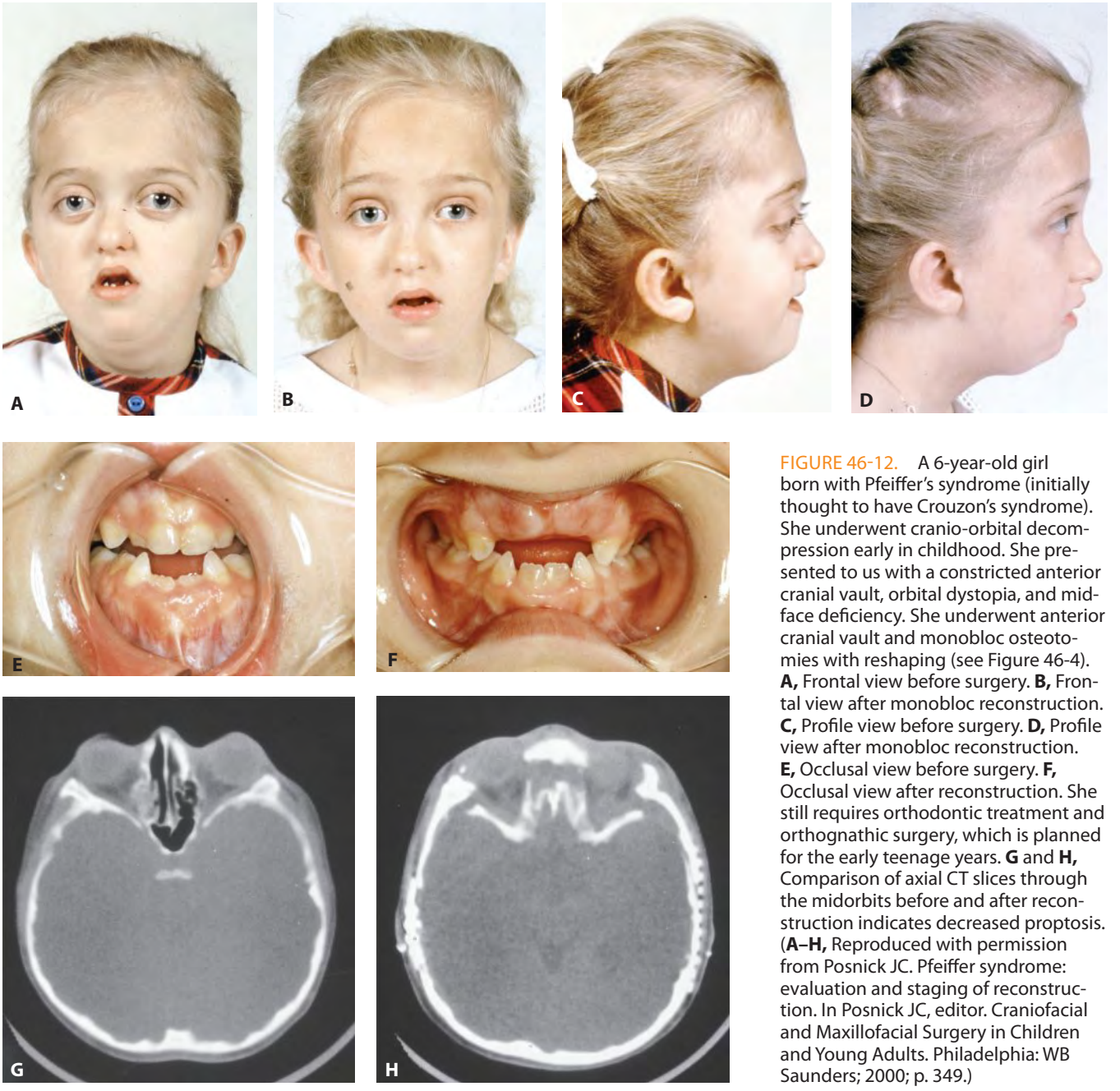


FIGURE 46-12. A 6-year-old girl born with Pfeiffer's syndrome (initially thought to have Crouzon's syndrome). She underwent cranio-orbital decompression early in childhood. She presented to us with a constricted anterior cranial vault, orbital dystopia, and midface deficiency. She underwent anterior cranial vault and monobloc osteotomies with reshaping (see Figure 46-4). **A,** Frontal view before surgery. **B,** Frontal view after monobloc reconstruction. **C,** Profile view before surgery. **D,** Profile view after monobloc reconstruction. **E,** Occlusal view before surgery. **F,** Occlusal view after reconstruction. She still requires orthodontic treatment and orthognathic surgery, which is planned for the early teenage years. **G** and **H,** Comparison of axial CT slices through the midorbits before and after reconstruction indicates decreased proptosis. (**A–H,** Reproduced with permission from Posnick JC. Pfeiffer syndrome: evaluation and staging of reconstruction. In Posnick JC, editor. Craniofacial and Maxillofacial Surgery in Children and Young Adults. Philadelphia: WB Saunders; 2000; p. 349.)

similarities in certain patients with Pfeiffer's syndrome, Crouzon's syndrome, and Jackson-Weiss syndrome, the three disorders are nosologically distinct.[47,111] According to Cohen and Kreiborg,[111] the phenotypes of the three conditions do not correlate well with the known molecular findings.[102] Patients with these three syndromes may have similar or even identical mutations in exon B of *FGFR2*, yet they breed true within families, an observation that is as yet unexplained by the molecular findings.[112–119]

Current thinking suggests that Pfeiffer's syndrome is heterogeneous because it is caused by a single recurring mutation (Pro252Arg) of *FGFR1* and by several different mutations affecting *FGFR2*.[120,121] Cohen and Kreiborg[112] have reviewed the literature and further subgrouped Pfeiffer's syndrome according to clinical features, associated low-frequency anomalies, and outcome. According to Cohen and Kreiborg, type 1 corresponds to the classic Pfeiffer syndrome and is associated with satisfactory prognosis. The type 2 subgroup is associated with the cloverleaf skull anomaly whereas type 3 is not. Both types 2 and 3 have a less favorable outcome, with frequent death in infancy. The type 1 variant frequently presents with bicoronal craniosynostosis and midface involvement. The longitudinal evaluation and staging of reconstruction depend on individual variations but is similar to that described for Crouzon's syndrome.

CARPENTER'S SYNDROME

Carpenter's syndrome is characterized by craniosynostosis often associated with preaxial polysyndactyly of the feet, short fingers with clinodactyly, and variable soft tissue syndactyly, sometimes postaxial plydactyly, and other anomalies such as congenital heart defects, short stature, obesity, and mental deficiency.[24] It was first described by Carpenter in 1901 and was later recognized to be an autosomal recessive syndrome. In general, the reconstructive algorithm described for Crouzon's syndrome can be followed.

SAETHRE-CHOTZEN SYNDROME

Saethre-Chotzen syndrome has an autosomal dominant inheritance pattern with a high degree of penetrance and expressivity.[122,123] Its pattern of malformations may include craniosynostosis, low-set frontal hairline, ptosis of the upper eyelids, facial asymmetry, brachydactyly, partial cutaneous syndactyly, and other skeletal anomalies. As part of the reconstruction, cranio-orbital reshaping will almost certainly be required and is similar to that described for Crouzon's syndrome. Evaluation and management of the total midface deficiency and orthognathic deformities as decribed for Crouzon's syndrome should be followed.

CLOVERLEAF SKULL ANOMALY

Kleeblattschädel anomaly (cloverleaf skull) is a trilobular-shaped skull secondary to craniosynostosis (Figure 46-13).[124,125] The cloverleaf skull anomaly is known to be both

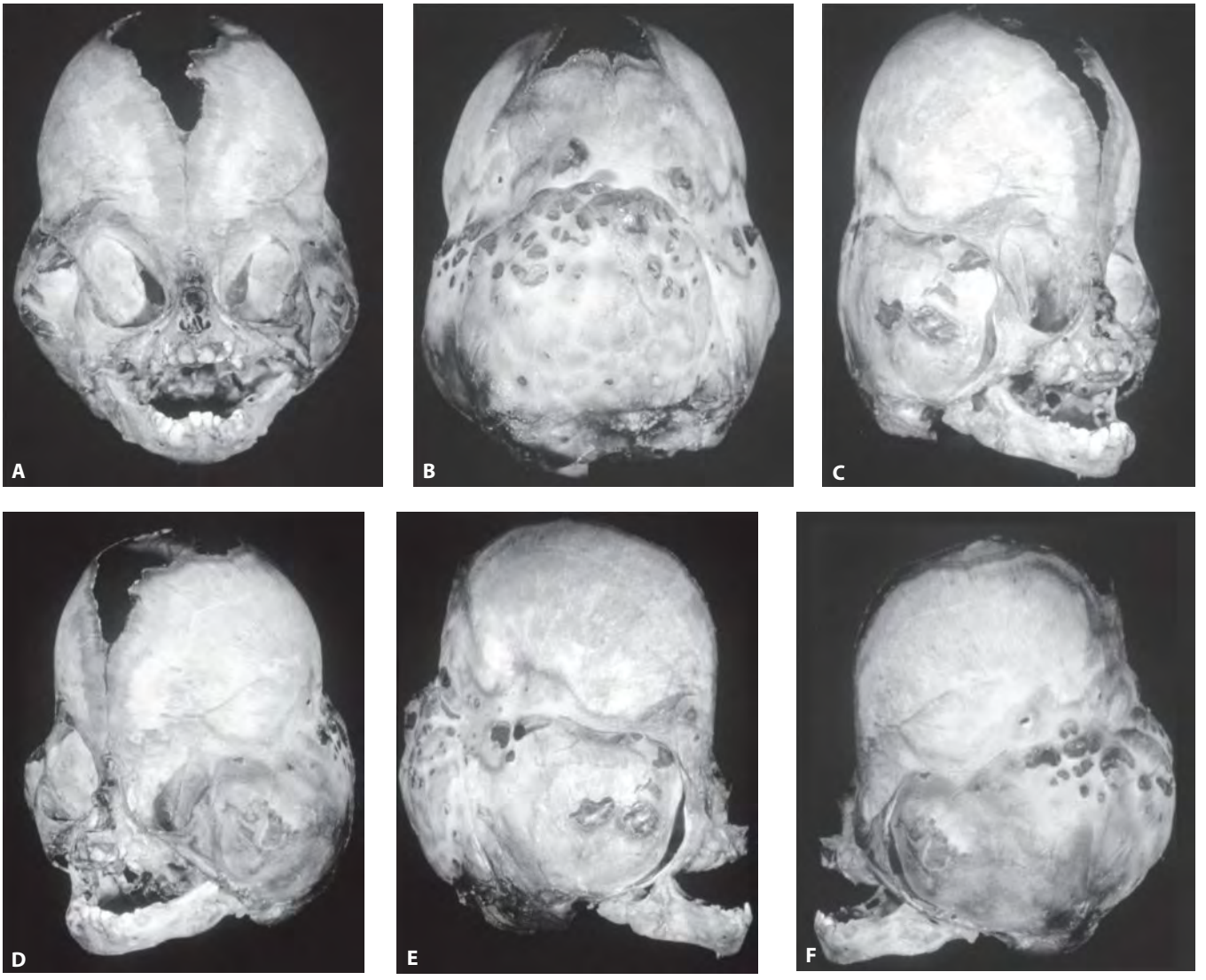


FIGURE 46-13. The craniofacial skeleton of a 6-month-old child born with a cloverleaf skull anomaly. He underwent tracheostomy and gastrostomy shortly after birth and died of pneumonia before craniofacial reconstruction could be undertaken. **A,** Frontal view. **B,** Posterior view. **C,** Right oblique view. **D,** Left oblique view. **E,** Left profile view. **F,** Right profile view. (**A–F,** Reproduced with permission from Cloverleaf skull anomalies: evaluation and staging of reconstruction. In Posnick JC, editor. Craniofacial and Maxillofacial Surgery in Children and Young Adults. Philadelphia: WB Saunders; 2000; p. 364.)

etiologically and pathogenetically heterogeneous. This anomaly is also nonspecific: it may occur as an isolated anomaly or together with other anomalies, making up various syndromes (i.e., Apert's, Crouzon's, Carpenter's, Pfeiffer's, and Saethre-Chotzen).[126] The extent and timing of anterior cranial vault or upper orbital, posterior cranial vault, and midface reconstruction will be dependent on individual variation in the presenting deformity. In general, the protocol described for Crouzon's syndrome can be followed.

SUMMARY

Details of the timing and techniques for correction of the varied forms of CFD syndromes differ from center to center. However, an essential element of successful rehabilitation is the delivery of care by committed, experienced, and technically skilled clinicians. The combined expertise of an experienced craniofacial surgeon and pediatric neurosurgeon working together to manage the cranio-orbital malformation and the experienced maxillofacial surgeon and orthodontist working together to manage the orthognathic deformity are essential to achieve maximum function and facial aesthetics for each patient.

Our preferred approach for management of the CFD syndromes is to stage the reconstruction to coincide with facial growth patterns, visceral (brain and eye) function, and psychosocial development. Recognition of the need for a staged reconstruction serves to clarify the objectives of each phase of treatment for the craniofacial surgeon, team, and most important, the patient and patient's family.

By continuing to define our rationale for the timing and extent of surgical intervention and then evaluating both function and aesthetic outcomes, we will further improve the quality of life for the many hundreds of children born with syndromal forms of craniosynostosis. Our objective is to see each individual achieve personal success in life without special regard for the original malformation.

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SECTION 6

Temporomandibular Joint Disease

47

CHAPTER

Anatomy and Pathophysiology of the Temporomandibular Joint

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CLASSIFICATION

The temporomandibular joint (TMJ) is a complex structure composed several components including the glenoid fossa of the temporal bone, the condylar head of the mandible, and a specialized dense fibrous connective tissue structure, the articular disk, as well as several ligaments and associated muscles. The TMJ is a specialized joint that can be classified by anatomic type as well as by function.

From an anatomic standpoint, the TMJ is classified as a *diarthrodial joint*, which is a discontinuous articulation of two bones allowing freedom of movement that is dictated by the associated muscles and limited by the associated ligaments.[1] The fibrous connective tissue capsule is well innervated, well vascularized, and tightly attached to the bones at the edges of their articulating surfaces on the medial an lateral aspects of the TMJ. The TMJ is also a *synovial joint*, lined on its inner aspect by a synovial membrane, which secretes synovial fluid. The fluid acts as a joint lubricant and supplies the metabolic and nutritional needs of the nonvascularized internal joint structures.

From a functional standpoint, the TMJ is classified as a compound joint, composed of four articulating surfaces: the articular facets of the temporal bone (1) and of the mandibular condyle (2) and the superior (3) and inferior (4) surfaces of the articular disk. The articular disk divides the TMJ into two compartments. The lower compartment permits hinge motion or rotation and, hence, is termed *ginglymoid*. The superior compartment permits sliding (or translatory) movements and is, therefore, called *arthrodial*. As a result, the TMJ as a whole can be termed a *ginglymoarthrodial joint*.

BONY STRUCTURES

The articular portion of the temporal bone (Figure 47-1) is composed of three portions. The largest part is the *articular*,

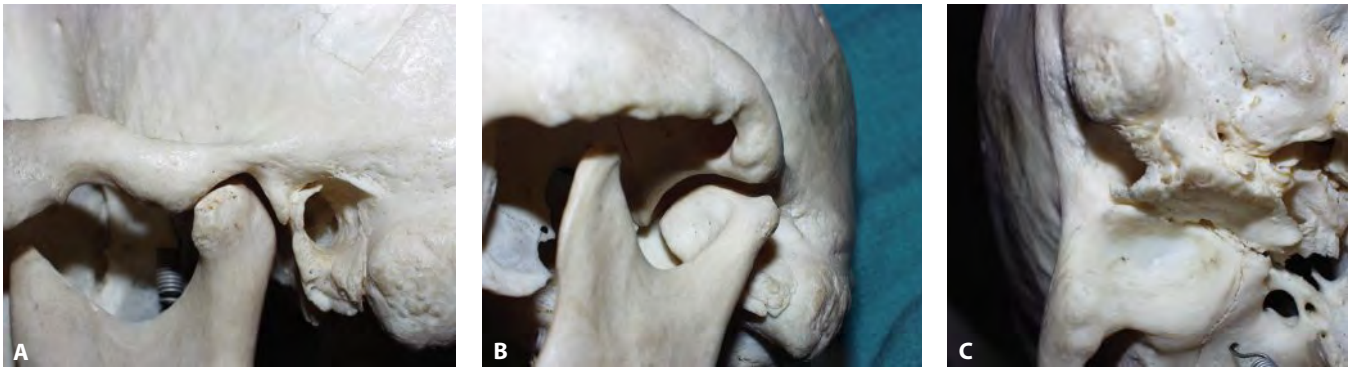


FIGURE 47-1. **A,** The left temporomandibular mandibular joint (TMJ) viewed from the sagittal aspect on a dry skull. **B,** The left TMJ viewed from the oblique/coronal aspect on a dry skull. **C,** The left glenoid fossa and articular eminence.

or *mandibular, fossa,* a concave structure extending from the posterior slope of the articular eminence to the postglenoid tubercle, which is a ridge between the fossa and the external acoustic meatus. The surface of the articular fossa of the temporal bone is very thin and may appear translucent when viewed through a dry skull specimen. As a result, this area is not a major stress-bearing region for the TMJ. The second portion, the *articular eminence,* is a transverse bony prominence that is continuous across the articular surface mediolaterally. The articular eminence is usually thick and serves as a major functional component of the TMJ. The articular eminence is distinguished from the articular tubercle, a nonarticulating process on the lateral aspect of the zygomatic root of the temporal bone, which serves as a point of attachment of collateral ligaments. The third portion of the articular surface of the temporal bone is the *preglenoid plane,* a flattened area located anterior to the articular eminence.

The mandible is a U-shaped bone that articulates with the temporal bone by means of the articular surface of the condyles, which are paired structures forming an approximately 145- to 160-degree angle to one another. The mandibular condyle (Figure 47-2) is approximately 15 to 20 mm in transverse width dimension and 8 to 10 mm in an anteroposterior dimension. The condyle tends to be rounded mediolaterally and convex anteroposteriorly. On its medial aspect just below its articular surface is a prominent depression, known as the *pterygoid fovea,* which is the site of insertion of the inferior head of the lateral pterygoid muscle that assists in protrusion of the mandible during condylar translation.

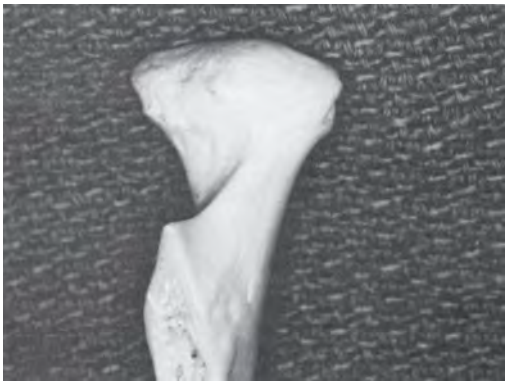


FIGURE 47-2. The mandibular condyle.

CARTILAGE AND SYNOVIUM

Lining the inner aspect of all synovial joints, including the TMJ, are two types of tissue: *articular cartilage* and *synovium* (Figure 47-3). The space bounded by these two structures is termed the *synovial cavity,* which is bathed in synovial fluid. The articular surfaces of both the temporal bone and the condyle are covered with dense articular fibrocartilage, a fibrous connective tissue. This fibrocartilage covering layer has the capacity to regenerate and to remodel under functional stresses. Deep to the fibrocartilage layer, particularly on the condylar head, is a proliferative zone of cells that may develop into either cartilaginous or osseous tissue, based upon functional loads. Therefore, major changes

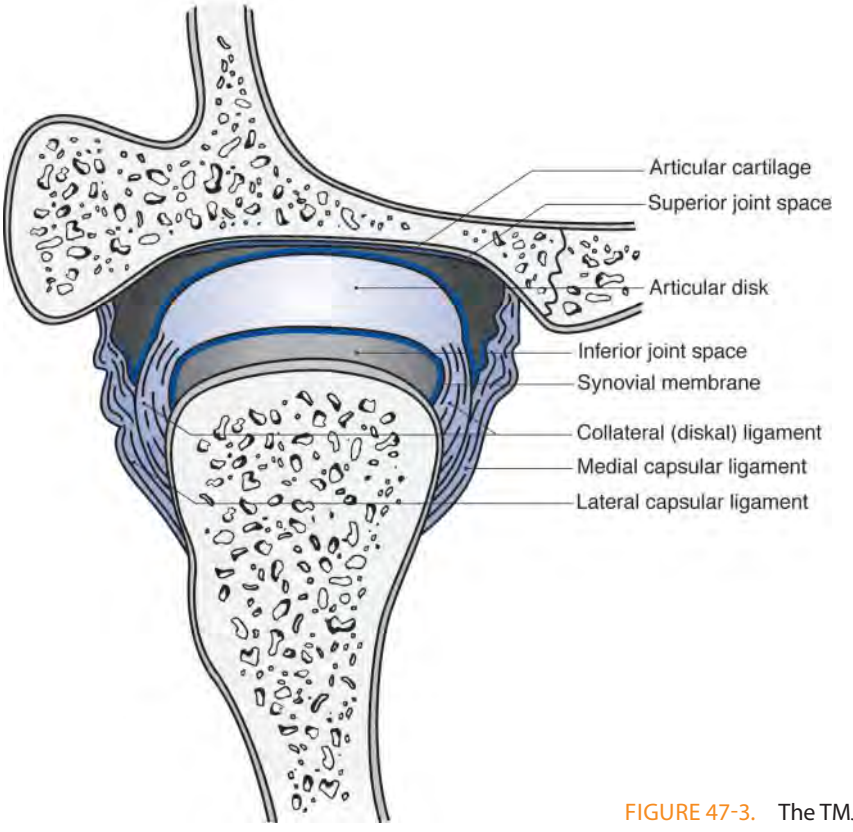


FIGURE 47-3. The TMJ (coronal view).

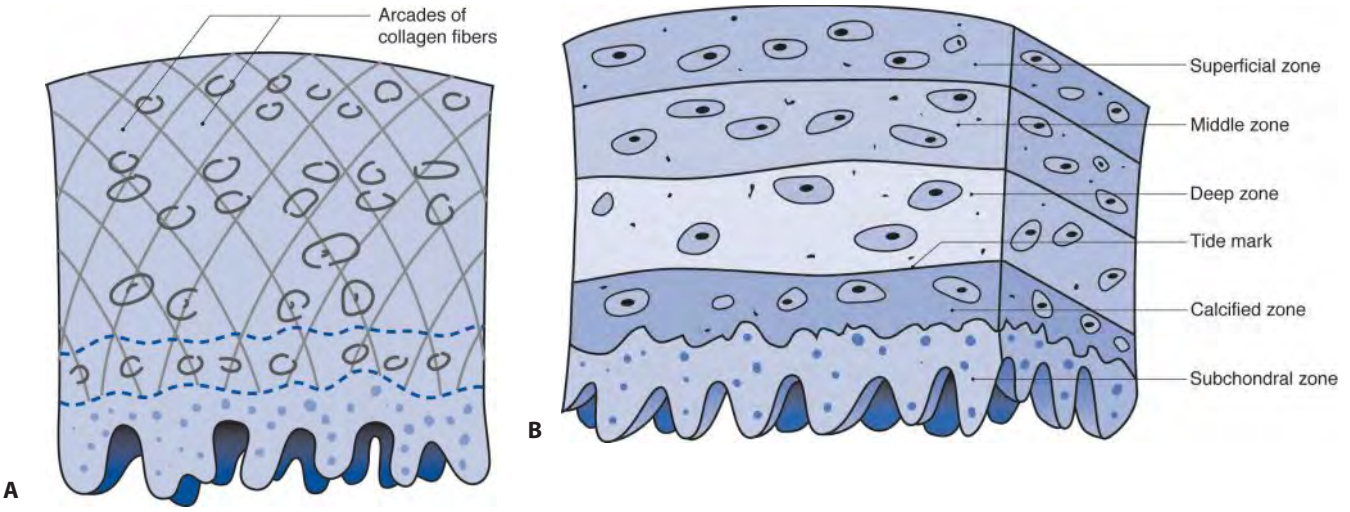


FIGURE 47-4. The articular cartilage. (Adapted from Albright JA, Brand RA. The scientific basis of orthopedics. 2nd ed. Norwalk, CT: Appleton and Lange; 1987; pp. 373–386.)

in morphology resulting from alterations in joint function are typically seen in this layer.

Articular cartilage is composed of chondrocytes and an intercellular matrix of collagen fibers, water, and a nonfibrous filler material, termed *ground substance.* Chondrocytes are enclosed in otherwise hollow spaces, called *lacunae,* and are arranged in three layers characterized by different cell shapes (Figure 47-4A). The *superficial zone* contains small, flattened cells with their long axes parallel to the surface.[2] In the *middle zone,* the cells are larger, rounded, and oriented in a columnar fashion perpendicular to the surface. The *deep zone* contains the largest cells and is divided by the "tide mark" below which some degree of calcification has occurred. There are few blood vessels in any of these areas, so the cartilage receives nourishment primarily by diffusion from the synovial fluid.

Collagen fibers are arranged in arcades with an interlocking meshwork of fibrils parallel to the articular surface joining together as bundles and descending to their attachment in the calcified cartilage between the tide mark (see Figure 47-4B). Functionally, these arcades provide a framework for interstitial water and ground substance to resist compressive forces encountered during joint loading. Because it is formed by an intramembranous process, the articular cartilage of the TMJ contains a greater proportion of collagen fibers (fibrocartilage) than other synovial joints, which are typically covered instead by hyaline cartilage.

The ground substance contains a variety of plasma proteins, glucose, urea, and salts as well as proteoglycans, which are synthesized by the Golgi apparatus of the chondrocytes. Proteoglycans are macromolecules consisting of a protein core attached to many glycosaminoglycan chains of chondroitin sulfate and keratan sulfate. Proteoglycans play a role in the diffusion of nutrients and metabolic breakdown products. Ground substance permits the entry and release of large quantities of water, an attribute thought to be significant in giving cartilage its characteristic functional elasticity in response to deformation and loading.

Lining the capsular ligament is the synovial membrane, a thin, smooth, richly innervated vascular tissue without an epithelial lining. Synovial cells appear somewhat undifferentiated and serve both a phagocytic and a secretory role and are thought to be the site of production of hyaluronic acid, a glycosaminoglycan found in synovial fluid. Some synovial cells, particularly those in close approximation to the articular cartilage, are thought to have the capacity to differentiate into chondrocytes. The synovium is capable of rapid and complete regeneration after injury. Recently, synovial cells (as well as chondrocytes and leukocytes) have been the focus of extensive research regarding the production of anabolic and catabolic cytokines within the TMJ.[3]

Synovial fluid is considered an ultrafiltrate of plasma.[2] The fluid contains a high concentration of hyaluronic acid, which is responsible for the fluid's high viscosity (resistance to flow). The proteins found in synovial fluid are identical to plasma proteins; however, synovial fluid has a lower total protein content, with a higher percentage of albumin and a lower percentage of α_2-globulin. Alkaline phosphatase, which may also be present in synovial fluid, is produced by chondrocytes. Leukocytes are also found in synovial fluid, with the cell count being less than 200/mm_3 and with less than 25% of these cells being polymorphonuclear leukocytes (PMNs or neutrophils). Only a small amount of synovial fluid, usually less than 2 mL, is present within each healthy functional TMJ.

Functions of the synovial fluid include lubrication of the joint, phagocytosis of particulate debris, and nourishment of the articular cartilage. Joint lubrication is a complex function related to the viscosity of synovial fluid and to the ability of articular cartilage to allow the free passage of water within the pores of its glycosaminoglycan matrix. Application of a loading force to an area of the articular cartilage causes a

deformation at that specific location. It has been theorized that water is extruded from the loaded area into the synovial fluid adjacent to the point of contact. The concentration of hyaluronic acid and, therefore, the viscosity of the synovial fluid is greater at the point of maximal load, thus resulting in protection of the articular surfaces. As the load is distributed to adjacent areas, the deformation is transferred as well, whereas the original point of contact regains its shape and thickness through the reabsorption of water from the synovial fluid. The exact mechanisms of fluid balance and flow between the articular cartilage and the synovial fluid are unclear. Nevertheless, the net result is a coefficient of friction for the normally functioning joint of approximately 14 times less than that of a dry joint.

THE ARTICULAR DISK

The articular disk (Figure 47-5) is composed of a dense fibrous connective tissue, and it is nonvascularized and noninnervated; this is an adaptation that allows the disk to resist pressure.[4] Anatomically, the disk can be divided into three general regions as viewed from the lateral perspective: the anterior band, the central intermediate zone, and the posterior band. The thickness of the disk appears to be correlated with the prominence of the eminence, such that proportionally, the anterior band (3, thickest), intermediate zone (1, thinnest), and posterior band (2, middle thickness) have relative thicknesses. The intermediate zone is thinnest and is generally the area of maximum function between the mandibular condyle and the temporal bone. Despite the designation of separate portions of the articular disk, it is in fact a homogeneous tissue and the three bands do not consist of specific anatomic structures. The disk is flexible and adapts to functional demands of the articular surfaces.[5] The articular disk is attached to the capsular ligament anteriorly, posteriorly, medially, and laterally.[6] Some fibers of the superior head of the lateral pterygoid muscle insert on the disk at its anteromedial aspect, serving to stabilize the disk to the mandibular condyle, via the medial and lateral collateral ligaments, during function.

RETRODISKAL TISSUE

Posteriorly, the articular disk blends with a highly vascular, highly innervated structure known as the *retrodiskal tissues.* Anatomically, the retrodiskal tissues are referred to as the *bilaminar zone* (superior and inferior retrodiskal laminae), which is involved in the production of synovial fluid. The superior aspect of the retrodiskal tissue contains elastic fibers and is termed the *superior retrodiskal lamina*, which attaches to the tympanic plate and functions as a restraint to disk displacement in extreme translatory movements.[5] The inferior aspect of the retrodiskal tissue, termed the *inferior retrodiskal lamina*, consists of collagen fibers without elastic tissue and functions to connect the articular disk to the posterior margin of the articular surfaces of the condyle. It is thought to serve as a check ligament to prevent extreme rotation of the disk on the condylar head in rotational movements.

LIGAMENTS

Ligaments associated with the TMJ are composed of collagen and act predominantly as restraints to motion of the condyle and the disk. Three ligaments—collateral, capsular, and temporomandibular ligaments—are considered functional ligaments because they serve as major anatomic componentsof the joints. Two other ligaments—sphenomandibular and stylomandibular—are considered accessory ligaments because, although they are attached to osseous structures at some distance from the joints, they serve to some degree as passive restraints on mandibular motion.

The collateral (or diskal) ligaments (see Figure 47-3) are short paired structures attaching the disk to the lateral and medial poles of each condyle. Their function is to restrict movement of the disk away from the condyle, thus allowing

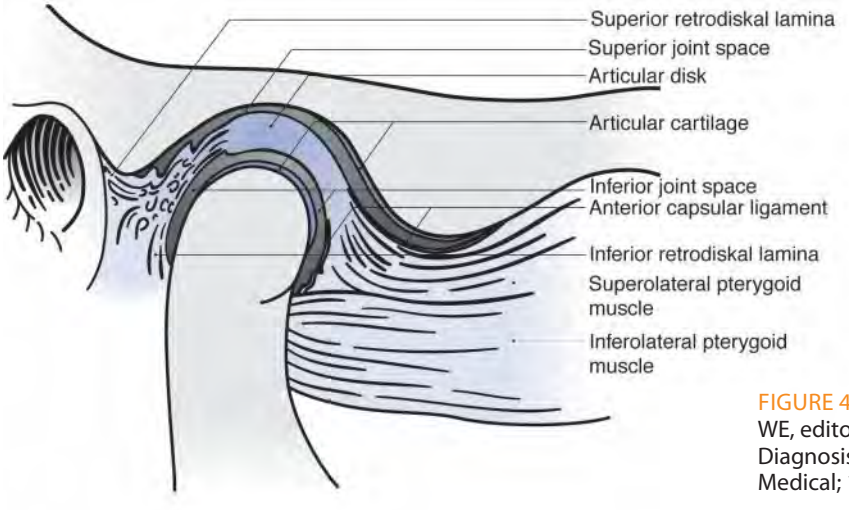


FIGURE 47-5. The TMJ (lateral view). (Adapted from Bell WE, editor. Temporomandibular Disorders: Classification, Diagnosis and Management. 2nd ed. Chicago: Year Book Medical; 1986; pp. 16–62.)

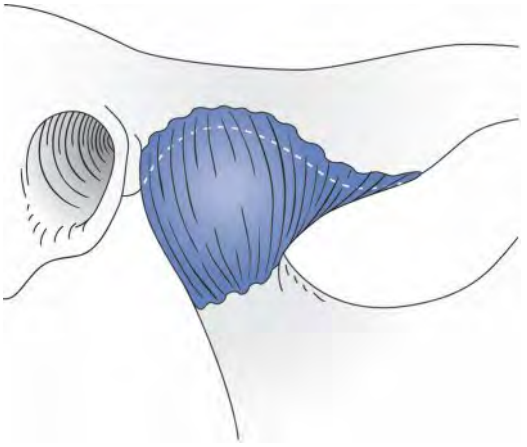


FIGURE 47-6. The capsular ligament (lateral view).

smooth synchronous motion of the disk-condyle complex. Although the collateral ligaments permit rotation of the condyle with relation to the disk, their tight attachment forces the disk to accompany the condyle through its translatory range of motion.[6]

The capsular ligament (Figures 47-6 and 47-7; see also Figures 47-3 and 47-5) encompasses each joint, attaching superiorly to the temporal bone along the border of the mandibular fossa and eminence and inferiorly to the neck of the condyle along the edge of the articular facet. It surrounds the joint spaces and the disk, attaching anteriorly and posteriorly as well as medially and laterally, where it blends with the collateral ligaments. The function of the capsular ligament is to resist medial, lateral, and inferior forces, thereby holding the joint together. It offers resistance to movement of the joint only in the extreme range of motion. A secondary function of the capsular ligament is to contain the synovial fluid within the superior and inferior joint spaces.

The temporomandibular (lateral) ligaments (see Figure 47-7) are located on the lateral aspect of each TMJ.[5] Unlike the capsular and collateral ligaments, which have medial and lateral components within each joint, the temporomandibular ligaments are single structures that function in paired fashion with the corresponding ligament on the opposite TMJ. Each temporomandibular ligament can be separated into two distinct portions that have different functions.[6] The outer oblique portion descends from the outer aspect of the articular tubercle of the zygomatic process posteriorly and inferiorly to the outer posterior surface of the condylar neck. It limits the amount of inferior distraction that the condyle may achieve in translatory and rotational movements. The inner horizontal portion also arises from the outer surface of the articular tubercle, just medial to the origin of the outer oblique portion of the ligament, and runs horizontally backward to attach to the lateral pole of the condyle and the posterior aspect of the disk. The function of the inner horizontal portion of the temporomandibular ligament is to limit posterior movement of the condyle, particularly during pivoting movements, such as when the mandible moves laterally in chewing function. This restriction of posterior movement serves to protect the retrodiskal tissue.

The sphenomandibular ligament (Figure 47-8) arises from the spine of the sphenoid bone and descends into the fanlike insertion on the mandibular lingula as well as on the lower portion of the medial side of the condylar neck.[1] The sphenomandibular ligament serves to some degree as a point of rotation during activation of the lateral pterygoid muscle, thereby contributing to translation of the mandible.

The stylomandibular ligament (see Figure 47-8) descends from the styloid process to the posterior border of the angle of the mandible and also blends with the fascia of the medial pterygoid muscle. It functions similarly to the sphenomandibular ligament as a point of rotation and also limits excessive protrusion of the mandible.

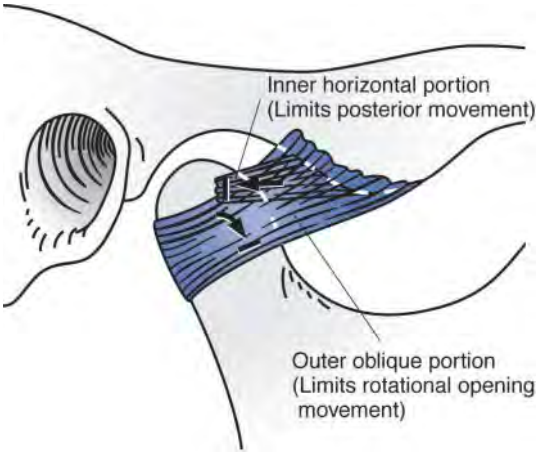


FIGURE 47-7. The TMJ (lateral aspect). (Adapted from Okeson JP, editor. Management of Temporomandibular Disorders and Occlusions. 2nd ed. St. Louis: CV Mosby; 1989; pp. 3–26.)

VASCULAR SUPPLY AND INNERVATION

The vascular supply of the TMJ arises primarily from branches of the superficial temporal and maxillary arteries posteriorly and the masseteric artery anteriorly. There is a

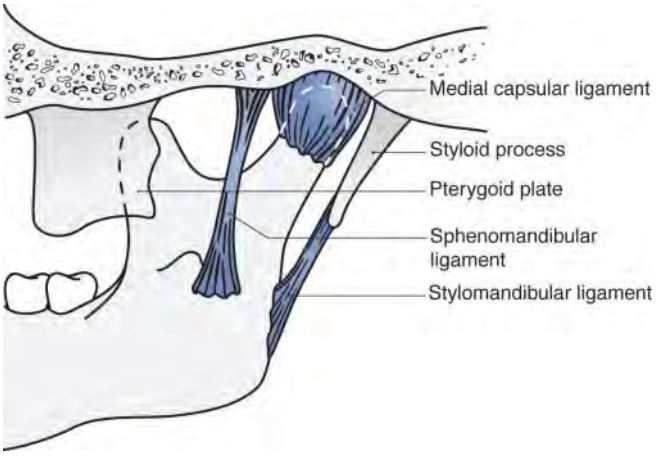


FIGURE 47-8. The temporomandibular ligament (medial view).

rich plexus of veins in the posterior aspect of the joint associated with the retrodiskal tissues, which alternately fill and empty with protrusive and retrusive movements, respectively, of the condyle-disk complex and which also function in the production of synovial fluid. The nerve supply to the TMJ is predominantly from branches of the auriculotemporal nerve with anterior contributions from the masseteric nerve and the posterior deep temporal nerve.[1] Many of the nerves providing innervations to the joint appear to be vasomotor and vasosensory, and they may have a role in the production of synovial fluid.

MUSCULATURE

All muscles attached to the mandible influence its movement to some degree. Only the four large muscles that attach to the ramus of the mandible are considered the muscles of mastication; however, a total of 12 muscles actually influence mandibular motion, all of which are bilateral.[1] Muscle pairs may function together for symmetrical movements or unilaterally for asymmetrical movement. For example, contraction of both lateral pterygoid muscles results in protrusion and depression of the mandible without deviation, whereas contraction of one of the lateral pterygoid muscles results in protrusion and opening with deviation to the opposite side.

Muscles influencing mandibular motion may be divided into two groups by anatomic position. Attaching primarily to the ramus and condylar neck of the mandible is the supramandibular muscle group, consisting of the temporalis, masseter, medial pterygoid, and lateral pterygoid muscles. This group functions predominantly as the elevators of the mandible. The lateral pterygoid does have a depressor function as well.[7] Attaching to the body and symphyseal area of the mandible and to the hyoid bone is the inframandibular group, which functions as the depressors of the mandible. The inframandibular group includes the four suprahyoid muscles (digastric, geniohyoid, mylohyoid, and stylohyoid) and the four infrahyoid muscles (sternohyoid, omohyoid, sternothyroid, and thyrohyoid). The suprahyoid muscles attach to both the hyoid bone and the mandible and serve to depress the mandible when the hyoid bone is fixed in place. They also elevate the hyoid bone when the mandible is fixed in place. The infrahyoid muscles serve to fix the hyoid bone during depressive movements of the mandible.

Supramandibular Muscle Group

The temporalis muscle (Figure 47-9) is a large, fan-shaped muscle taking its origin from the temporal fossa and lateral aspect of the skull, including portions of the parietal, temporal, frontal, and sphenoid bones. Its fibers pass between the zygomatic arch and the skull and insert on the mandible at the coronoid process and anterior border of the ascending ramus down to the occlusal surface of the mandible, posterior to the third molar tooth.[1] Viewed coronally, the temporalis muscle has a bipennate character in that fibers arising from the skull insert on the medial aspect of the coronoid process whereas fibers arising laterally from the temporalis fascia insert on the lateral aspect of the coronoid process. In an anteroposterior dimension, the temporalis muscle consists of three portions: the anterior, whose fibers are vertical; the middle, with oblique fibers; and the posterior portion, with semihorizontal fibers passing forward to bend under the zygomatic arch. The function of the temporalis muscle is to elevate the mandible for closure. It is not a power muscle. In addition, contraction of the middle and posterior portions of the temporalis muscle can contribute to retrusive movements of the mandible. To a small degree, unilateral contraction of the temporalis assists in deviation of the mandible to the ipsilateral side.

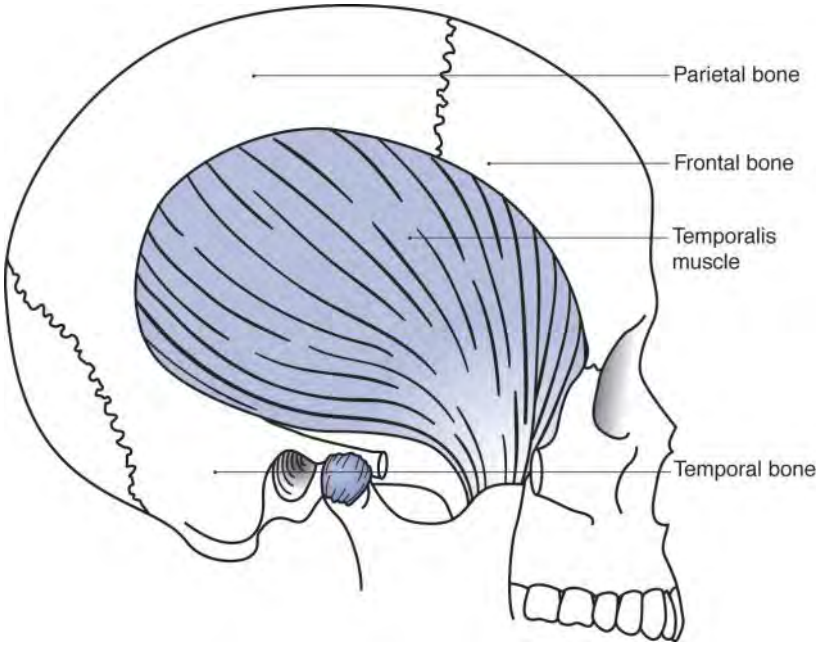


FIGURE 47-9. The temporalis muscle with zygomatic arch and masseter muscle removed. (Adapted from Clemente CD, editor. Gray's Anatomy of the Human Body. 30th ed. Philadelphia: Lea & Febiger; 1985; p. 451.)

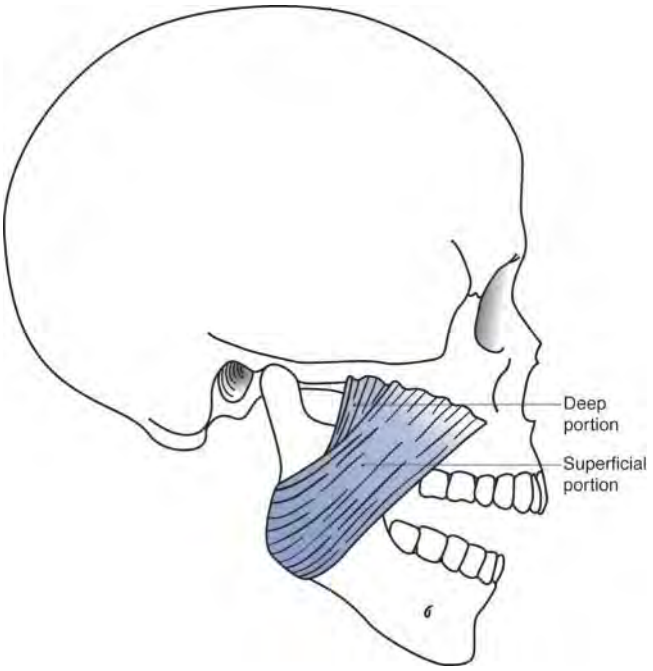


FIGURE 47-10. The masseter muscle.

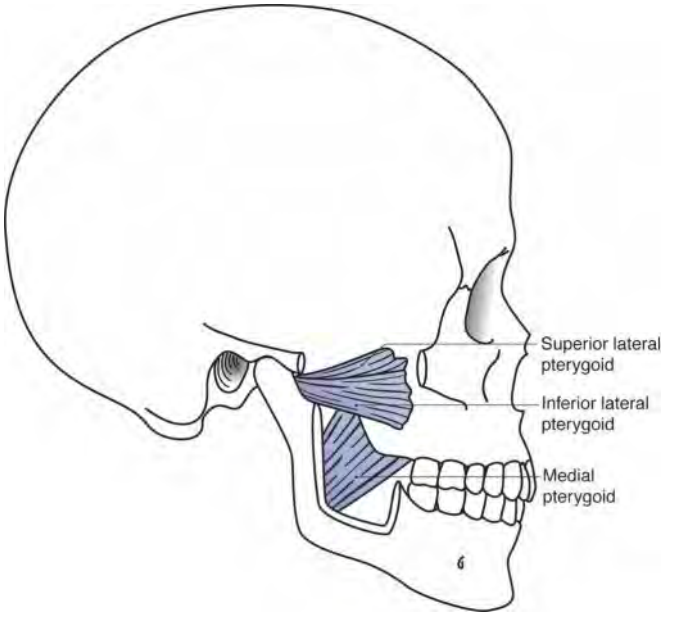


FIGURE 47-11. The medial and lateral pterygoid muscles. (Adapted from Clemente CD, editor. Gray's Anatomy of the Human Body. 30th ed. Philadelphia: Lea & Febiger; 1985; p. 451.)

The masseter muscle (Figure 47-10), a short rectangular muscle taking its origin from the zygomatic arch and inserting on the lateral surface of the mandible, is the most powerful elevator of the mandible and functions to create pressure on the teeth, particularly the molars, in chewing motions. The masseter muscle is composed of two portions, superficial and deep, which are incompletely divided, yet have somewhat different functions. The superficial portion originates from the lower border of the zygomatic bone and the anterior two thirds of the zygomatic arch and passes inferiorly and posteriorly to insert on the angle of the mandible. The deep head originates from the inner surface of the entire zygomatic arch and on the posterior one third of the arch from its lower border. The deep fibers pass vertically to insert on the mandible on its lateral aspect above the insertion of the superficial head. The superficial portion in particular has a multipennate appearance with alternating tendinous plates and fleshy bundles of muscle fibers, which serve to increase the power of the muscle. Both the superficial and the deep portions of the masseter muscle are powerful elevators of the mandible, but they function independently and reciprocally in other movements. Electromyographic studies show that the deep layer of the masseter is always silent during protrusive movements and always active during forced retrusion, whereas the superficial portion is active during protrusion and silent during retrusion.[8] Similarly, the deep masseter is active in ipsilateral movements but does not function in contralateral movements, whereas the superficial masseter is active during contralateral movements but not in ipsilateral movements.

The medial pterygoid muscle (Figure 47-11) is rectangular and takes its origin from the pterygoid fossa and the internal surface of the lateral plate of the pterygoid process, with some fibers arising from the tuberosity of the maxilla and the palatine bone. Its fibers pass inferiorly and insert on the medial surface of the mandible, inferiorly and posteriorly to the lingual. Like the masseter muscle, the medial pterygoid fibers have alternating layers of fleshy and tendinous parts, thereby increasing the power of the muscle. The main function of the medial pterygoid is elevation of the mandible, but it also functions somewhat in unilateral protrusion in a synergism with the lateral pterygoid to promote rotation to the opposite side.

The lateral pterygoid muscle (see Figure 47-11) has two portions that can be considered two functionally distinct muscles. The larger inferior head originates from the lateral surface of the lateral pterygoid plate.[9] Its fibers pass superiorly and outward to fuse with the fibers of the superior head at the neck of the mandibular condyle, inserting into the pterygoid fovea. The superior head originates from the infratemporal surface of the greater sphenoid wing, and its fibers pass inferiorly, posteriorly, and outward to insert in the superior aspect of the pterygoid fovea, the articular capsule, and the articular disk at its medial aspect, as well as to the medial pole of the condyle. Anatomic studies have shown that the majority of the superior head fibers insert into the condyle rather than the disk.

The inferior and superior heads of the lateral pterygoid muscle function independently and reciprocally.[8,10] The primary function of the inferior head is protrusive and contralateral movement. When the bilateral inferior heads function together, the condyle is pulled forward down the articular eminence, with the disk moving passively with the condylar head. This forward movement of the condyle down the

TABLE 47-1. **Contributions of the Supramandibular Muscles of Mastication to Movements of the Jaw as Confirmed by Electromyography**

Muscles of Mastication	Resultant Jaw Movement
Medial pterygoid	Closure, protrusion
Lateral pterygoid (inferior head)	Protrusion, opening contralateral
Lateral pterygoid (superior head)	Retrusion, closure, ipsilateral
Masseter, superficial layer	Protrusion, closure contralateral
Masseter, deep layer	Retrusion, closure ipsilateral
Temporalis, anterior portion	Closure
Temporalis, posterior portion	Retrusion, closure ipsilateral

inclined plane of the articular eminence also contributes to opening of the oral cavity. When the inferior head functions unilaterally, the resulting medial and protrusive movement of the condyle results in contralateral motion of the mandible. The function of the superior head of the lateral pterygoid muscle is predominantly involved with closing movements of the jaw and with retrusion and ipsilateral movement. A summary of the movements of the lateral pterygoid muscle and the other supramandibular muscles is given in Table 47-1.

Inframandibular Muscle Group

The inframandibular muscles can be subdivided into two groups: the suprahyoids and the infrahyoids. The suprahyoid group consists of the digastric, geniohyoid, mylohyoid, and stylohyoid muscles; lies between the mandible and the hyoid bone; and serves to either raise the hyoid bone, if the mandible is fixed in position by the supramandibular group, or depress the mandible, if the hyoid bone is fixed in position by the infrahyoids. The infrahyoid group, consisting of the sternohyoid, omohyoid, sternothyroid, and thyrohyoid muscles, attaches to the hyoid bone superiorly and to the sternum, clavicle, and scapula inferiorly. This group of muscles can either depress the hyoid bone or hold the hyoid bone in position, relative to the trunk, during opening movements of the mandible.

BIOMECHANICS OF TMJ MOVEMENT

Complex free movements of the mandible are made possible by the relation of four distinct joints that are involved in mandibular movement: the inferior and superior joints—bilaterally. Two types of movement are possible: rotation and translation.

The inferior joints, consisting of the condyle and disk, are responsible for rotation, a hingelike motion. The center of rotation is considered to be along a horizontal axis passing through both condyles.[4,5] In theory, pure hinge motion of approximately 2.5 cm measured at the incisal edges of the anterior teeth is possible. Nevertheless, most mandibular movements are translatory as well, involving a gliding motion between the disk and the temporal fossa, which are the components of the superior joints. The mandible and disk glide together as a unit because they are held together by the collateral ligaments. The maximum forward and lateral movement of the upper joint in translation is approximately 1.5 cm.

All movements of the mandible, whether symmetrical or asymmetrical, involve close contact of the condyle, disk, and articular eminence. Pure opening, closing, protrusive, and retrusive movements are possible as a result of bilaterally symmetrical action of the musculature. Asymmetrical movements, such as those seen in chewing, are made possible by unilateral movements of the musculature with different amounts of translation and rotation occurring within the joints on either side.

The positioning of the condyle and disk within the fossa, as well as the constant contact between the condyle, disk, and eminence, is maintained by continuous activity of the muscles of mastication, particularly the supramandibular group. The ligaments associated with the TMJ do not move the joint. Although they can be lengthened by movements of muscles, they do not stretch (i.e., do not have an elastic recoil that returns them to a resting position automatically).

Instead, the role of the ligaments is that of a passive restriction of movement at the extreme ranges of motion. During normal function, rotational and translational movements occur simultaneously, permitting the free range of motion necessary in speaking and chewing.

PATHOLOGY OF THE TMJ

The demand for treatment of temporomandibular joint dysfunction (TMJD) is well known. Most studies estimate the prevalence of clinically significant TMJ-related jaw pain to be at least 5% of the general population. Approximately 2% of the general population seeks treatment for a TMJ-related symptom.[11,12] TMJD may be the result of muscular hyperfunction or parafunction and/or underlying primary or secondary degenerative changes within the joint. It is important to note, however, that no single causative factor leading to TMJD has been unequivocally demonstrated in scientifically based studies.[13] Classification of TMJD is separated into nonarticular and articular categories and has been eloquently described by de Bont and colleagues.[13]

Nonarticular disorders include muscle disorders such as myofascial dysfunction, muscle spasm (with splinting, pain, and muscle guarding), and myositis. Articular disorders, often accompanied by internal derangement, include noninflammatory and inflammatory arthropathies, growth disorders, and connective tissue disorders. In diagnosing and treating TMJD, it is helpful to assess patients with the previous classification as a frame of reference Table 47-2. Treatment modalities can vary significantly depending on this classification.

Nonarticular Temporomandibular Disorders

Nonarticular TMJ disorders most commonly manifest as masticatory muscle dysfunction. Approximately one half or more of all TMJDs are forms of masticatory myalgia.[14,15]

TABLE 47-2. **General Classification of Synovial Disorders**

Articular Disorders
Noninflammatory Arthropathies
Primary idiopathic osteoarthrosis
Secondary osteoarthrosis (trauma, prior surgery, avascular necrosis)
Mechanical derangements
Bone and cartilage disorders, with articular manifestations
Inflammatory Arthropathies
Synovitis
Capsulitis
Rheumatoid arthritis
Juvenile rheumatoid arthritis
Seronegative polyarthritis
Ankylosing spondylitis
Psoriatic arthritis
Reactive arthritis (bacterial, viral, fungal)
Growth Disturbances
Non-neoplastic: developmental (hyperplasia, hypoplasia, dysplasia)
Non-neoplastic: acquired (condylolysis)
Neoplasms
Pseudotumors (synovial chondromatosis)
Benign (chondroma, osteotoma)
Malignant (primary, metastatic)
Diffuse Connective Tissue Disorders
Miscellaneous Articular Disorders
Nonarticular Disorders
Muscular Disorders
Muscle spasm (strain)
Myofascial pain and dysfunction
Fibromyalgia
Myotonic dystrophies
Myositis ossificans progressiva
Growth Disturbances

They include such conditions such as acute muscle strain and spasm, myofascial pain and dysfunction (MPD), chronic conditions such as fibromyalgia, and less commonly, myotonic dystrophies and myositis ossificans. They invariably contribute to decreased mandibular range of motion and pain. The important role of the supramandibular and inframandibular muscle groups on mandibular movement and function is evident in these conditions. Other nonarticular disorders include growth disorders affecting TMJ function and miscellaneous factors such as heterotopic bone formation leading to TMJD.

MPD is most commonly related to masseter or temporalis muscle spasm.[16] In addition, it can involve the pterygoids or any combination of the supramandibular or inframandibular muscle groups. Parafunctional habits such as bruxism and jaw clenching are thought to be the main contributors to MPD and have also been found to be causative in acute closed-lock conditions. The literature is replete with various treatment modalities for MPD. Such treatments include occlusal adjustments (for gross discrepancies), night-guard appliances (for joint unloading, jaw repositioning, and occlusal protection), nonsteroidal anti-inflammatory medications, muscle relaxants, and physical therapy. These treatment modalities, alone or in combination, remain the standard of care for the treatment of nonarticular TMJD, particularly MPD.

Fibromyalgia is a systemic condition marked by poor sleep, generalized pain with absence of localization to joints, and a history of somatization in other organ systems such as irritable bowel syndrome and headaches.[17] It is typically observed in an older population than MPD and has a female predilection. Fibromyalgia is often difficult to differentiate from MPD and is treated in similar fashion; that is, nonsurgically, with anti-inflammatory medications, dietary modifications, home-care techniques, bite appliances, and physical therapy. There appears to be a poorer overall response to the treatment of fibromyalgia when compared with MPD.

Rarely, other nonarticular conditions such as myotonic dystrophy and myositis ossificans progressiva can lead to significant loss of function and pain in the TMJ region. Myotonic dystrophy is a dominantly inherited multisystem disorder that may affect facial muscles in fully developed disease states.[18] This condition may contribute to atrophy and fibrosis of the supramandibular and inframandibular musculature. Clinically, there are a variety of types of myotonic dystrophies. They tend to exert their pathologic effects in similar fashion, sometimes resulting in trismus, loss of function, and pain. Myositis ossificans progressiva is a rare condition resulting in fibrosis of soft tissues after apparent minor trauma. This condition has been reported to affect TMJ function after local trauma, including surgery, and can result in significant loss of mandibular range of motion, trismus, and pain.[19] Soft tissue ossification can sometimes occur after head trauma, severe burns, or neurogenic stimulus. In these cases, heterotopic bone formation is observed and can lead to ankylosis in multiple joints throughout the body including the TMJ.[20]

Articular TMJ Disorders

Noninflammatory articular disorders of the TMJ, the most common of which is osteoarthrosis, are often idiopathic. Osteoarthrosis can manifest as chondromalacia (softening of the cartilage), temporary or permanent disk displacement, degenerative changes within bone and cartilage often with osteophyte formation and remodeling, fibrosis, or any combination of these. Noninflammatory articular disorders may also be secondary to trauma, infection, previous surgery, crystal deposition disorders (gout and pseudogout), avascular necrosis, or structural damage to joint cartilage resulting in disk displacement and/or perforation (Figure 47-12). TMJ disk displacement has been categorized through a widely accepted staging system by Wilkes,[21] using such criteria as severity of displacement and chronicity (Table 47-3).

Noninflammatory arthropathies are distinctly limited in their amount of overt inflammation and may be clinically silent or focal in nature. Alternatively, if the condition becomes more severe, symptoms will ensue. If degenerative changes

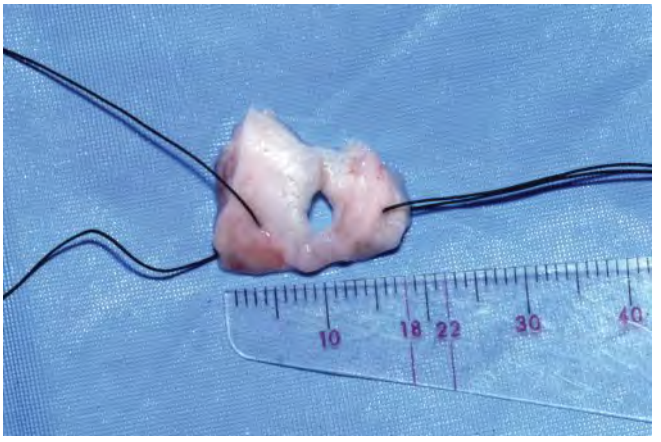


FIGURE 47-12. Perforated disk (Wilkes stage V).

TABLE 47-3. **Wilkes Classification**

Stage I	Early reducing disk displacement
Stage II	Late reducing disk displacement
Stage III	Nonreducing disk displacement: acute/subacute
Stage IV	Nonreducing disk displacement: chronic
Stage V	Nonreducing disk displacement: chronic with osteoarthritis

progress to synovitis, joint effusion (secondary to increased vascular permeability), or capsulitis, it is then considered to have transformed into an inflammatory arthropathy.

Inflammatory arthropathies are primarily due to such conditions as rheumatoid arthritis, juvenile rheumatoid arthritis, ankylosing spondylitis, psoriatic arthritis, or arthritis resulting from infectious causes (see Table 47-2). Secondary causes of inflammatory arthropathies include synovitis, capsulitis, traumatic arthritis, or acute inflamed crystal-induced arthritis, such as gout. As discussed previously, noninflammatory arthropathies can progress to the inflammatory types through increasing concentrations of degradation products within the joint. Degenerative changes resulting in the release of inflammatory mediators have been demonstrated to worsen the degree of tissue destruction and dysfunction within the TMJ. This pathologic inflammatory cascade has been the primary focus of current TMJ research.

Historically, the gross evaluation of disk position and disk integrity has been the mainstay of internal derangement diagnosis and management. More recently, the physiologic activity of synovial cells, chondrocytes, and inflammatory cells in symptomatic joints has been associated with pathogenesis. This fundamental shift in focus has changed the primary treatment approach from open-joint surgery aimed at restoring the functional anatomy of the TMJ to less invasive techniques directed toward lysis of adhesions and intracapsular lavage.[22] TMJ arthrocentesis and arthroscopy are thought to achieve an alteration in the joint milieu favoring a reduction in symptoms and improved joint function. Open-joint surgery, nonetheless, may still have a role in severe degenerative disease when preoperative criteria are met and surgery is indicated.

Milam and Schmitz[23] have proposed a variety of molecular biologic mechanisms for TMJD. Synovoid cells, chondrocytes, and inflammatory cells in the TMJ produce a physiologic balance between anabolic and catabolic cytokines.[23] Anabolic cytokines such as insulin-like growth factor-I and transforming growth factor-beta are instrumental in the formation of extracellular joint matrix molecules. Collagen, proteoglycans, and glycoproteins are essential in load-bearing joints like the TMJ. Alternatively, catabolic cytokines such as interleukin-1 (IL-1), IL-6, and tumor necrosis factor-alpha (TNF-α) have been identified with the formation of proteases within the TMJ. These proteases (aspartic, cysteine, serine, and metalloproteases, among others) operate at low and neutral pH to exert their pathologic effects leading to degenerative changes.

Oxidative stress, often found associated with pathologic joints, is thought to contribute to free radical formation in the TMJ. The presence of free radicals has been postulated as an amplifying factor in the activation of cytokines, enzymes, neuropeptides, and arachidonic acid metabolites leading to degenerative joint disease.[24] Nitric oxide, a free radical involved in regulating vascular tone, has been observed at higher concentrations in arthritic joints. Nitric oxide has direct effects on prostaglandin synthesis and cyclooxygenase-2 enzymes leading to synovial inflammation and tissue destruction.[25] In a normal functioning joint, a delicate balance is maintained between anabolic and catabolic mechanisms. In symptomatic joints, catabolic processes have been found to exert greater overall effects, thus disrupting the balance between anabolic physiologic maintenance and the negative effects of catabolic cytokines.

TMJ synovial fluid analysis has proved to be an excellent vehicle for evaluating the proposed contribution of cytokines, proteinases, and other catabolites to TMJD. Multiple independent studies support the hypothesis of catabolic imbalance within the joint. Kubota and coworkers[26] demonstrated increased levels of IL-1β, IL-6, and active matrix metalloproteinases in TMJs with internal derangement and osteoarthritis when compared with control samples. This study suggests the presence of elevated concentrations of these cytokines and proteinases serving as potential catabolic markers for cartilage degradation in the human TMJ. Murakami and associates[27] reported high concentrations of chondroitin-4 and chondroitin-6 sulfates compared with hyaluronic acid in the TMJ synovial fluid of patients with internal derangement suggesting glycosaminoglycan components as markers of joint pathology. Israel and colleagues[28] demonstrated the prevalence of synovitis and osteoarthritis through arthroscopic evaluation in symptomatic TMJs. These findings correlated with increased levels of keratan sulfate in the synovial fluid of these joints, suggesting its role as a potential biochemical marker for articular cartilage degradation.[28] Recently, osteoclastogenesis inhibitory factor/osteoprotegerin

(OCIF/OPG), a member of the TNF receptor family, has been studied in synovial fluid samples of TMJD patients.[29] Increased osteoclastic activity has been seen histologically in diseased mandibular condyles. Osteoclast differentiation requires cell-to-cell contact between osteoclast progenitors and bone marrow stromal cells. The presence of OCIF/OPG is thought to inhibit osteoclast differentiation by preventing the cell-to-cell contact needed for such activity. Synovial fluid samples in this study demonstrated decreased amounts of OCIF/OPG in osteoarthritic and internally deranged joints, suggesting its physiologically important function in healthy joints. Although further investigation of synovial fluid components in TMJD is necessary to formulate definitive conclusions, it continues to shed new light on the pathogenesis and treatment of such disorders.

Treatment of patients with internal derangement of the TMJ typically begins with nonsurgical treatment modalities. Bite appliance therapy, diet modifications, nonsteroidal anti-inflammatory medications, muscle relaxants, moist heat or ice, and physical therapy have been found to be efficacious.[30] Surgical intervention is typically employed only after failure of nonsurgical treatment objectives.

A variety of surgical treatment modalities have been used in the treatment of articular TMJD. Arthrocentesis and TMJ arthroscopy have been found to be minimally invasive effective treatments for articular TMJD by decreasing pain and increasing mandibular range of motion. (Surgical techniques for arthroscopy are addressed in Chapter 49). Indications for these modalities include, but are not limited to, acute closed-lock degenerative joint disease accompanied by pain and limited range of motion and joint effusion. Arthrocentesis and arthroscopy have also been reported to be useful in severe, often sudden-onset, closed-lock disease due to an anchored or "stuck disk" phenomenon. This proposed phenomenon involves the disk becoming adherent to the glenoid fossa through increased intra-articular friction, with or without the formation of adhesions within the joint. Lysis of adhesions with joint lavage has been reported efficacious in restoring mandibular range of motion and decreasing pain in these clinical scenarios.[31,32] TMJ arthrocentesis and arthroscopy show promising results using the previously discussed criteria of pain symptoms and mandibular range of motion.[33] Based on the pathophysiology discussed in this chapter, a hypothesis explaining the efficacy of joint lavage relates to a proposed alteration in the biochemical constituents of the joint fluid, thus shifting the balance toward anabolic processes while reducing the amount of active catabolites contained within the joint.[34]

Indications for open arthrotomy include, but are not limited to, joint ankylosis, the need for reconstruction owing to condylar resorption or growth disturbance, history of previous surgery, removal of foreign bodies, neoplasia, trauma, or severe degenerative disease precluding less invasive interventions. (Indications and techniques for open TMJ surgery are addressed more thoroughly in Chapters 51 and Chapter 52). Open-joint surgery is primarily based on restoration of the functional anatomy of the TMJ when less invasive techniques are not feasible or unsuccessful. Data suggest comparable outcomes between open and closed surgery in the TMJ with lower morbidity associated with the latter.[35] Open TMJ surgery remains a viable treatment option at the end of the surgical treatment algorithm. New insight into the pathogenesis of TMJD has opened the door to less invasive (albeit equally effective) treatment options for a large number of TMJD patients.

Infections of the TMJ

Infections of the TMJ are not common. Prompt diagnostic and therapeutic intervention is required when an infection of the TMJ is suspected because joint distention is usually painful and permanent changes in joint function can occur. On examination, patients usually exhibit a posterior open bite on the ipsilateral side as a result of the increased joint fluid. The patient will also maintain a posture toward the contralateral side.[36] The surface overlying an infected joint is often warm, and fluctuance is occasionally felt.

The bacteria causing an infected joint are usually spread through a hematogenous route. The synovium is vascular and lacks a basement membrane, which permits bloodborne bacteria to gain access to the joint space.[37] Joints with underlying arthritic disease tend to be more susceptible to distant infection. Although the source of the bacteria is usually at a distant site, spread from dental infections of maxillary teeth has been reported in which the bacteria are thought to spread through the pterygoid plexus of veins to the joint.[38] Direct innoculation of a joint area after a traumatic injury is also possible. Complications of infections of the TMJ include fistula formation, fibrosis or bony ankylosis, temporal bone osteomyelitis, and intracranial abscess formation.

A thorough history and review of systems aids in the diagnosis of acute infectious arthritis of the TMJ. Active infection in adjacent sites, especially the ipsilateral maxillary molars, should be searched for. Other joints must be assessed to determine whether they are involved. Initially aspiration of the joint should be considered to both relieve the pain from the joint capsular distention and help in the identification of the infecting organism(s). The aspiration is performed by using a 20-gauge or larger needle under sterile conditions. The synovial fluid should be Gram-stained and cultured for both aerobic and anaerobic bacteria. Sedation or general anesthesia may be required for the arthrocentesis.

In sexually active adults, 60% of general acute infectious arthritis is due to *Neisseria gonorrhoeae.*[39] The majority of these patients have a prodrome of malaise, anorexia, headaches, fever, and chills. A few days of migratory arthritis usually precedes the localization of infection in one or two joints. Markowitz and Gerry[40] reported a TMJ involvement rate of 3% in patients with disseminated gonococcal arthritis. In children younger than 2 years, almost 50% of acute infectious arthritis is due to *Haemophilus influenzae.* No reports of TMJ involvement are available. Other gram-positive cocci have

been isolated from TMJ infections in all age groups, including staphylococci (particularly in the elderly) and β-hemolytic streptococci. The adherence characteristics of *Staphylococcus aureus* and *N. gonorrhoeae* to synovium account for their prevalence.[41] Thus, the best choice for initial empirical antibiotic therapy for an acute infectious TMJ arthritis is an agent that combines a penicillin with a β-lactamase inhibitor. The combination of ampicillin and sulbactam will cover infections from the staphylococcal and streptococcal groups. Sulbactam, a derivative of penicillin, inactivates bacteria-produced β-lactamase and also has direct bactericidal activity against the *Neisseria* organisms. Therefore, this combination may have an advantage over the combination of a penicillin and clavulanic acid. It should be noted that bacterial resistance has become increasingly more problematic. Reference to up-to-date antibiotic regimens is recommended.

Lyme disease, due to infection with *Borrelia burgdorferi*, has been documented to cause acute infection of the TMJ. The incidence of this is greater in the Northeastern United States where this tick-borne disease is more common and the authors have treated five patients with this disorder. This disorder has been reported even in Slovenia, so it should be considered as part of the differential diagnosis of patients with TMJ disorders.[42] Patients present with acute pain directly in the TMJ region, which may or not be bilateral in distribution. The findings of limitation in opening along with pain on chewing should lead the practitioner to consider this as a cause. Serum titers for antibodies for *B. burgdorferi* become positive in the later phases of this disease and can assist with the diagnosis. Patients may often present with migratory arthritis affecting the large joints such as the knee, but other and multiple joint involvement are reported. Treatment is with a 1-month course of doxycycline or amoxicillin. In more acute or recalitrant cases, a 2-week course of intravenous ceftriaxone is utilized.[43] Cultures of *B. burgdorferi* from joint aspirates have not been documented, but the response to antibiotics along with the findings of antibodies in serum samples lead to the diagnosis.

Effective treatment of other forms of septic arthritis by oral antibiotics has not been well studied; therefore, the parenteral administration of antibiotics should be used initially.[37] Choices include ampicillin and sulbactam (Unasyn) 3 g intravenously (IV) every 6 hours or clindamycin 600 mg IV every 6 hours in penicillin-allergic patients. A third-generation cephalosporin, cefotaxime 6 to 12 g IV per day, could be used for a gram-negative infection in a nonhospitalized patient.[37] Tobramycin 3 mg/kg/day in four doses should be considered to treat a possible presence of *Pseudomonas aeruginosa* in infections that develop in hospitalized or immunocompromised patients.

The duration of treatment depends on the clinical response and the organism isolated. Based on information available for treatment of septic arthritis involving *N. gonorrhoeae*, the patient with a septic TMJ could be placed on oral ampicillin or tetracycline after a 2-week course of intravenous therapy. Reportedly infections involving *S. aureus* and gram-negative bacilli require 4 weeks of total therapy, and 2 to 3 weeks of therapy is adequate for streptococci and *Haemophilus* species.[44] Thus, it appears that a 30-day total course of antibiotic therapy for acute TMJ infection is appropriate.

In addition to culture and sensitivity testing, the aspirate from the infected joint should be submitted for white blood cell (WBC) count and differential and examined for the presence of crystals and fibrinogen. Fibrinogen is usually present in the synovial fluid of acutely infected joints. Therefore, some of the synovial fluid collected should be placed into a heparinized tube to prevent clotting. It is important to note that ethylenediaminetetraacetic acid (EDTA) interferes with crystal analysis; therefore, synovial fluid should not be placed in tubes containing it. The synovial fluid of an inflamed joint commonly contains greater than 2000 WBC/mm^3 (normal < 200 WBC/mm^3). Septic joints normally have WBC counts greater than 50,000/mm^3. The cells are primarily mononuclear, as opposed to a predominance of PMN cells in infected joint fluid. An exception to this occurs in fungal or mycobacterial joint infections in which the synovial fluid usually contains less than 20,000 WBC/mm^3 and shows a greater proportion of mononuclear cells.[45]

Following the institution of antibiotic therapy, lavage of the joint may be useful. Removing the joint fluid containing the products of the inflammation, reducing the bacterial load within the joint, and relieving the joint distention will usually markedly relieve the patient's symptoms and may also decrease the likelihood of spread to the temporal bone. Murakami and workers[38] have reported on the use of the arthroscope for monitoring and treating an acutely infected TMJ.

Following the resolution of an acute TMJ infection, a program must be started to minimize joint disability and to monitor for recurrence of infection. The acute inflammatory process that accompanies an infection can result in the deposition of fibrinogen and other products, which can predispose the joint toward a fibrous or bony ankylosis. Active range of motion exercises are started as soon as possible to prevent intra-articular adhesions. The patient's range of motion should be documented at weekly intervals. If the range of motion is still limited 1 month after the resolution of the infection, a brisement procedure or an arthroscopic procedure to lyse intra-articular adhesions should be considered. However, before this, extracapsular causes of limited opening, such as masseter muscle trismus, need to be differentiated from intracapsular disorders. Intracapsular restrictions are usually accompanied by restriction of lateral excursions to the contralateral side and deviation on opening. Recurrence of joint infection (of all joints) has been reported to occur at a rate of 10.5%.[46] Newman[46] noted that infection recurred as long as 1 year after the initial episode. The patient should be advised of this possibility.

Neoplastic Diseases

Tumors affecting the TMJ area are exceedingly rare. In one review, the authors report a fairly large incidence of 6 patients with TMJ tumors out of 621 patients presenting with initial

TMJD.[47] The tissues from which a neoplasm may arise include the synovium, bone, cartilage, and associated musculature. Neoplasms of this region can present with signs and symptoms similar to those occurring with internal derangement (preauricular pain and dysfunction) and, thus, can result in a delay in the diagnosis. Owing to the proximity of the cranial base, delay in treatment can be detrimental. The clinician should be aware of this when treating temporomandibular disorders, especially if the patient fails to respond to traditional therapy.

Benign Tumors

The most common benign bone tumors of the TMJ include the osteoma and condylar enlargement or condylar hypertrophy. Both present signs related to the increase in size of the condyle, a shift in the mandible to the contralateral side, and an ipsilateral open bite. Often, the range of motion is decreased as the increased size of the condylar head prevents normal translation. Radiographs, including tomograms and computed tomography scans, should be obtained to delineate the extent of the condylar growth and to determine involvement of the glenoid fossa and associated structures. Radionuclide scans should be performed to determine whether the process is still active and bone is being produced. Treatment includes a condylar head resection (partial or complete) for active lesions or a condyloplasty to reduce condylar size and restore the occlusion for nongrowing lesions. Condylar reconstruction is usually not necessary. The disk should be preserved or replaced (if it has been damaged) with a temporalis muscle flap or cartilage graft. Physical therapy is usually required to reduce dysfunction. Postoperative maxillomandibular fixation is not usually necessary, but guiding elastics may be helpful with muscle retraining. An active physical therapy program to reduce joint adhesions prevents permanent restriction of the joint.

Virtually all other benign bone tumors have been reported to occur in the TMJ. These bone tumors behave as they would in other areas of the mandible and, therefore, should be treated in a similar fashion.

Synovial Tumors

Synovial chondromatosis is the most commonly reported neoplasm of the TMJ synovium. Lustman and Zelster[48] reported a series of 50 cases in which the mean age was 47 years. This is in contrast to synovial chondromatosis involving other joints, which is more commonly found in the 20- to 30-year-old age group.[49] Pain and swelling of the preauricular area are the most common initial signs. Depending on the degree of calcification present, radiographs may reveal the presence of loose radiodense bodies within the joint. These loose bodies are formed by metaplastic synovial tissues. Foci of metaplastic synovium detach from the synovial lining and remain viable while suspended in the synovial fluid. While suspended, they form a perichondrium and continue to grow and enlarge. Although the reason is unknown, this process most frequently occurs in the superior joint space. The loose bodies are composed of cartilage containing multinucleated cells. The presence of cellular atypia and hyperchromatism is common, and a careful review of all histologic material removed is necessary to rule out the possibility of chondrosarcoma.

Treatment of synovial chondromatosis involves extirpation of the loose bodies and removal of the synovial lining. Lustman and Zelster[48] reported that a condylectomy was necessary in 13 of 47 cases to gain access to the anteromedial portion of the joint. The condyle itself is not involved and, therefore, should be removed only for access. Recurrence of synovial chondromatosis is quite rare and is thought to be caused by an incomplete excision of the original lesions. No cases of TMJ synovial chondromatosis transforming into chondrosarcoma have been reported, although this has been reported in the knee.[50]

Ganglion Cysts

Ganglion cysts have also been reported to occur in association with the TMJ. These are cystic structures that arise subcutaneously in association with the joint capsule or tendon sheaths. Histologic examination of a ganglion reveals a true cyst, containing a mucinous fluid and hyaluronic acid. These lesions present as a preauricular mass and may produce classic "TMJ symptoms," such as pain and limitation of function. The swelling produced by the ganglion in the preauricular region can be confused with a parotid mass. Surgery is indicated to remove the cyst and recurrences have not been reported.[51]

Malignant Tumors

Malignancies of the TMJ are very rare and are usually the result of direct extensions of primary lesions of adjacent structures. Metastatic disease has been reported to involve the TMJ but is more commonly found in the mandibular angle region. This may be due to the relative paucity of cancellous bone in the condylar head region.[52] The most common lesions to metastasize to the condyle are adenocarcinomas of the breast, kidney, and lungs. As with benign tumors, the early signs of malignant disease of the TMJ are pain and dysfunction. Primary malignancies of the TMJ have been reported as intrinsic tumors of the condylar bone, disk, synovium, and cartilaginous linking. Typically, patients with malignancies of the TMJ are older than the usual internal derangement patient. Patients with a history of preexisting malignant disease must undergo a thorough search for metastasis if TMJ symptoms develop. Radionuclide scans may be useful, although the inflammation from chronic synovitis can result in activity localizing in the condyle.[53] Patients presenting with a fracture of the condyle without a history of trauma should be suspect for the presence of a malignant lesion in the condyle.

Primary TMJ malignancies require aggressive therapy to prevent intracranial extension of the disease. Radiation, surgery, and chemotherapy are all appropriate means of treatment of diseases in this region. Radiation therapy can also be used for palliation in disseminated disease to control pain from the TMJ region and to prevent pathologic fractures.

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48

CHAPTER

Nonsurgical Management of Temporomandibular Disorders

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Temporomandibular disorder (TMD) is the general term used to describe the manifestation of pain and/or dysfunction of the temporomandibular joint (TMJ) and its associated structures. In general, up to 5% of the population is affected by TMD, with significantly more frequent and more severe signs and symptoms appearing in women and older adults.[1,2] The etiology of TMD may include trauma, parafunctional habits, malocclusion, joint overload, arthritides, psychological factors, and chronic nonergonomic positioning of the head and neck. The impact of psychological factors is difficult to determine, but approximately 10% to 20% of patients with TMD also manifest some form of psychiatric illness.[3] Because symptoms of TMD are quite variable and remain exceedingly difficult to attribute exclusively to one or more events (such as the true contribution or extent of involvement of muscles of mastication), the joint itself, or psychological factors, is best understood in terms of interdependence. When a diagnosis of TMD is suspected or confirmed, therapy should be directed toward improvement of function and reduction of pain and discomfort. Sufficient literature is available to suggest that nonsurgical treatment modalities may account for as high as a 74% to 85% favorable response rate in patients with TMD.[4,5] In one study, Suvinen and coworkers[6] reported that 81% of patients showed 50% or greater improvement after conservative physical therapy with a 6-month follow-up period, and the authors attributed the improvement to a possible placebo-type effect. Other sources report significant relief in 30% to 60% of patients when under some form of treatment.[7] In addition, long-term follow-up studies have suggested that almost all patients with TMD will improve over time, regardless of the type of treatment they may receive.[4,8–12] Thus, it appears well established in the literature that the majority of patients with TMD achieve some relief of symptoms with nonsurgical therapy. The dilemma for the surgeon is compounded by the broad spectrum of results and claims that use a seemingly endless variety of surgical and nonsurgical strategies in the management of TMD. Because the extent or severity of symptoms is apparently unrelated to the specific etiology, and the overwhelming number of symptoms respond to conservative management, the question of whether, and how, to incorporate surgical and nonsurgical treatment into the care of these patients becomes challenging for the oral and maxillofacial surgeon. There are absolute indications in which surgical intervention would be of primary benefit, and the question is whether there is still a role for nonsurgical therapy in these patients, and if so, when and how it should be instituted, as well as the length of treatment. One approach to this dilemma is to consider the concept of nonsurgical versus surgical therapy misleading and incomplete and not evidence-based in the literature. There are many times when it may be inappropriate to consider surgery, and at other times, nonsurgical therapy should precede, and almost always follow, surgical intervention. Therefore, it is essential for the surgeon to have a thorough appreciation of the available techniques and their limitations in order to know when and how to most appropriately manage TMD. The purpose of this chapter is to delineate those nonsurgical treatment modalities that are considered adjunctive to the available surgical options.

TREATMENT CONSIDERATIONS

The primary goal in the treatment of TMD is to alleviate pain and/or mandibular dysfunction. Pain and alteration in function (i.e., mastication and speech) can become quite debilitating, greatly affecting oral health care and diminishing the

quality of life for these individuals. Another critical objective relates to patient counseling and education regarding the predisposing factors for TMD. Depending upon the degree of impairment, patients can often be assured that TMD is a benign condition and that clinical improvement may be expected with compliance with appropriate therapeutic recommendations. However, it is prudent, if not incumbent upon the surgeon, to inform patients that complete elimination of symptoms is, in most cases, an unreasonable expectation. Nonsurgical techniques that can decrease unintentional overloading of the masticatory system, eliminate pain, reduce dysfunction, decrease chronicity, and promote healing are essential in all phases of therapy. A patient home care program may prevent further injury and allow for a period of healing. In general, patients can be instructed to limit mandibular function, modify habits, avoid stress, and institute a home exercise program.[8]

Clicking and popping of the TMJ are quite common in TMD as well as in patients with asymptomatic normal TMJs. Internal derangement of the TMJ is difficult to eliminate without surgery and usually recurs; there is inconclusive evidence to suggest whether or not this anatomic abnormality actually presents a problem for the patient, such as accelerated osteoarthritis or the development of TMD. There is considerable support in the literature that joint noise, without pain or dysfunction, requires no intervention or treatment (Table 48-1).

Once the diagnosis of TMD has been established, frequent follow-up appointments are necessary after therapy is initiated in order to determine whether there is any improvement in signs and/or symptoms. Initial results may require reassessment after several weeks of therapy, and further diagnostic procedures may be warranted to rule out vascular, neurologic, neoplastic, psychological, or otolaryngologic abnormalities. TMD is a complex multisystem disorder that is affected by many interacting factors, and strong consideration should be given toward a multidisciplinary diagnostic and treatment approach. The roles of the dentist, physical therapist, orthodontist, neurologist, psychologist/psychiatrist, anesthesiologist, and oral and maxillofacial surgeon cannot be understated and should be key constituents of any facial pain center or TMD team. The precise timing and length of therapy must be determined by the surgeon based upon the severity of clinical symptoms as well as other supporting diagnostic indicators. As with other major joints in the body, clinical suspicions should be directed to rule out pathology, decrease inflammation, allow unimpeded joint motion, and restore range of motion. To accomplish these goals in a ginglymodiarthrodial joint that is physcially connected to the opposite side, and is intimately involved in oral health and daily stresses of mastication, is indeed a clinical challenge. The remainder of this chapter provides basic guidelines for nonsurgical therapeutic modalities for TMD, and it is not intended to eliminate, or preselect, adjunctive dental or surgical treatment.

TABLE 48-1. **Goals of Nonsurgical Therapy for Temporomandibular Disorder**

Improvement in jaw function
Reduction in facial pain
Patient education and counseling
Provision of referrals for physical therapy, occlusal appliances, if indicated

NONSURGICAL THERAPY

Diet

A soft diet is is a critical component in the management of TMD in order to allow jaw rest, because this can prevent overloading of the TMJ and decrease muscular hyperactivity. The extent of time that a patient should maintain a soft food diet is dependent upon the severity of the clinical symptoms. Patients should be instructed to divide the foods into smaller pieces and abstain from eating chewy, hard, or crunchy foods, including uncooked vegetables and meats. Intermaxillary fixation is not typically used as a means of jaw rest, although it may be considered in extreme cases. A strict liquid or blenderized diet may be recommended for those patients who manifest severe TMD symptoms (Table 48-2).

Pharmacotherapy

Medications are often prescribed for managing the symptoms associated with TMD, and patients should understand that these medications may not offer a complete cure to their problem but can be a valuable adjunctive aid when prescribed as part of a comprehensive program. With pharmacotherapy, there is always a danger of drug dependency and abuse, particularly with the use of narcotics and tranquilizers. Because many TMD symptoms are periodic, there is a tendency to prescribe medications on an "as-needed," or PRN, philosophy. This type of regimen may provide brief periods of pain relief, but more frequent pain cycles can result in a lower effectiveness of the drugs, with resultant overuse or abuse of these medications.[12–15] The general recommendation for pharmacotherapy is that the medications should be prescribed at regular intervals and for a specific period of time (e.g., four times daily for 3 wk). The clinician must recognize the influence of personality traits in these patients that may contribute to drug dependency or abuse. Other obvious influential factors that must be considered in the choice of drug regimens are co-morbid medical disease, medications and potential interactions, patient age, occupation, and each

TABLE 48-2. **Soft Diet Benefits**

Decreased muscular hyperactivity
Decreased loading forces on TMJ
Controlled range of motion with hinge and sliding movements
Full liquid diet with elimination of hard chewy foods
Elimination of gum chewing

TMJ = temporomandibular joint.

patient's attitude toward pharmacotherapy in general. The most common pharmacologic agents used for the management of TMD are analgesics, anti-inflammatory agents, anxiolytic agents, antidepressants, muscle relaxants, antihistamines, and local anesthetics. Analgesics, corticosteroids, and anxiolytics are useful for the treatment of acute pain associated with TMD, whereas anti-inflammatory medications and antidepressants are primarily indicated for chronic pain management. Muscle relaxants, nonsteroidal anti-inflammatory drugs (NSAIDs), and local anesthetics can be used for both acute and chronic pain management.

Analgesics

Analgesic medications are available as either opiate or nonopiate preparations. Nonopiate analgesics (salicylates and acetaminophen) can be added to the anti-inflammatory regimen to assist in pain relief. The salicylates (acetylsalicylic acid [ASA]) are commonly used in TMD and are the benchmark medications to which other analgesics are usually compared. Salicylates are antipyretic, analgesic, and anti-inflammatory. For those patients who cannot take aspirin, a nonacetylated aspirin such as choline magnesium trisalicylate, or salsalate, may be effective. As with all salicylates, however, choline magnesium trisalicylate and salsalate should not be prescribed for children or teenagers with chickenpox, influenza, or flu symptoms or exposure. Opioid analgesics (oxycodone, propoxyphene, and hydrocodone) should be prescribed only for moderate to severe pain, and for a limited duration of treatment, owing to the high potential for addiction. These drugs are often administered in conjunction with NSAIDs or acetaminophen-containing drugs (e.g., Vicodin, Lortab, Percocet, or Darvocet). These medications act on opioid receptors in the central nervous system (CNS), producing analgesia and sedation. Because patients can become dependent rapidly on the narcotic analgesics, it is recommended that these drugs not be prescribed for longer than 2 to 3 weeks. Other side effects of this class of medications include constipation secondary to decreased gastrointestinal (GI) motility.

Anti-inflammatory Medications

Two types of anti-inflammatory medications are useful in treating TMD: NSAIDs and corticosteroids (Figure 48-1). Glucocorticoids prevent the release of arachidonic acid, a key

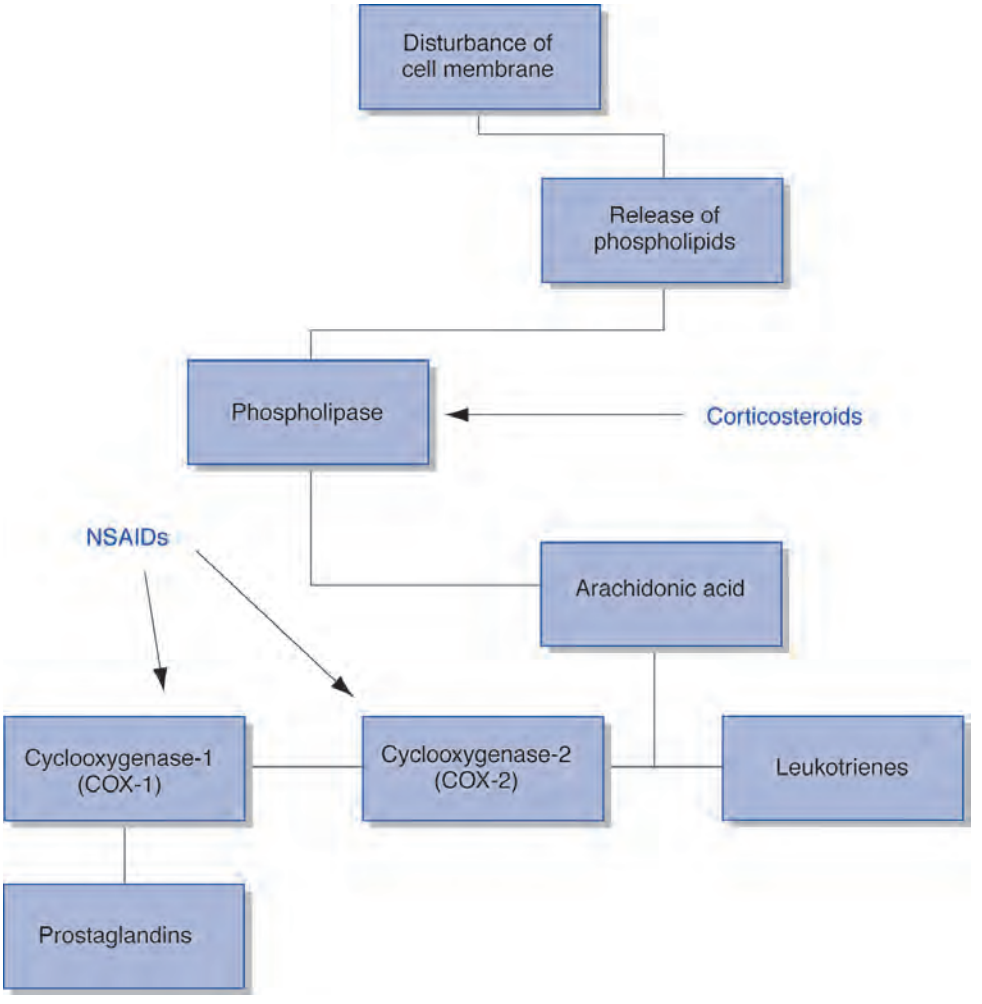


FIGURE 48-1. Arachidonic acid cascade. Corticosteroids prevent release of arachidonic acid and interrupt the cascade. Nonsteroidal anti-inflammatory drugs (NSAIDs) inhibit cyclooxygenase, which inhibits prostaglandin synthesis from arachidonic acid.

TABLE 48-3. **Commonly Used Nonsteroidal Anti-inflammatory Agents**

Category	Generic	Brand	Half-Life (hr)
Salicylates	Acetylsalicylic acid (aspirin)	Bayer	2.5
	Enteric-coated	Ecotrin	2.5
	Aspirin with buffering agent	Bufferin	2.5
	Aspirin with caffeine	Anacin	2.5
	Diflunisal	Dolobid	8–12
	Choline magnesium trisalicylate	Trilisate	9–17
	Salsalate	Disalcid	16
Propionic acid	Ibuprofen	Motrin, Advil, Nuprin, Rufen	1.8–2.5
	Fenoprofen	Nalfon	2–3
	Suprofen	Suprol	2–4
	Naproxen	Naprosyn	12–15
	Naproxen sodium	Anaprox	12–15
Acetic acid	Indomethacin	Indocin	4.5–6
	Sulindac	Clinoril	7.8 (16.4)*
	Tolmetin	Tolectin	1–1.5
Fenamic acid	Meclofenamate	Meclomen	2 (3.3)*
	Mefenamic acid	Ponstel	2
Pyrazolones	Phenylbutazone	Butazolidin	84
Oxicam	Piroxicam olamine	Feldene	30–86
COX-2 inhibitor	Celecoxib	Celebrex	11–12

COX-2 = cyclooxygenase-2.
*Active metabolite.
Adapted from Syrop SB. Pharmacological therapy. In Kaplan AS, Assael LA, editors. Temporomandibular Disorders—Diagnosis and Treatment. Philadelphia: WB Saunders; 1991; pp. 501–514.

component of the inflammatory cascade, whereas NSAIDs inhibit cyclooxygenase (COX), which inhibits prostaglandin synthesis from arachidonic acid.[16–18]

NSAIDs

The advantages of NSAIDs in TMD management result from their combined analgesic and anti-inflammatory properties (Tables 48-3 and 48-4). NSAIDs may offer relief for patients with synovitis, myositis, capsulitis, symptomatic disk displacement, and osteoarthritis.[19] This form of drug therapy helps to alleviate the inflammation, which thereby causes a decrease in pain perception. Typical side effects of NSAIDs include gastric irritation, allergies, and liver dysfunction. An ideal NSAID would be one that has minimal gastric effects, a quick onset with a long duration of action, low dosage requirements; is tolerated well even at high plasma drug levels; and is low in cost. NSAIDs are divided into seven groups based on their chemical structure: ASA, propionic acids (ibuprofen, naproxen), acetic acids (indomethacin, ketorolac), fenamic acids (meclofenamate), oxicams (piroxicam), and the COX-2 inhibitors (celecoxib [Celebrex] and rofecoxib [Vioxx was removed from the market in 2004]). The most common NSAIDs used are ibuprofen, diclofenac, and naproxen, but because of fewer GI side effects and minimal effect on platelets, COX-2 inhibitors became popular alternatives, although the potential significant cardiovascular effects (increase in thromboxane leading to thrombosis, myocardial infarction, stroke) need to be considered. Several studies have found that COX-2, an important inflammatory mediator, is present in the TMJ synovial tissue and synovial fluid of patients with internal derangement of the TMJ. This suggests that the COX-2 inhibitors may be more effective for TMJ pain and arthralgia than other analgesics.[20,21] A variety of methods may be used to reduce gastric irritation from NSAIDs including the use of enteric coating; use of prodrugs (nabumetone [Relafen]), taking the NSAID with, or after, meals, or in conjunction with antacids; and using gastric protective agents (ranitidine and sucralfate).[22] One typical initial pharmacologic regimen for the patient with TMD may include ibuprofen (Motrin, Advil) 400 to 600 mg orally four times daily (maximum dose, 3200 mg/day), with meals for a period of 3 weeks, with reassessment.

Corticosteroids

Corticosteroids result in the complete blockage of the arachidonic acid cascade, producing a more pronounced anti-inflammatory response than NSAIDs. Systemic steroids are indicated only for short-term therapy (5–7 days) owing to their long-term possible complications, including osteoporo-

TABLE 48-4. **Ideal Nonsteroidal Anti-inflammatory Drug Properties**

Minimal gastrointestinal irritation
Rapid onset
Long duration of action
Lower dosage for effect
Favorable therapeutic index
Well-tolerated at high serum levels
Readily available
Low cost

TABLE 48-5. **Commonly Used Benzodiazepines**

Generic	Brand	Usual Dosage (mg/day)	Elimination (Half-Life [hr])
Alprazolam	Xanax	0.5–1.5 (ddd)	12–15
Chlordiazepoxide	Librium	15–60 (ddd)	5–30
Diazepam	Valium	2–40 (ddd)	20–50
Flurazepam	Dalmane	30 (at bedtime)	47–100
Lorazepam	Ativan	2–6 (ddd)	10–18
Oxazepam	Serax	30–60 (ddd)	5–15
Prazepam	Verstran	20–40 (ddd)	30–100
Temazepam	Restoril	15–30 (at bedtime)	10–20
Triazolam	Halcion	0.25–0.5 (at bedtime)	1.5–5

ddd = divided daily doses
Adapted from Syrop SB. Pharmacological therapy. In Kaplan AS, Assael LA, editors. Temporomandibular Disorders—Diagnosis and Treatment. Philadelphia: WB Saunders; 1991; pp. 501–514.

sis, diabetes, hypertension, electrolyte abnormalities, and clinical Cushing's disease.[23] Steroid preparations have been directly injected into the TMJ in an attempt to decrease inflammation or mediate the inflammatory response (e.g., after arthrocentesis or arthroscopy), but long-term, or excessive, use is associated with condylar hypoplasia through inhibition of chondroblastic activity and increasing loss of calcium in the urine and GI tract.[24]

Anxiolytics

Anxiolytic medications reduce the anxiety, insomnia, and muscle hyperactivity associated with TMD (Tables 48-5 and 48-6). These drugs often help the patient reduce the perception of, or reaction to, stress. Benzodiazepines (e.g., diazepam) decrease anxiety, relax skeletal muscle, and cause sedation and may be preferable owing to their more desirable characteristics (e.g., less associated sedation). The muscle relaxant properties may be used to decrease the effects of bruxism secondary to hyperactivity of muscles of mastication. It is recommended that the benzodiazepines not be used for more than a 2-week period because of the high potential for dependency, although this length of time may be increased up to 3 weeks only at bedtime to control bruxism.[19] Buspar (azaspirode-canedione) is an anxiolytic drug; however, it does not produce either sedation or muscle relaxation, and it may be used to control anxiety in TMD patients without producing drowsiness.

TABLE 48-6. **Antianxiety Medications: Benzodiazepines**

GABA receptors agonists
5-HT agonists in the amygdala
Useful for anxiety, insomnia, muscle hypertactivity
High abuse potential
Avoid short-acting or high-potency drugs (triazolam, alprazolam, lorazepam)
Taper gradually to avoid withdrawal, rebound anxiety

GABA = γ-aminobutyric acid; 5-HT = 5-hydroxytryptamine (serotonin).

Antihistamines

Antihistamines (promethazine and hydroxyzine) antagonize central and peripheral H_1 receptors and have a sedative effect as well as anxiolytic properties. Antihistamines, unlike the benzodiazepines, do not have the potential for abuse. They can be used more safely in children and the elderly and for the treatment of vertigo and nausea that may accompany TMD.[25]

Antidepressants

Antidepressants that are useful in the management of TMD include monoamine oxidase inhibitors (MAOIs), tricyclic antidepressants (TCAs), and selective serotonin reuptake inhibitors (SSRIs; Tables 48-7 and 48-8). They are prescribed for chronic pain, headaches, sleep disorders, obsessive-compulsive disorders, and central-mediated pain disorders. The relationship between pain and depression is a challenge that often necessitates treatment of both of these clinical entities simultaneously. Depression in the TMD or chronic pain population is greater than in the general population, and studies report that up to 30% of TMD patients have major

TABLE 48-7. **Commonly Used Antidepressants**

Generic	Brand	Dosage (mg/day)	Side Effects
Amitriptyline	Elavil	10–300	High
Desipramine	Norpramin	50–300	Moderate
Doxepin	Sinequan	25–300	High
Imipramine	Tofranil	20–300	Moderate
Nortriptyline	Pamelor, Aventyl	25–150	Moderate
Fluoxetine	Prozac	5–20	Moderate

TABLE 48-8. **Antidepressant Medications**

Tricyclics antidepressants are most commonly used for chronic pain and depression.
MAOIs have many adverse reactions.
SSRIs (fluoxetine [Prozac]) may increase anxiety, bruxism.

MAOIs = monoamine oxidase inhibitors; SSRIs = selective serotonin reuptake inhibitors.

depression at the time of presentation for TMD treatment and up to 74% of patients with chronic TMD have had at least one episode of major depression.[26,27]

MAOIs are not routinely prescribed for TMD owing to the numerous side effects, drug interactions, and dietary restrictions. The benefits of TCAs have been well documented in chronic pain or depression populations and are probably due to analgesic and antidepressant actions. The analgesic properties are independent of the antidepressant effect, which requires higher doses, and it has been shown that low doses of amitriptyline (amitriptyline [Elavil] 10 mg) before sleep can have an analgesic effect on chronic pain but have no relationship to the antidepressant actions that require doses up to 20 times greater.[28] TCAs may also help treat nocturnal bruxism and any sleep disturbance associated with TMD,[29] and the side effects of TCAs are related to anticholinergic activity causing xerostomia, constipation, blurred vision, and urinary retention. SSRIs can also be used for treating the TMD patient with depression. These medications often need to be taken for several months and patients must be counseled appropriately; fluoxetine (Prozac) may, in fact, increase bruxism and anxiety and should be carefully monitored.

Muscle Relaxants

Centrally acting muscle relaxants (cyclobenzaprine, methocarbamol, and carisoprodol) may be used to relax a hyperactive musculature associated with TMD (Tables 48-9 to 48-11). These relaxants may also act as sedatives, and they are commonly combined with the use of NSAIDs. Cyclobenzaprine (Flexeril) has a similar chemical structure to the TCAs and, if given over an extended period of time, will produce antidepressant and sedative actions, as well as the anticholinergic side effects of the TCAs. Central muscle relaxants can be very effective for acute myofascial pain (e.g., secondary to trauma). Baclofen, a peripheral muscle relaxant, has been used in myofascial pain but is most appropriately reserved for severe muscle spasm or neurogenic pain. Botulinum toxin has been used to treat severe bruxism, and by providing muscle relaxation, inflammation of the masseter muscle and TMJ capsule may be reduced.[30,31]

Local Anesthetics

Local anesthetics act on the nerve cell membrane to prevent generation and conduction of impulses (Table 48-12). Local anesthetics can be used as diagnostic blocks intra-articularly and/or intramuscularly to alleviate pain and increase range of motion. For example, local anesthetic injection posterior to the maxillary tuberosity will anesthetize the lateral pterygoid muscles, which if in spasm, will allow maximal protrusion of the mandible without pain. A local anesthetic without vasoconstrictor should be used, because the decrease in blood flow may exacerbate muscular pain. The intrinsic vasodilatory effect of the local anesthesia may improve vascular tissue perfusion and, thereby, further alleviate pain. It has been shown that an intra-articular injection of mepivacaine, along with physiotherapy in patients with TMJ internal derangement (anterior disk displacement), results in pain relief and improved masticatory efficiency.[32]

Physical Therapy

Many factors contribute to limited range of mandibular motion, including muscular pain or spasm, anterior disk displacement (closed lock), pathologic lesions, traumatic injuries (e.g., mandible or zygoma fracture), and bony or fibrous

TABLE 48-9. **Commonly Used Muscle Relaxants**

Generic	Brand	Usual Dosage (mg/day; divided doses)
Carisoprodol	Rela, Soma	1000–1400
Chlorzoxazone	Paraflex, Parafon Forte D.S.C.	750–3000
Meprobamate	Miltown, Equanil	1200–1600
Methocarbamol	Robaxin	1500–4500
Cyclobenzaprine	Flexeril	5–30
Orphenadrine	Norflex, Disipal	150–300
Diazepam	Valium	2–40
Combination Fixed Dosage		
Meprobamate Aspirin	Equagesic	1–2 tablets three or four times daily
Orphenadrine Aspirin Caffeine	Norgesic	1–2 tablets three or four times daily

Adapted from Syrop SB. Pharmacological therapy. In Kaplan AS, Assael LA, editors. Temporomandibular Disorders—Diagnosis and Treatment. Philadelphia: WB Saunders; 1991; pp. 501–514.

TABLE 48-10. Central Muscle Relaxants

Central Muscle Relaxants
Carisoprodol (Rela, Soma)
Chlorzoxazone (Paraflex)
Methocarbamol (Robaxin)
Cyclobenzaprine (Flexeril)
Effects
Tranquilizing effects
General sedative effect on central nervous system
No specific neurotransmitter
No effect on skeletal muscle, motor endplate, or nerve fiber

TABLE 48-11. Peripheral Muscle Relaxants

Peripheral Muscle Relaxants
Baclofen (lioresal) derivative of GABA that blocks spinal cord contraction; reserved for severe muscle spasm, or neurogenic pain
Botulinum toxin (Botox) is useful for management of oromandibular dystonia
Effects
Block synaptic transmission at neuromuscular junction
Block muscle contraction

GABA = γ-aminobutyric acid.

ankylosis preventing rotatory and/or translational movements of the mandible. It is well known that prolonged jaw immobilization may have deleterious effects on both the TM joints and the facial muscles. Immobilization may cause degenerative cartilagenous and osseous changes to the articulating joint surfaces, significant synovial fluid alterations, and changes to the surrounding tissues. Prolonged reduced jaw motion results in rapid muscle fatigue, muscle weakness, and fibrous tissue contractures. The health and nourishment of the synovium depend upon normal joint function, and synovial fluid generation is reduced, or halted, when the TMJs are immobile. In addition, it has been observed that the synovial fluid of patients with TMD pain and limited jaw motion often contains a variety of inflammatory byproducts demonstrated by synovial fluid analysis (SFA). Kaneyama and colleagues[33] listed a variety of cytokines, such as interleukin (IL)-1β, tumor necrosis factor (TNF)-α, IL-6, and IL-8 in symptomatic TMJs, that were not observed in asymptomatic TMJs. This high level of cytokine activity in SFA is believed to be related to the underlying inflammatory pathogenesis of TMD, because cytokines such as IL-6 and IL-1β may induce the "inflammatory cascade." As a result of the release of selective proteinases, there may be destruction of articular cartilage and underlying osseous destruction. Each cytokine has unique properties that not only affect the surrounding tissues but also aid in the release of other cytokines.[33] As a result, the role of functional mobility on the synovium may be an indeterminate factor in the overall health of the TMJs (Table 48-13).

TABLE 48-12. Local Anesthetics

Act on nerve cell membrane to prevent generation and conduction of impulses.
Useful for diagnostic blocks.
Muscular injections to increase range of movement.

TABLE 48-13. Physical Therapy

Home Treatment Program (For Mild Acute Symptoms)
Soft diet
Jaw rest (decreased jaw function)
Heat and ice packs
Jaw/tongue posture opening exercises
Lateral jaw movement exercises
Controlled passive jaw motion exercises (Therabite)
Office Treatment (For Reduction of Pain and Inflammation)
Ultrasonography
TENS
Range of motion exercises
Soft tissue manipulation procedures
Trigger point injections
Acupuncture (± electric current or heat applied to acupuncture needles)

TENS = transcutaneous electrical nerve stimulation.

Exercise Therapy

Physical therapy and exercise are important components of any TMD program. Initial, mild, or acute symptoms can be managed with soft diet, jaw rest, alternating heat and ice packs, jaw/tongue posture opening exercises, lateral jaw movements, and passive stretching exercises. Once again, the precise sequence of exercise therapy is determined on an individual basis as well as the experience of the clinican or physical therapist and is usually based upon the chronicity of the problem, the degree of pain, and the magnitude of limitation of jaw function. Further reduction of pain and inflammation may require an office-based physical therapy program. Ultrasonography, transcutaneous electrical nerve stimulation (TENS), soft tissue manipulation, trigger point injections, iontophoresis, and acupuncture have also been advocated as effective in the management of the TMD patient.

Jaw exercise therapy can be described as passive, active, or isometric. *Passive jaw exercise* allows the patient to manually, or with a device such as Therabite Jaw Motion Rehabilitation System (Atos Medical, Milwaukee, WI), progressively increase interincisal opening (Figure 48-2). Passive jaw exercise has received a great deal of attention, and many authors have reported significant reduction in pain and improvement jaw mobility during the nonsurgical phase of TMD treatment, as well as for the postoperative care after surgical intervention (arthrocentesis, arthrocopsy, or open TMJ surgery).[34–37] Passive jaw exercise is also very effective for patients who experience muscular-associated trismus and myofascial pain and dysfunction (MPD). Jaw exercises should be used with caution or may be contraindicated in patients with severe TMJ disk displacement without reduction owing to the possibility of further damage to the disk or



FIGURE 48-2. Passive jaw exercise device. (TheraBite, Atos Medical, Milwaukee, WI.)

retrodiskal tissues, and the physical therapist should be made aware of any limitations to physiotherapy.

Active jaw exercises, which are initiated by the patient's jaw musculature as opposed to a jaw opening device that allows the muscles to be "passive," may be incorporated into a home therapy program for TMD. One regimen allows the patient to activate, for example, the suprahyoid muscles (geniohyoid, mylohyoid, digastric, and stylohyoid), thereby inactivating the elevators of the jaw (medial pterygoid, masseter, temporalis). This may allow for relaxation of hyperactive muscles of mastication and may assist in increasing maximal interincisal opening. In the active stretch phase, patients are advised to keep their mouth open for several seconds and then relax the muscles. They are instructed to open their mouth until just before they perceive pain, and then to hold for several seconds, and repeat this exercise several times a day, with a gradual increase in the magnitude of mouth opening. An active lateral stretch exercise, which permits the contralateral lateral pterygoid to be stretched, may be accomplished by visualizing the movements in a mirror. In the active protrusion exercise, also performed in front of the mirror, the mandible is protruded forward while stretching the lateral pterygoid muscles bilaterally. All active movements or excursions are maintained for several seconds, and then slowly released, in order to effect a physiologic "stretch" of the muscles.

Isometric exercises have been recommended for patients with severe pain and trismus, and there is no movement during isometrics while the depressor muscles are activated, allowing for relaxation of the opposing elevator musculature (medial pterygoid, masseter, and temporalis). These exercises are typically performed by holding the mandible in a stationary position as the muscles are activated isometrically. The lateral pterygoid muscles may also be exercised in a similar isometric fashion.

Mongini[38] describes a three-stage office technique for mandibular manipulation in patients with pain, decreased mobility, and disk displacement without reduction. Right and left lateral movements are actively initiated by the patient. The patient continues the movement while the clinician applies light pressure in the same direction, and in the last stage of the technique, the mandible is moved to the opposite side with patient assistance.[38] Kurita and associates[39] described a technique of placing one thumb on the last molar on the affected side while the other hand supports the head in the temporal region. The mandible is then moved downward and forward, while the patient is instructed to protrude and move the jaw laterally, and open the mouth while the clinician manipulates the jaw. After this movement, the mandible is manipulated posteriorly so that the condyle is positioned in a posterosuperior position within the glenoid fossa. Only 18% of the patients received significant benefit from the manipulation, and the more advanced the disk displacement the lower the success of the treatment.[39] Yuasa and Kurita[40] suggested that physical therapy, along with administration of NSAIDs (for a 4-wk period), is a more effective way to treat TMJ disk displacement without associated osseous changes. Nevertheless, there is no shortage of recommended jaw exercises, and care must be taken to first do no harm (Table 48-14).

Thermal Agents

Thermal treatment modalities are often incorporated into the management of the patient with TMD. The use of both cold and heat can alleviate muscle pain and play an equal role during jaw stretching and muscle strengthening exercises.[41–43] Heat therapy has been reported to reduce muscle pain by increasing nerve conduction velocity and causing local vasodilation and increased blood flow, thereby clearing out local inflammatory mediators.[43] Superficial heat therapy can be implemented with conductive (gel-type heat packs, paraffin, whirlpool) or radiant (infrared) modalities. The most common types of heat used include a moist hot washcloth, heating pad, or hydrocollator, a pad filled with clay and heated in a water bath to 70°C to 88°C, or a commercially available gel pack that can be used in either a hot or a cold mode. The heat pack chosen is placed on the site (e.g., over the masseter muscle) for 15 to 20 minutes, causing a transient rise in skin temperature to approximately 42°C. The use of a moist, as opposed to dry heat, heating pad (gel pack)

TABLE 48-14. **Manual Physical Therapy**

Soft tissue techniques.
Massage, relaxation, stimulation, release of adhesions, muscle stretching.
Manipulative therapy, cervical spine realignment (chiropractics).
Rapid, passive, short duration, and amplitude thrusting forces to position the TMJ beyond its normal limits.
Patient has no control over treatments.
Pain relief is immediate, but transient.

TMJ = temporomandibular joint.

appears to be an effective modality of treatment for myofascial pain associated with TMD.[44]

Cryotherapy is often used by physical therapists as an aid in muscle stretching exercises in an attempt to increase jaw opening that is limited by muscular pain.[41] The pain perception model described by Melzack and Wall[43] explains why cold therapy stimulates the small myelinated A-delta fibers responsible for thermal discrimination and inhibites pain perception, which is mediated by the small unmyelinated C fibers. A physical therapist typically applies refrigerant sprays superficially on the skin surface in a sweeping motion followed by active stretching of the jaw musculature. Cold therapy should be used with caution because of the potential for increased joint stiffness, muscle contracture, and decreased jaw mobility. Cold can also have analgesic effects when applied immediately after a therapeutic exercise regimen. Ice wrapped in a towel, fluoromethane spray, and reusable ice packs can all be used to deliver cryotherapy to the TMJ and related facial and neck muscles. The "stretch and spray technique," initially described by Modell and Simons,[42] and later modified by Travell and Simons,[41] remains a mainstay of office physiotherapy. The therapist holds the fluoromethane spray approximately 30 to 45 cm from the patient and sprays in a sweeping motion multiple times; this is then followed by active jaw stretching exercises. Possible side effects include superficial skin frostbite and, as mentioned, the potential for increased joint stiffness limiting the jaw exercise regimen. Many physical therapists follow the cryotherapy sprays with moist heat to prevent the jaw muscles from undesirable contracture.

Ultrasonograpy and Phonophoresis

Deep heat may also be delivered by ultrasonography or phonophoresis. The ultrasound machine operates above audible frequency sound waves (0.75–1.0 MHz), which convert to heat while traveling through the soft tissues. The ultrasound probe is applied to the skin using an acoustic conductive gel, then moved slowly over the affected area in small, circular movements. The operator must be careful not to keep the machine in one place for too long as it may cause overheating of the connective tissue, causing structural damage. The deep heat is intended to increase blood perfusion to the area via vasodilation, clearance of inflammatory mediators from the area, decreasing pain and increasing jaw mobility.[45] Reported effects of ultrasound therapy include altered cell membrane permeability, intracellular fluid absorption, decreased collagen viscosity, vasodilation, and analgesia. The beneficial effects to joints include reduced capsular contracture, breakdown of local calcium deposits, and decreased hyaluronic acid viscosity of the joint synovial fluid with improved mobility.[46] Because ultrasonography delivers heat to the deeper soft tissues, it may have some advantages over the superificial heat delivery techniques mentioned previously in managing tendinitis, capsulitis, muscle spasm, and tight, or restricted, ligaments.

Phonophoresis is an application of ultrasound heat therapy that incorporates a pad filled with a steroid or anesthetic cream that is placed over the affected area. As the ultrasound waves are applied, the medications perfuse into the tissues. The most common indication for phonophoresis is synovitis associated with painful jaw hypomobility. Contraindications for the use of ultrasonography and phonophoresis include areas that may have a reduced blood circulation, fluid-filled organs, eyes, radiation therapy sites, and malignant tissues, and ultrasound therapy should be used with caution over active bone growth centers.[47]

Electrical Stimulation (TENS)

TENS has become a viable home therapy option in the management of TMD. The precise mechanism of action is unknown, although it has been suggested that the gate control theory, counterirritation, neurohumoral substance release, and peripheral blockade mechanisms are all involved.[48] TENS uses a low-voltage electrical current that is designed for sensory counterstimulation in painful disorders. It is used in an attempt to decrease muscle pain and hyperactivity and for neuromuscular reeducation.[49,50] TENS units are small and portable, and the electrodes are placed along specific dermatomes or over acupuncture and trigger point sites. The patient can control the settings with variable frequency, amplitude, waveform, width, and pulse modes. Treatment may last for several hours and relies on biofeedback from the patient in the control of the specific delivery of the stimulation. TENS emits an asymmetrical biphasic wave of 100- to 500-msec pulse. The efficacy of TENS for analgesia and muscle relaxation in myofascial pain has been well documented.[51] Electrode placement is contraindicated over the carotid sinus, transcranially, directly over the cervical spine, on a pregnant abdomen, or on patients with demand-type pacemakers.[52]

High-Voltage Stimulation

High-voltage stimulation units deliver currents of positive and negative polarity with voltages greater than 100 V, which may be administered in a constant, or intermittent, pattern. The positive polarity produces vasoconstriction, whereas the negative polarity produces vasodilation. The positive polarity reduces nerve irritability, and negative polarity enhances nerve conduction and also softens the affected soft tissues. thereby decreasing muscle tension or spasm. Treatment with high-voltage stimulation has been shown to improve jaw mobility and relieve pain intensity in TMD patients.[53] It can also be used for pain relief, reduction of edema, and neuromuscular stimulation.[53]

Iontophoresis

Iontophoresis and phonophoresis are techniques that are capable of enhancing drug penetration through the skin. As mentioned previously, phonophoresis uses ultrasonic waves to transmit molecules of drug through the skin, whereas iontophoresis uses low-level electric current. Iontophoresis transfers ions from a solution through intact skin by passing a direct current between two electrodes.[54] Positive ions are transmitted at the cathode, and negative ions are transmitted at the anode. Examples of negatively ionizing drugs are

TABLE 48-15. **Electrical Stimulation Techniques**

TENS.
Phontophoresis: ultrasound heat drives drugs (steroids, anesthetic crème) into tissues.
Iontophoresis: electric current drives drugs into tissues.
High-volume stimulation (100 V): pumping effects of muscle contraction increase circulation.

TENS = transcutaneous electrical nerve stimulation.

dexamethasone and methylprednisolone. Other drugs used in iontophoresis include lidocaine and salicylates. Iontophoresis was introduced in treating TMD and postherpetic neuralgia in 1982.[55] It appears to be most effective against inflammation, muscle spasm, and calcium deposits. The deep penetration of the medication also aids in the treatment of severe joint inflammation and pain (Table 48-15).

Trigger Point and Muscle Injections

A trigger point is an area of hyperirritability in a tissue that, when compressed, is locally tender and hypersensitive and results in referred pain and tenderness.[56] The development of trigger points may be due to trauma, sustained contraction or spasm, or acute muscle strain. When a needle penetrates this area, it may cause a twitch response and referred pain.[56] Injection of local anesthetic agents without epinephrine may cause a temporary anesthesia, which enables the clinician to stretch the muscles maximally in the affected area. A vasodilator effect of the local anesthetic may improve vascular perfusion to the area, thereby allowing harmful metabolites or inflammatory mediators, which may induce pain, to be more readily removed by the increased blood flow to the area.

Stress-Reduction Techniques

Relaxation and Biofeedback

Relaxation and stress-reduction techniques for patients with TMD can be very effective treatment modalities. A wide variety of techniques exist, and these typically involve a coordinated sequence of skeletal muscle contracture and release, beginning with the feet and proceeding cranially toward the head and neck region. Patients may also use audiotapes that attempt to train certain synchronized breathing patterns as well as specific relaxation techniques. Biofeedback techniques may incorporate the use of electromyography (EMG) and skin temperature to measure the patient's physiologic function and response to treatment. This information is then conveyed back to the patient by a meter or sound, so that the patient can gauge the level of relaxation, adjust the level of relaxation, and measure progress accordingly.[57] The aim is to achieve psychological self-regulation and to monitor the relationship between muscular tension and pain. In a review of the literature, Crider and Glaros[58] reported 69% of subjects rated their symptoms as improved, or symptom-free, after biofeedback and relaxation treatments, whereas only 35% of patients receiving placebo intervention showed improvement. Furthermore, on follow-up examination, the patients showed no decline from immediate post-treatment levels of improvement.[58] Scott and Gregg[59] advocate that relaxation techniques and EMG feedback can yield good results, especially in patients who have no clinical depression and have had TMJ pain for only a short period of time. The major limitation of the use of these techniques is the difficulty required to motivate patients with pain to engage in jaw exercise regimens (Table 48-16).

TABLE 48-16. **Behavioral Therapy**

Components of Behavioral Therapy
Patient education in recognition of stress, anxiety, and depression
Relaxation training programs
Biofeedback exercises
Self-hypnosis techniques
Meditation techniques
Cognitive therapy programs
Types of Behavioral Therapy
Psychiatric therapy
Pain clinic management

Acupressure and Acupuncture

Acupressure and acupuncture are alternative treatment options that may be implemented along with other treatment modalities during the course of nonsurgical treatment for TMD. Acupuncture uses the relationship between energy flow through meridians, natural elements, and positive and negative life forces. Fine needles are used to reestablish proper energy flow in these areas. There are several theories regarding the mechanism of action of acupuncture and acupressure. The first proposed mechanism is the gate control theory, which states that the needle produces a painless stimulation, causing neuronal gates to close and prevent signal propagation of pain to the spinal cord.[60] Other explanations include the release of endorphins from the pituitary gland, which blocks pain sensation, promotion of alpha waves (associated with stress reduction and relaxation), and rebalancing the electric ion flow pattern, which when disrupted, may elicit pain.[60] There are several different acupressure techniques including Jin Shin (two acupressure points held for 30 sec–5 min), Shiatsu (more rapid techqniue, held for 3–10 sec), reflexology (acupressure on feet, hands, and ears corresponding to areas of the body), Do-In (self-acupressure and breathing exercises), and G-Jo (acupressure for first aid purposes). Some studies have reported favorable results when these techniques are combined with other modalities (e.g., occlusal splint therapy), but overall data are limited,[61,62] and therefore, these alternative therapies may be offered as an adjunct to more conventional well-established therapeutic options for TMD management.

Psychotherapy

In some cases, TMD may be the somatic expression of an underlying psychiatric or psychological disorder such as

depression or conversion syndrome.[63,64] The clinician should screen for a personal or familial history of psychiatric disease, physical or sexual abuse, and substance abuse. Anxiety disorders occur much more commonly in patients with chronic pain syndromes.[65] Once a psychiatric component is identified, patients should be referred for psychiatric consultation for adjunctive treatment of these contributing factors. Psychological treatments may include behavioral therapy, cognitive-behavioral therapy, and self-management/support groups. Psychiatric treatments may also include pharmacotherapy along with behavioral therapy. In many cases, the TMD patient may be referred to specialized pain centers for assessment and management, especially when long-term pharmacologic therapy is indicated or when other nonsurgical methods have failed to improve the symptoms and the patient is not a surgical candidate.

Occlusal Appliance Therapy

An occlusal bite appliance is a removable device, usually made of hard acrylic, which is custom fit over the occlusal surfaces of the mandibular or, more commonly, the maxillary teeth. The splint is constructed so that there is bilateral symmetrical posterior occlusal contacts with the teeth of the opposing arch in centric occlusion (flat plane splint), or there may be anterior contacts in lateral and protrusive excursions of the mandible (anterior repositioning splint). The physiologic basis of treatment is not well understood, but the effectiveness of the occlusal splint has been attributed to a decreased loading on the TMJs and reduction of the neuromuscular reflex activitivation. Alleviation of bruxism and MPD may be due to the change in the vertical dimension of occlusion that alters proprioception in the postural position of the mandible.[66–68] There are generally two types of appliances: stabilization, or flat-plane devices, and anterior repositioning splints.

Stabilization (Flat-Plane) Appliance

A stabilization appliance covers all the teeth in one arch, typically the maxilla, and is indicated to relax the muscles of mastication, aid in joint stability, and protect teeth from bruxism (Table 48-17 and Figures 48-3 and 48-4).[68,69] Additional indications for stabilization appliances may include myalgia, inflammation, and retrodiskitis secondary to trauma. Commonly, stabilization devices are used for several weeks after arthrocentesis and arthroscopy procedures and after open joint surgery as well. With a stabilization appliance, the condyles are repositioned into the most muscularly stable position, while the teeth have even symmetrical bilateral simultaneous contacts.[70] There must be bilateral equal posterior contacts so that an environment of stable physiologic posture is achievable. Canine guidance may be created during protrusive and lateral excursions, or there may be anterior disocclusion during these jaw movements, depending upon the prexisting occlusal realtionships. As the patient's symptoms improve, the bite splint should be continually adjusted to maintain even contacts bilaterally. Many practicioners often prefer the splint to be fabricated on the maxillary arch because it covers more enamel area, especially with class II or class III patients in whom fabrication of a mandibular appliance can be difficult. The major advantages of a mandibular stabilization appliance, over a maxillary splint, are better speech and less visibility, which may contribute to improved patient compliance with the therapy.[70] The appliance should be worn up to 24 hr/day and may or may not be taken out at mealtimes, because this is the time when masticatory forces and TMJ stresses are at the highest levels. In general, stabilization appliances

TABLE 48-17. **Occlusal Appliances: Stabilization Appliance**

Hard acrylic flat-plane device
Full coverage maxillary occlusal arch
Worn 24 hr/day (± during meals)
Bilateral even distribution of occlusal forces
Provides stabilization of the TMJ apparatus
Relaxation of masticatory muscles

TMJ = temporomandibular joint.

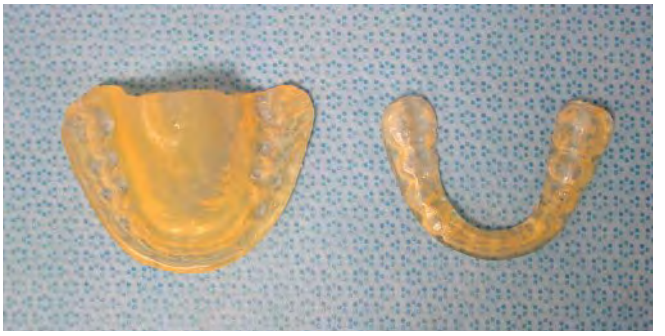


FIGURE 48-3. Stabilization appliance. Hard acrylic full-coverage occlusal splint.



FIGURE 48-4. Maxillary hard acrylic stabilization appliance does not alter anterior/posterior jaw position.

should be weaned as soon as possible after symptoms improve, or after arthrocentesis or arthroscopy procedures, in order to prevent dental movements and the development of iatrogenic malocclusions.

Major and Nebbe[71] reported effective reduction in headaches and muscle pain using stabilization appliances, but occlusal stabilization appliances have limited value in reducing joint pain. Lundh and coworkers[72] concurred that the stabilization splints have little value in relieving painful disk displacement without reduction. Kai and colleagues[73] reported that after treatment with a stabilization occlusal splint on the maxillary arch, clinical signs and symptoms of nonreducing anteriorly displaced TMJ disks decreased but osteoarthritic radiographic findings increased.

Anterior Repositioning Appliance

The anterior repositioning splint is an interocclusal appliance that guides the mandible into a more anterior position than usual during mandibular protrusion (Table 48-18 and Figures 48-5 and 48-6). The purpose of these appliances is to alter the structural condyle-disk-fossa relationship in an effort to decrease TMJ loading.[74] Indications for this device are primarily anatomic internal disk derangement disorders. The maxillary appliance is preferred, and it is fabricated with a guide ramp that permits gradual anterior repositioning of the mandible.[73] Anterior repositioning appliances are used less frequently because repositioning of the mandible over a period of time can result in irreversible occlusal changes such as posterior open bites, which could require extensive dental restorative and prosthetic rehabilitation.

Occlusal Adjustment

There is a limited role and lack of supporting evidence for occlusal adjustment or selective enamel grinding in the treatment of TMD.[75] The purpose of selective occlusal adjustment is to permanently reposition the dentition into a better occlusal relationship. This is an irreversible process and is most appropriately utilized for acute TMD symptoms that arise from recent dental treatment resulting in overcontoured dental restorations or after orthognathic surgery with isolated areas of occlusal interferences. In these select cases, occlusal equilibration may allow for proper condylar positioning and prevents reflexive muscular problems associated with improper dental interferences.

TABLE 48-18. **Occlusal Appliances: Anterior Repositioning Appliance**

Hard acrylic device.
Maxillary or mandibular occlual coverage splint with anterior inclined plane.
During jaw closure, mandible moves anteriorly.
Useful for anteriorly displaced disk to change condylar position and recapture disk.
Possible need for occlusal equilibration and splint readjustment.

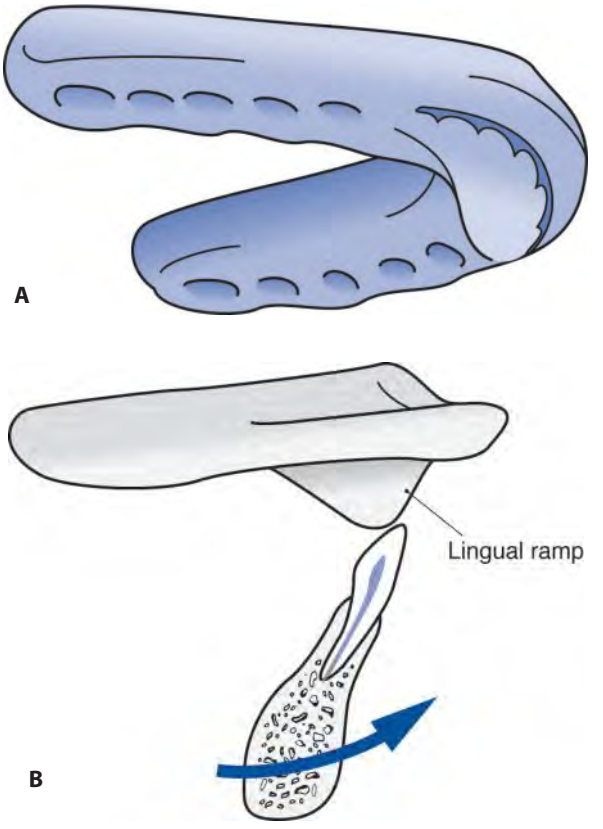


FIGURE 48-5. **A,** Maxillary repositioning appliance. **B,** Lingual ramp engages the mandibular incisors to reposition the lower jaw anteriorly.

Additional Strategies

As mentioned, the concept that nonsurgical therapy is employed solely as an alternative to surgical intervention is flawed, and more accurately, the techniques described should be used whether surgical intevention is indicated or not, in an attempt to resolve the signs and symptoms of TMD. Two clinically relevant examples help to illustrate this philosophy



FIGURE 48-6. Mandibular hard acrylic repositioning appliance may alter anterior/posterior jaw position.

of treatment: the use of preemptive analgesia and the prevention of ectopic or heterotopic bone formation.

Preemptive Analgesia

Effective postsurgical pain control begins before the surgery itself, because it is recognized that methods that limit CNS neuronal activation by surgical stimulation may significantly reduce pain in the postsurgical period and may also reduce the possibility of development of some persistent pain syndromes. The term *preemptive analgesia* is used to describe methods that apparently reduce postsurgical pain by protecting the CNS from surgical stimulation. The concept is based upon observations that collectively indicate that nociceptive processing is a highly dynamic process. Nociception, and subsequent pathway sensitization, likely involves de novo protein synthesis and establishment of novel connections made by neurons in the affected pathway.[78–80] The old hypothesis that these pathways are merely static conductors of neural signals generated by noxious stimuli appears to be invalid because we know now that gene transcription is induced in stimulated neuronal populations.[81,82] Some neuropeptides that are translated from these genes may facilitate future neural activities by receptive field expansion or by facilitation of specific interneuronal interactions. The term *neuroplasticity* is often used to refer to the dynamic state of stimulated neural pathways. These, and perhaps other, more ominous changes (i.e., neuronal death from excessive stimulation), may be fundamental to the development of some chronic pain states. Fortunately, these CNS responses may be significantly attenuated by preemptive analgesic techniques.[79,83] From animal and clinical studies, there is evidence that protracted neural responses to painful stimuli can be modified or prevented by the following: (1) neural blockade with local anesthetics,[84–86] (2) administration of opioids,[87–91] (3) administration of *N*-methyl-D-aspartate receptor antagonists (e.g., MK801, ketamine, dextrophan),[92–94] or (4) administration of ketorolac, both a peripheral and a central-acting nonopioid analgesic.[95–97] These agents must be administered *before* noxious stimulation to prevent CNS changes that may be related to the development of postsurgical pain and perhaps persistent pain. It is interesting to note that a general anesthetic state does not prevent neuroplastic changes induced by surgical stimulation, unless the general anesthetic technique employs high-dose opioids or ketamine. Neural impulses from surgical stimulation apparently reach the CNS, evoking sensitization, despite the fact that overt signs of surgical stimulation (e.g., patient movement, elevated heart rate and systemic blood pressure) are blocked by general anesthesia. This stimulus-dependent neural sensitization, characterized by receptive field expansion and the "wind-up" phenomenon,[78,79] has been attributed to postsurgical hyperesthesia and pain. When regional anesthesia is employed as an adjunct to general anesthesia, there is strong evidence that postoperative pain is significantly reduced, and this is consistent with current models of central sensitization and neuroplasticity. However, to ensure that neural activities induced by surgical stimulation are fully blocked, the surgeon must administer regional anesthesia *before* surgical stimulation. If necessary during long procedures, the surgeon should reanesthetize the operative field, and not rely solely on a general anesthetic state for CNS protection. Jebeles and associates[98] studied the effects of preemptive regional anesthesia on postsurgical discomfort associated with tonsillectomy and adenoidectomy performed under general anesthesia. Twenty-two children were given either bupivacaine or saline infiltrations in the peritonsillar regions before surgical stimulation. For this study, postsurgical analgesics were standardized for all subjects and postsurgical pain was assessed over a 10-day period by three dependent measures: constant pain, pain evoked by swallowing a standard volume of water, and the time required to drink 100 mol of water based on the assumption that the rate-limiting factor for this activity is throat pain. All three dependent measures confirmed that subjects given bupivacaine regional anesthesia with general anesthesia experienced significantly less pain over a 10-day postsurgical period than the saline-injected group.[22] Other studies have provided similar evidence that regional anesthesia, provided before surgical stimulation, can significantly reduce pain after craniotomy and bone harvesting from the iliac crest.[99,100] To date, no research has documented the efficacy of preemptive regional anesthesia on postoperative pain, or on the subsequent development of chronic pain, in the operated patient with TMD. Nevertheless, existing evidence strongly suggests that the use of regional anesthesia as an adjunct to general anesthesia will significantly reduce postsurgical pain in this group of patients. Opioids administered *before* surgical stimulation may also reduce postsurgical hyperesthesia believed to be due to central sensitization. For example, isoflurane or isoflurane, and nitrous oxide, administered in a concentration sufficient to suppress cardiovascular responses to surgical stimuli (i.e., minimum alveolar concentration that blocks adrenergic responses [MACBAR]), do not inhibit formalin-induced hyperesthesia.[101] Formalin injected intradermally provides a potent noxious stimulus that results initially in a volley of neural activity that sensitizes central nociceptive neurons in the pathway. This observation reinforces previous studies indicating that general anesthesia alone does not offer protection against central sensitization. However, morphine administered *before* formalin injection significantly reduces postinjection hyperesthesia in this model.[101] In fact, a significant reduction in formalin-induced hyperesthesia was also observed even if morphine was reversed by naloxone shortly after the formalin injection was administered, indicating that even a brief exposure to an opioid before noxious stimulation is sufficient to prevent or significantly reduce stimulation-induced hyperesthesia.[101] Likewise, alfentanil reduces capsaicin-induced hyperalgesia in human subjects, but only if this agent is administered before capsaicin administration.[102] Capsaicin is a vanilloid extracted from chili peppers that is known to selectively stimulate C-fiber neurons expressing the vanilloid receptor (VR-1). Stimulation of C-fiber neurons by capsaicin

administration produces receptive field expansion and hyperesthesia by mechanisms that involve central sensitization.[79] Notably, alfentanil did not effectively reduce pain scores, flare response, or secondary hyperalgesia when administered *after* an intradermal injection of capsaicin in these human studies.[102] These studies highlight the fact that as with regional anesthesia, opioids are only optimally effective as modulators of central sensitization and subsequent hyperalgesia if they are administered before surgical stimulation.

Ketorolac is a peripherally and centrally acting nonopioid analgesic that is also an effective preemptive analgesic.[95–97] In a randomized, double-blind trial involving 48 patients undergoing ankle fracture surgery, ketorolac 30 mg administered parenterally before surgical stimulation significantly reduced postsurgical pain relative to the same amount of ketorolac administered after surgical stimulation.[96]

Preemptive analgesia and persistent pain Some preemptive analgesia techniques significantly reduce postsurgical pain, and there is evidence that preemptive analgesia may provide some protection against the development of some chronic pain states. Bach and coworkers[103] reported one of the few investigations designed to assess the impact of preemptive analgesia on the evolution of a chronic pain state. In this clinical study, 25 elderly patients scheduled for a below-the-knee amputation received either treatment with epidural bupivacaine and/or morphine to produce a pain-free state for 3 days before surgery or no pretreatment (control group). All patients subsequently underwent amputation under spinal anesthesia. After 6 months, none of the patients assigned to the presurgery analgesia group experienced phantom limb pain.[103] However, 38% of the subjects who did not receive the presurgical pain treatment had phantom limb pain at the 6-month postamputation period. Furthermore, 27% of these subjects experienced persistent phantom limb pain at the 1-year follow-up period.[103] Based upon this data regarding preemptive analgesia, it is recommended that when feasible, an opioid-based or opioid-supplemented general anesthetic technique be employed for TMJ surgery. Also, a regional anesthetic should be administered to cover the entire surgical field *before* surgical stimulation. During prolonged surgical procedures, the surgical site should be reanesthetized periodically. If there are no contraindications (e.g., bleeding concerns), some consideration can also be given toward ketorolac administration (i.e., 0.5 mg/kg or 30 mg intravenously) *before* surgical stimulation. Finally, postoperative pain should also be well controlled with a combination of regional anesthesia and opioid or ketorolac analgesia in the postoperative period, because these regimens may significantly reduce postsurgical pain, facilitating a shortened convalescence period, and may also protect the patient from CNS responses to surgical stimulation that may be involved in the genesis of some chronic pain syndromes.

Nonsurgical Therapy for TMJ

Periarticular Ectopic Bone Formation

Periarticular ectopic bone formation is viewed as a significant postsurgical complication with a negative impact on functional outcomes in some patients with end-stage TMD. Ectopic bone may form in adjacent native periarticular tissues or in the vicinity of alloplastic materials used to reconstruct the TMJ. In either instance, periarticular ectopic bone formation is viewed as a pathologic entity because it typically restricts normal joint movement and may contribute to chronic facial pain. The pathogenesis of periarticular ectopic bone formation is poorly understood. It has been suggested that displaced osteogenic precursor cells are stimulated to form ectopic bone by inflammatory mediators formed in response to surgical insult.[104] Alternatively, osteoinductive molecules (e.g., bone morphogenetic proteins) may be dispersed into periarticular tissues during surgery, resulting in the stimulation of local pluripotent cells and subsequent ectopic bone synthesis. In addition, other factors, such as genetic influences, sex hormones, systemic disease (e.g., ankylosing spondylitis, Paget's disease), or other local conditions, could also contribute to the formation of ectopic bone in periarticular tissues of the TMJ. Two strategies have been employed to attempt to prevent or minimize periarticular ectopic bone formation in either orthopedic surgery patients or those with TMJ disease. These include low-dose radiation therapy and NSAID therapy.

Low-dose radiation therapy Some clinicians have advocated the use of low-dose radiation to prevent or minimize postsurgical fibro-osseous ankylosis of the TMJ.[105–108] This approach is based on an earlier report indicating that low-dose radiation may be effective at preventing the formation of ectopic bone after hip arthroplasty.[109] For prevention of periarticular ectopic bone formation of the TMJ, fractionated total doses of 10 to 20 Gy have been used. One study reported that 10-Gy dosing was as effective as higher-dose (50-Gy) regimens.[106] Timing appears to be critical for optimum results from low-dose radiation therapy, because it is believed that low-dose radiation therapy elicits its effect on ectopic bone formation by prohibiting the proliferation of pluripotent cells that are precursors to osteoblasts. Therefore, it is recommended that low-dose radiation therapy be initiated within 4 days of surgery to provide optimum suppression of ectopic bone formation. Although there is some concern that early postsurgical radiation may have a detrimental impact on wound healing, one study of the efficacy of a single dose of 600 cGy administered between postsurgical days 2 and 4 (mean, 3.2 days), radiation did not appear to significantly affect wound healing after hip arthroplasty.[110] Radiation therapy was used to prevent ectopic bone formation in the periarticular region of the TMJ in a 53-year-old man[107] who sustained mandible fractures in an automobile accident and subsequently underwent five operations to correct TMJ ankylosis suffered as a complication of his injury. Over an 18-year period, the patient suffered from significant limitation of jaw movement, with reported maximum interincisal movements as low as 6 mm. After his final TMJ arthroplasty, the patient underwent fractionated cobalt radiation therapy consisting of 10 sessions beginning on the first postoperative day, for a total radiation

dose of 20 Gy in equal fractions, and at 3-year follow-up, the patient had sustained a maximum interincisal distance of 25.5 mm.[107]

Schwartz and Kagan[108] provided a report describing a similar beneficial effect of fractionated radiation (i.e., 20 Gy in 10 fractions) in a 51-year-old man who experienced a zygomatic-coronoid ankylosis after a depressed fracture of the zygomatic arch. This condition was surgically treated with a 5-mm gap arthroplasty with placement of an intervening sheet of silicone rubber, and postsurgical radiation was initiated 1 week after the operation. At a 19-month follow-up, the patient exhibited a 40-mm maximum interincisal distance, and although the patient initially complained of xerostomia and some loss of facial hair, there was no significant morbidity from this treatment. Experiences with 10 patients suffering from bony ankylosis of the TMJ was reported by Durr and colleagues,[106] consisting of 4 men and 6 women (median age 32.5 yr, range 14–59 yr) with a previous history of TMJ ankylosis who underwent TMJ arthroplasties and immediate postsurgical (within 1–3 days) radiation therapy consisting of 10 to 11.2 Gy in five fractions over a 5-day period. The median follow-up was 19 months (range 7–31 mo). Only 3 patients (30%) were followed for more than 2 years postoperatively, and 40% of these patients experienced some recurrence of ectopic bone formation assessed radiographically. A parotitis was identified in 30% of the patients; however, the radiation therapy did not appear to interfere with healing, and there were no other reported complications.

Reid and Cooke[105] have reported the largest case series to date involving postoperative radiation therapy to manage ectopic bone formation of the TMJ in 14 patients with histories of multiple TMJ surgeries. Each patient underwent TMJ arthroplasty with total joint reconstruction using an alloplastic prosthesis. The majority of these patients received a fractionated 10-Gy radiation dose beginning on the first postoperative day. However, some patients treated early in the series received a fractionated 20-Gy radiation dose. There was a mean follow-up of 4.2 years (range 1–9.6 yr), and the recurrence rate for ectopic bone formation at the 1-year follow-up was 21%. However, long-term follow-up revealed ectopic bone formation in 75% of the patients seen at 5 years and 100% (n = 2) of patients examined at the 9-year follow-up (Figure 48-7). Consistent with earlier reports, no significant persistent side effects of radiation therapy were noted.

A major concern with the use of low-dose radiation for the treatment of ectopic bone formation in the TMJ area is the potential for induction of neoplasia. Despite the fact that there have been no reported cases of malignant transformation associated with low-dose radiation therapy used to manage periarticular ectopic bone formation in the TMJ region, this concern seems justified based on a report by Ron and associates,[111] who examined 10,834 patients who had undergone low-dose radiation therapy for the treatment of ringworm infection of the scalp (tinea capitis). All irradiated subjects received treatment (mean radiation dose of 1.5 Gy) before the age of 16 years. Control subjects included 10,834

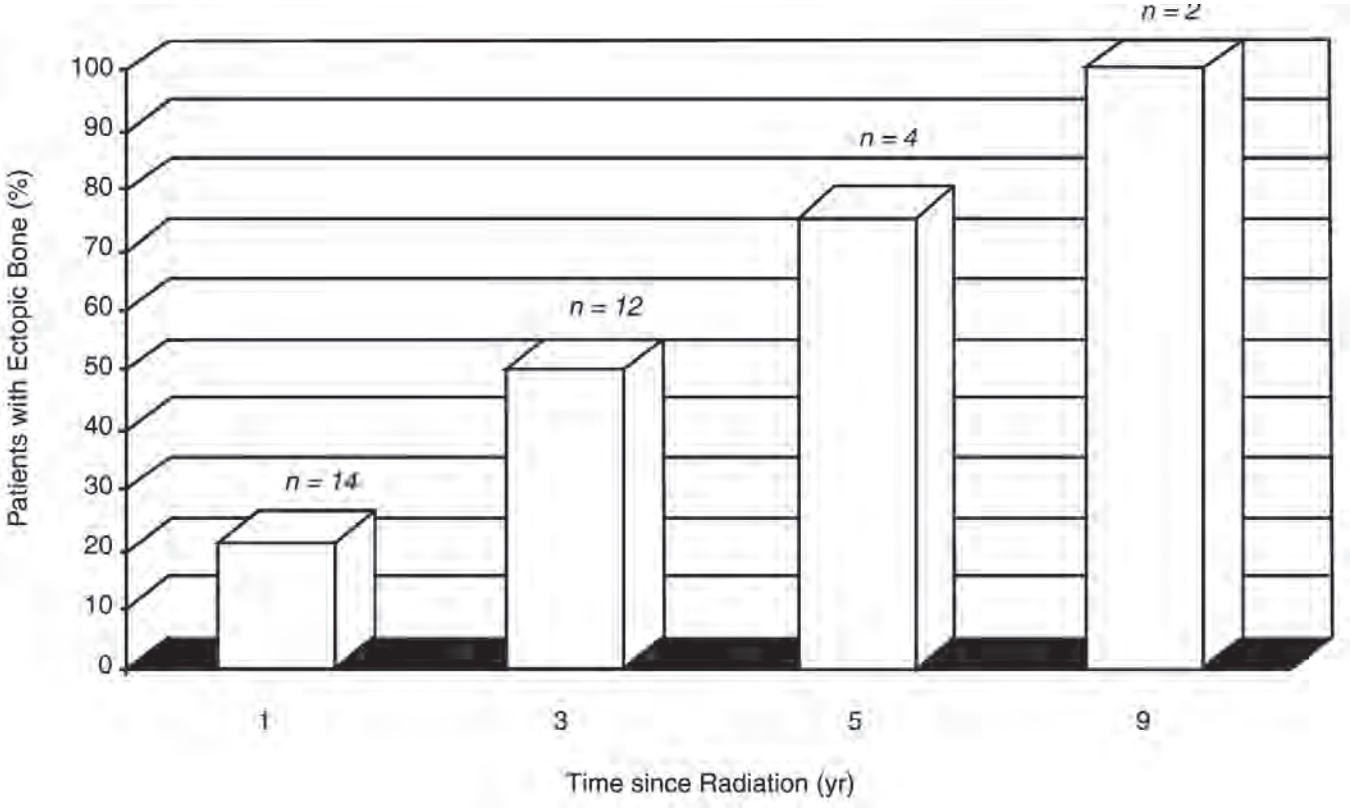


FIGURE 48-7. Recurrence of ectopic bone in patients treated with low-dose radiation. Data adapted from Reid R and Cooke H.[135]

nonirradiated age- and sex-matched individuals not related to the radiated subjects, serving as a general population control, and 5392 nonirradiated siblings of the subjects. The subjects were monitored for up to 33 years for the development of benign and malignant neural tumors. Tumors developed in 73 individuals, 60 among irradiated subjects, 8 in the general population control group, and 5 among siblings of irradiated subjects. Overall, there was a sevenfold increase in neoplasms of the nervous system in individuals who had undergone low-dose radiation therapy. Twenty-four malignant neoplasms were identified in this study, with 18 occurring in irradiated patients, 4 in the general population control group, and 2 in siblings of irradiated subjects. There was a 4.5-fold increase in the incidence of malignant neoplasms in patients who underwent low-dose radiation therapy relative to the control groups. The cumulative risk of developing a neural tumor over a 33-year period was significantly higher in the irradiated group (0.84% ± 0.16%) than in the controls (0.09% ± 0.03%). It should be noted that there was a prolonged latency of tumor occurrence (mean 17.6 yr after radiation exposure). From this study, it appears that the risk of radiation-associated tumors was highest between 15 and 24 years postradiation. This is significant in light of the fact that the longest published follow-up for any case series reporting the effects low-dose radiation therapy for management of ectopic bone formation in the TMJ area is 9 years (only 2 patients).[105] It should be noted that patients receiving radiation therapy for tinea capitis in this study were significantly younger (<16 yr of age) than the TMJ patients reported in the other case series studies; therefore, it is possible that the risk of radiation-associated neoplasia may be age-dependent. However, this assumption has not yet been validated in appropriately designed clinical studies.

In summary, the evidence supporting the use of low-dose radiation therapy to prevent or minimize ectopic bone formation in periarticular regions of the TMJ is supplied by case summaries that report beneficial effects with 10- to 20-Gy exposures in fractionated doses initiated within 4 days of surgery. However, it should be recognized that definitive studies, appropriately blinded and controlled, have not been reported and that in the absence of these studies, the true efficacy of this therapy remains unknown. Furthermore, there is evidence that such therapy may pose significant long-term health risks as well as the uncommon possibility of induction of neoplasia.[141]

NSAID therapy Several studies have provided evidence that NSAIDs may be effective retardants of ectopic bone formation.[110,112–115] The mechanism(s) by which these drugs elicit this effect is currently unknown; however, the effect is believed to be secondary to the ability of these drugs to inhibit prostanoid, and perhaps leukotriene, synthesis associated with normal inflammatory responses to injury. Kienapfel and coworkers[110] compared the effects of indomethacin, a nonselective COX inhibitor (also capable of inhibiting lipoxygenase), with those of low-dose radiation therapy employed to prevent ectopic bone formation after hip arthroplasty. For this study, 154 patients scheduled for hip arthroplasty to treat various degenerative arthritides were randomly assigned to one of three groups: (1) low-dose radiation treatment (600 cGy administered as a single dose between postoperative days 2 and 4); (2) indomethacin treatment of 50 mg administered orally twice daily beginning on postoperative day 1 and continuing until postoperative day 42; or (3) control group, with no postoperative radiation or NSAIDs. Patients assigned to the indomethacin treatment group who were either at risk for NSAID-induced GI disease or who developed dyspepsia with the therapy were administered cimetidine 200 mg (H_2 receptor antagonist) concomitantly. All subjects enrolled in the study were assessed clinically and radiographically 18 months after surgery.[110] Ectopic bone formation was significantly inhibited by both treatment conditions relative to the control group, and furthermore, both treatments were found to be equally effective. Surgical wound site secretions were more persistent in the radiated subjects postoperatively, but neither treatment group subsequently exhibited signs of poor wound healing that were significantly different from the controls. The incidence of dyspepsia was higher in the indomethacin-treated group, but GI bleeding was not detected in this group. Other NSAIDs have been used to prevent, or minimize, ectopic bone formation after hip arthroplasty, including ibuprofen, ketorolac, and diclofenac.[112,113,115,145] All of these agents are nonselective COX inhibitors that block COX-1 and COX-2 receptors, and it is not known whether the selective COX-2 inhibitors are effective retardants of ectopic bone formation. To date, there have been no reported studies of NSAID use for the management of ectopic bone formation of the TMJ, so the efficacy of these agents for this specific application remains unclear; however, based upon the orthopedic surgery literature, it may be prudent to consider NSAIDs for patients who are at risk for ectopic bone formation after TMJ surgery. The potential GI and renal complications associated with this approach should not be underestimated, and patients undergoing NSAID therapy to prevent or minimize ectopic bone formation should be properly monitored. If indicated, an H_2 receptor antagonist should be administered to reduce the potential for serious GI complications.

SYMPATHETICALLY MAINTAINED PAIN

Sympathetically maintained pain (SMP), reflex sympathetic dystrophy, causalgia, or *complex regional pain syndrome (CRPS)* are terms used to describe a syndrome(s) characterized by continuous burning pain associated with abnormal nociception affected by activity in the sympathetic nervous system, and SMP typically follows traumatic injury to the affected area. Some multiply operated patients with end-stage TMJ disease with localized tactile or mechanical allodynia and burning pain complaints may be suffering from SMP; however, the incidence of SMP in patients with end-stage TMJ disease is unknown. The typical clinical presentation of SMP may include any of the following signs and symptoms

that occur with an incidence of 75% or greater—weakness (95%), pain (93%), altered skin temperature (92%), skin color change (92%), limited range of motion (88%), and hyperesthesia (75%)[116]—whereas less common findings include edema, altered hair growth, tremor, hyperhidrosis, muscle/skin atrophy, and bone resorption.[116]

The mechanism(s) underlying the development of SMP is unknown, but evidence suggests that this condition may result from neuroplastic changes induced by peripheral sensory nerve injury. In animals, the sprouting of sympathetic neurons into sensory dorsal root ganglia has been observed after injury to peripheral sensory nerves, and this sprouting may be induced by a neurotrophic substance, nerve growth factor (NGF), that is released into injured surrounding tissues,[117–121] and a similar response is seen in animals in which NGF is administered intrathecally.[121] These data are consistent with the belief that SMP results from an abnormal sympathetic input to sensory ganglia after peripheral sensory nerve injury that permits the development of physical connections between sympathetic neurons and primary afferent sensory neurons, and this injury may elicit this abnormal response via molecular intermediates, specifically NGF. Clearly, this phenomenon does not occur in all individuals who sustain injuries to peripheral sensory nerves, and further research is needed to investigate this model and to determine risk factors for susceptibility to the development of SMP. Over 25 treatment options for SMP have been reported in the literature,[122] and the most common therapeutic approach has involved the interruption of sympathetic activity via a stellate ganglion anesthetic block. An effective stellate ganglion block may produce protracted pain relief, lasting longer than the duration of anesthetic blockade, although this effect is usually transient. Other pharmacologic interventions (e.g., phentolamine, prazosin, bretylium, guanethidine, calcitonin, nifedipine, gabapentin) as well as surgical sympathectomy have also been used with inconsistent results.[122] With new information concerning the molecular events that may underlie the development of SMP, it is anticipated that more effective therapies will be developed in the future.

MECHANISMS OF FAILURE OF NONSURGICAL THERAPY

Nonsurgical therapy for TMD is composed of a wide variety of management options without consistency and with variable responses reported in the literature. In addition, the diagnosis of TMD is also not based upon strict, univerally accepted criteria, which makes the application of treatment regimens difficult in a heterogenous group of patients. Based upon these factors, nonsurgical therapy as a sole treatment modality, it is very difficult to assess the clinical success or failures of nonsurgical treatment for TMD over time. DeLeeuw and colleagues[76] reported long-lasting satisfactory results for patients treated with nonsurgical therapy for internal derangements and osteoarthrosis with a 30-year follow-up, with persistence of symptoms such as joint noise and resolution of pain and discomfort. Several possibilities could explain the causes of failure of nonsurgical therapy alone for TMD management including improper data gathering, diagnosis, and treatment planning; lack of patient compliance with treatment recommendations; and difficulties with emotionally debilitated patients or patients with major depression or other significant coexisting morbidities.[77] When significant symptoms persist after 3 to 6 months of nonsurgical therapy, alternative therapies and/or diagnostic modalities should be considered, including radiographic studies (magnetic resonance imaging [MRI]) and possibly TMJ arthrocopic or open joint surgery. In many cases, a critical component of nonsurgical failure is patient noncompliance, because the basic home care modalities require diligence and lack of rapid improvement in symptoms often leads to patient abandonment of the treatment protocol including soft diet, moist heat, the use of NSAIDs, bite splint therapy, and physical therapy. Other possible problems include patients who may experience GI upset and not investigate other pharmacologic options; patients who may not be able to find a dentist to fabricate an occlusal appliance, or the cost of such an appliance may be prohibitive; and patients who may not be able to locate a physical therapist who has experience with the management of TMD. Remember that patients who have had chronic problems with TMD may become easily frustrated when initial nonsurgical modalities fail to result in improvement or if treatment exacerbates their symptoms (e.g., bite splint frequently worsens pain before improvement); patients should be motivated to continue their efforts with nonsurgical treatment, especially because the majority of these patients will never become surgical candidates.

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49

CHAPTER

Arthroscopy and Arthrocentesis of the Temporomandibular Joint

Joseph McCain, DMD, and Luciano Stroia, DDS

The management of temporomandibular joint (TMJ) disorders from a surgeon's perspective has improved significantly since the advent of arthroscopy. Arthroscopic observations have provided an in vivo examination of the joint. Disk position and quality, texture of articular cartilage, and vascularity and redundancy of synovium can be observed clearly and precisely. Prior to arthroscopy, joint pathology and surgical correction efforts were targeted toward disk displacement, condyle dislocation, and osteoarthrosis with osteophyte formation.

Currently, the emphasis has shifted to the surgical management of the chemistry of joint space inflammation. This refocus of priorities has been successful in reducing joint pain and increasing joint mobility. Structural joint reconstruction is completed secondarily following reduction of pain, when indicated. This chapter offers an arthroscopic cascade in the overall management of the orthopedic TMJ patient.

HISTORY

The first TMJ arthroscopy was performed by Masatoshi Ohnishi in 1974. Murakami and coworkers, Holmund, McCain, Sanders, Koslin, Moses, and others soon followed. This history has been well documented in the literature. Interestingly, arthrocentesis evolved from, rather than preceded, arthroscopy. Lack of proper equipment, poor insurance reimbursement, and good results despite poor arthroscopic technique led surgeons to explore joint lavage and manipulation in lieu of actual more invasive arthroscopic surgery, and positive results have been reported by Nitzan and others.

Currently, there are two tiers of arthroscopic surgeons: those who are capable of single-puncture basic procedures and those who are capable of multiple punctures and triangulation with capability for advanced arthroscopic surgical procedures. Those who perform single-puncture arthroscopy can perform diagnostic and basic interventions. Reconstructive procedures on refractory cases are then managed with open arthrotomy techniques. Advanced arthroscopic procedures may be done with multiple puncture techniques, in lieu of open surgery, by surgeons whose expertise is at a higher level. There still remains the need for open arthroplasty procedures because even advanced arthroscopy has its limitations.

GOALS

As with other surgeries that have converted over the past decade from open to closed, less invasive techniques, the subspecialty of TMJ arthroscopy, has become popular once again. The goals include the ability to establish an accurate diagnosis, restore function, reduce pain, and diminish joint noise with a minimally invasive, safe, effective, and repeatable procedure with acceptable long-term outcomes. In addition, a reduction in the catabolic environment by the preliminary procedures places the joint in an anabolic state. If a reconstructive procedure such as diskopexy does need to be performed after arthroscopy, it is done in a joint space that can better accept the surgical insult with a minimal amount of bleeding or scar formation, thereby yielding a more favorable outcome.

The ability to refine and improve upon current successful open arthroscopic procedures continues to motivate the advanced TMJ arthroscopist.

INDICATIONS

The indications for arthroscopic intervention include patients who have pain and jaw dysfunction not responsive to nonsurgical dental or medical management. The general indications are shown in Table 49-1. The most common presenting clinical signs and symptoms include limited mouth opening (Figure 49-1), painful joint noises, and mandibular dislocation. Other candidates for at least a diagnostic arthroscopy procedure include patients who have persistent preauricular pain and the diagnosis of intra-articular TMJ pathology cannot be ruled out by clinical or imaging examination.

The clinician should formulate a preoperative differential diagnosis. The accepted terminology for articular disorders includes the Wilkes classification system (Table 49-2). This classification is based upon maximum interincisal opening (MIO), joint noise, and radiographic findings.

CONTRAINDICATIONS

The contraindications to arthroscopy and arthrocentesis include bony ankylosis, advanced fibrous ankylosis, ankylosing osteoarthritis, and overlying skin infection.

Bony ankylosis typically requires autogenous or alloplastic joint replacement. Advanced fibrous ankylosis and ankylosing osteoarthritis will only temporarily respond to advanced arthroscopic techniques. These cases respond to open joint débridements, interpositional grafting, or joint replacement. Puncturing through an area of infected skin increases the potential complication of a septic joint postoperatively.

TABLE 49-1. **General Indications for Arthroscopy**

1. Articular disorders (Wilkes classification)
2. Mandibular dislocation
3. Arthralgia
4. Preauricular atypical facial pain

TABLE 49-2. **Wilkes Classification**

I: Clicking without pain
II: Clicking with pain
III: Locking closed without bone changes
IV: Locking with bone changes
V: Crepitation

ADVANTAGES

The advantages of minimally invasive endoscopic interventions in the TMJ area are

1. No significant skin incision or facial scar.
2. Reduced incidence of VII nerve injury.
3. Diagnostic and therapeutic capabilities.

The disadvantages of minimally invasive procedures are

1. Steep learning curve.
2. Cost of equipment.

PATIENT EVALUATION

The evaluation of the TMJ patient can be an arduous task for the clinician. One needs to have a system to access the nature of the problem and its severity. The surgeon must evaluate the need for surgery. A problem-oriented history and physical examination must be completed. This skill must be developed at the risk of burnout and disinterest driving the provider away from participating in the care of these patients. Unfortunately, the characteristic traits of the temporomandibular disorder (TMD) patient, although not misinterpreted, have led to a certain "labeling" of those patients suffering from these disorders or diseases. Notwithstanding the nonmalicious nature of these prejudices, they will significantly diminish the clinician's capability to appropriately diagnose these conditions. TMJ patients come from all walks of life, males and females, young and old, married, widowed, and divorced, those with



FIGURE 49-1. Patient with limited opening and deviation (right Wilkes III with lateral deviation to the affected side).

children, and those without. Many have the myofascial pain primarily or secondarily to a joint problem, and the astute clinician must be able to discern the clinical differences in order to most appropriately plan for treatment.

McCain TMJ Assessment Standardized Forms

An analogue-driven TMJ practice is recommended. The first document filled out by the patient, after proper introductions and instructions, is the pain and functional analogue (Figure 49-2). This form is completed at the beginning of each visit to monitor progress.

Anamnesis

Chief Complaint

This extremely important step of patient assessment is probably the most easily dismissed area of anamnesis. Over the years, numerous options/windows have been added to a standardized TMJ assessment form, and the list remains fluid, because TMD symptomatology may present with a novel array of complaints that have not yet prompted in-depth exploration by the clinician. As surgeons, our focus of interest is directed more at the orthopedic complaints because they are more likely to require an intervention. However, the patient may report only one, or a variety of, symptoms. 'I can't open my jaw," "My joint clicks and hurts," "I have ear pain," "I have headaches." Whether the otalgia is TMJ referred pain or not is an extremely important step in diagnosis. There are a significant number of patients with tinnitus or other acoustic complaints referred by ear-nose-throat/otorhinolaryngology (ENT/ORL) colleagues to rule out TMD. Some patients can accurately describe the joint noise that has prompted them to seek care (e.g., clicking, popping, grinding, grating). Whereas most patients will describe some form of masticatory dysfunction, some patients will be specific in their description of progressive restriction of mouth opening, transient jaw locking, and so forth, or they will need only a few targeted questions from the examiner in order to help them to delineate their condition.

Parafunction and Associated Causes

The clinician is often tempted to overemphasize parafunctional habits when examining the patient. Masticatory mus-

TO OUR TMJ PATIENTS:

Please take a minute to answers these questions. We ask all of our patients these questions every time they come in. However, in some cases, we feel the answers may not be honest because one of the doctors may be present when you answer. We want a sincere, honest opinion from you because it helps you and those TMJ patients who we will treat in the future, as well as our colleagues who seek to help their patients with arthroscopy.

(Place an "X" at the point along the line which answers the question best).

1-What is your overall level of pain in your jaw joint(s) today?

I ———————————————— X————————————————I

most intense pain imaginable — no pain

2-What is your overall level of jaw function today?

I ———————————————— X–————————————————I

can't use jaw at all — no problems with jaw use

3-How do you feel now compared to your first visit here?

I ——————————N/A——————————I————————————————I

much wrose — same — much better

Please indicate on the drawings below where you have pain:

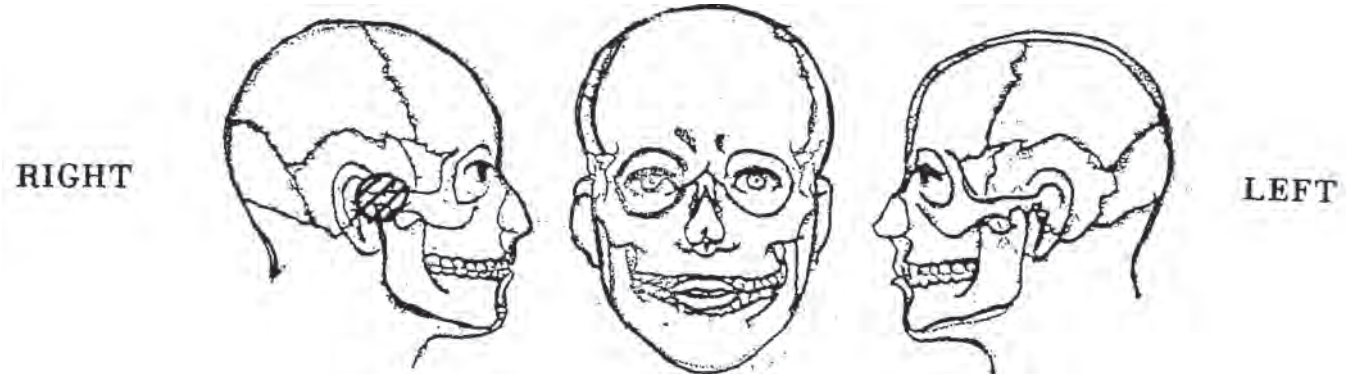


FIGURE 49-2. Functional and pain analog.

culature spasm ("jaw stiffness"), matinal or vesperal, is a common complaint. However, unless the patient admits to vigil bruxing or clenching or the habit is witnessed, parafunction should not be attributed to etiopathogenicity in the particular TMJ condition. Mental or physical stress should definitely be taken into consideration. The surgeon must remain aware of the symbiotic interrelation between parafunction, stress, and the development of TMD.

Analog Scales

The visual analogue scale (VAS) provides a real sense of the acuteness and urgency of the patient's perception of the problem. Remember that arthrocentesis and arthroscopy are elective procedures, so every patient must have insight into her or his condition and be motivated enough to improve her or his present status. The condition should be severe enough for the surgeon to believe it can be ameliorated or suppressed with surgical intervention. Preventive goals are secondary motivators.

History

A pertinent documentation of events should take place in accordance with patient's narrative description of the disease progression. The association between traumatic events and various TMJ conditions cannot be overemphasized. Whether direct (when the mandible has been involved primarily) or indirect (e.g., after a dental appointment, extractions, endodontic treatment, oral surgical procedures, endotracheal intubation maneuvers), the patient may not correlate the events with this specific condition. The clinician should not indiscriminately dismiss minor indirect trauma as a factor in triggering TMD. An interesting fact is that the patient may omit reporting even direct trauma events to the jaw if they occurred in the distant past. The surgeon's keen sense of observation should allow documentation of even small facial cicatricial marks, especially in the inconspicuous submento-submandibular area from prior chin trauma. When a history of motor vehicle accident (MVA) has been established, additional information may be gained from the details of the event (e.g., restrained vs. unrestrained, driver vs. passenger, deployment of airbag). The majority of patients will be able to provide pertinent descriptions of the specific TMJ-related problems, including jaw clicks or pops, crepitus, locking (open or closed), and dislocation as well as the circumstances of each occurrence, frequency, remission/relapse sequence, aggravating and alleviating factors, pain radiation, and so forth.

Diet

The consistency of the patient's diet is an important indicator of the level of TMJ dysfunction and the necessity to consider surgical management. The surgeon must correlate this information with the analogue scales.

Treatment

All prior attempts to treat the TMJ condition both medically and surgically should be recorded. A previous ENT/ORL or neurologic evaluation will, in most instances, provide the surgeon the results of acoustic-vestibular or neuropathic pain workups, helping the specialist to "zero-in" on the TMJ workup of the condition. It is important to know whether the entire spectrum of conservative treatment has been exhausted. The surgeon must ascertain whether the noninvasive therapy (e.g., Boering modifications, orthotics, over-the-counter [OTC]/prescription medications) or surgical treatment has been appropriate and evaluate the outcomes of these treatments and, additionally, the compliance of the patient with these previous regimens.

PHYSICAL EXAMINATION

Mouth Opening and Range of Motion

The physical examination should be problem-focused and brief, but pertinent. Ask the patient to open the mouth and observe the velocity of opening and measure the MIO. A rapid opening to 40 mm is not a very exciting finding for the surgeon in order to motivate a patient toward the operating table, unless you are assessing an undiagnosed facial pain syndrome. A patient who opens to 30 mm then catches and clicks to gain further opening certainly should get the attention of the surgeon. Regardless of their subtlety, lateral deviations should be measured and noted in the record. Complete documentation of range of motion (ROM) in lateral excursive movements and during jaw protrusion follows next. The lack of translation of the condyle may imply a nonreducing disk. Condyle subluxation or dislocation may be palpated in the preauricular region or within the external auditory canal.

Joint Noise

A correlation must be made next between the ROM and associated joint noise/noises. Joint sounds may be audible and/or palpable. Reciprocal clicking may reflect a recapturing of the disk. Opening clicks may suggest a fixated, or stuck, disk or subluxating condyle. A grating sound is the result of the condyle pressing against a fragmented edge of disk. The edge of disk can also be compressed against the fossa and/or eminence. Crepitation may not be accompanied by pain, and loss of function is only a clinical sign and requires periodic follow-up, not necessarily invasive surgery.

Pain Assessment of the Joint and Adjacent Musculature

For the purposes of access and feasibility, only the lateral and posterior poles of the condyle are palpated. Pain at the lateral

pole may indicate synovitis, osteoarthritis, joint effusion, or disk dislocation. The latter is best elucidated by palpating superiorly on the neck of the condyle, posterior and slightly inferior to the prominent lateral pole itself. This particular area of the posterolateral pole of the condyle is the site of the attachment of the disk to the lateral capsule and condylar neck. Both acutely and chronically inflamed disk dislocations may provoke a pain response upon palpation. Intrameatal pain suggests a posterior capsule retrodiskal inflammatory process. Both lateral capsular and endaural joint tenderness to palpation are common findings in TMD. The closed-lock patients will invariably demonstrate a "soft tissue crepitance" in at least a portion of their range of normal mouth motion. This bruit is caused by the bilaminar retrodiskal tissues rolled between the condyle and the eminence. Reciprocal, mediolateral, and late opening clicks, although less frequent, may still be encountered. The muscular examination is limited to the superior portion of the anterior margin of masseter muscle and the insertion of the temporalis tendon on the coronoid process. These particular elevator muscles of the mandible provide the most precise information regarding myofascial pain dysfunction syndrome (MPD) and are most likely to be involved with that process. All clinical findings should be recorded on the checklist form (Figure 49-3).

Joint Locking or Dislocation

Mahan's Sign

The most important pathognomonic sign is the "Mahan sign," named after Parker Mahan who described this sign in 1990 (Figure 49-4). The working condyle is loaded by biting on blades with the contralateral canines. If positive with elicitation of pain, there is generally an orthopedic problem in the joint. The sign is usually positive in Wilkes II, III, and IV patients. Some Wilkes II patients will not be positive and that affects the choice of intervention. Noninflamed (negative Mahan's sign) Wilkes II patients may be managed better with diskopexy, and Mahan-positive Wilkes II patients are managed with nonsurgical interventions before diskopexy.

The reason that Wilkes V patients will not have a positive Mahan sign is because the condyle will traverse the perforation and not compress the retrodiskal tissues. Wilkes V patients, however, will have the second important pathognomonic sign, crepitation.

Head, Eyes, Ears, Nose, and Throat

Examination of the head, eyes, ear, nose, and throat (HEENT) is performed next, with special focus on the external ear examination, facial symmetry, and dentoskeletal classification. Also, the Ellis occlusal class, dental interferences during function, and evidence of occlusal wear should be noted.

Panoramic Radiograph

The Panorex is then evaluated with a focus on the condyles and joint spaces. Maintenance of the oval morphology is considered normal. Flattening of the head, loss of cortical detail, osteosclerosis, osteophyte formation, narrowing of the joint space, and subcortical cysts are important bone changes to factor into the Wilkes classification.

Magnetic Resonance Imaging

On occasion, the patient may present with a copy of MRI results, as a part of a pertinent referral from another clinician. Whereas the surgeon should become familiar with the radiology interpretation of the study, the arthroscopist should "re-read" the MRI and interpret it according to his or her clinical and surgical experience. In the most stable relationship, the rather thick posterior band of the disk occupies the greatest concavity of the glenoid fossae (Figure 49-5). Henceforth, a closed-mouth MRI view showing the posterior band in the 10:30 to 11:00 position should be read as abnormal. Preoperative changes on the MRI mandate close consideration of surgical detail and postoperative physical therapy. As long as the disk itself is salvageable, disk form should not preclude disk suturing and/or stabilization. The open-mouth view MRI (Figure 49-6) will give the practitioner the most accurate perspective of the disk position with function (i.e., recapture of the disk in the case of disk displacement), especially in cases that correlate with reciprocal or double clicking on physical examination. Although T1-weighted open- and closed-mouth MRI views are paramount for confirming the clinical diagnosis, the T2-weighted images more accurately reflect an associated inflammatory component of the joint disease, such as the presence of joint effusion (Figure 49-7). This completes the patient assessment.

Etiology

The etiologic factors are organized into five categories/classes.

I. **Parafunction** (direct microtrauma, associated MPD). The effects of psychological stress may cause the masticatory system to spasm, thus compressing and disrupting normal joint articulation. Bruxism and clenching inflict repetitive and abnormal overloading of the joint and muscular system, causing spasm and muscle pain to develop with intermittent clicking.

II. **Dentofacial deformities and nalocclusions** (direct microtrauma). Articular dysfunction occurs from disharmonious motion between the maxillary and the mandibular arches. The most common patient in this category is the one with vertical maxillary excess (VME) in combination with mandible retrognathia/hypoplasia. A majority of internal derangements may be caused by various components of mandibular retrusion. A class I



T.M.J CONSULTATION

NAME: JANE DOE

AGE: 24

GENDER: Female

MARITAL STATUS:
SIGNIFICANT OTHER
PARTNER
WIDOWED
MARRIED
DIVORCED
SINGLE.

CHILDREN:
NONE
ONE
TWO
THREE
FOUR
MORE THAN FOUR.

OCCUPATION:
RETIRED
BUSINESS
OTHER
STUDENT
ADMINISTRATION
HEALTHCARE
LAWYER
EDUCATION
SALES
LAW ENFORCEMENT.

MEDICAL HISTORY INFORMATION IS COMPLETED, REVIEWED AND INCLUDED IN THE PATIENT'S CHART.

CHIEF COMPLAINT:
PAIN
JOINT NOISE
MIGRAINE
GRINDING
TINNITUS
OTALGIA
RESTRICTED RAMGE OF MOTION
MASTICATORY DYSFUNCTION
DISLOCATION
CLICKING
MUSCLE SORENESS
OTHER
TRANSIENT LOCKING.

HISTORY:

BRUXISM:
PM JAW STIFFNESS
PATIENT HAS A HISTORY OF BRUXISM
PATIENT HAS NO HISTORY OF BRUXISM.
WITNESSED
AM JAW STIFFNESS.

STRESS:
YES
NO.

CLENCHING:
YES
NO.

ANALOGS
LEFT
RIGHT.

TRAUMA:
INDIRECT
DIRECT
NONE.

MVA:
NO
YES
DATE:
SEATBELT
DRIVER
PASSENGER.

JAW CLICK:
RIGHT NO
RIGHT YES
LEFT YES
LEFT NO.

CREPITUS:
LEFT YES
RIGHT NO
RIGHT YES
LEFT NO.

LOCK:
LEFT YES
RIGHT YES
RIGHT NO
LEFT NO
YES
NO
RIGHT
LEFT.

DISLOCATION:
RIGHT YES
RIGHT NO
LEFT NO
LEFT YES
NO
YES
RIGHT
LEFT.

DIET:
REGULAR
REGULAR COMPROMISED
SOFT
LIQUID.

TREATMENT:
DIET
OTC MEDS
RX MEDS
ORTHOTIC
SURGERY
NEUROLOGIC EVAL
ENT EVAL
OTHER
NONE.

VERTICAL OPENING:
.

PHYSICAL EXAM

ROM:
LL:NTB
PR:JTB
LL: PB
PR:NTB
RL:PB
RL:JTB
RL:NTB
LL: JTB
PR:PB.

JOINT NOISE

CLICK:
RIGHT YES
LEFT YES
LEFT NO
RIGHT NO
YES
NO
RIGHT
LEFT.

CREPITUS
LEFT YES
LEFT NO
RIGHT NO
RIGHT YES
YES
NO
RIGHT
LEFT.

PAIN:
NO
IM RIGHT NO
IM RIGHT YES
MASSETER RIGHT NO
MASSETER LEFT NO
IM LEFT YES
PALPATION CONDYLE LP LEFT NO
PALPATION CONDYLE LP RIGHT NO
MASSETER RIGHT YES
PALPATION CONDYLE LP LEFT YES
MASSETER LEFT YES
IM LEFT NO
PALPATION CONDYLE LP RIGHT YES.

LOCK:
RIGHT YES
RIGHT NO
LEFT NO
LEFT YES
YES
NO
RIGHT
LEFT.

DISLOCATION:
RIGHT NO
LEFT NO
LEFT YES
RIGHT YES
YES
NO
RIGHT
LEFT.

MAHAN DIRECT:
RIGHT YES
LEFT NO
LEFT YES
RIGHT NO
YES
NO
RIGHT
LEFT.

MAHAN INDIRECT:
RIGHT YES
LEFT NO
RIGHT NO
LEFT YES
YES
NO
RIGHT
LEFT.

EARS:
NOT EXAMINED
NORMAL
ABNORMAL.

FACE:
SYMMETRICAL
ASYMETRICAL
MANDIBLE LEFT
MANDIBLE RIGHT.

SKELETAL CLASS:
I
II
III.

HEENT:
OTHERWISE NORMAL.

OCCLUSION:
I
II
III.

INTERFERENCES:
NONE
(LEFT) BALANCING
(LEFT) WORKING
(LEFT) PROTRUSIVE
(RIGHT) BALANCING
(RIGHT) WORKING
(RIGHT) PROTRUSIVE
(PROTRUSION) RIGHT
(PROTRUSION) LEFT
(PROTRUSION) ANTERIOR.

WEAR:
SEVERE
MODERATE
MILD
NORMAL
NONE.

IMAGING:

PANOREX:
WISDOM TEETH YES, WISDOM TEETH NO, ANTIGONIAL NOTCHING, INFERIOR BORDER SCALLOPING, RIGHT, NORMAL, OSTEOARTHRITIS, 1, 2, 3, LEFT, NORMAL, OSTEOARTHRITIS, 1, 2, and 3.
MRI :
EFFUSION
DISC SIGNAL CHANGES
NORMAL
RIGHT
DISC DISPLACEMENT WITH REDUCTION
DISC DISPLACEMENT WITHOUT REDUCTION
OSTEOARTHRITIS
LEFT
NORMAL
DISC DISPLACEMENT WITH REDUCTION
DISC DISPLACEMENT WITHOUT REDUCTION
OSTEOARTHRITIS.

ETIOLOGY:
V RHEUMATOID ARTHRITIS/OTHER GENERAL
IV INDIRECT TRAUMA
III DIRECT TRAUMA
II OCCLUSAL (DENTO-SKELETO-FACIAL CONGENITAL/AQUIRED MALFORMATIONS)
I PARAFUNCTION

DIAGNOSIS:
OSTEOARTHRITIS RIGHT
OTALGIA
ANKYLOSIS
ANKYLOSIS O.A
OSTEOARTHRITIS LEFT
ARTHRALGIA
HEADACHE
RIGHT WILKES I
LEFT WILKES V
RIGHT WILKES II
RIGHT WILKES III
RIGHT WILKES IV
RIGHT WILKES V
LEFT WILKES I
LEFT WILKES II
LEFT WILKES III
LEFT WILKES IV
RIGHT
NORMAL
MPD
DISLOCATION
INTERNAL JOINT DERRANGEMENT
LEFT
NORMAL
MPD
DISLOCATION
INTERNAL JOINT DERRANGEMENT .

ENTRY LEVEL:
(IV) MEDICAL
(III) SURGICAL
(II) ORTHOTIC
(I) BOERING
(V) NONE.

MEDICATIONS:
MEDROL DOSE PACK
FLEXARIL 10 MG ADVISED
NAPROSYN 375 MG ADVISED.

DISCUSSION:
THE FOLLOWING TREATMENT HAS BEEN RECOMMENDED.

FIGURE 49-3. The McCain TMJ Assessment Standardized Form.

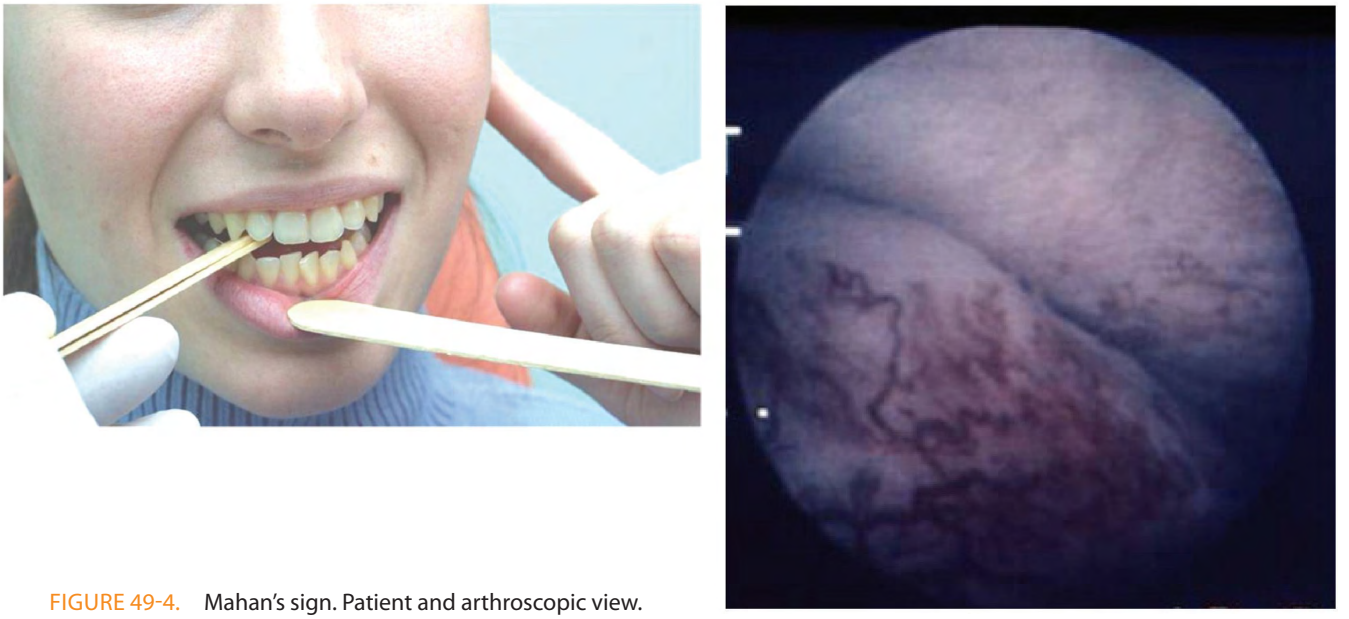


FIGURE 49-4. Mahan's sign. Patient and arthroscopic view.

occlusion in some patients may, however, exhibit vertical maxillary deficiency (VMD), causing early loss of posterior dentition with subsequent posterior or vertical collapse and bite closure. In this case, the dysfunction is the result of the collapse of the vertical dimension, with a direct microtrauma effect on the joint over an extended time.

III. Direct macrotrauma. Direct trauma involves an acute direct trauma to the mandible with or without fracture. Most of the time, patients can specifically recall the event because their TMJ problems developed immediately after the event. In some situations of direct trauma to the mandible, there are no immediate effects on the joint. However, as years progress, the classic symptoma-



FIGURE 49-5. Magnetic resonance imaging (MRI) closed-mouth–normal disk position



FIGURE 49-6. MRI open-mouth–normal disk position or disk recapture.

tology develops in the joint and no other finding of anamnesis indicates any other etiologic factor.

IV. **Indirect macrotrauma.** These are patients reporting injuries of the acceleration-deceleration type phenomenon, followed by progressive symptoms consisting of muscle splinting that advances to arthralgia and joint noise.

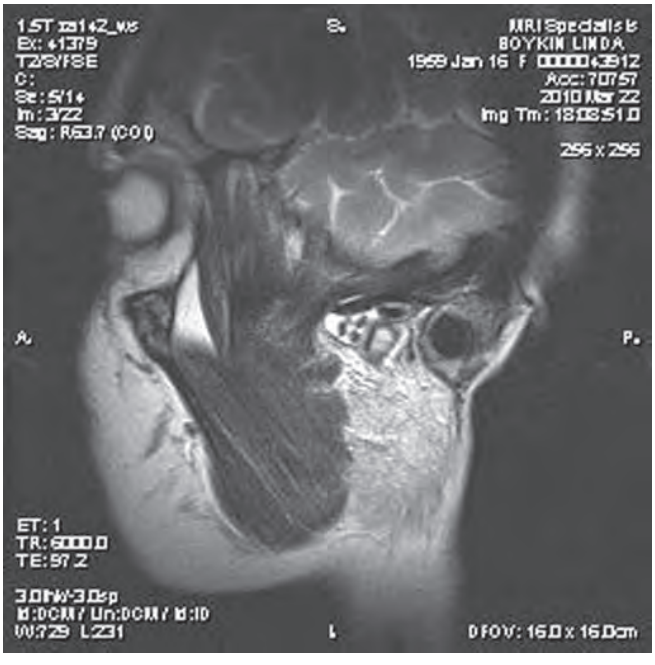


FIGURE 49-7. T2 weighted MRI—joint effusions.

V. **Systemic disease.** Most of the diseases with TMJ involvement fall into the categories of rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), and benign or malignant tumors, although the latter two are rather infrequent occurrences.

Diagnosis

In most cases, the patient will present without prior advanced imaging; however, a comprehensive history and clinical examination will allow a diagnosis to be formulated, and an MRI is indicated only if the clinical examination is equivocal. A diagnostic staging should be made for each patient according to the Wilkes classification system.

Discussion

The conclusion of the TMJ consultation form is the formulation of an initial treatment plan. A few remarks are in order after the completion of the standardized form. If the TMJ anamnesis examination is performed correctly, it will definitely elucidate the necessary information about the patient. This can be achieved only by developing an accurate, concise, and repeatable technique for collection of data. A complete TMJ workup (including diagnosis and initial treatment plan) could be completed in 15 to 30 minutes, using standardized forms and clinical experience.

Rapid TMJ Assessment

As the clinician becomes more experienced, more accurate and more comfortable with all the rigors of the TMJ examination, the assessment procedures can be expedited. Eventually, the TMJ surgeon performs the same rapid TMJ assessment for the initial assessment of a new patient as well as for the follow-up patients. This rapid assessment helps the surgeon to conduct a problem-focused assessment using a standardized form (see Figure 49-3), thus further increasing the speed of evaluation. The TMJ follow-up form (Figure 49-8) gives the practitioner all the pertinent information with regard to the patient's condition, progress, future course of action, and follow-up. As a quick review, the clinician should assess ROM of the mandible in all excursions, while listening/auscultating and palpating the joints, periarticular structures, the masseter and temporalis muscles as previously described. Also, a check should be performed for Mahan's sign, and all findings should be correlated with those from previous visits.

ARTHROCENTESIS

In 1991, Nitzan and Dolwick identified a unique clinical situation of closed lock of the TMJ that could not be explained by conventional methods of diagnosis of the

Miami Oral & Maxillofacial Surgery

PATIENT NAME: JANE DOE
TODAYS DATE: July 21, 2010
PATIENT ID: 23066

S/P:
STEROID INJECTION
MEDICAL MANAGEMENT
BOERING
ORTHOTIC
DISCOPEXY SCREW
DISCOPEXY SUTURE
EMINECTOMY
PHYSICAL THERAPY
OFFICE ARTHRSCOPY
DISCOPEXY
DEBRIDMENT
SILASTIC PULL OUT
HOSPITAL LYSIS AND LAVAGE
TOTAL JOINT
CHRISTENSEN FOSSA
CONTRACTURE

MEDICATIONS:
ULTRAM
OTC
STEROIDS
ELAVIL
NO
NARCOTICS
NON NSAIDS
MUSCLE RELAXANT
NON STERIODAL
OTHER
YES.

DIET:
REGULAR:
SOFT.

ANALOGS:

LEFT
RIGHT

MIO:

MAHAN DIRECT:
RIGHT YES
LEFT NO
RIGHT NO
LEFT YES

JOINT NOISE:
LEFT YES
LEFT NO
RIGHT NO
RIGHT YES.

MUSCLE PAIN:
LEFT YES
LEFT NO
RIGHT NO
RIGHT YES.

JOINT PAIN:
LEFT YES
LEFT NO
RIGHT NO
RIGHT YES.

OCCLUSION:
UNSTABLE
STABLE.

FIGURE 49-8. The McCain TMJ Follow-up Standardized Form.

disease process. Prior to their description, Murakami in 1986 identified the ability to recapture a persistently displaced anterior disk displacement with a single-puncture technique to accomplish a pumping action and the use of hydraulic pressure in the superior joint space. It was Nitzan and Dolwick, however, who questioned the usual mechanisms of development of disk displacement and identified a condition of severe, limited mouth opening (<30 mm) without translation, no joint noise, that could occur at any age, was not a result of macrotrauma, was unrelated to disk shape or position, was unresponsive to nonsurgical TMJ therapy, and was responsive to the technique of joint pressure lysis and lavage (arthrocentesis). They were convinced that this condition was different from classic disk displacement. The lack of condylar translation was attributed to adherence of the disk to the fossa due to a "vacuum" or "suction-cup" effect of the convex disk pressed against the anterior slope of the glenoid fossa, and this situation is more likely to occur in joints with decreased synovial fluid volume and increased viscosity. Several studies showed the efficacy of dual-puncture arthrocentesis in relieving "disk adhesion" (the stuck disk) with acute painful limited opening and allowing a rapid improvement in mouth opening and decrease in pain. It is important to consider performing the procedure early after the onset of symptoms (<3 mo) and consideration of the use of bite splint therapy postoperatively to unload the joint space. Whereas corticosteroids have been used routinely in the joint space after the arthrocentesis procedure, several studies have indicated that the use of sodium hyaluronate may have better short- and long-term benefits. Over the years, studies have shown comparable results of arthroscopy and arthrocentesis primarily due to the lysis and lavage portion of the technique, resulting in clearance of inflammatory mediators and relief of a vacuum effect in the joint space. It has been concluded that arthrocentesis could be considered as an intervening treatment between nonsurgical management and arthroscopy, especially in cases that could be identified as disk adhesion with acute onset of symptoms. The procedure is relatively simple to perform in office with a low cost and minimal chance of complications (fluid extravasation into soft tissues, transient facial nerve weakness) and potentially high benefit if it can resolve the clinical symptoms. Of course, the major benefits of arthroscopy over arthrocentesis include direct visualization of the joint space and the ability to perform a variety of invasive procedures as described in detail in this chapter, whereas arthrocentesis does little to address the specific etiology of the disk problem or prevent recurrence. Whereas treatment decisions must be individualized in each patient, arthrocentesis should be considered before more invasive techniques.

The Arthroscope

The arthroscope is a specific endoscope designed to extend the eye of the arthroscopist to the tip of the instrument, inside the intra-articular space.

Optical Characteristics

Field of View

The field of view (FOV) of the arthroscope is the included angle drawn from the tip of the scope to the extreme edges of the field or object space. When the scope is used in a fluid medium, the FOV angle is approximately 40% less than the one measured in the air with the same arthroscope (Figure 49-9).

Direction of View

The direction of view is the angle projected between the normal axis of the arthroscope and a line through the center of the image viewed through the arthroscope. Direction and FOV are the most common angles used in the description of the arthroscope. In order to determine the extreme edge of field of a scope, add the direction of view angle plus half the FOV angle. The sum of the two numbers equals the extreme angle from the axis that can be seen (see Figure 49-9).

Apparent FOV

The apparent FOV is the included angle drawn from the observer's eye to the extreme edges of the field or object space.

Optical Parameters

Stigmatism of the Image

This parameter refers to focusing a joint in the field identically to a joint in the apparent field. Lack of stigmatism is indicative of either design or manufacturing flaws in the arthroscope.

Distortion

This parameter correlates with a straight line in the image. The "shop-terms" "pin-cushion" or "barrel" distortion refer to the sides of a square symmetrically situated in the FOV that has the very sides curved inward or outward, respectively. Unless significant, distortion is of no consequence in endoscopy.

Chromatic Correction

Images may be displaced axially as a function of wavelength or color or may be different sizes as a function of color. The chromatic correction of an instrument is much more critical for photography than for visual use. As far as the TMJ arthroscopy is concerned, white-balancing the scope before intra-articular introduction will address the chromatic correction.

Vignetting

Vignetting translates into a defect stemmed in the optical design, manufacturing, or misalignment caused by bending or displacement of the optical elements. It results in a loss of brightness of the peripheral area of the image, secondary to light obstruction by mounts, stops, or element cells. On occasion, vignetting may present as an oval rather than a round FOV.

Transmission

Each surface of an optical system reflects a fraction of the incidence of the luminous ray beam; and each element

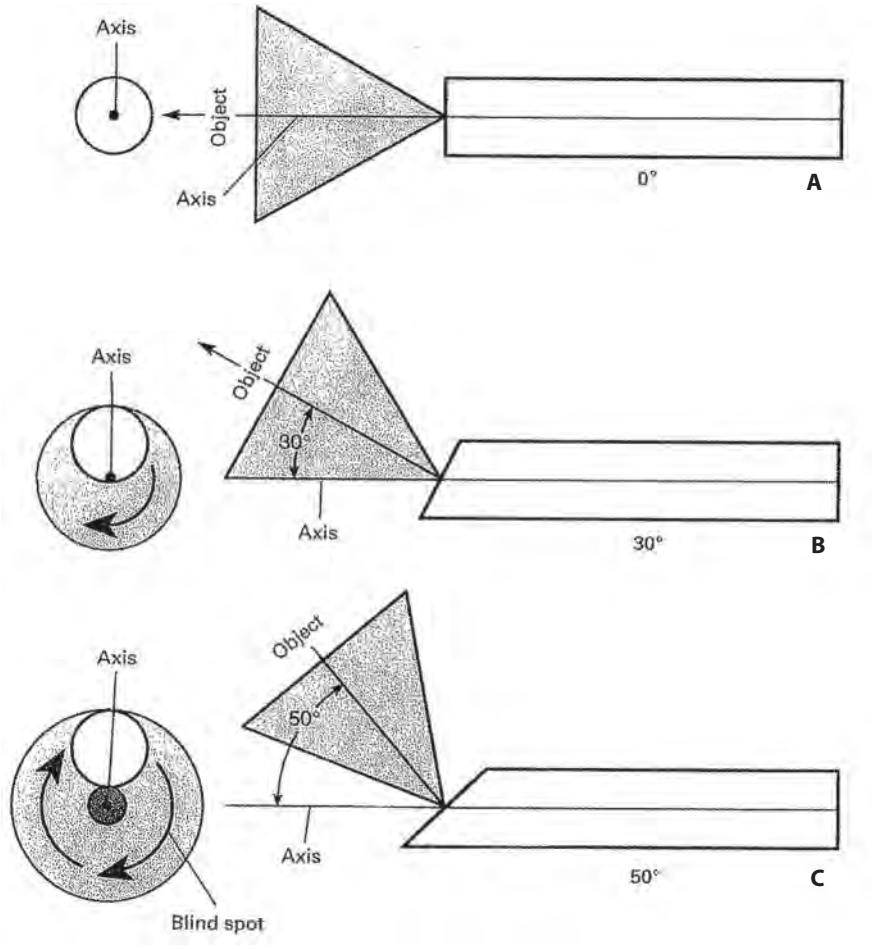


FIGURE 49-9. Direction of view of three arthroscopes, each with a field of view angle of 60 degrees and an effective field of view (left of each scope) if the arthroscope is rotated 360 degrees about its axis. **A,** The 0-degree scope: the field of view remains the size of the diameter of the scope as it is rotated. **B,** The 30-degree scope: the field of view is greater with this scope as it is rotated. **C,** The 50 degree scope: the field of view is even greater; however, the offset is such that a blind spot *(black circle)* exists as the endoscope is rotated.

of the optical system will absorb a certain fraction. The effectiveness of transmission of an endoscopic system is approximately 90%.

Veiling Glare

Image-forming light reflected by each surface is, in turn, re-reflected, in part, by all surfaces preceding it in the system. Some of this light, together with a small amount scattered by each surface due to imperfections in polish and debris on the surfaces, reduces the contrast in the image and the ability of the endoscopist to resolve fine detail.

Technology of Video Arthroscopy

The video camera is routinely used in TMJ arthroscopy for viewing the surgical field indirectly on a monitor. The advantages of its use are better operative field sterility (less action in and around the operative field), surgeon comfort, and most important, a high-quality image magnified on the monitor, enhancing visibility and engagement of the entire surgical team (Figure 49-10). It is invaluable in the evaluation of intra-articular pathology by both surgeon and assistant as well as a teaching modality. Recorded media allows for review of surgical findings during the postsurgical visit. The patient can now be educated by virtual means regarding her or his joint disease process. Also, the medicolegal record-keeping is very accurate. The disadvantage of the entire system is, obviously, pecuniary. The sophisticated miniaturized video camera, high-resolution monitors, as well as the quality recording equipment necessary for video arthroscopy may be rather expensive. In addition, this quite

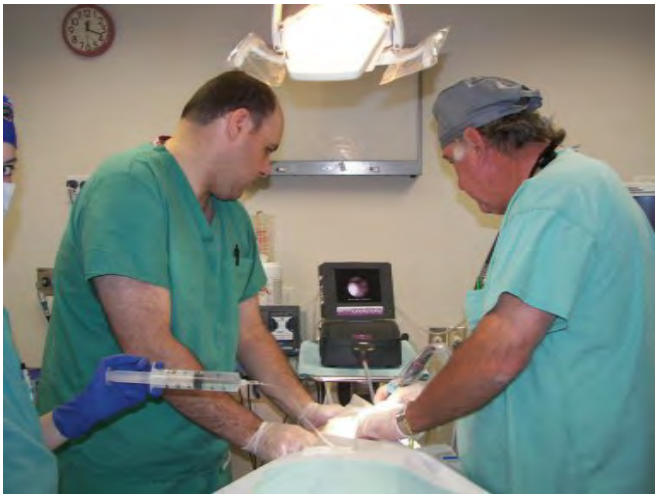


FIGURE 49-10. Temporomandibular joint (TMJ) arthroscopy.

complicated equipment requires basic understanding of electronics and video electronics in order to set up and operate appropriately.

A video camera is composed of a lens system that receives the image from the arthroscope and focuses the image on a light-sensitive electronic device that translates this image into an electronic signal. The core of the camera is a silicon wafer with discrete light-sensing elements called *pixels*. The pixels have the ability to translate color and light intensity into electronic signals that, in turn, can be recorded from the camera on a DVD, displayed on a TV monitor, and/or processed into a still image.

The chip sensor determines the quality of the image and specifications of the video camera. The numbers and size of pixels correspond directly to the detail of image reproduction (camera resolution). Out of the charged couple device (CCD) and metal oxide semiconductor (MOS) pixels, only the CCDs are at present used, because their high color sensitivity, color rendition capabilities, and broad dynamic range for various light intensities has permitted a significant reduction of the video camera size while increasing the number of pixels.

A camera produces color by selectively filtering certain pixels on the sensor for one specific color. Electronic signals for red, green, and blue are produced separately. Red, green, and blue, or the complementary colors magenta, yellow, and cyan, can be either added or subtracted to produce any single color. Masks of these colors, in either a striped or a mosaic pattern, filter the individual pixels. These filtered pixels are electrically sampled and the actual color is determined. The latest advancements in technology have permitted the use of a different method of color determination that employs a prism system within the camera, which separates the colors, then directs them to three separate sensors. Despite an increase in the quality of the image, this system was, until recently, technique prohibitive because it promoted an increase in the size of the camera.

Specifications

The quality of the image produced is a factor of several camera specifications, including horizontal and vertical resolution, minimum light sensitivity, color sensitivity, and signal-to-noise ratio. Other factors such as type of signal produced, scanning systems, and color systems also influence the characteristics of the image and its ability to be used with other equipment.

Resolution is the degree of picture detail that can be perceived and is comparable with good vision, allowing the surgeon to visualize more detail or to discern more features. A video image is composed of a series of lines, both horizontal and vertical, scanned in the camera and drawn by the monitor to produce the image. The initial factor in determining the resolution capabilities of the video camera is the actual number of pixels in the camera. Subsequently, resolution is determined by the ability of the electronic equipment to transmit, record, or reproduce the picture elements. The least sensitive piece of equipment in the system will, finally, determine the observed resolution. In general, the camera should be the least sensitive part in the circuit.

The size of the arthroscopic image is determined by the optics of the scope and lens system on the camera magnifying the image focused on the sensor in the camera. Cameras used for smaller size scopes have a greater lens magnification system in order to adequately enlarge the image. If the image is too reduced on the sensor, the camera's resolution will be underemployed.

The *alignment* and *ability* of the camera to accurately reproduce color are set by the manufacturer. Adjustments in the electronic interpretation of color are imminent due to the variations in the light sources used to illuminate the joint. White light is made of all spectral colors. Certain light sources will emit more blue or yellow light, thus influencing the appearance of joint structures and color of the image. When viewing a white object illuminated by different light sources, the signal from the camera is adjusted to equalize the red, green, and blue electronic signals using the "white balance" settings. Most cameras white-balance automatically or with minimal operator intervention.

A *color temperature* of 5000°K is ideal for accurate color reproduction and corresponds most closely to natural daylight illumination. Further adjustment for color may also be made on the monitor if necessary. A color pattern can be generated by the camera, whereas color reproduction can be altered on the monitor using brightness, tint, and hue adjustments.

The *scanning system* is the means by which the video image is assembled from information provided by the sensors.

The TMJ arthroscopy requires a cold white bright light source to adequately illuminate the joint cavity. The light source must be variable in intensity to adjust for changes in illumination requirements for different parts of the joint. This has been accomplished by controlling the quantity of light delivered at the light source with a variable iris aperture, via an autosensing light source that determines the strength of the video signal and increases or decreases the iris aperture accordingly, thus controlling the quantity of light delivered to the light cable. The arthroscopic system also employs an automatic gain control system of the video signal that leaves the light intensity constant and varies the strength of the video signal proportional to the required illumination.

Light sensitivity is the degree to which the camera determines an image in low-light situations. It measured in lux. The *signal-to-noise ratio* is a measure of the video signal to the background electronic noise. A larger ratio denotes a better, clearer picture. It is measured in decibels.

On Point System

One of the developments in technology of the later years is the On Point system (Biomet, Jacksonville, FL) (Figure 49-11). This diagnostic arthroscopy system uses a 1.2-mm, 0° scope. The high-quality resolution and portable surgical



FIGURE 49-11. The On Point System (Biomet).

unit make this operation amenable to the outpatient setting, with significant cost savings for the patient. The armamentarium includes an ergonomic handpiece containing a camera and light source, enabled to capture still images and record videos. The all-digital imaging platform includes a 6.4-inch LCD monitor that combines the performance of the Xenon Fiber Optic Light Source (delivering a 175-W illumination performance) and the High-Resolution Camera System (480-line digital camera) to deliver the image. The 1.2-mm disposable scope is the size of an 18-gauge needle. The other instruments included are a single-use cannula, trocar, obturator, and cannula plug (Figure 49-12). The cannula has an outside diameter of 1.9 mm and a Luer port on the body to connect an irrigation source. The port also permits intra-articular injection.



FIGURE 49-12. Sterile single-use instruments: trocar, cannula, obturator, cannula-plug.

Smith-Nephew System

The data acquired by the 560 Series 3-CCD High Definition Camera (HD 1080i) (Figure 49-13) are conveyed to the CCU via high-speed digital-interface. The image data is processed and converted into HDTV images, which are displayed on flat-panel HD monitors. A 1.9-mm-diameter 30-degree scope, with a 75-degree FOV angle and a 65-mm working length, is connected to the camera head. The scope J-locks into a 2.0-mm cannula with flowport. The cannula is versatile for the 2.0-mm short trocar as well as the 2.0-mm short obturator with a conical tip.

Armamentarium

Hand Instrumentation

Cannulas

The delivery system facilitates not only passing of hand instruments into the joint but also irrigation and maintenance of joint insufflation. The markings on the cannula begin at 15 mm from the tip and continue in 5-mm increments (Figure 49-14). Similar cannulas must be used in double-puncture arthroscopy to allow for the interchanging of instruments and scope between portals. The 1.7-mm-diameter hand instruments leave a 0.3-mm space for irrigation outflow. All instruments are marked in 5-mm increments. For larger-diameter instruments (i.e., 2.7 mm), this author has designed a switch-stick, facilitating the interchange between the 2.0- and the 3.0-mm-diameter cannulas.

Probes

The **straight probe** is the most basic arthroscopic hand instrument. It is used for palpation, severing adhesions, and mobilization/temporary immobilization of tissue (i.e., disk). The **hooked probe** is similar to the latter except for a small terminal hook. It is the preferred instrument for palpation in cases of chondromalacia. The typical use of this probe is to elevate the anterior aspect of the disk after anterior releasing procedures and to complete the dissection of the disk from capsule and pterygoid muscle. The hooked probe is also preferred in difficult cases of disk reduction, with lax/redundant retrodiskal tissue, where a straight probe may lacerate the structure. Hooking of the oblique protuberance before disk reduction enables reduction without herniation of tissue (Figure 49-15).

Biopsy Forceps

The serrated type (Figure 49-16) has cupped beaks and is used for small biopsy samples and for the débridement of pathologic or fragmented tissues. The basket type (Figure 49-17) harvests mostly full-thickness biopsy specimens (i.e., synovial tissue).

The French no. 5 myringotomy suction tip will evacuate clots or heme during arthroscopy (Figure 49-18). The 2.0-mm cannula easily accommodates this suction.

Meniscus Mender

This packaged set consists of straight and curved spinal needles with stylet and a suture loop used to snare the suture

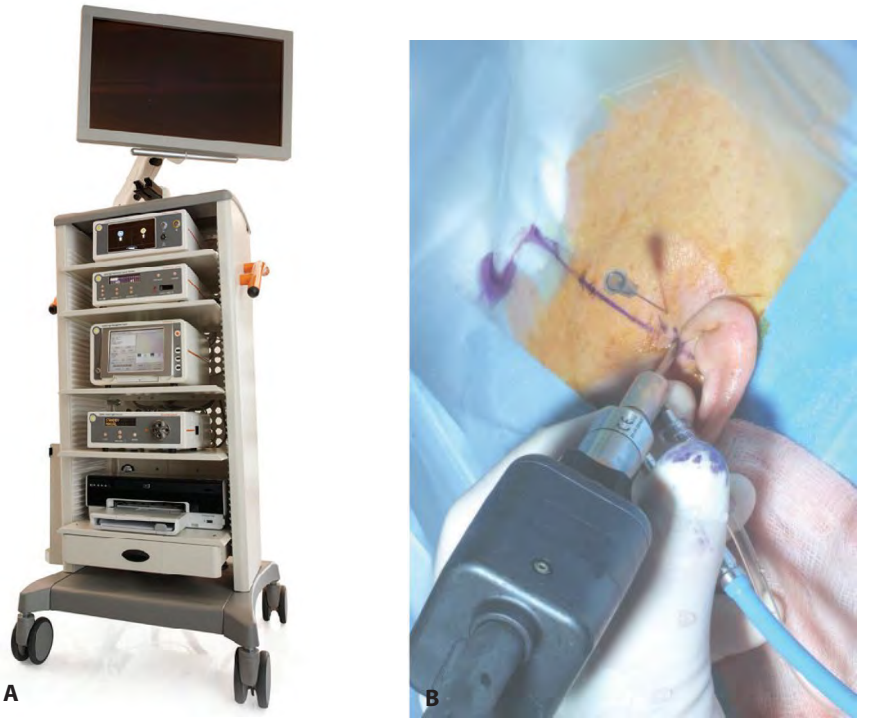


FIGURE 49-13. **A** and **B,** The Smith-Nephew System 560 Series 3-CCD.

once passed through the needle (Figure 49-19). While in the process of designing a less invasive replica of Meniscus Mender I and II, this author uses, for most situations, a 22-gauge needle to pass the suture for diskopexy procedures.

Other Hand Instruments

In the advent of the latest advancements in motorized, electrical, but most of all, laser technology, a number of hand instruments have become obsolete. The suction punch, bone rasps, curettes, banana blades, forked blades, sickles, and others are only rarely used and in very specific situations. Two instruments in this category should, however, never be missing from the arthroscopist's arsenal: the obturator and the **golden retriever** (Figure 49-20). The latter is an invaluable magnetized instrument specialized in the apprehension and delivery of intra-articular broken instruments.



FIGURE 49-14. **Left to right,** Scored cannula, sharp trocar, blunt obturator with J-locking system.

Motorized Instruments

The unique concept of motorized shavers and abraders enables suctioning of the arthroscopic field while cutting and removing tissue in an efficient manner. Four factors are paramount for these instruments to function efficiently: (1) the design of the cutting blade, (2) the pressure balance between the suction and the continuous irrigation fluid, (3) the revolution speed (rpm) of the instrument, and (4) the type of tissue that the surgeon is attempting to cut. This author has discovered that the ultimate parameters of efficiency for the shaver are reached at the lower spectrum rpm-range speed. After developing a refined intra-articular tactile sense for the instrument, the author has noticed that the most efficient "shaving" occurs with a repeated pistoning motion.

Shavers

The larger of the two shavers in use at present, 2.9 mm in diameter, is used for more aggressive arthroscopic arthroplasty. A 2.9-mm-diameter suction punch may become necessary with the use of this shaver. For the sake of maintaining the procedures in the least invasive manner, this author has, lately, used only the 1.9-mm shaver.

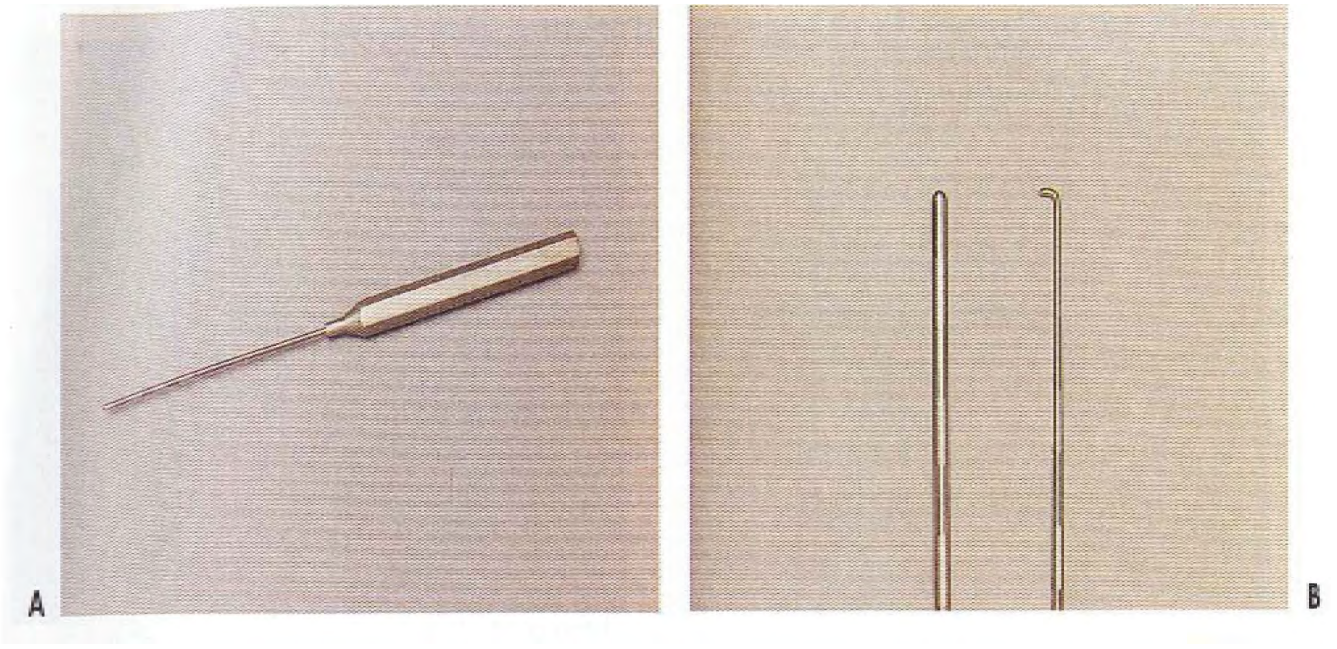


FIGURE 49-15. Probes. **A,** Straight probe (5-mm-increment gradations). **B,** Straight and hooked probes (active ends).

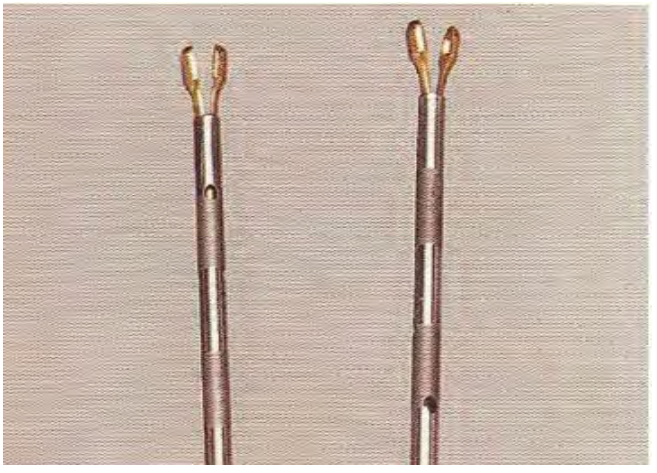


FIGURE 49-16. Nonserrated and serrated biopsy forceps.

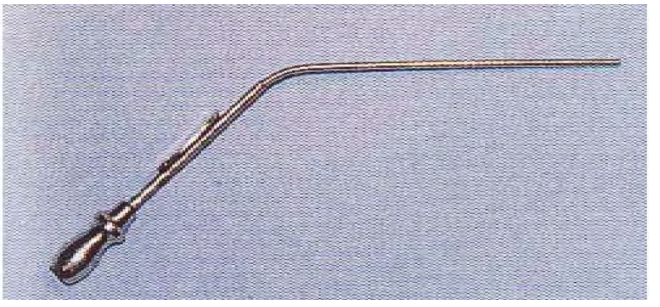


FIGURE 49-18. French no. 5 myringotomy suction tip.

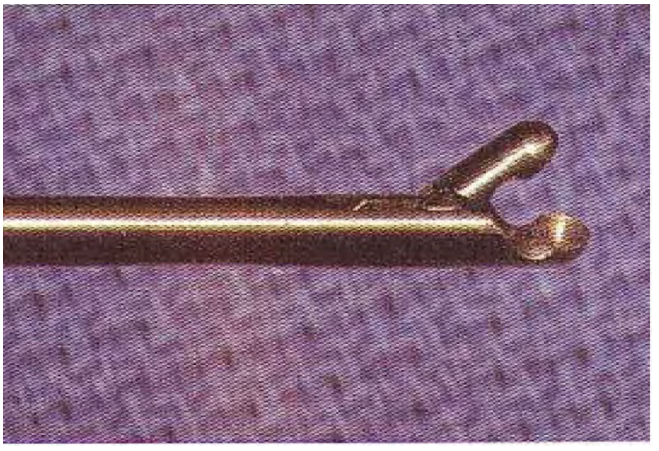


FIGURE 49-17. Basket biopsy forceps.

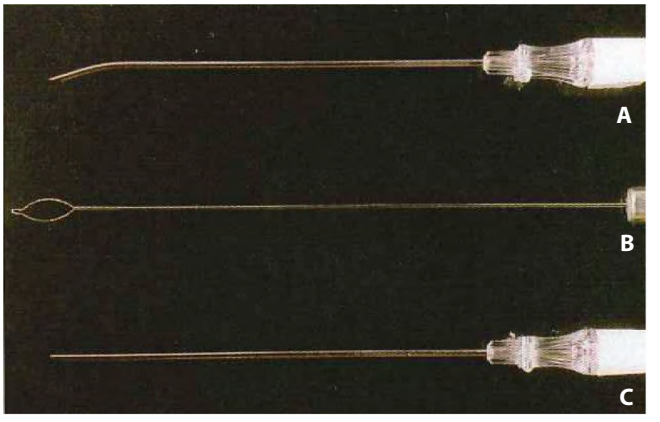


FIGURE 49-19. Meniscus mender set. **A,** Curved needle with stylet. **B,** Suture loop/snare. **C,** Straight needle



FIGURE 49-20. Golden retriever.

Whisker Shaver

The one disadvantage of this shaver is its ability to generate intra-articular iatrogeny if not controlled appropriately. It can scuff articular surfaces and cause cartilaginous defects transfixiant to the subjacent osseous structures. The instrument has been so profiled that the articular surfaces are completely protected from the cutting portion of the active part (Figure 49-21). The perforations in the external sleeve of the instrument promote a suction effect on the soft tissue that the inner blade cuts next. The whisker shaver has been designed for the débridement of fine fragmented fibrocartilage from articular surfaces, small fibrous adhesions, or small fragments of the disk periphery. It may remove fibrotic or dessicated synovial tissue; however, its use on intact/vascular synovium is discouraged.

Full-Radius Shaver

Available in 1.9 and 2.9 mm diameters, it is a more aggressive instrument designed to resect tissue of an even more fibrotic consistency, such as fibrous bands, adhesions, large areas of dessicated synovium, and fragmented edges of meniscus.

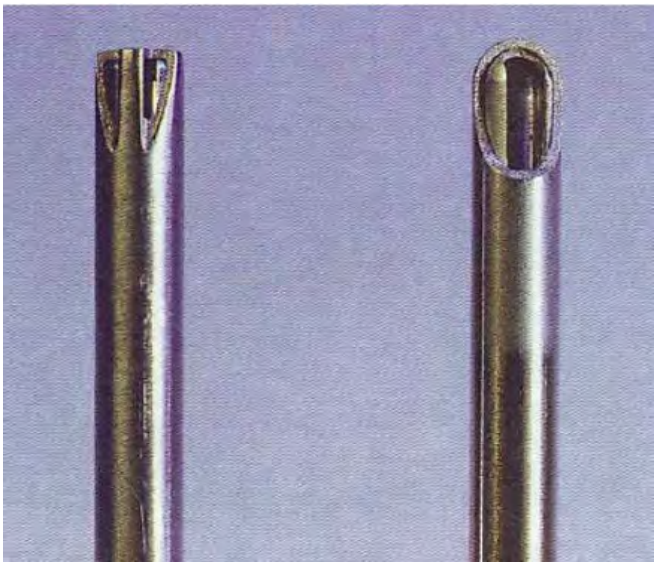


FIGURE 49-21. Whisker and full-radius shavers.

Abraders

These bone drills (the round and barrel-shaped abraders) are used to decorticate the osseous structures and create pinpoint areas of microhemorrhage in order to promote cartilaginous regeneration.

Electrosurgery

Electrosurgery is the use of high-frequency electric current to facilitate a tissue change. The human body's electrolyte composition makes it a conductor of electricity. During electrocautery, alternating current is passed to the surgical electrode probe in the form of heat. The manner in which the tissue responds to the electrothermal energy depends on the waveform of the current, the power at the electrode tip, the time of exposure at the electric tip, and cooling of tissue with blood circulation. The waveform determines the type of current used. The continuous sinusoidal waveform is used for the cutting mode, because it, mechanically, disrupts cells. The attenuated/"dampened" waveform, eventually oscillating down to resting potential, is used for the coagulating mode.

The McCain monopolar and bipolar electrocautery probes (Figure 49-22) are all insulated with the exception of the tips of the instruments. The safer, more rigid probes have the ability to manipulate tissue while delivering the electrical energy. In order not to compromise the insulation of the probes, the recommended settings of the generator should not be exceeded. Whereas common settings, based on clinical experience, exist for each tip, the individual settings may change. The intro-/extromission of the electrocautery probe at the working cannula is a very deliberate motion. Abrasion of insulation, in the process, should be avoided at all costs because it can expose another potentially active area on the

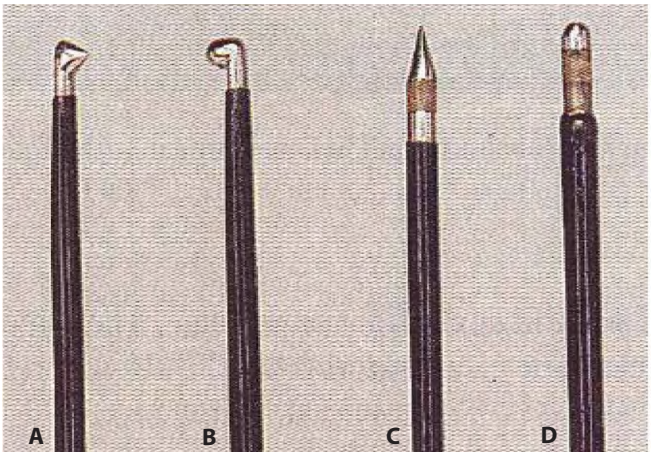


FIGURE 49-22. McCain monopolar and bipolar probes. **A,** Monopolar sharp hook. **B,** Monopolar blunt hook. **C,** Bipolar sharp hook. **D,** Bipolar blunt hook.

tip of the instrument. In this particular situation, an area of the joint can be iatrogenically injured while outside the arthroscopic FOV.

A grounding pad should be used at all times. It should self-adhere to the skin. If a grounding discontinuity occurs, the entire system shuts down. The thousands of arthroscopic procedures performed by this author, such as posterior synovial pouch cauterization, electrosurgical anterior release (McCain technique), electrosurgical synovectomy, and electrocautery for hemostasis, have afforded the experience necessary to understand the effects of electrocautery on intra-articular temperature. Extensive intra-articular thermocoupling studies have enabled the author to elaborate an irrigation protocol for these procedures. Except for the case of monopolar cautery, in which the irrigating agent is sterile water, all other procedures will undergo irrigation with lactated Ringer's solution. The reason behind using sterile water (theoretically, the safest, nonconducting medium) only for monopolar procedures stems in the hyposmolar absorption and lack of support for proteoglycan synthesis that will, eventually, translate in chondrocytomegaly. This phenomenon would very likely occur in the case of intra-articular tissues that have sustained injury and, subsequently, would maintain a higher rate of matrix synthesis than normal tissue.

Laser

Laser (light amplification by stimulated emission of radiation) technology transmits energy in the form of light beam. The laser beam is columnated, equally spaced apart and coherent in the same time and space. Laser energy is delivered in a continuous or a pulsated wave. The wavelength of a particular laser determines the physical properties of a laser in the clinical use setting.

Carbon Dioxide Laser

Laser energy in the far infrared spectrum (CO_2 laser) is avidly absorbed by water. Because tissues have a high water content, the CO_2 laser energy is readily absorbed within the superficial layer of tissue with a very localized tissue effect. However, owing to the very long wavelength, this laser cannot be passed through a flexible quartz fiber without undergoing distortion.

Holmium:Yttrium-Aluminum-Garnet Laser

This laser's wavelength is 2140 nm, making it somewhat similar to the CO_2 laser with regards to its high hydroabsorbability. The holmium laser, however, has a limited depth of penetration (0.3–0.5 mm), making it very useful intra-articularly. In this author's hands, this laser variety is the most versatile for arthroscopic techniques on the TMJ, such as anterior release, synovectomy, posterior scarification, and débridement of fibrocartilage. The small size of the delivery tip facilitates excellent access in limited spaces, while its metal encasement prevents the breakage of this quartz fiber. To this date, the holmium:yttrium-aluminum-garnet (Ho:YAG) laser is considered the most safe and effective modality for intra-articular TMJ delivery of energy.

Portals of Entry and Danger Zones

Fossa Portal

This puncture site has to be situated inside a 20-mm-diameter circle centered over the glenoid fossa (Figure 49-23A). The osseous boundaries of this circle are the apex of the articular eminence anteriorly, the middle of the acoustic meatus posteriorly, the posterior portion of the temporal fossa at the anterior segment of the supramastoid crest of the temporal squama, and finally, the posterosuperior border of the ramus at condylar neck level inferiorly. The superficial correspondent of the posterior osseous landmark is the middle of the tragus, because it is situated 5 mm anterior to the tragal apex and 5 mm posterior to the anterior wall of the osseous external auditory canal. This author's observations have placed the peak of the cartilaginous wall of the meatus at the exact level of the posterior aspect of the TMJ capsule. The neurovascular bundle (auriculotemporal nerve, superficial temporal vein and artery) is, in 80% of cases, 5 to 8 mm anterior to the midportion of the tragus. Within the limits of the glenoid fossa circular trough, the anatomic structures are, from lateral to medial, dermis, subcutaneous tissue, superficial parotid fascia, superior and posterior parotid lobule, auriculotemporal nerve, superficial temporal vein, and superficial temporal artery (see Figure 49-23B).

Anterior Eminence Portal

For teaching visualizing purposes, the same 20-mm-diameter circle can be superimposed over the anterior eminence region in the same fashion as for the fossa portal (Figure 49-24A). The boundaries for this circle are the zygomatic process of the temporal arch anteriorly, the apex of the articular eminence of the zygomatic process of the temporal bone posteriorly, the temporal fossa superiorly, and the mandibular sigmoid notch inferiorly. The anatomic structures at this site are, from lateral to medial, the tegument, the superficial parotid fascia, the superoanterior parotid gland lobule, the deep parotid fascia, and the most posterior point of intersection of the frontal branch of the facial nerve, the masseter, the masseteric nerve, artery, and vein, the anterolateral aspect of the TMJ capsule, and the lateral pterygoid (see Figure 49-24B). Measuring from the midportion of the tragus anteriorly, the frontal branch of the facial would be found ranging 13 to 40 mm with a mean of 20 to 25 mm. The zygomatic orbital branch lies anterior to the frontal branch.

Danger Zones

Before proceeding with the fossa puncture, then the diagnostic sweep, the surgeon must be cognizant of the "danger zones" and proximity of vital structures associated with this procedure. The three "danger zones" are the dura mater and temporal lobe situated immediately cranial to the glenoid fossa, with a thickness of 0.5 to 1.5 mm (Figure 49-25); the

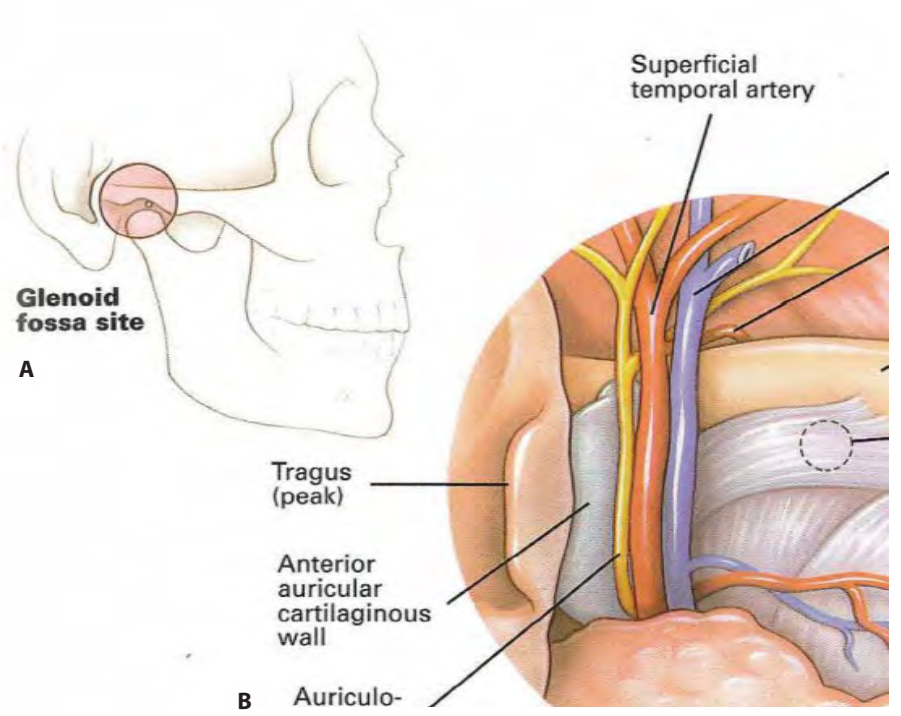


FIGURE 49-23. Fossa portal. **A,** Lateral view of the glenoid puncture site with the most important adjacent structures. **B,** Coronal view of the TMJ (lateral-to-medial) with the adjacent anatomic structures.

tympanum ossicles, and middle ear; and the entire length of the medial aspect of the capsule represented by the lateral pterygoid. The vital structures that have to be given special consideration are the mandibular division of the trigeminus, especially the lingual and inferior alveolar branches, the ATN and the internal maxillary artery together with all the small branches supplying the capsule (Figure 49-26).

Internal Arthroscopic Anatomy

Posterior Recess

The first structure encountered upon entering the joint is the synovium. The synovial membrane is mesenchymal in origin and a continuation of the cambium layer of the periosteum. Similar to other joints, the TMJ synovium covers the capsule and periarticular disk tissues, with the exception of the

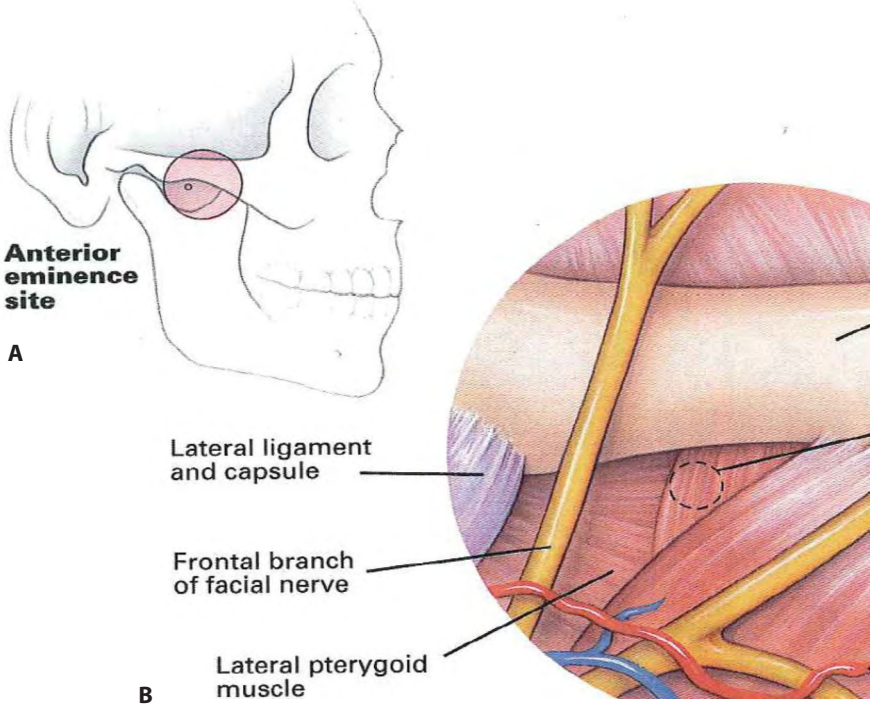


FIGURE 49-24. Anterior eminence portal. **A,** Eminence puncture site with the adjacent structures. **B,** Coronal view at the level of the articular eminence (lateral-to-medial) with the adjacent anatomic structures.

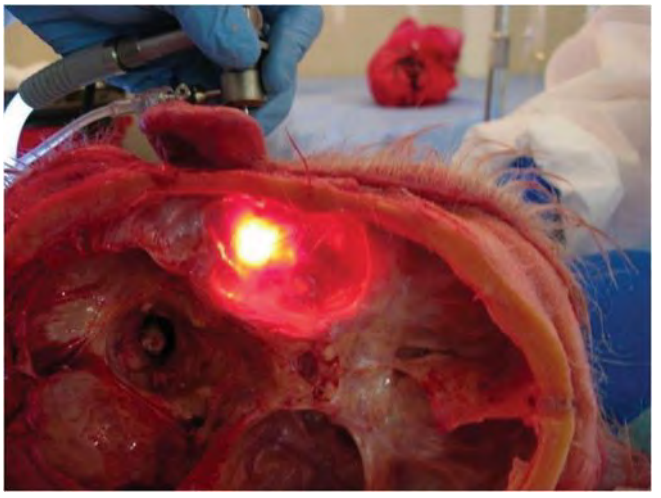


FIGURE 49-25. Danger zones. Meticulous cadaver middle cranial fossa dissection reveals the proximity of the articular glenoid fossa to the temporal lobe dura mater. Courtesy of Gary Warburton, DDS, MD

articular surfaces of the cartilages. The synovial lining is grayish with a translucent background and soft in consistency. The normal synovium has a mild amount of capillary proliferation, diffuse throughout the lining. With the condyle in a forward position (Figure 49-27), the synovium of the posterior pouch lies flat and tight over the retrodiskal tissue. The oblique protuberance is a classic structure of the posterior pouch. With condyle forward, the oblique protuberance is a fibroelastic band that protrudes into the retrodiskal tissue. It is located two thirds medial from lateral to medial into the joint. With the condyle seated, the synovium "buckles and ruffles" like an accordion being closed (Figure 49-28). The synovial tissue attaches from the back portion of the disk. The posterior band of disk running over the retrodiskal tissue attaches to the posterior aspect of the of the glenoid fossa at approximately midportion or the superior one third of the glenoid posterior wall of the glenoid fossa. The medial synovial drape appears normally as a gray, translucent synovial lining. Very distinct striae run superior to inferior, defining the medial synovium. It serves as another classic landmark in the arthroscopic examination. In the background of the medial aspect of the drape, the pterygoid shadow reflects with a red/purple tinge.

Intermediate Zone

In the middle of the joint, the fibrocartilage of the glenoid fossa is visible, thin, white, and not very reflective. Anterior to the glenoid fossa and along the back slope of the articular eminence, the fibrocartilage becomes whiter and more reflective of light and also takes on a classic appearance of striae formation that runs anterior to posterior. Changing the angle of view inferiorly, with the condyle forward, part of the disk can be seen as milky white, highly reflective of light, and without striations. With the condyle forward, the disk completely covers the condyle. The clear junction between the synovium and the posterior band of the disk is represented by a red-white line where the capillary proliferation stops and the disk begins. A U-shaped depression or flexure between the synovial juncture and the posterior band of the disk can be observed. With the condyle seated, the disk covers the condyle and the flexure deepens significantly to the normal anatomic position of the disk. Toward the most lateral depth of the joint are the articular eminence and the trough within the eminence where the disk moves with the condylar translation.

Anterior Recess

In the anterior pouch, the anterior slope of the articular eminence is evident. The fibrocartilage and anterior band of the

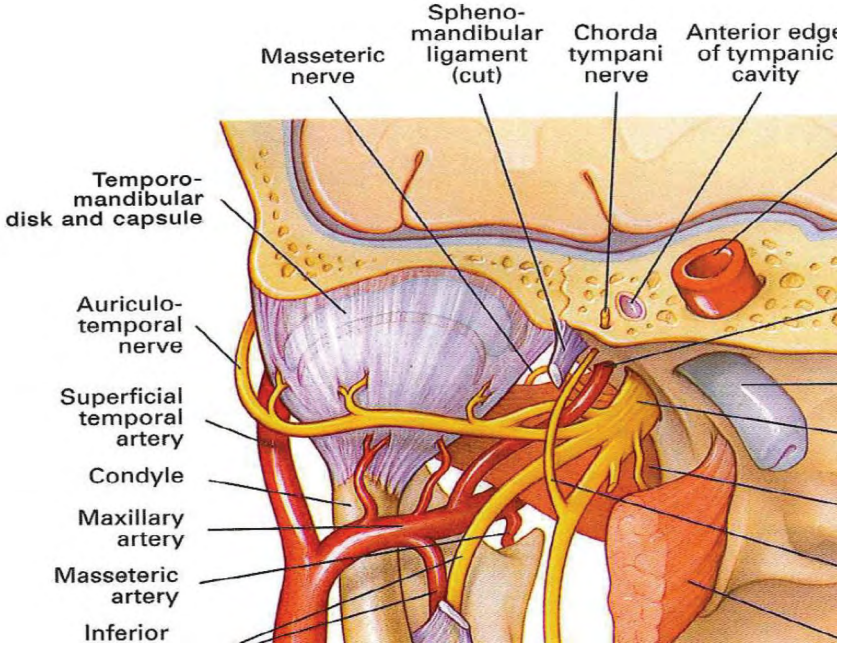


FIGURE 49-26. Posteroinferior view of the TMJ. Entry portals and reports with adjacent anatomic structures.

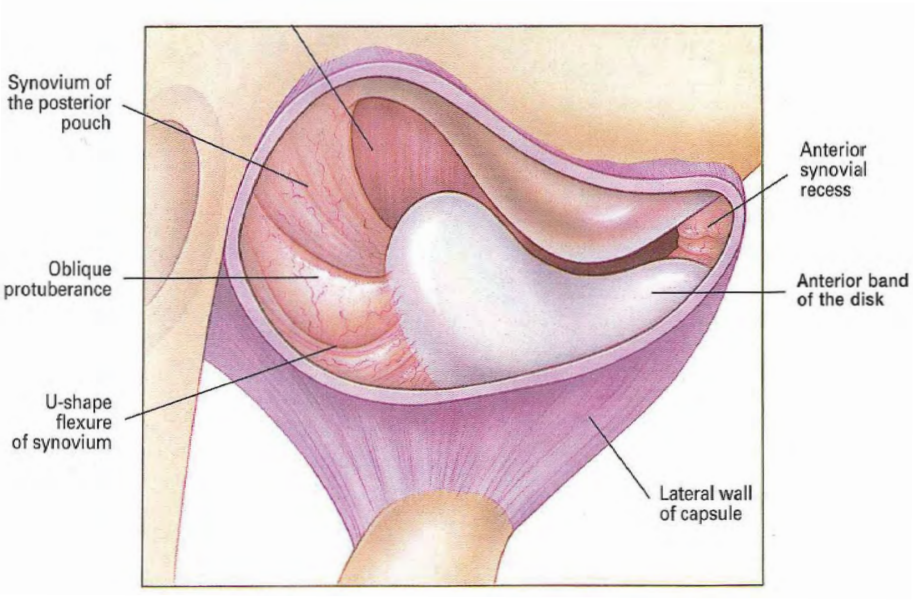


FIGURE 49-27. Intra-articular structures of the TMJ with the condyle held in the forward position (lateral view).

disk are both white, reflective, and without striae. The synovium is tightly attached and has a gray, translucent background with mild capillary proliferation widely dispersed throughout. The synovium is also attached tightly to the anterior aspect of the ascending aspect of the articular eminence. Looking medially, the continuation of the medial synovial drape can be seen. The vertical striae of the drape are nonexistent, and the background consistency is deep purple secondary to the reflection of the pterygoid muscle fibers, much closer in this area. The lateral synovium is not tightly bound down but maintains the same color as the medial synovium. The posteroanterior trough on the medial aspect of the joint is where the synovium descends off the medial synovial drape and then attaches to the disk in the same fashion as the medial trough.

Inferior Joint Space Anatomy

With the exception of very specific situations, such as existing disk perforation permitting the inferior joint space exploration without inducing any additional surgical trauma to the joint, this author does not advocate routine inferior joint space arthroscopic exploration.

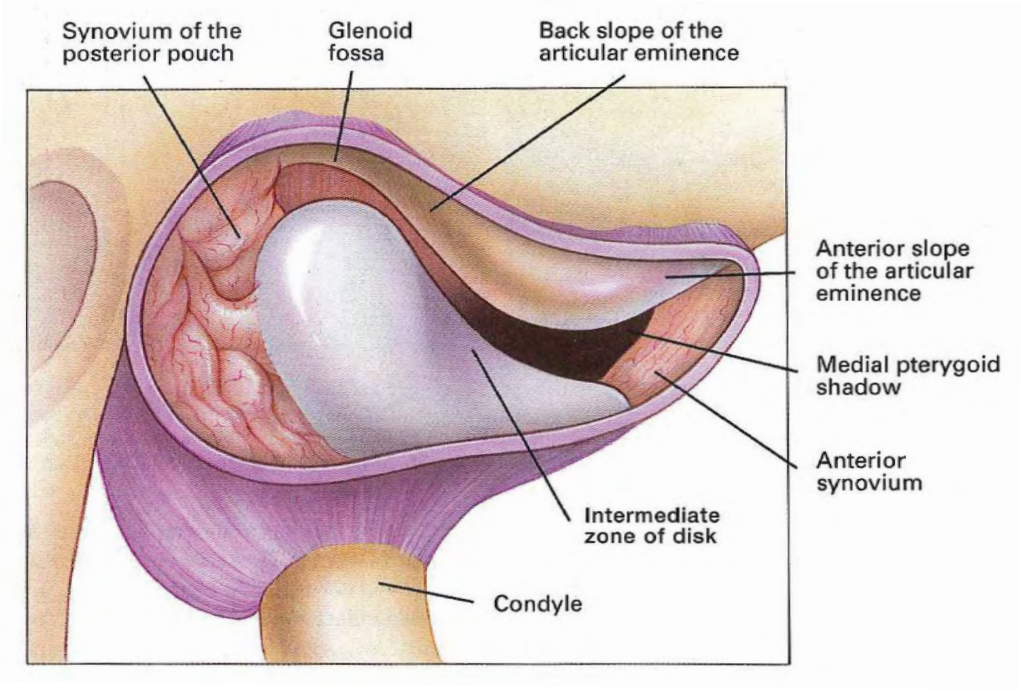


FIGURE 49-28. Intra-articular structures of the TMJ with the condyle seated into the glenoid fossa (lateral view).

Posterior Recess

In the posterior pouch of the inferior joint space, the synovium is similar in appearance, color, and capillary proliferation as previously described in this chapter. However, the consistency is softer and the synovium is not as adherently attached to the capsule.

Intermediate Zone

The inferior aspect of the disk has a more grayish appearance than the superior aspect. The striae are absent. The disk is similarly reflective. The fibrocartilage is glistening white and highly reflective, without striae.

Anterior Recess

The synovial tissue has identical consistency. It is more attached to and less mobile onto the subjacent capsule.

TMJ Arthroscopic Technique

Immediate Preoperative Steps

Examination under Anesthesia

The importance of this initial step of the procedure is paramount. It provides the surgeon with valuable information both pre- and postoperative. The preoperative examination ascertains joint mobility, articular bruits, and most important, the degree of difficulty of the upcoming punctures. Once the anesthesiologist administers the muscle relaxant, all myofascial factors related to condylar motion or disk function are eliminated. The clinician should be aware of the conditions that do not affect the mandibular range of motion independent of muscular tonus (e.g., advanced muscle fibrosis, extra-articular osseous impingements such as coronoid hyperplasia, Eagle's syndrome). Each joint is palpated separately. Normally, the condyle should subluxate the articular eminence. An inconsistent but common finding in patients with non-reducing disk derangements is the absence of this normal eminence subluxation. The postoperative dictation needs to include this particular observation pertaining to the examination under anesthesia. Bruits have to be elicited and described. By firmly pressing the condyle anterosuperior against the posterior slope of the eminence, then translating moving the condyle in all excursions, simulating mastication mechanics, clicking and crepitus can easily be achieved. A "seating-click," for instance, is used to describe a reducing disk displacement situation, when it is elicited during examination under general anesthesia.

Palpation of TMJ Anatomy

The surgeon palpates the lateral joint anatomy in preparation for the puncture while the assistant manipulates the jaw. With the condyle seated, the assistant's thumb rolls into the buccal fold away from the occlusal surfaces to allow proper seating of the condyle in maximum occlusal intercuspation. The areas to be palpated are the superficial temporal artery preauricular (puncture must be anterior to the artery), the condyle in back-and-forth and side-to-side motion of mandible, the zygomatic process of the temporal, particularly the maximum concavity of the glenoid fossa (the soft tissue depression for the fossa portal puncture is located in this area), and the articular eminence with the condyle seated.

Marking the Fossa Portal Puncture Site

The Holmlund-Helsing line is drawn with marker between the lateral canthus and the apex of tragus. A marking point is made at about the midportion of the external tragus. From this point, approximately 10 mm anterior and 2 mm inferior to the line, the maximum concavity of the fossa is located.

Insufflation

The purpose for distention of the joint in this particular case is to expand the target area. In this process, 3 mL 0.5% bupivacaine in a 3-mL syringe with a 25-gauge, $1^{1}/_{2}$-inch needle are used. A vasoconstrictor should not be used because it could mask a correct diagnostic evaluation of engorgement of capillaries or reperfusion hypoxia phenomenon reflecting synovitis. From a caudolateral position, the needle penetrates the tegument in the preauricular crease, approximately 10 mm inferior to the Holmlund-Helsing line, at the junction of the tragus and pina. The needle is aimed at the central portion of the back slope of the eminence. Bone is contacted with the tip of the needle. The average joint will take approximately 3 mL. A plunger rebound greater than 0.5 mL indicates sufficient insufflation. Adequate joint distention is indicated by the amount of pressure on the plunger. Stenosed or fibrotic joints will typically take less fluid and the pressure required to insufflate the joint is increased ("early rebound"). Hypermobile joints or joints with disk perforations without adhesions may require more fluid.

Operative Steps

Fossa Puncture

The puncture is placed at maximum concavity of the glenoid fossa. The cannula is held in the right hand for a right joint puncture or in the left hand for a left joint puncture. The index controls the tip, and the palm of the hand controls the base of the cannula. With the condyle forward, the pollicis of the nondominant hand palpates the stable zygomatic process corresponding to the maximum concavity of the glenoid fossa immediately caudal. The trocar penetrates the skin with a rotational motion. This puncture is performed in a deliberate and careful fashion, attempting one pass through the lateral capsule into the joint space. Multiple lacerations of the capsule from multiple attempts cause problems with extravasation during the course of the operation. The trocar is then advanced until contact is felt with the osseous structure superiorly. The instrument is never to be passed straight through the capsule without locating the bone. The trocar is used almost in the same fashion as a periosteal elevator after it perforates the temporalis and the periosteum at the level of the zygomatic bone (Figure 49-29). The zygomatic arch is felt between the pollicis of the nondominant hand and the index finger of the dominant hand. The trocar then steps off the ledge (Figure 49-30). The distance from the tegument surface to the ledge varies between 5 and 10 mm. If this dis-

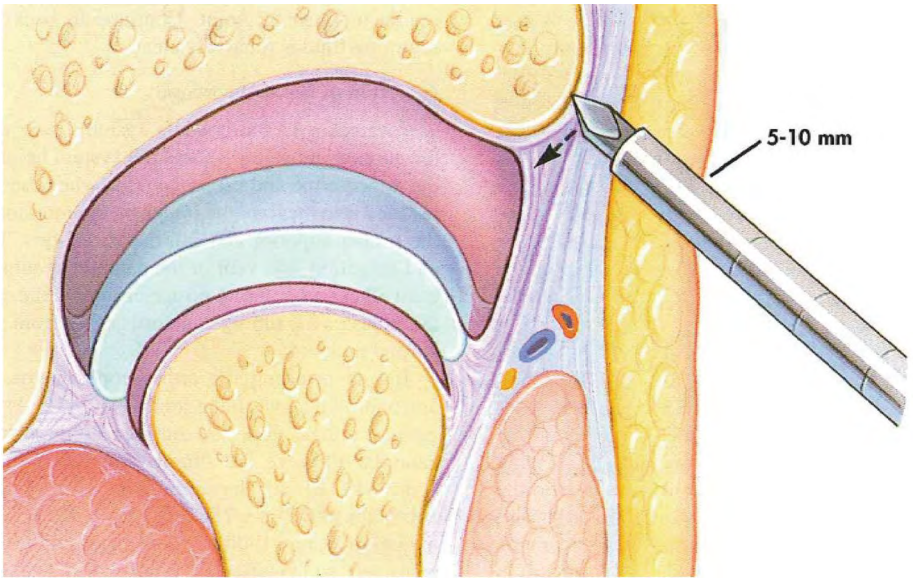


FIGURE 49-29. Cannula and trocar are advanced to the inferolateral aspect of the zygoma, then inferiorly stepped off the osseous ledge.

section is not accomplished properly, there is a high probability of invading the posterolateral subsynovial tissue. The trocar is then rotated until a slight pop is felt (Figure 49-31). It is then inserted approximately 10 to 15 mm. In the resting position, the cannula should be angled anterosuperiorly. A posterior or straight angulation could result in the laceration of the cartilaginous anterior wall of the external auditory meatus and, possibly, the perforation of the tympanic membrane and violation of the middle ear. Upon removal of trocar from the cannula, the reflux of fluid confirms perforation of the capsule. A blunt obturator is inserted and locked into the cannula. The cannula is then angled toward the open joint space. The middle portion of the cannula should, at this point, lever off the lateral aspect of the lateral margin of the glenoid fossa. The cannula should not be inserted more than 20 to 25 mm from tegument to the center of the joint (Figure 49-32). Before inserting the scope, the surgeon backwashes the joint in order to remove all blood and synovial fluid. The backwashing is continued until the return fluid is clear. The scope can be inserted next. The image on the monitor will confirm correct entry into the joint space. The image may not be very clear due to the absence of outflow.

Outflow Needle Puncture

With the mandible protruded, the scope is directed to the center of the fossa area of the joint. The assistant insufflates the joint with 2 to 3 mL of fluid in order to maintain joint distention. The purpose of the outflow needle is to establish a patent irrigation needle and, at the same time, maintain the joint adequately distended for intra-articular instrumentation. A 22-gauge, $1^{1}/_{2}$-inch needle is inserted approximately 5 mm anterior and 5 mm inferior to the fossa puncture site, under joint insufflation. Irrigation system should now be patent. An improvement in the quality of the image should be apparent.

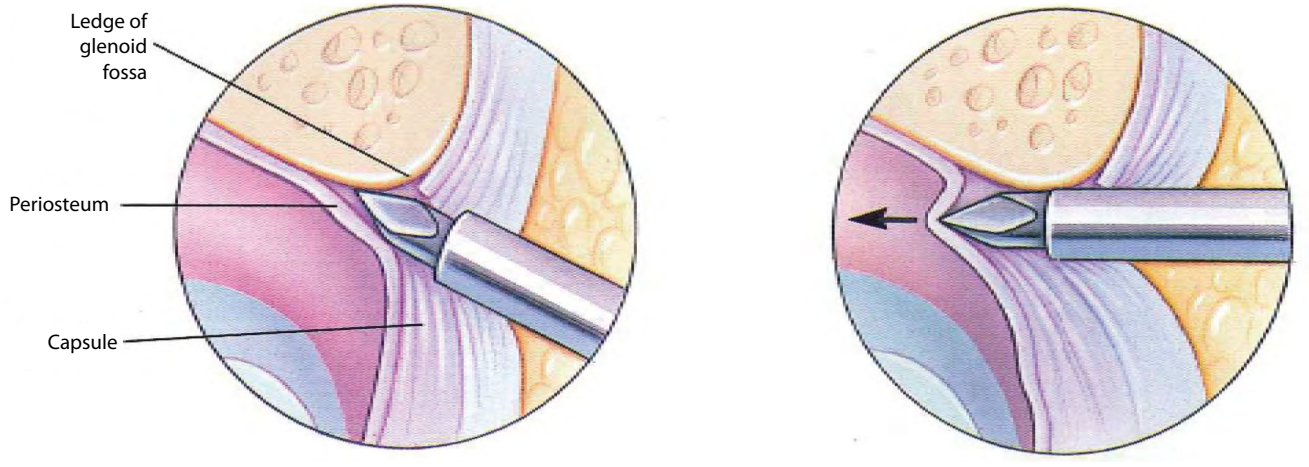


FIGURE 49-30. Cannula and trocar rotated through the capsule. Instrumentation maintains continuous osseous contact throughout this part of the procedure.

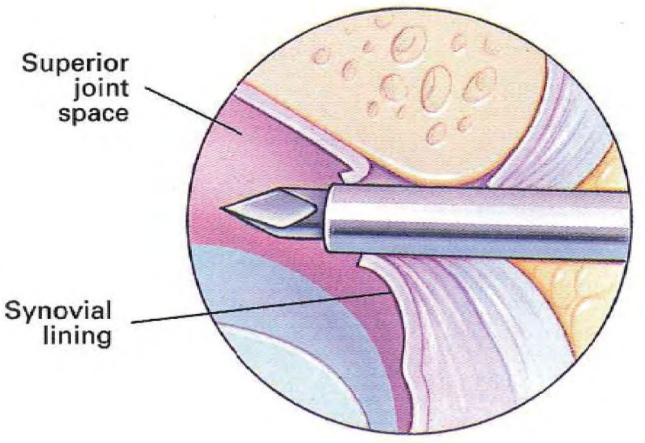


FIGURE 49-31. Capsule penetration (strictly transsynovial) using a rotation/advancement motion.

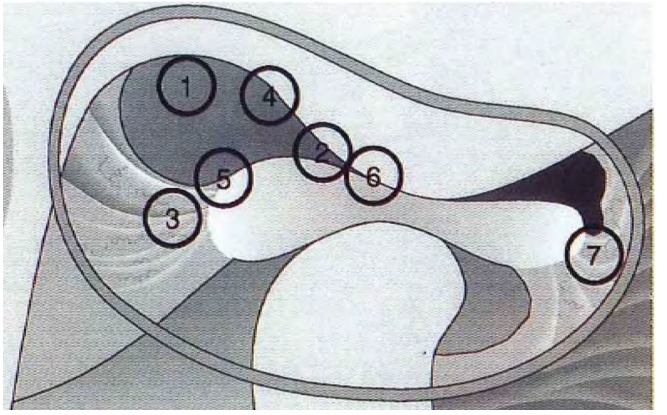


FIGURE 49-33. The seven points of interest of the TMJ arthroscopic examination. **1.** Medial synovial drape. **2.** Pterygoid shadow. **3.** Retrodiskal synovium. **4.** Posterior slope of the articular eminence and glenoid fossa. **5.** Articular disk. **6.** Intermediate zone. **7.** Anterior recess.

The system is switched next to continuous irrigation from the lactated Ringer's solution hanging bag.

Diagnostic Sweep (Seven Points of Interest of TMJ Arthroscopic Examination)

Seven anatomic areas are examined during the sweep: medial synovial drape, pterygoid shadow, retrodiskal synovium (where the oblique protuberance, the retrodiskal synovial tissue attached to the posterior glenoid process, and the lateral recess of the retrodiskal synovial tissue have to be closely inspected), posterior slope of the articular eminence and glenoid fossa, articular disk, intermediate zone, anterior recess (with special consideration for the disk-synovial crease, the midportion, the medioanterior corner and the lateroanterior corner) (Figure 49-33). Losing orientation inside the joint, even for a short time can be a very frustrating experience for the novice arthroscopist. Adding to the obstacles of inexperience, intra-articular pathology can deepen the confusion. The easiest method of preventing this occurrence is for the operator to be comfortable with the four classic intra-articular anatomic landmarks: medial synovial drape with its distinct superior-to-inferior striae, oblique protuberance of the retrodiskal synovium, posterior slope of the articular eminence with distinct anterior-to-posterior striae and anterior disk-synovial crease, and juncture of anterior synovium and anterior band of disk, which is the area for placement of a second or working cannula.

Area 1. Medial synovial drape (Figure 49-34) In many situations, the surgeon will run into one of the classic landmarks, typically, the oblique protuberance or the posterior slope of the articular eminence. The drape can be reached by

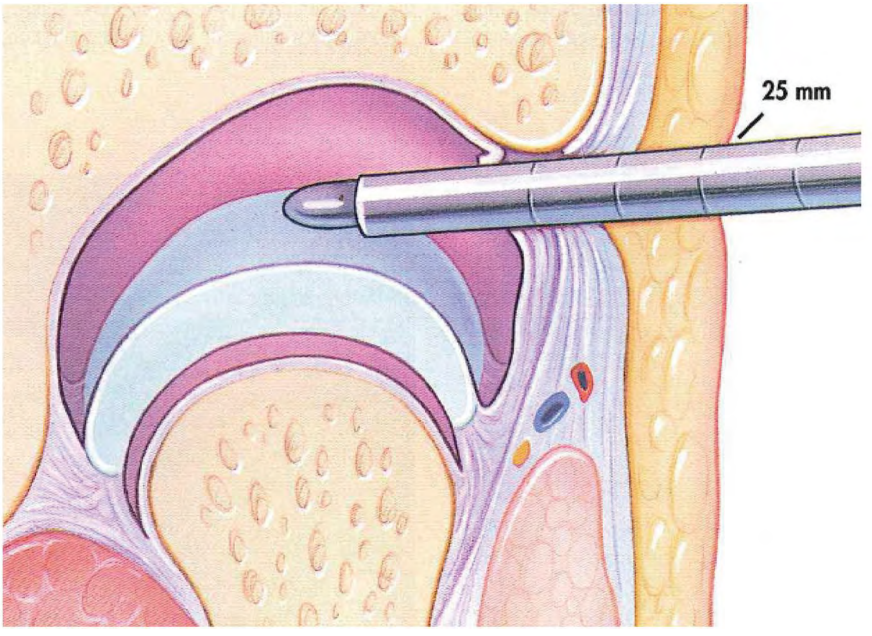


FIGURE 49-32. Cannula advancement to the open intra-articular space to the 25-mm mark.

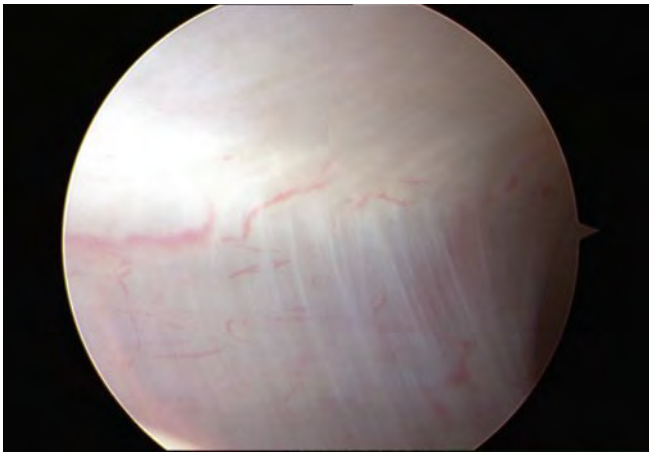


FIGURE 49-34. Medial synovial drape (normal arthroscopic appearance).

swiveling and pistoning the arthroscope until the drape becomes visible. This particular articular entity represents one of the most important barometers of TMJ synovitis. In acute inflammatory states, capillary proliferation with hyperemia of the medial synovial drape is increased (Figure 49-35). In addition, erythematous patches (petechiae) may be seen on the drape (Figure 49-36), or the entire drape may have an erythematous appearance (Figure 49-37). Occasionally, an in-bulging or prolapsing of the drape into the joint space occurs. Adhesive phenomena can also be seen (Figure 49-38). In chronic synovitis, the drape has a fibrotic or whitish appearance (Figure 49-39).

Area 2. Pterygoid shadow This second area to be examined will be reached by swiveling the scope anteriorly and pistoning medially until the shadow comes into view. A medial trough leads from the medial synovial drape anterior to the pterygoid shadow. The normal-appearing pterygoid shadow has a purple tinge attributed to the superior head of the lateral pterygoid subjacent to the synovial lining (Figure 49-40). Also, an obvious, well-delineated color difference is present between the drape and the shadow. During pathologic states, the pterygoid shadow takes on the appearance of marked erythema and marked hypervascularization and becomes quite thin (Figure 49-41). The synovial lining can

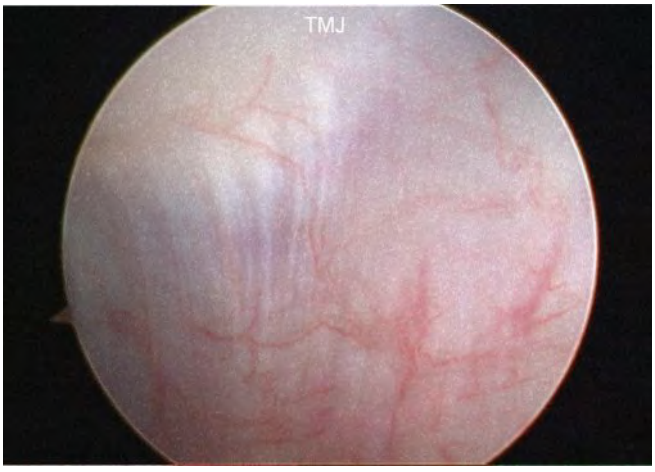


FIGURE 49-35. Acute synovitis with capillary proliferation and hyperemia at the medial synovial drape.

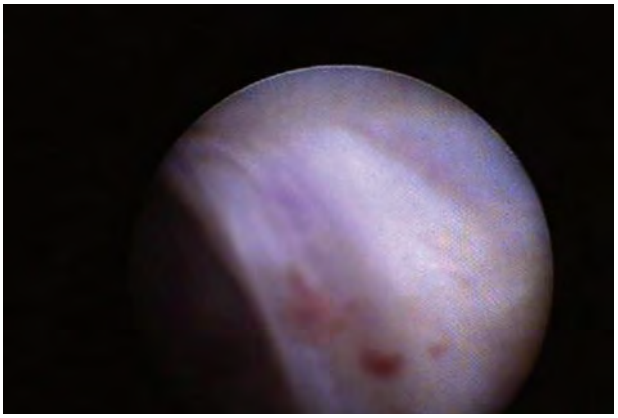


FIGURE 49-36. Petechial synovitis at the medial synovial drape.

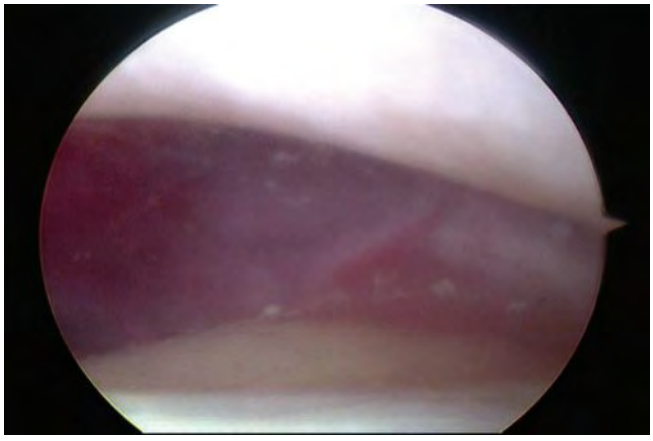


FIGURE 49-37. Medial synovial drape erythema.

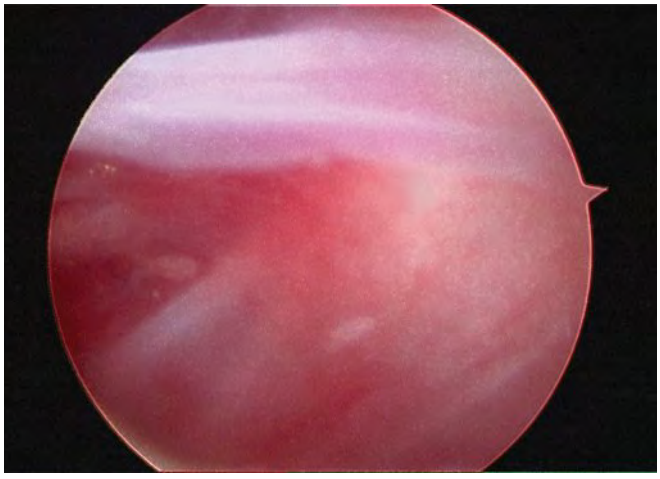


FIGURE 49-38. Adhesive phenomenon and acute synovitis at the medial synovial drape.

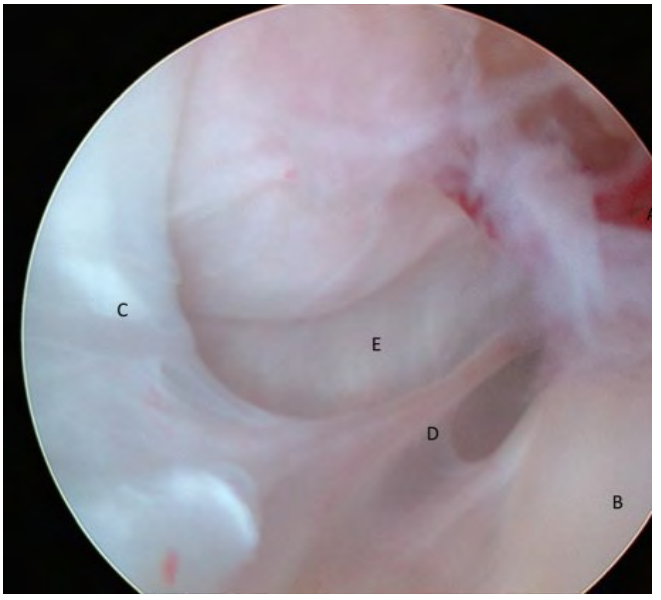


FIGURE 49-39. Chronic synovitis with a fibrotic medial synovial drape. **A,** Scuff (1–2 o'clock). **B,** Posterior band of disk (4–5 o'clock). **C,** Fibrotic retrodiskal synovium (7–11 o'clock). **D,** Horizontal adherence (band) from retrodiskal tissue across the medial synovial drape. **E,** Fibrotic medial synovial drape.

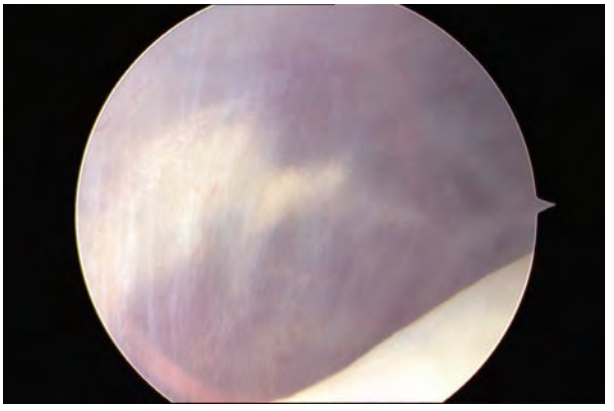


FIGURE 49-40. Pterygoid shadow (normal arthroscopic appearance).

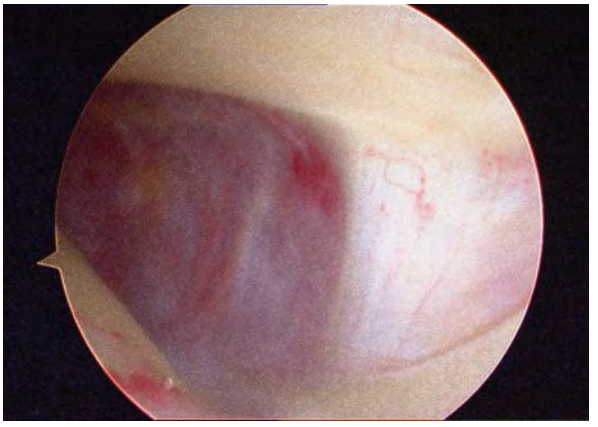


FIGURE 49-41. Erythema at the pterygoid shadow.

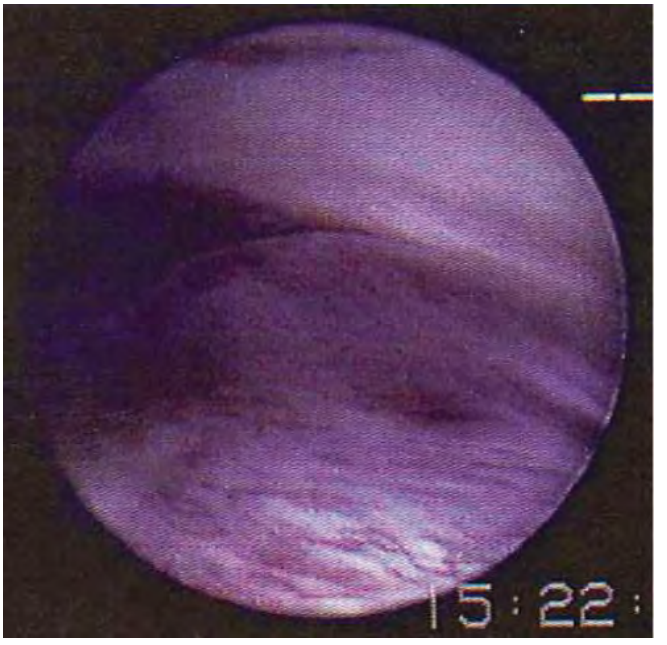


FIGURE 49-42. Intra-articular prolapse/herniation of the lateral pterygoid secondary to erosion/perforation of inflamed synovial lining at area no. 2.

thin to the extent at which it causes a perforation of this area and herniation of the pterygoid muscle directly into the anteromedial aspect of the superior joint space (Figure 49-42).

Area 3. Retrodiskal synovium This third area to be examined is reached by backtracking the initial path of the scope. Once the medial synovial drape is visible, the scope is pistoned-out (lateral) and swiveled minimally to bring into view both anterior and posterior components of the retrodiskal synovium (Figure 49-43). The arthroscopically normal retrodiskal synovium with pertaining structures have been previously described. In inflammatory pathologic states, the synovial tissue takes on the appearance of increased hypervascularity and erythema along with a redundant pattern.

Zone 1: Oblique protuberance: This area is visualized by pistoning-out the scope. It is located about one third of the way lateral from the drape (Figure 49-44).

Zone 2: Retrodiskal tissue attached to posterior glenoid process: This area takes a superior swivel of the scope to visualize (Figure 49-45).

Zone 3: Lateral recess of the retrodiskal synovial tissue: This area can be accessed by pistoning-out from the oblique protuberance (see Figure 49-45). In this zone as well, the pathologic states induce a hyperemic or petechial appearance of synovium (Figure 49-46).

Area 4. Posterior slope of the articular eminence and glenoid fossa This fourth area of the intra-articular examination is reached in the following manner. From the lateral recess the scope is pistoned-out until the periphery of the capsule is visible. From there, the scope is advanced so



FIGURE 49-44. Oblique protuberance. **A,** Oblique protuberance. **B,** Articular disk. **C,** Articular tubercle.

that the capsular fragmentation is no longer visible. To examine the fibrocartilage of the back slope of the eminence, the scope is pistoned-in to the most medial aspect of the articular eminence then slowly pistoned-out, backtracking the last travel path. The fibrocartilage of this area has a distinct, classic white and highly reflective appearance with anteroposterior striae within the fibrocartilage (Figure 49-47). As it tapers toward the glenoid fossa, the fibrocartilage becomes darker (more brownish) and thinner. The arthroscopist has to give special consideration to this area because it is the most prone to iatrogenic injury (scuffing) during intra-articular scope manipulation. The fibrocartilage over the bulk of the posterior slope of the articular eminence is significantly thicker compared with the glenoid fossa. The pathology occurring often in this area is various stages of

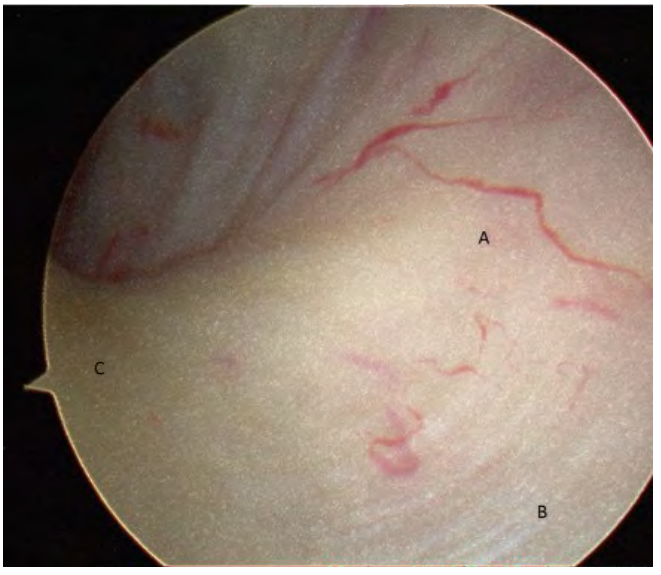


FIGURE 49-45. Retrodiskal tissue. **A,** Retrodiskal tissue of the left TMJ. **B,** Lateral recess of retrodiskal tissue. **C,** Posterior edge of disk.



FIGURE 49-46. Petechial synovitis in the lateral recess.

chondromalacia. This important entity is discussed in detail later in this chapter. To complete the examination of this area, the scope is swiveled superiorly and posteriorly. From that position, pistoning-out to the joint periphery will permit visualization of the glenoid fossa. Normal fibrocartilage over the glenoid fossa is thin, white, and without striae. When destruction (thinning) of the fibrocartilage is advanced, the underlying bone appears slightly yellow or brownish. In inflammatory states, creeping of the synovial tissue can be observed in the glenoid fossa (Figure 49-48) and the posterior slope of the eminence (Figure 49-49).

Area 5. Articular disk When the examination of the glenoid fossa is complete, the scope should be at the extreme periphery of the joint and in position to examine the fifth area. From this posterolateral position, the posterior band of disk is located. The posterior band should always be visible in the posterior recess. With the condyle forward, the inspection proceeds in an anterior and inferior direction from

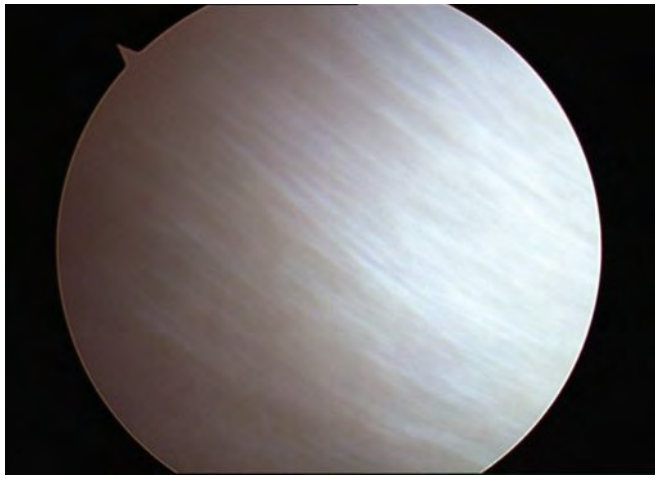


FIGURE 49-47. Posterior slope of the articular eminence.

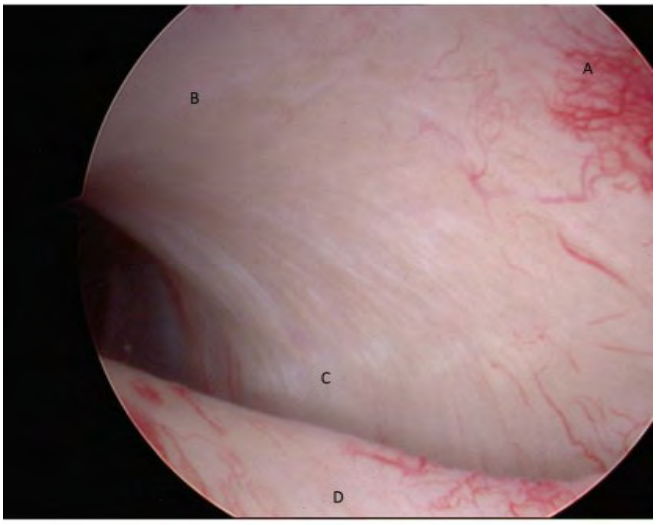


FIGURE 49-48. Glenoid fossa creeping synovitis phenomenon. **A,** Creeping synovitis at the fossa. **B,** Posterior slope of the eminence fibrocartilage of normal aspect. **C,** Medial synovial drape. **D,** Retrodiskal tissue with synovitis.

this peripheral position. The normal disk anatomy has been previously described (Figure 49-50). In pathologic states, the synovium creeps onto the surface of the disk (Figure 49-51). Fragmentation of the disk surface (Figure 49-52) is usually an indication that a perforation of the disk is either imminent or present (Figure 49-53). In cases of disk perforation, the inferior joint space can be examined by introducing the scope through the perforation into the inferior joint space.

Joint dynamics and disk mobility: The next step of the diagnostic sweep is to examine dynamics and mobility from the posterolateral position. The scope needs to be at the most peripheral posterolateral position, almost exiting the joint space. If the operator does not ensure the appropriate position of the scope in this instance, damage to the retrodiskal tissues and the disk may occur along with scuffing of the

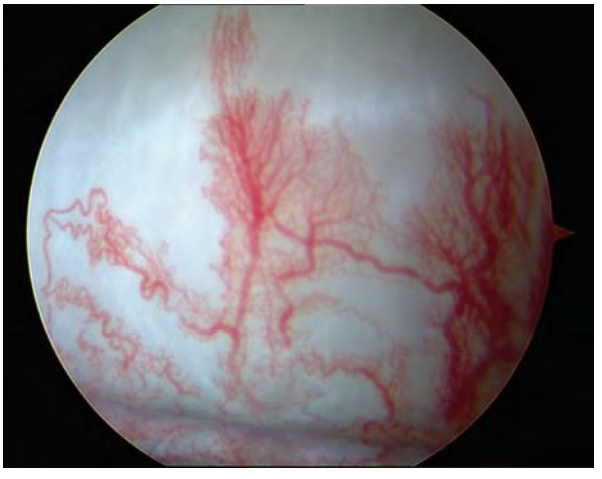


FIGURE 49-49. Creeping synovitis phenomenon on the fibrocartilage of the posterior slope of the articular eminence.

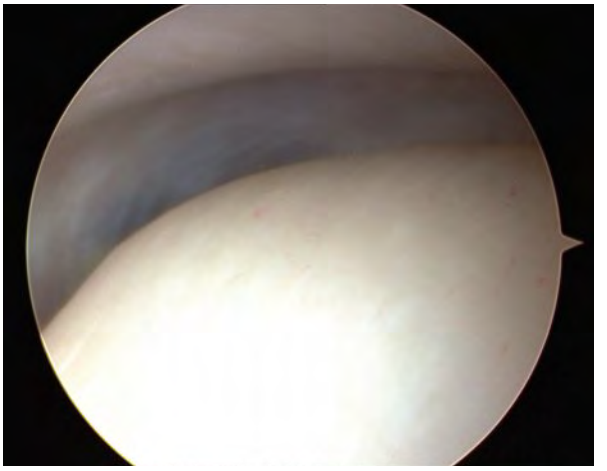


FIGURE 49-50. Normal articular disk.

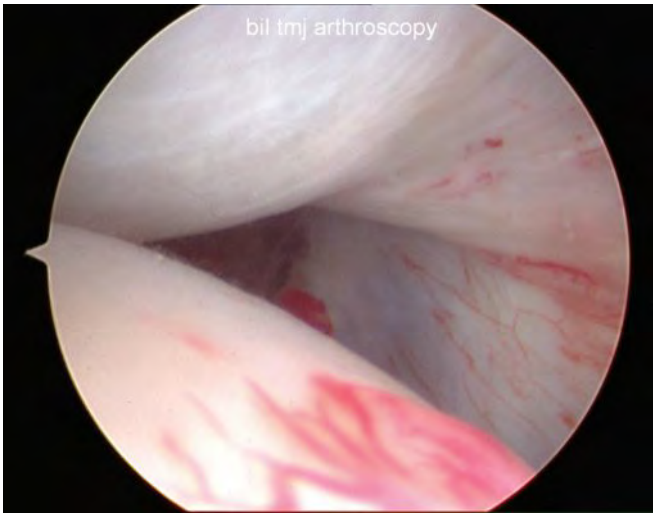


FIGURE 49-51. Posterior band of disk creeping synovitis phenomenon (at 6 o'clock). Less prevalent creeping synovitis at the drape and fossa is noted in the same image.

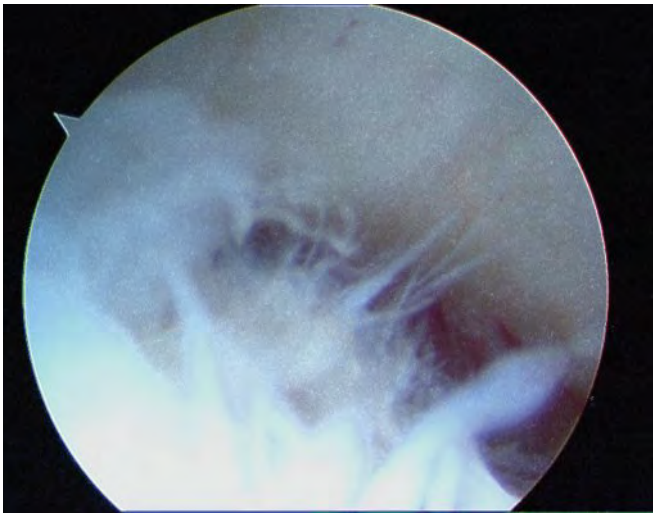


FIGURE 49-52. Disk surface fragmentation (leading to perforation).

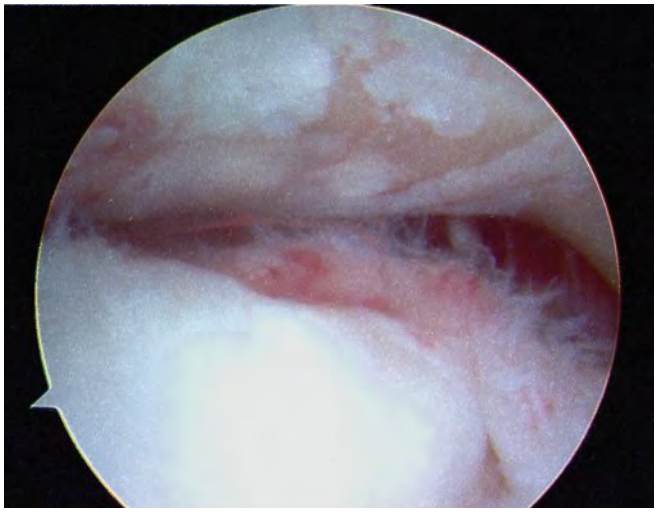


FIGURE 49-53. Disk perforation (also note the denuded osseous structure on the posterior slope of the eminence and fossa, consistent with stage IV chondromalacia).

glenoid fossa and the back slope of the eminence. At this stage in the operation, the assistant manipulates the condyle in a seated position in a reciprocal fashion, together with the surgeon**.** Observation of disk mobility between the fossa and the disk can now be ascertained and disk mobility between the condyle and the disk can be inferred. In nonpathologic states, the disk should glide smoothly along the articular eminence in a fluid movement, without any anteroposterior or mediolateral erratic movements. If an erratic movement is noted in the anteroposterior direction with a simultaneous audible or palpable clicking phenomenon, then a reducing disk is the most likely situation.

Arthroscopic assessment of disk position: This is possible whether or not the disk is in a normal position. In normal arthroscopic anatomy, the posterior band of the disk lies adjacent to the back slope of the fibrocartilage of the articular eminence and the glenoid fossa when the condyle is in the forward and seated positions, respectively. To indentify the reducing disk, arthroscopic observation is performed first with the condyle forward. In the seated position, the retrodiskal synovium comes into view. The non-reducing disk is determined arthroscopically by observing the roofing of the disk with the condyle forward. In non-roofing situations, the disk is not reducing.

Roofing: This concept was developed to evaluate the covering of the articular disk over the condyle when the condyle is either forward or seated. An attempt is made to specifically grade the amount of displacement by arthroscopic observation of the disk. The first observation of roofing is made with the condyle forward (Figures 49-54 to 49-56). When viewed in physiologic disk position, with the condyle 100% roofed by the disk, the posterior band of the disk can be seen lying adjacent to the posterior slope of the articular eminence, thus giving a white-on-white appearance. The disk flexure with its U-shape is prominent; the retrodiskal synovial tissue appears

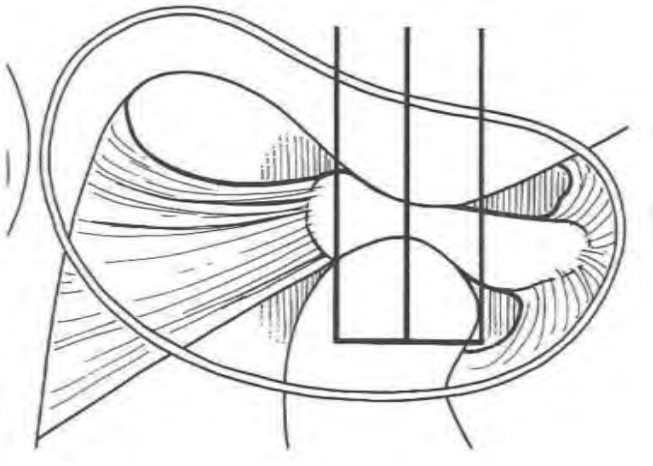


FIGURE 49-54. Condyle forward with 100% roofing.

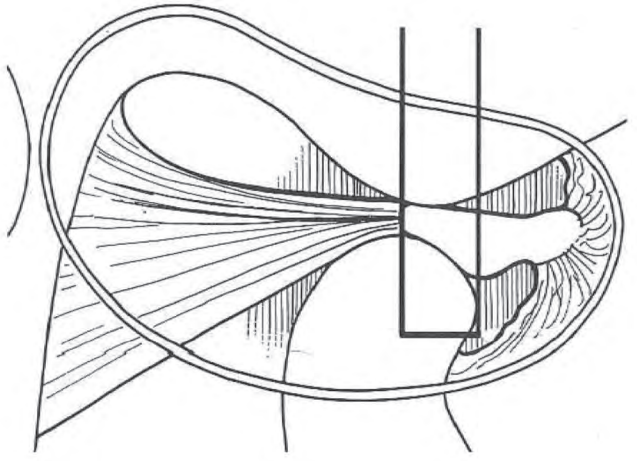


FIGURE 49-55. Condyle forward with 50% roofing secondary to anterior disk displacement.

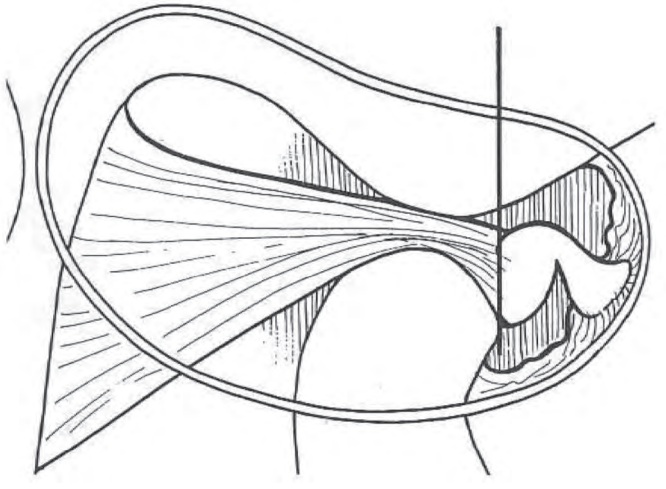


FIGURE 49-56. Condyle forward with no roofing (0%) secondary to complete disk dislocation.

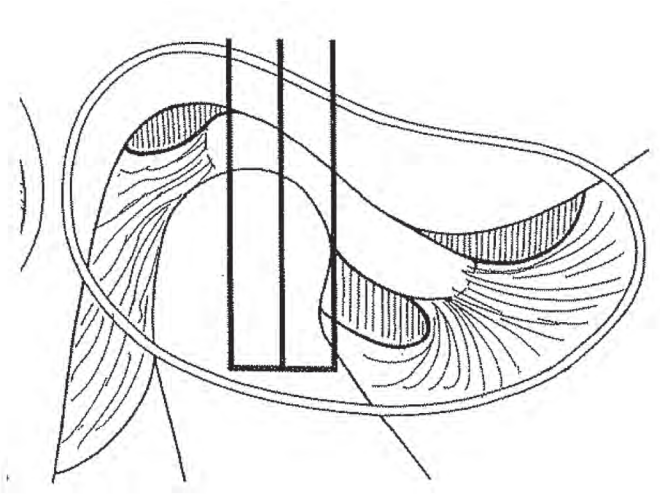


FIGURE 49-57. Condyle seated with 100% roofing.

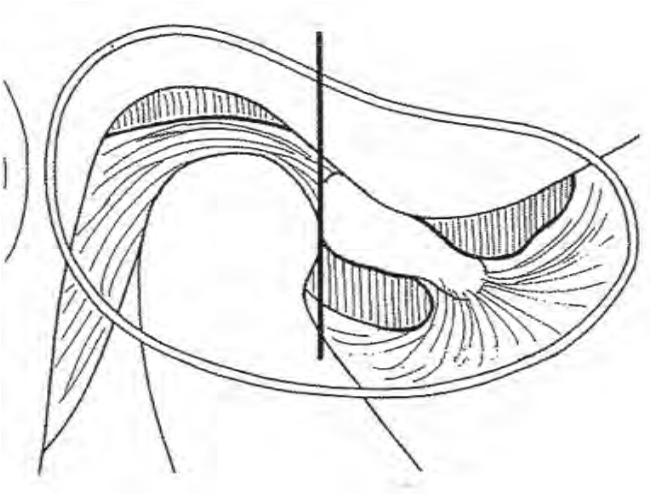


FIGURE 49-59. Condyle seated with 0% roofing (complete disk displacement).

normal, without hypervascularity. The second observation of roofing is made with the condyle seated (Figures 49-57 to 49-59). This assessment describes the degree of displacement in reducing the disk. Gradations of roofing are ascertained by the arthroscopic view of the posterior band of disk as it abuts the midportion of the glenoid fossa and the articular eminence. In nonpathologic states, with the condyle 100% roofed, the posterior band of the disk abuts at approximately the midportion of the glenoid fossa or just at the beginning of the back slope of the articular eminence. When evaluating the degree of roofing with the condyle seated, occasionally there are some technical difficulties. In certain instances, the condyle cannot be completely seated into the glenoid fossa because it is obstructed by the tip of the arthroscope, notwithstanding the capsular peripheral position of the scope. In abnormal situations of disk displacement and marked redundant retrodiskal synovium, the arthroscopic view quickly becomes obstructed by the redundant synovium, even though the condyle is seated and the position of the scope maintained. These factors are valuable diagnostic indicators that disk displacement in the seated position is occurring.

Area 6. Intermediate zone In order to examine this sixth area, with condyle forward, the scope is pistoned to facilitate placement at approximately 1 mm away from the interface between the juncture of the articular eminence and the articular disk. From this 11 to 1 o'clock position, the scope contours the path of the articular eminence. In normality, this area should have a complete white-on-white appearance, with the fibrocartilage, cranial, white and the disk, caudal, also white (Figure 49-60). The scope is swiveled, first anteriorly, contouring down the articular eminence, then laterally, positioning it in the lateral synovial trough while observing the intermediate zone. With the condyle forward, the scope is pistoned and swiveled anteriorly as far as permitted. With disk displacement without reduction, it is paramount to note when disk tissue is first observed. The degree of roofing can be assessed by comparing the white

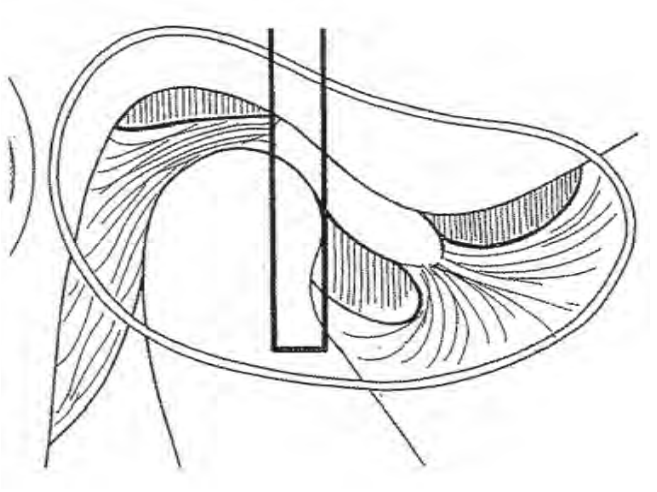


FIGURE 49-58. Condyle seated with 50% roofing (anterior disk displacement).

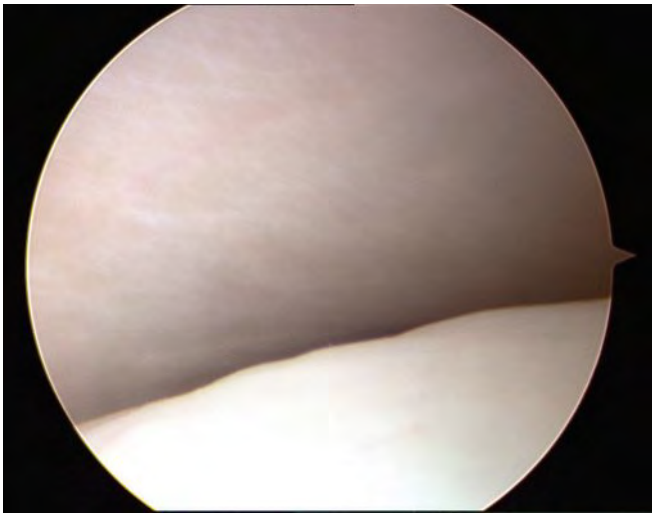


FIGURE 49-60. Intermediate zone.

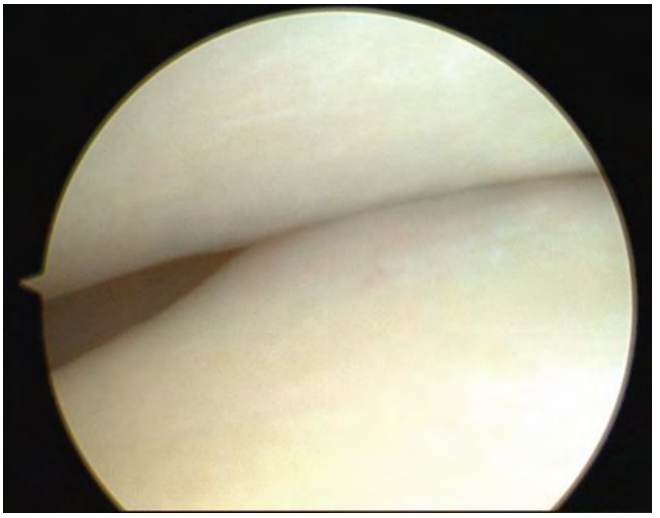


FIGURE 49-61. Anterior triangle of the intermediate zone and anterior recess.

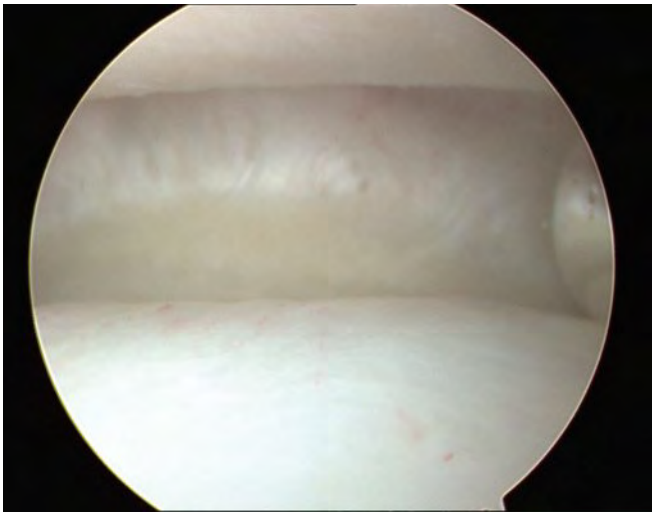


FIGURE 49-63. Anterior recess anterior limit (note puncture bubble at 3 o'clock).

fibrocartilage, cranial, and the red retrodiskal synovium, caudal. The scope is then pistoned as far anterior as permitted to the apex of the articular eminence. When the arthroscope cannot be negotiated any more anterior, the condyle is seated. A triangle reflecting an open space and pathway to the anterior recess should be visualized. The triangle is limited by the apex of the anterior slope of the articular eminence, the articular disk inferiorly, and the beginning of the anterior synovium anteriorly (Figures 49-61 and 49-62). Seating the condyle and pistoning the scope into the anterior recess must be precisely coordinated.

Area 7. Anterior recess This seventh area is the last one to be examined. Examination can only begin after the condyle is seated and the anterior triangle identified. The arthroscope is pistoned directly into the anterior recess until the midportion of the anterior synovium is visible. At this point, by swiveling and pistoning the scope slightly posteriorly as well as inferiorly, the anterior disk-synovial crease is identified. The crease is examined by following it to the terminal medial point, the most extreme medioanterior corner of the crease and the pterygoid shadow (Figure 49-63). The scope is then pistoned-out along the synovial crease to the periphery of the capsule. At the lateral and anterior limit, the juncture between the anterior disk-synovial crease and the lateral synovial capsule can be viewed. In pathosis, the vascularity of the anterior synovial pouch increases and all characteristics of inflammation of synovium are present. Occasionally, synovial redundancy and synovial plicae (Figure 49-64) are also present. Creeping of synovium onto the articular eminence can also be seen. The disk below in the anterior recess may appear "buckled," with the anterior band becoming prominent (Figure 49-65).

Manipulation under anesthesia: A slight increase in the level of sedation will be necessary for this step of the

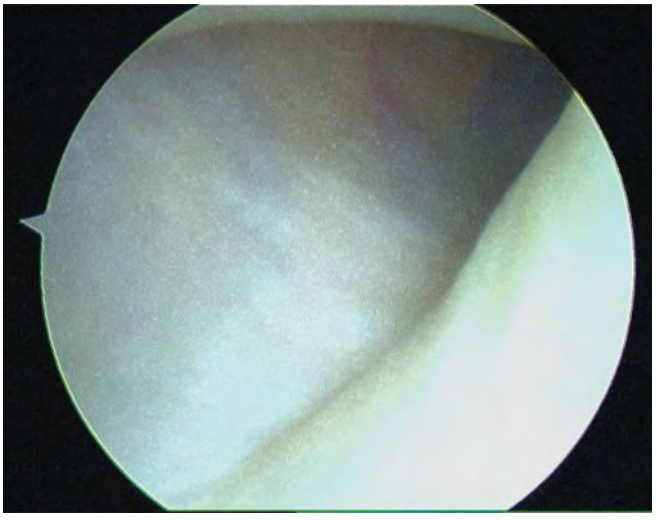


FIGURE 49-62. Anterior triangle (close up).

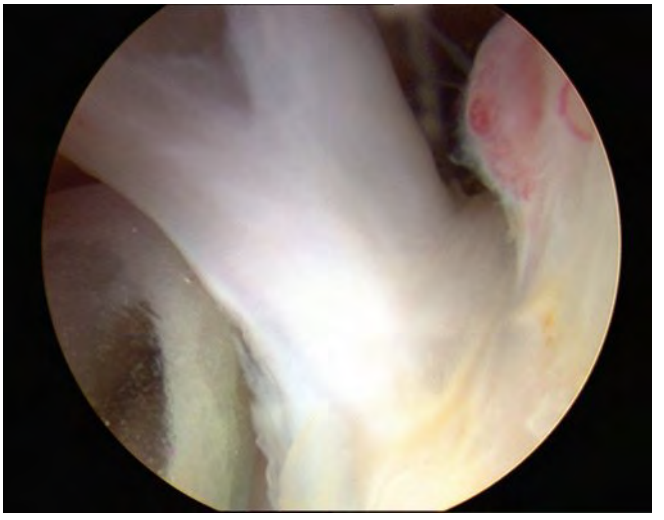


FIGURE 49-64. Fibrous bands/adhesions.

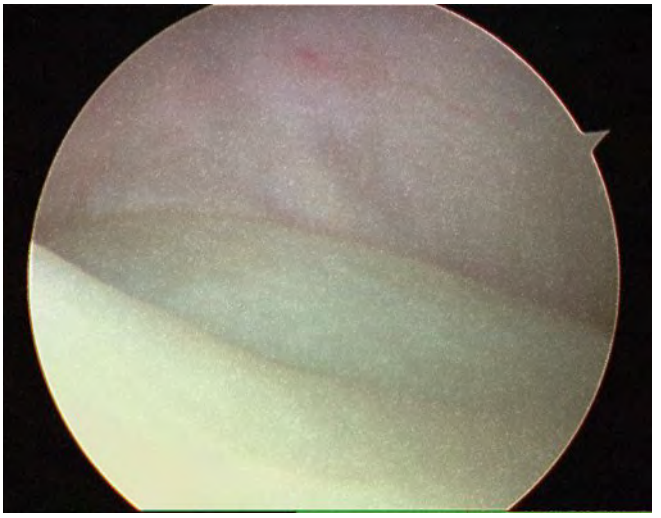


FIGURE 49-65. Anteromedial disk displacement/"buckled" disk.

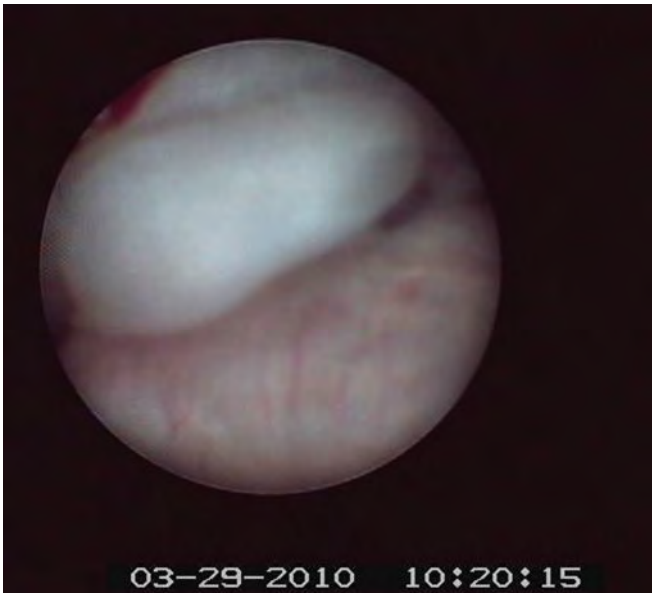


FIGURE 49-66. Villonodular synovitis (On Point Biomet).

procedure for most patients. A relatively cooperative patient is preferred. Unassisted first, then assisted maximal MIO are performed. When the patient fails to open at least 35 to 40 mm, the surgeon executes a very gentle "pry bar" assisting in the achievement of an acceptable MIO. This manipulation not only provides the initial step in the patient's postoperative rehabilitation/physical therapy (PT) but also represents a prediction factor for the postoperative course of the case. One of the pertinent observations we have made over the years is that, in cases of Wilkes III and IV, pain at the end-range of opening is a high predictive factor for diskopexy. In other words, notwithstanding the reversal of these patients to "no clicking" or a Wilkes I, these particular entities usually relapse to a stage II, III or II to IV, making disk repositioning/diskopexy a necessity as the next step of the arthroscopic cascade.

Intra-articular Pathology

Before continuing with the description of surgical therapeutic arthroscopic techniques, a review of intra-articular pathology entities is necessary to correlate with the diagnostic findings.

Synovitis

The acute form consists of acute inflammation with dilated superficial capillaries without hyperemia in the early stages, progressively increasing hyperemia in the early stages, and total obliteration of the superficial vascularity in the most severe stages. For clinical description purposes, it has been staged 1 to 4, with 2 and 3 for the intermediate stage. The chronic form is characterized by synovial hyperplasia with increasing proliferation of tissue folds, particularly in the retrodiskal area.

Fibrosis

Intra-articular hemorrhage results in the formation of a fibrin scaffold for the fibroblasts to migrate upon and produce fibrous adhesions. In addition, pseudodigit villous proliferations are frequently present. Fibrosis can present as fibrous adhesions or total synovial fibrosis.

Villonodular Synovitis

This entity consists of diffuse pigmented multiple nodular villi, which can involve the entire synovium (Figure 49-66). Typically, it occurs after prolonged inflammation stages, most commonly seen in conjunction with osteoarthritis.

Synovial Chondromatosis

This rare disease of the synovial membrane presents with multiple osteocartilaginous bodies attached to the synovium and others floating freely in the joint space. This rare entity deserves to be documented with the appropriate imagery: Panorex (Figure 49-67), computed tomography (CT) views (Figures 49-68 and 49-69), three-dimensional reconstruction images (Figures 49-70 and 49-71), and arthroscopic images of the three stages of the disease (Figures 49-72 to 49-74).

Rheumatoid Arthritis

RA is a systemic disease of undetermined etiology. It is considered an autoimmune disease with a rheumatoid antibody factor, anti-immunoglobulin G (IgG), present in 85% to 90% of patients. From a TMJ perspective, it is characterized by



FIGURE 49-67. Synovial chondromatosis (Panorex view).

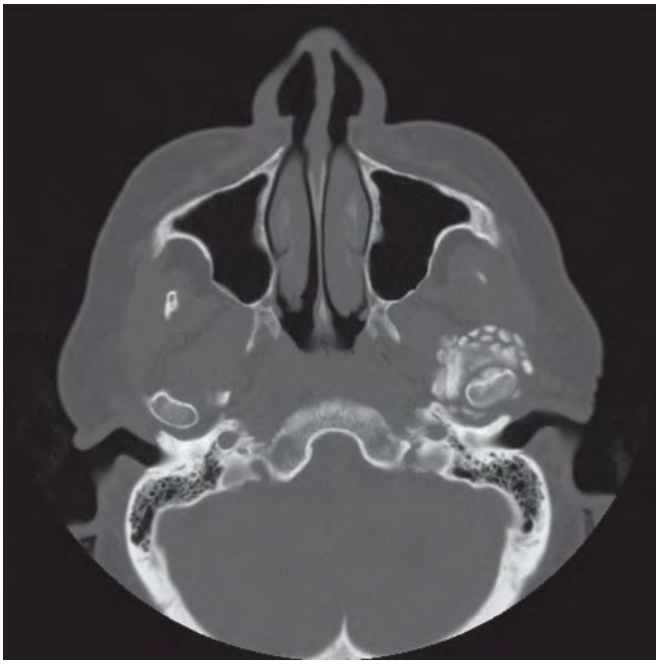


FIGURE 49-68. Synovial chondromatosis (axial computed tomography [CT]).

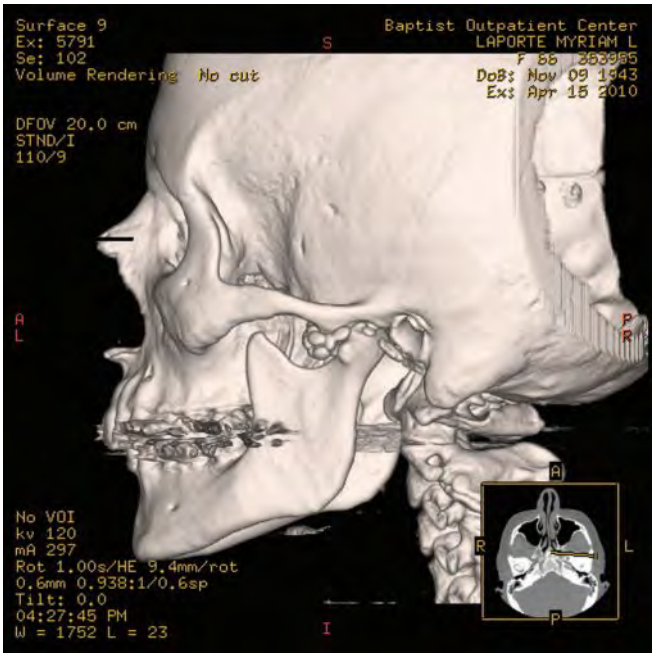


FIGURE 49-70. Synovial chondromatosis (three-dimensional recon, lateral view).

synovial hypervascularity with elongated villous lesions and aggregates of lymphocytes, as well as dilated capillaries. More than 50% of the RA patients have TMJ involvement.

Pseudogout/Chondrocalcinosis

As opposed to gout, where the crystals are deposited in the synovium, synovial villi, fibrillated and fragmented fibrocartilage (chondromalacia grade III), chondrocalcinosis is characterized by calcium pyrophosphate crystals in the synovium with an unaltered articular cartilage.

Joint Stenosis

In cases of synovitis without any disk or cartilage pathology, some joint stenosis accompanies the condition along with generalized capsulitis. This will cause a restriction of the ROM of the joint. Small intrasynovial adhesions are not

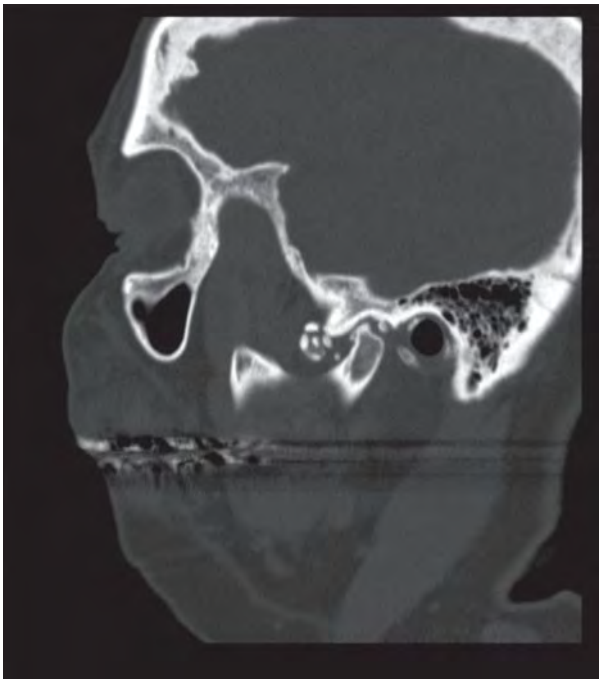


FIGURE 49-69. Synovial chondromatosis (lateral CT).

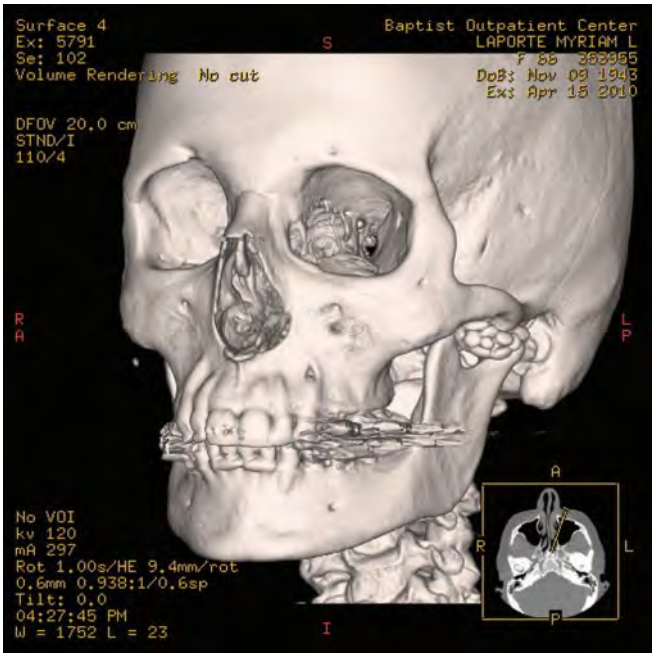


FIGURE 49-71. Synovial chondromatosis (three-dimensional Recon, half profile view).

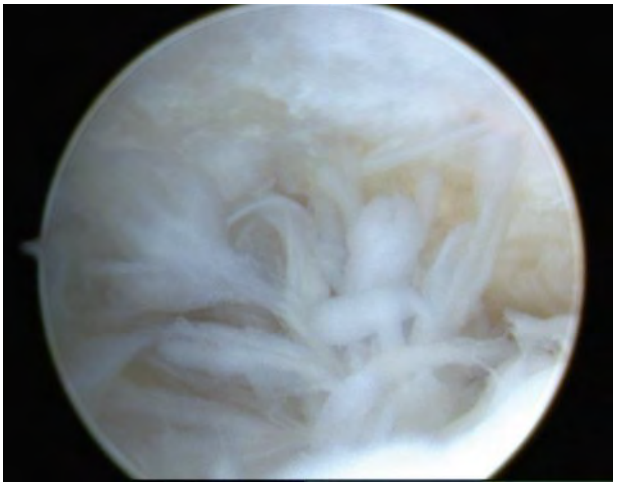


FIGURE 49-72. Synovial chondromatosis (arthroscopic image, stage I of the disease).



FIGURE 49-73. Synovial chondromatosis (arthroscopic image, stage II before loose body).



FIGURE 49-74. Synovial chondromatosis (arthroscopic image, stage III loose bodies, in this particular situation completely obturating the articular space).

likely to restrict joint motion; however, the combination of these adhesions, alteration of synovial fluid mechanics, and generalized capsulitis and stenosis will restrict condylar motion from both a functional and a pain standpoint. In this author's experience, adhesive synovitis is most commonly diagnosed in the anterior recess, with the posterior recess being the second most common area. Less frequent, lateral recess adhesions are identified, whereas medial wall and medial trough adhesions are rather infrequent. It is important to mention that lateral adhesions cannot be determined without an arthroscopy. Characteristically, patients with lateral adhesions present with limited opening status posttrauma, essentially negative disk mechanic problems, and negative arthritic changes in the joint. During the diagnostic sweep, when the arthroscope is contoured toward the front, the mechanical blockage is encountered, which prohibits the scope's advancement. This obstacle is a fibrotic adhesion in the lateral recess that most often begins to occur at approximately the peak or anterior portion of the articular eminence. This fibrous and synovial adhesive phenomenon restricts translation. At this point, the scope should not be levered or forced any farther. On the monitor, a partial eclipse of the normal arthroscopic view is visible, indicating bending of the arthroscope, with imminent risk of dislodging the lens. After appropriate documentation of the problem, the procedure should be discontinued and, pending symptomatology, the joint should be arthroscopically reentered in the operating room with a double- or multiple-puncture procedure.

Articular Dysfunction

From an arthroscopic perspective, the discussion revolves around reducing and non-reducing disks. In the situation of a reducing disk, the posterior band of the disk is visible and accessible for suturing when the condyle is in the forward position. In the non-reducing disk, no posterior band is seen with the condyle forward. Also, the entire anterior band may be obscured with fibrosis. Obviously, the cases of chronic disk dislocation associated with fibrosis and pseudowall formation will obliterate the disk-synovial crease.

Osteoarthritis

The studies of Boering and Rasmussen seem to support the idea of a natural process of joint aging. Whether this process occurs slowly or rapidly, the operative procedures to treat this condition are limited by joint size, stenosis, and pathologic state, not to mention the technical limitations of the surgeon and instrumentation. One of the typical arthroscopic findings when dealing with this particular entity is articular cartilage degeneration that can be observed along the posterior slope and peak of the articular eminence. A brief discussion of chondromalacia at the same time with the classification of this pathognomonic finding follows.

Chondromalacia Grading

Grade I: Softening of Cartilage

Softening of cartilage is caused by digestion of proteoglycan collagenases from injured chondrocites. Clinically, the

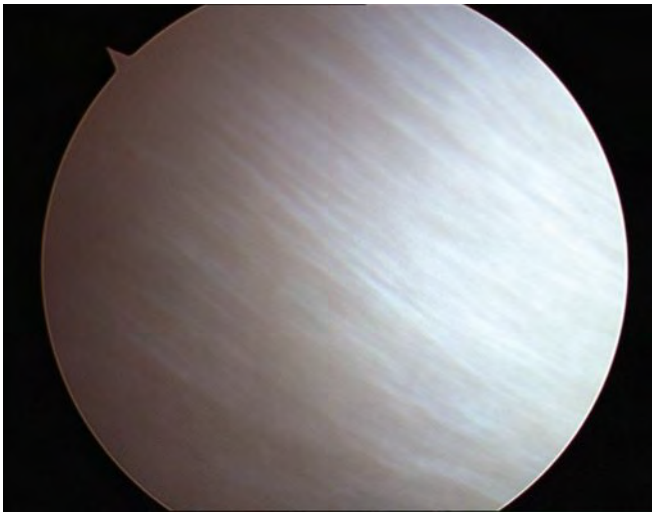


FIGURE 49-75. Normal appearance of the posterior slope of the eminence (chondromalacia stage 0) of the right TMJ.

cartilage turns opaque white, as opposed to a tannish normal (Figure 49-75) appearance. The difference is quite subtle; however, palpation of the articular cartilage, very gently with the scope, will determine its compressibility and friability, as well as the dimpling or pitting effect of this edematous tissue, evident mostly on the posterior slope of the eminence (Figure 49-76).

Grade II: Furrowing

Furrowing is the result of disruption of some of the deep-zone collagen fibrils at the calcified and noncalcified cartilage attachment and hydrated swelling of the proteoglycan-depleted areas along the fibrils in the TMJ (Figure 49-77).

Grade III: Fibrillation and Ulceration

Fibrillation and ulceration are caused by rupture of the deeper collagen fibrils from their calcified and noncalcified cartilage attachment and then the disruption of the parallel articular surface fibrils. These disruptions result in fibrillar

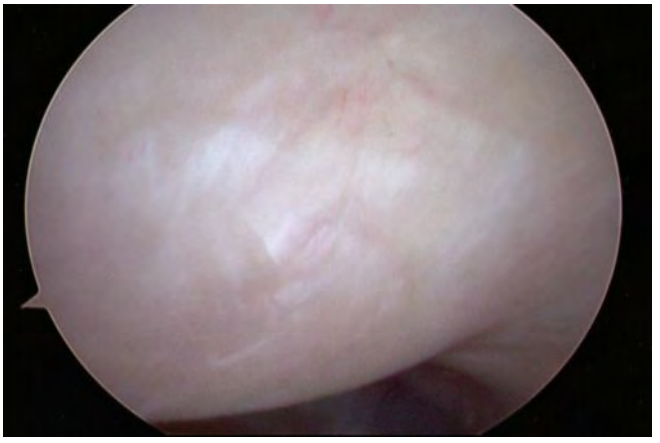


FIGURE 49-76. Chondromalacia stage I of the left TMJ.

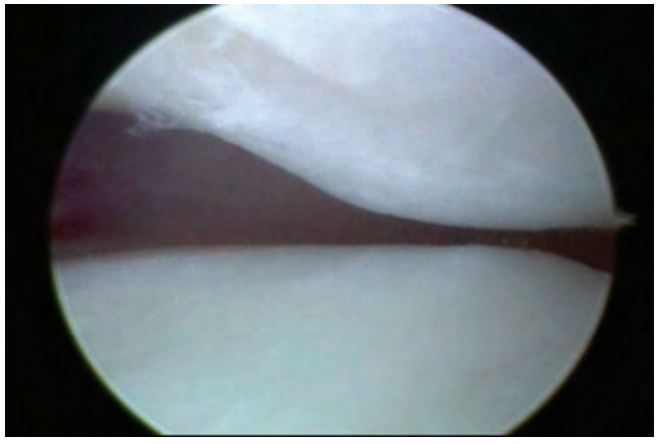


FIGURE 49-77. Chondromalacia stage II (furrowing) of the right TMJ (note the "opacification" of the posterior slope cartilage and furrowing/incipient fibrillation between 10 and 11 o'clock).

strands of degenerating cartilage that can be observed hanging from the posterior slope and peak of the articular eminence when suspended in the Ringer's lactate arthroscopic medium (Figure 49-78).

Grade IV: Crater Formation and Subchondral Bone Exposure

Crater formation and subchondral bone exposure are a result of a progressive breakdown of the deep and superficial fibrils followed by a breakdown of the intermediate cartilage fibrils (Figure 49-79).

Arthrofibrosis

We chose to describe this condition separately from the "fibrosis" discussed previously for the severity of this condition and its association with the severe hypomobility of Wilkes stage IV and V. It also represents one of the most

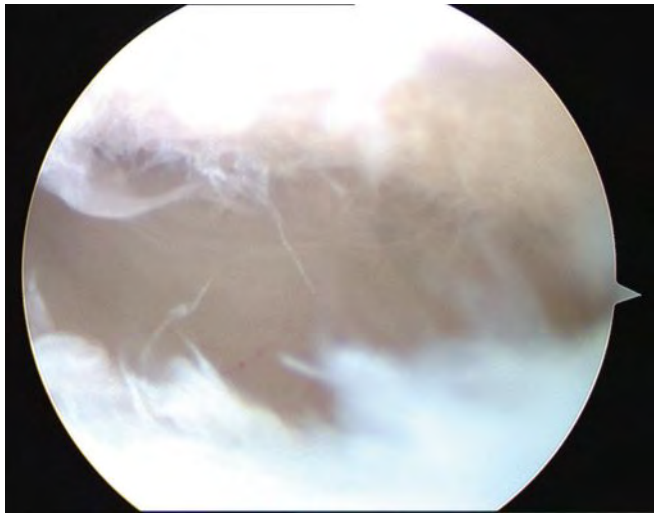


FIGURE 49-78. Chondromalacia stage III (fibrillation and ulceration) of the right TMJ (note also typical presentation of disk perforation inferior to and opposing the area of chondromalacia).

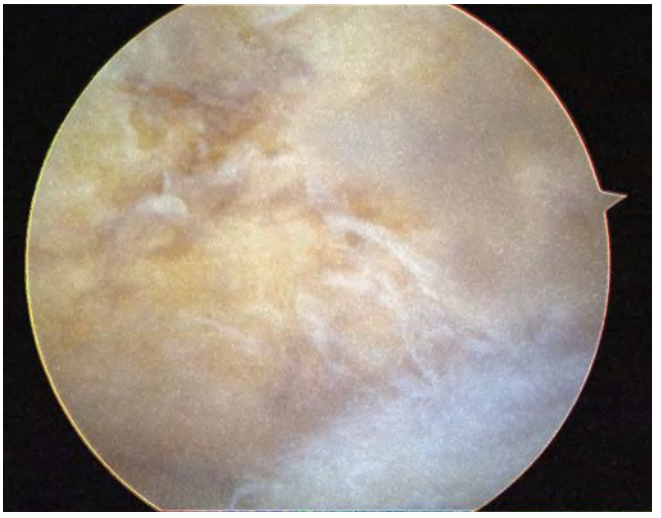


FIGURE 49-79. Chondromalacia stage IV (crater formation and subchondral osseous structure exposure).

difficult surgical challenges for the TMJ arthroscopist. Correlation with clinical findings compels us to reiterate that severe trismus in the presence of good excursive joint motion should prompt the surgeon in diagnosing an extra-articular condition. Hence, the focus should be more on myotomy/fasciotomy procedures, followed by aggressive physical therapy and counseling. The patient with intra-articular degeneration and hypomobility presents typically with minimal opening, minimal excursive motion, and focal joint pain. Documentation of baseline interincisal opening should be done under sedation/anesthesia immediately before the procedure. The entire diagnostic arthroscopy for this particular entity becomes very different from what we have presented earlier. Particular consideration in puncturing into the hypomobile joint has to increase the awareness of the surgeon. Frequently, the greatest concavity of the glenoid fossa is less evident, whereas the condyle becomes difficult to palpate, particularly if the lateral pole is degenerated or absent. Also, insufflation of the superior joint compartment is made difficult by the inability to palpate the lateral capsular wall distention and the absence or reduced capability of ascertaining plunger rebound. These are typical signs of joint stenosis and intra-articular fibrosis or, more concisely, arthrofibrosis. The diagnostic sweep is attempted. However, a "white-out" of fibrillar tissue makes visualization of structures very difficult. A lysis maneuver is performed blindly to increase the working space. During this initial sweep, it is paramount that the contours of the fossa and eminence are palpated at all times. The joint is again backwashed and the scope reinserted. The glenoid fossa and eminence should be visible at this point. Sometimes, the condyle is visible as well. As previously described, the next step of the diagnostic sweep is the positioning of the scope medial into the joint to identify the medial synovial drape. Extreme caution should be exercised so as not to violate this structure and venture too medially, where the internal maxillary artery as well as other vital structures could be found. This complication can be avoided by continuous awareness of cannula depth.

ARTHROSCOPIC OPERATIVE PROCEDURES

Second Puncture

The variations in second-puncture site and technique are dictated by the anterior recess volume and condition of the joint. Conditions such as synovitis, disk displacement with or without reduction, and osteoarthritis will present with a normal or reasonable (increased or slightly decreased) anterior pouch volume. The second puncture (Figure 49-80) is performed with the condyle seated in the fossa. The irrigation needle is removed, then the puncture site is located according to the triangulation principles. The vectors of instrument orientation create an equilateral triangle, facilitating a repeatable and safe pattern of placement for the second punctures. The depth of the arthroscope can be assessed on the cannula. A second measuring cannula is positioned flat against the tegument with the tip (0-mm marking) contiguous with the scope at the point of entry (skin) and continuous (in a straight line) with the plane of the arthroscope. The depth of scope penetration is now translated to the cannula. Depending on the angle formed by the arthroscope and the tegument, 1 to 3 mm can be added to the previous measurement. The site for the second puncture has now been established. The ideal position of the working cannula is directly parallel to the disk-synovial crease in the anterior recess in order to facilitate the operative procedures. In a fashion similar to the fossa puncture, the assistant insufflates the joint with 2 mL of irrigation fluid. The trocar/cannula penetrates perpendicular to the tegument, then continues in the very same direction, ensuring the appropriate geometry and orientation of triangulation described previously. The tendency to direct the cannula posteriorly has to be avoided. The trocar is rotated through the skin and advanced transfixiant to the osseous structure. The attempted point of contact is the juncture between the anterior aspect of the anterior slope of the articular eminence and the continuation of the zygoma. As opposed to the fossa puncture, no vigorous dissection of the periosteum is performed at this level. The frontal ramus of the temporofacial division of cranial nerve VII crosses the anterior aspect of the eminence in close proximity to the second puncture site. Only the tip of the trocar should touch the subjacent cortical plate. Next, the trocar/cannula is rotated through the capsule and synovium. The trocar is observed on the monitor entering the joint space. Once intra-articular, the trocar is removed and drainage of the irrigating fluid is noted through the cannula. The assistant stabilizes the working cannula while the surgeon proceeds with instrumentation. In the case of fibrosis or advanced arthrosis, the anterior recess is very difficult to negotiate. The scope will be maneuvered as far as possible during the diagnostic sweep. When the arthroscope cannot be advanced or laterally positioned any further and the condyle has been seated, the

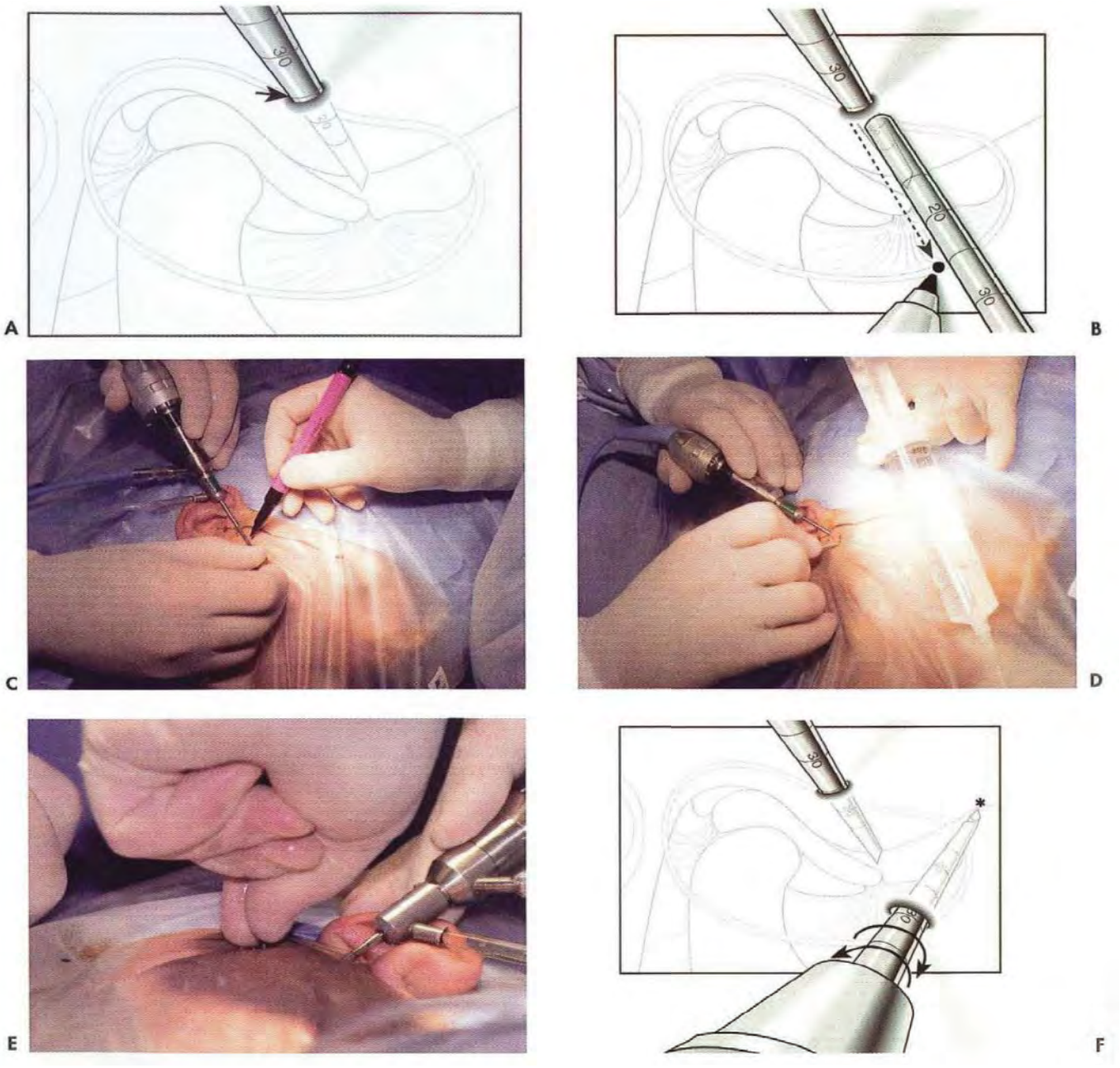


FIGURE 49-80. Second puncture technique clinical steps. **A,** Scope with intra-articular depth of penetration ideal for second puncture. **B,** Vector/triangulation system and utilization of measurements. **C,** Marking the second puncture site. **D,** Trocar/cannula at the second puncture site, ready for puncture. **E,** Trocar/cannula perpendicular to the skin. **F,** Osseous landmark for second puncture (target area).

second puncture is placed at the most anterolateral aspect permitted by the pathology of that particular joint. The joints with advanced arthrofibrosis, stenosis, or fibrous ankylosis present a distorted intra-articular anatomy. Henceforth, the second puncture is placed in the most open area of the joint. When no open space can be visualized arthroscopically, confirmation of the arthroscopic cannula in the superior joint space is confirmed by palpation. Once the cannula position is confirmed, the scope is removed and the obturator inserted. With the blunt obturator, a blind lysis maneuver is performed until osseous contact is perceived (Figure 49-81). This will create the opening necessary for the placement of the second puncture.

Lysis and Lavage

Although there are still practitioners performing the traditional lysis and lavage procedure (Figure 49-82) with a certain degree of success, the limitations of the procedure are obvious.

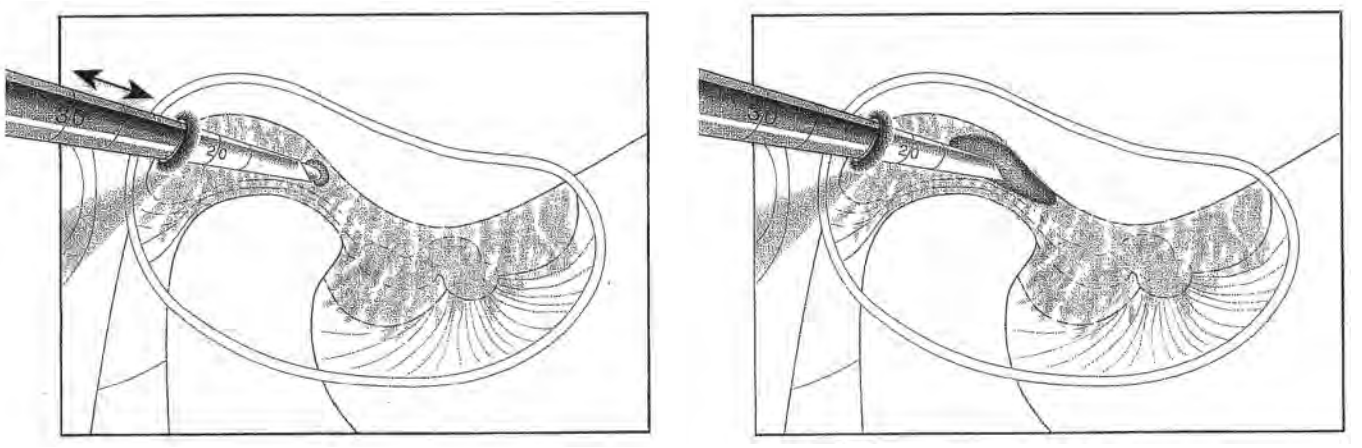


FIGURE 49-81. Blind lysis maneuver technique.

In addition to the diagnostic lysis and lavage, the double-puncture arthroscopic lysis and lavage emphasizes more on the lytic aspect. Utilizing a straight probe, the pouches and recesses of the superior joint space can be directly manipulated. The lysis procedure is designed to distend all pouches and recesses along the capsule in order to give the physical therapist a head start in postoperative joint manipulation.

Anterior and Posterior Recess Adhesions

The sequential lysis of adhesions in the superior joint space follows an anterior-to-posterior pattern, avoiding repeated motion of instrumentation from the back to the front of the joint, increasing the risk of unnecessary articular surface scuffing. The lysis maneuvers with a probe under direct arthroscopic visualization are performed according to the triangulation techniques. The purpose of the procedure is to restore the volume and architecture of the joint. The probe is first placed in the most medial aspect of the joint and then swept laterally along the disk-synovial junction with an inferoanterior maneuver. Then, by sweeping medially-to-laterally along the articular eminence, a superoanterior maneuver is executed to complete the lysis (Figure 49-83). These maneuvers are repeated until an adequate recess volume is restored. In many instances, the anterior recess lysis is followed by posterior recess lysis. Under direct observation, the probe and the scope are translated back into the posterior pouch. Upon release of lateral adhesions, a marked increase in the ROM of the condyle occurs immediately. There is one critical observation this author has made over thousands of procedures performed. Medial wall synovitis is, in many instances, related to the inflammation of the insertion of the superior belly of the lateral pterygoid, causing myalgia, restricted ROM of the mandible, and pain.



FIGURE 49-82. Traditional arthrocentesis.

Lateral Recess Adhesions

Lateral adhesions can be identified only during the diagnostic arthroscopic examination. Typically, the patients with lateral adhesions present with posttraumatic restricted ROM, essentially negative disk mechanical problems, and negative arthritic changes. During the diagnostic sweep, when the scope is contoured toward the front, the mechanical obstruction encountered will prohibit the advancement of the arthroscope. This blocking fibrotic adhesion in the lateral recess is part of an adhesive phenomenon that restricts translation. In most cases, it occurs at the peak or anterior portion of the articular eminence. A partial eclipse of the arthroscopic view will become apparent on the monitor, indicative of bending of the arthroscope. Excessive bending of the instrument can break or dislodge the lens, rendering the scope unusable. In order to access this particular pathology, the second puncture is positioned at the posterior limit of the adhesion. When a blunt probe lysis does not suffice, a small motorized whisker shaver will perform a débridement of fragments of adhesions from the joint. If necessary, bipolar electrocautery or laser will execute the lysis of more resilient fragments. If this particular articular area is not given the appropriate consideration, there is an increased likelihood of postoperative crepitation during function. The

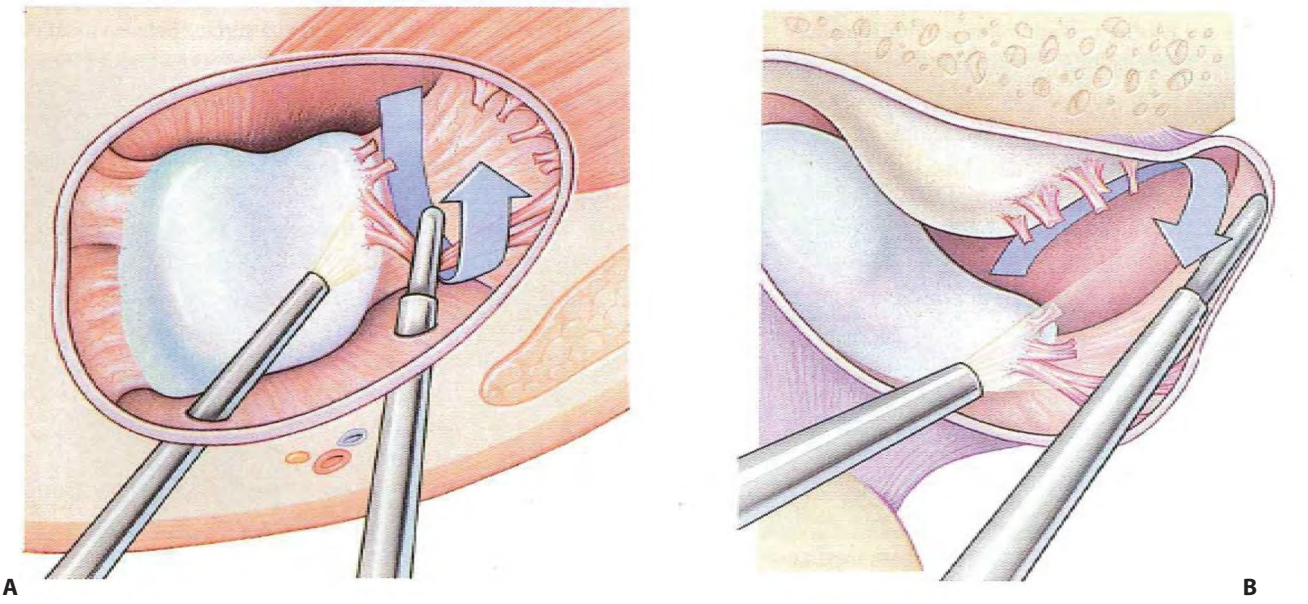


FIGURE 49-83. Anterior recess lysis maneuver. **A,** Medial-to-lateral sweep (axial view). **B,** Second medial-to-lateral sweep (superoanterior motion) (sagittal view).

release of lateral adhesions facilitates an immediate increase in the ROM.

Intra-articular Medications

Steroids

Before the advent of arthroscopy, intra-articular steroids were injected via blind technique. The double-puncture arthroscopic technique, however, has made possible the injection of medications specifically targeting various anatomic articular sites. Although this author has a tremendous amount of firsthand experience with the use of intra-articular steroids, he does not advocate their routine administration. In the past, some beneficial results have been shown in cases of marked synovitis and/or marked redundancy, particularly in stage III and IV medial wall synovitis. This author's experience and research support the theory that medial wall synovitis is related to inflammation of the insertion of the superior head of the lateral pterygoid, which, in turn, causes restricted opening pain. The benefit of the steroid injection is the reduction of muscular irritation and spasm, thus decreasing joint pain with function. The technique employs a 3-mL syringe with a 25-gauge spinal needle to inject a combination of 1 mL of 2 mg/mL dexamethasone and 1 mL of 6 mg/mL betamethasone.

Botulinum Toxin A

The positive therapeutic effect of botulinum toxin type A (Botox) on functional disorders and symptomatology in connection with the treatment of cervical dystonia is very well documented. The later studies of von Lindern, Israel, Mendes, and others have explored local injection of Botox as treatment method for chronic facial pain associated with hyperactivity of the masticatory muscles, with very promising outcomes. We are currently conducting a study on the efficacy of arthroscopically assisted direct injection of Botox into the superior head of the lateral pterygoid at the pterygoid shadow.

Hyaluronic Acid

This polysaccharide of the glucosaminoglycans family is a component of many extracellular tissues including synovial fluid and cartilage. It is a product of the articular chondro- and synoviocytes. The concept behind TMJ injection of hyaluronate is the stimulation of the endogenous synthesis of hyaluronic acid by the exogenous hyaluronic acid. Hyalgan is a 500- to 730-kDa molecular weight fraction of highly purified avian sodium hyaluronate buffered (pH 6.8–7.5) in physiologic saline. This author believes hyaluronate to be an excellent intra-articular lubricating agent that facilitates navigation while minimizing iatrogenic intra-articular injury (scuffing).

Débridement

There are instances when adhesions cannot be overcome with the probe or the breaking of adhesions results in fragments tenaciously attached to the walls. A motorized whisker shaver is then used to débride these fragments. Sometimes, it is necessary to use an alternating sequence of whisker shaver and bipolar electrocautery to first desiccate and then remove some of the more resilient fragments. In the absence of the débridement, there is a high chance of postoperative crepitation in the area.

Synovectomy

The most effective technique of reduction of redundant synovium is bipolar electrocautery. Redundant synovium most often occurs in the posterior pouch, especially after disk reduction procedures. Occasionally, it may be encountered in the anterior recess. Hypervascularity and redundancy can effectively be reduced by means of bipolar electrocautery. The McCain-Leibinger bipolar electrocautery easily fits through a 2.0-mm working cannula and is used for synovium reduction purposes. The synovial clinical response is a change in color from bright red to off-white or even a light brown. On occasion, the synovial tissue cauterized and desiccated fragments fail to vaporize with bipolar electrocautery. For these situations, the Ho:YAG laser will effectively perform the synovectomy.

Anterior Release

Conditions such as chronic disk dislocation, fibrosis, adhesive bands, or pseudowall formation can obliterate the disk-synovial crease. Using a blunt probe, a lysis procedure is performed, if necessary, in the anterior recess until the entire disk-synovial crease can be visualized from medial to lateral. Just before commencing the release, the disk-synovial crease is confirmed with a hook probe. If the crease is not properly identified, iatrogenic disk perforation may occur during the anterior release procedure. If the anteroposterior disk dimension appears adequate and a relatively normal disk shape is ascertained, the release starts in the medial one half of the disk-synovial crease (Figure 49-84). Using an Ho:YAG laser, the synovial capsule is incised just anterior and parallel to the anterior margin of the disk (Figure 49-85). The surgeon observes the muscle fibers inserting into the disk and capsule. Dissection should be performed to a depth no greater than 5 mm or until the anterior band is freely mobile and without tethering (Figure 49-86). Caution must be observed when making the myotomy at the most anteromedial corner. When the anteromedial synovial drape is incised at the junction with the disk (Figure 49-87), an artery, approximately 1 to 2 mm in diameter, is usually found directly subjacent to the junction. Arthroscopically, it appears as a white tubular structure. If this vessel is inadvertently incised, copious intra-articular hemorrhaging will occur. This vessel cannot be cauterized or tied off by any current means. To tamponade the bleeder, all instruments must be removed and constant lateral pressure maintained on the joint for 5 minutes by the clock, while the condyle is held in a forward and contralateral position. The puncture techniques are then repeated and the joint is lavaged and suctioned free of clots. No further releasing is done in that area and the release is completed laterally. Identification of this artery will avoid this problem and the myotomy can be carefully completed around the artery. The other potential bleeding points encountered in the

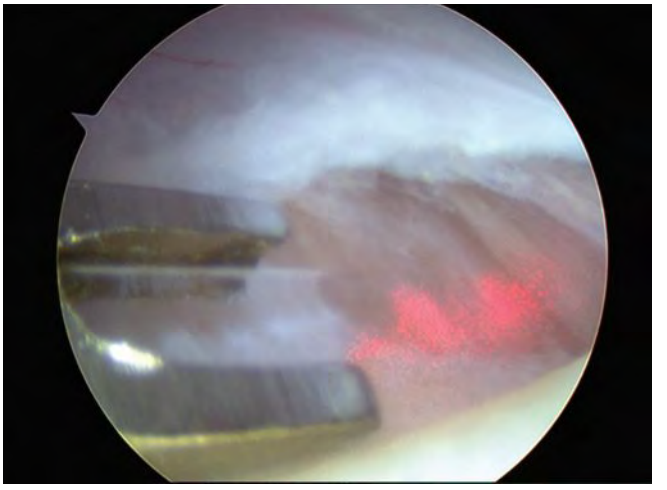


FIGURE 49-85. Anterior release (holmium laser incision of synovial capsule just anterior and parallel to the margin of the disk).

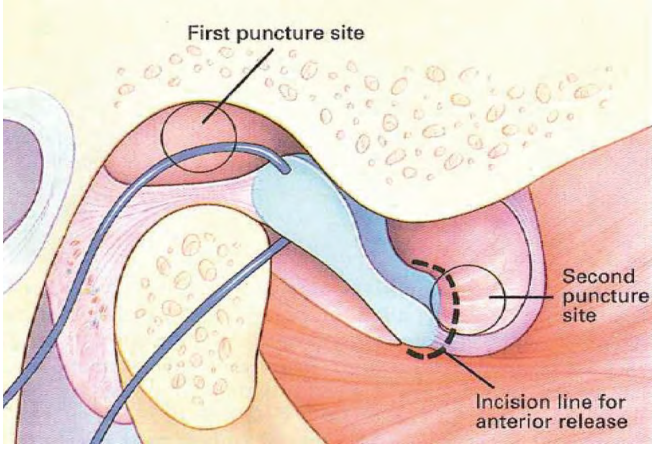


FIGURE 49-84. Anterior release along the entire medial-to-lateral extent of the anterior recess, 1 to 2 mm anterior to the anterior band of disk *(dotted line)*.

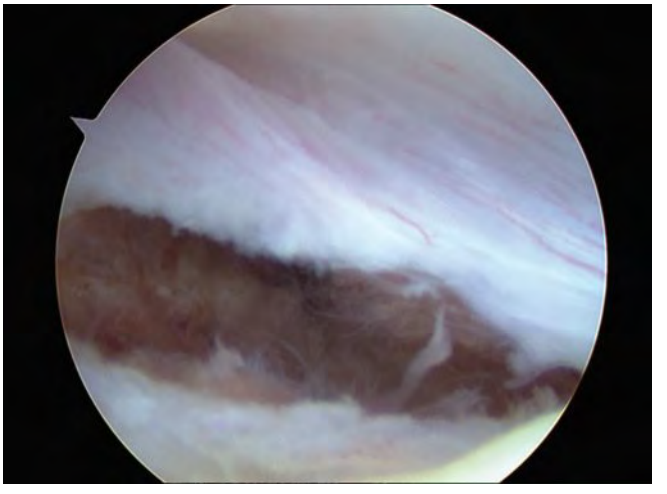


FIGURE 49-86. Anterior release (dissection 5 mm deep, completely freeing the anterior band of the disk).

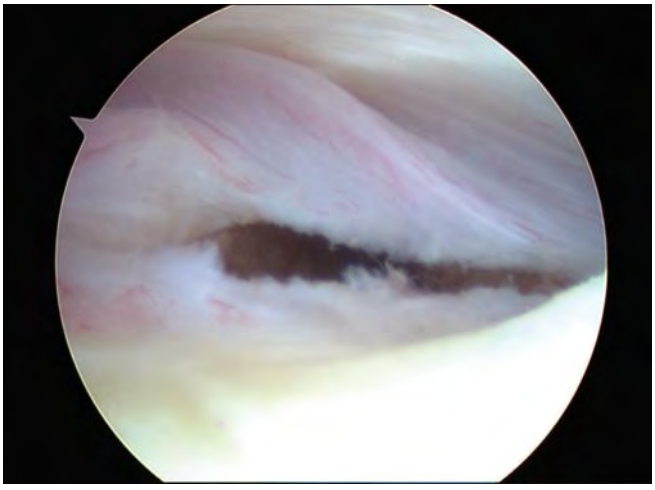


FIGURE 49-87. Anterior release (completed).

lateral pterygoid can be relatively easily controlled with bipolar cautery. The lateral one third to two thirds of the anterior release may be performed more expediently with less concern for hemorrhage. One of the concerns with an excessively deep anterior release is that of avascular necrosis that has been documented on postoperative MRIs. The most plausible explanation for this complication is that the fibers inserting in the pterygoid fovea (of the condyle) have been transected, causing subsequent venous congestion in the condylar head and, ultimately, changes on the MRI. In light of these observations, this author surmises that the myotomy should be confined to a depth of 5 mm, to only release the superior head insertion. Probing to assess anterior band mobility should be routinely performed during the anterior release. Obviously, the message here is to always err on the conservative side.

Disk Reduction

Once the anterior release is achieved, the disk reduced in the anatomic position. To begin the reduction, the blunt probe is introduced and passed posteriorly by contouring the intermediate zone. Simultaneously, the condyle is distracted anteriorly and to the contralateral side. Once the probe is positioned in the posterolateral recess, the retrodiskal tissues are depressed in a posteroinferior direction. Disk reduction should be felt by the surgeon and confirmed arthroscopically. The disk is held in reduced position with the probe and the working cannula, while the scope is retracted to a lateral position. Then the probe pressure is released in order to ascertain passive reduction/positioning of the disk in the anatomic position.

Posterior Scarification/Cauterization

Once the disk reduction is achieved, an increased amount of redundant synovium is noted in the retrodiskal flexure. Even with minor disk repositioning procedures and less evidence of redundancy of posterior synovium, in the absence of diskopexy, this procedure should be performed. Special consideration should be given to this step in the APS procedure. Postoperative MRIs have shown thickening of the retrodiskal tissue in a number of cases. This author's extensive experience has confirmed the most plausible theory explaining this occurrence as a certitude. It is mostly the inadequate ablation and, to a much lesser extent, the hypertrophic scar that cause the postoperative redundancy of the retrodiskal tissue. Typically, a "reefing" phenomenon occurs, in which the retrodiskal tissue is bunched up and needs to be reduced in bulk with bipolar cautery. If not, this fairly large amount of tissue can fibrose during healing. During postoperative settling, the disk may then be displaced by this potential fibrotic mass. Retrodiskal ablation should, therefore, be performed until a normal-shaped retrodiskal flexure is sculpted (Figure 49-88 and 49-89). The inflamed synovium is cauterized and the areas of excess tissue bulk ablated with bipolar cautery, laser, or both. Deep electrocautery of the oblique protuberance should then be performed. An examination of the anterior and posterior pouches is then completed.

Disk Function

Before making the decision to exit the TMJ, joint mechanics have to be reascertained. If disk reduction is inadequate or clicking is noted, the anterior release is revisited with the blunt probe. If tethering is observed, it is most likely in the medial muscle insertion area or in the most lateral extent of the crease. Once the anterior release is extended, disk function is rechecked. If clicking persists or the disk position does not present satisfactory stability, the diskopexy procedure needs to be seriously contemplated.

Arthroscopic Diskopexy

This procedure was developed with the thought that restoration of functional intra-articular anatomy, especially in

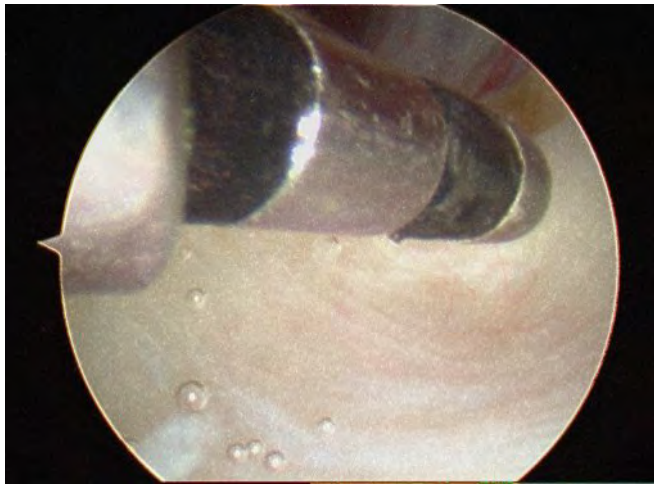


FIGURE 49-88. Posterior scarification (bipolar retrodiskal ablation).



FIGURE 49-89. Appropriate contouring of retrodiskal tissue (after ablation).

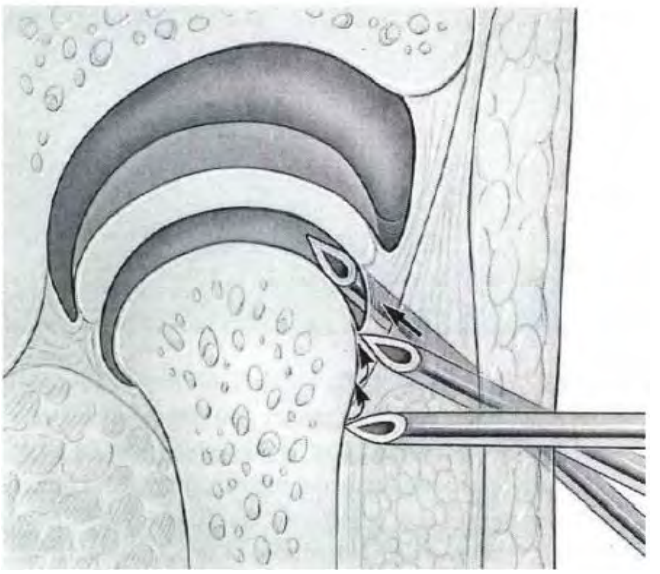


FIGURE 49-90. Technique of placement of the suture passing needle.

arthroscopic fashion, is the best treatment alternative for managing articular dysfunction. This author believes that symptomatic internal derangements have to be restored to a normal condyle-disk relationship, arresting the natural course of osteoarthritis. With this in mind, the diskopexy is performed to achieve and maintain this end point. This technique addresses both the reducing and the non-reducing disk.

Diskopexy for the Reducing Disk

Placement of the Suture Passing Needle

Since it is visible, the surgeon scans the posterior band of the disk in order to identify the ideal area in the disk for placement of suture. The best place is at the junction between the middle and the medial thirds of the disk. From this point, the arthroscope is swiveled into the lateral recess in preparation for suture passing, while the assistant maintains the condyle in a forward position. The vector technique is applied to determine the exact point of entry of the suture passing needle through the skin. The scope is positioned in the lateral recess directly lateral to the posterior band of the disk. This location will correlate with the lateral pole of the condyle. The depth of the scope is noted, then using an empty cannula, this distance is marked off on the skin at this point. The suture passing needle enters the skin at this point. A 20- gauge $1^1/_4$-inch needle threaded with a single no. 1 polydioxanone suture is passed perpendicular to the skin and advanced until the lateral pole of the condyle is felt with the needle. The needle-tip is then "walked" superiorly until it slides off the head of the condyle (Figure 49-90). An upward bulging of the posterior band will be noted at the point where the needle is passing through the disk. The suture passing needle is redirected to engage the desired amount of posterior band tissue (a 2- to 3-mm bite of disk fibrocartilage). The needle tip is then advanced and observed in the greatest concavity of the fossa. This area provides the greatest accessibility for suture retrieval.

Placement of the Suture Catching Needle

This needle is passed percutaneous from the preauricular skin crease at the level of the pinna. This particular instrument is called Meniscus Mender II and is provided with an interchangeable obturator and a lasso-type suture retriever. Once it enters the skin, the mender is advanced anteromedially and toward the maximum concavity of the glenoid fossa. As it enters the superior joint space, particular consideration is given to avoiding disk incorporation as the mender passes. The scope is fixed on the greatest concavity of the glenoid fossa, and once the mender is visualized, it is positioned in close proximity to the suture passing needle (Figure 49-91). At this point, the obturator is removed and the retriever is inserted. The lasso portion is slipped over the 20-gauge needle and the needle is advanced slightly (Figure 49-92). The suture is advanced through the passing needle and retrieved with the lasso (Figures 49-93 and 49-94). The suture passing needle and the meniscus mender are removed, leaving the two free ends of the suture exiting the skin. The scope is also removed.

Retrograde Passing of the Anterior Suture End

With a no. 11 blade, two small incisions (~5 mm each) are placed in the preauricular skin crease, away from each other, extending superiorly from the suture exit point. Using a fine straight hemostat, a blunt dissection is performed adjacent to the suture ends and along the tragal cartilage, down to the joint capsule. The anterior suture end must now be passed posteriorly for final tying. Using a no. 3 half-circle, tapered, French spring-eye needle, the anterior suture (lower end) is passed back down its needle tract to the capsule. The suture needle is then redirected posterosuperiorly along the extracapsular fatty tissue. The tip should emerge in the center of the superior blunt dissection wound and is retrieved with an

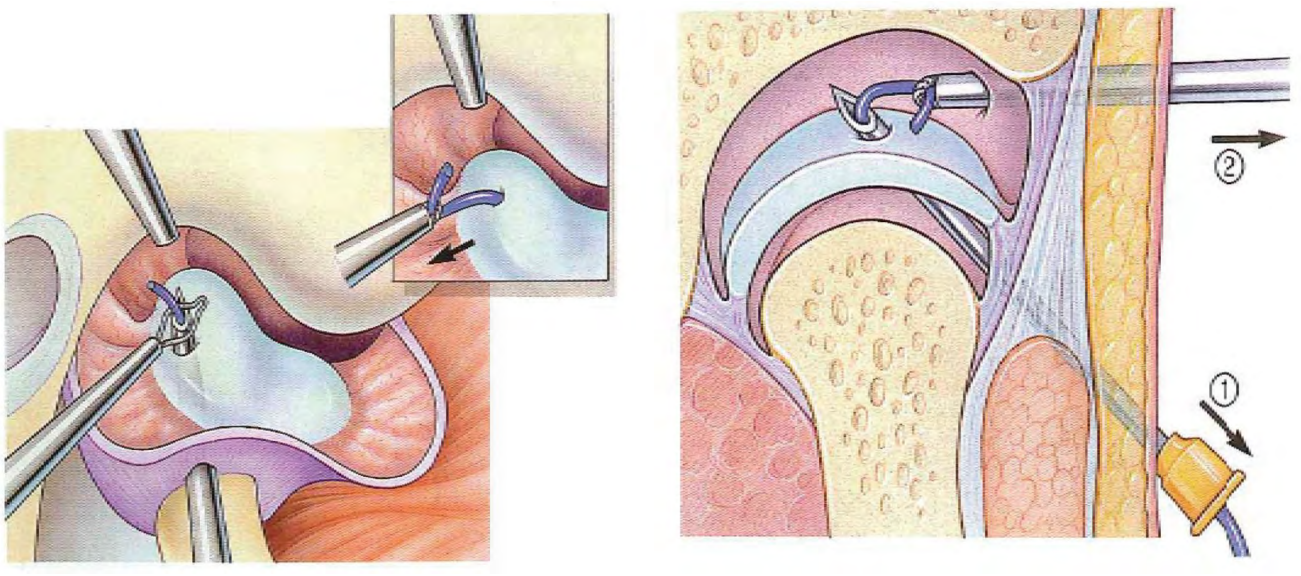


FIGURE 49-91. Suture diskopexy technique.

Adson. The passing of the needle through the middle of both blunt dissections is a very deliberate move, avoiding any impingement on epidermal or dermal tissue layers. If impingement occurs, a dimple in the skin will be noted and a less ideal disk traction results. This may also lead to frontal branch impingement. The suture is tied down in the extracapsular fatty tissue (Figure 49-95). A "surgeon's knot" is used to prevent slippage when cinching the knot down. Once the knot has been cinched down for the first time, the condyle is seated by the assistant and the knot is cinched again. Additional ties are placed in the suture and the suture is trimmed at the knot. The skin incisions are closed with 6-0 nylon or 5-0 fast gut, one interrupted suture per incision.

Diskopexy for the Non-reducing Disk

In this situation, with the condyle forward, the posterior band is not visible. This is a double-puncture technique. After performing the anterior release, posterior cauterization, and disk reduction in the same fashion described previously, the operator can proceed with the diskopexy. Here are the fine points of this procedure in the non-reducing disk. First, the disk is held in the reduced position with the working cannula and probe, with the scope pistoned laterally. In preparation for suturing, the surgeon hands the assistant the blunt probe. The assistant firmly stabilizes the disk in the reduced position. The scope is carefully swiveled laterally from the desired disk suturing point to the lateral recess. In general, this will locate the FOV over the lateral pole area, the work-

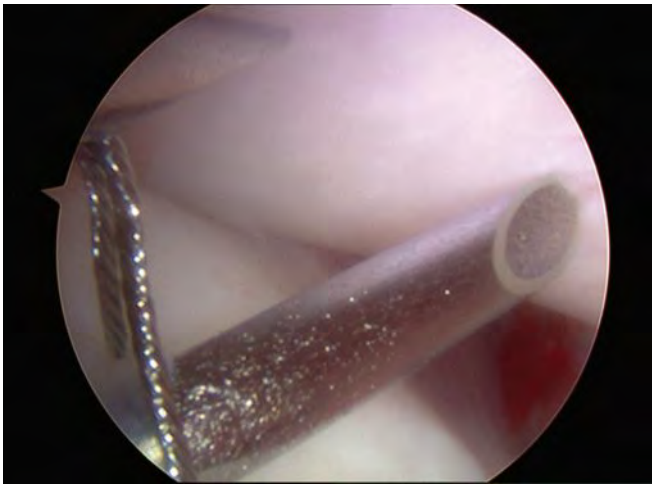


FIGURE 49-92. Placement of the suture passing needle in diskopexy (suture passing needle engaged by snare/lasso).

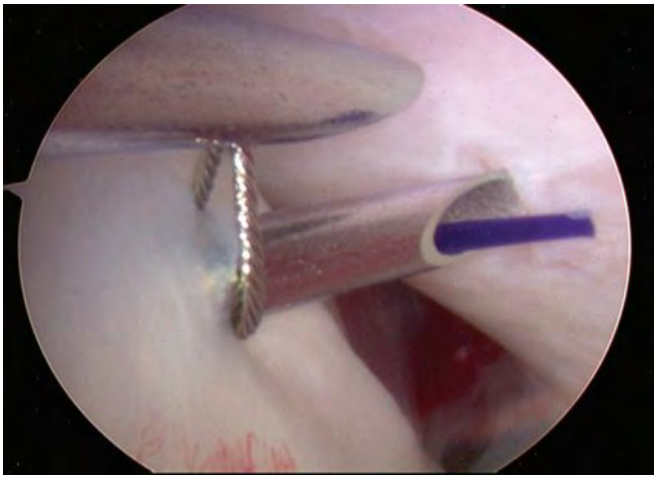


FIGURE 49-93. Intra-articular positioning of suture passing needle with protruding suture engaged by lasso.

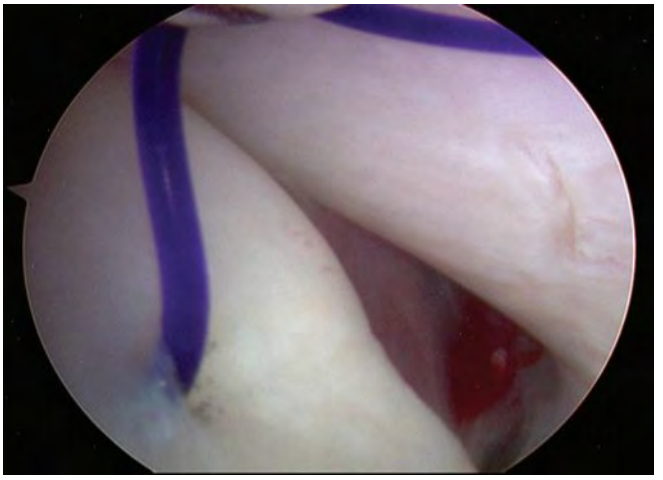


FIGURE 49-94. Suture apprehended into the meniscus mender/suture catching needle.

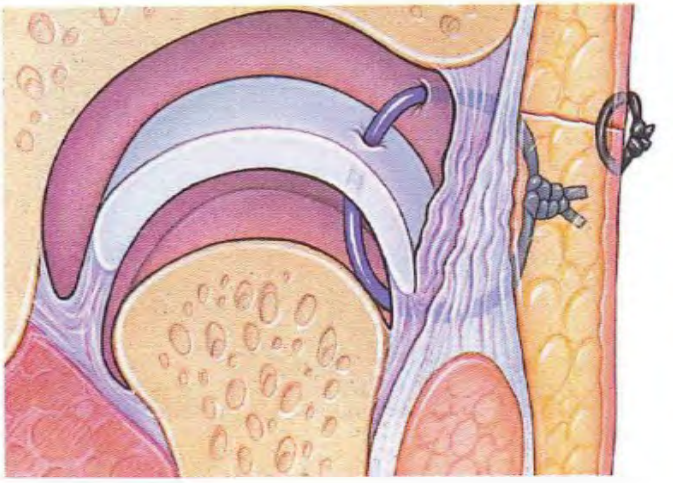


FIGURE 49-95. Suture diskopexy closure.

ing cannula occupying the lower portion of this field. The suture is passed as previously described. Once the suture passes through the disk, the assistant takes charge of the scope and the reduction probe while the surgeon retrieves the suture with the meniscus mender. All the other steps of the procedure are performed as described previously. Despite some limitations, such as inferior joint space adhesions or anchored disk phenomenon, the arthroscopic surgical techniques for disk repositioning and fixation are predictably repeatable.

Arthroscopic Rigid Disk Fixation

The normal anatomic picture of an articular disk is that of a lateral pole ligament and posterior capsule of the inferior joint space acting similarly to a shock cord on an aircraft carrier. The concept for arthroscopic rigid disk fixation originates in the Walker procedure, designed specifically to repair these ligaments. Essentially, the procedure calls for creating an anchor or a cavity at the posterolateral pole of the condyle to facilitate rigid or semirigid fixation of the disk to the condyle. The author has pioneered the arthroscopic technique for rigid disk fixation, involving three different products: the Osteomed 2-mm cannulated screw, the Linvatec bicortical "smart-nail," and the Inion screw. Regardless of the device employed, this three-puncture technique places the third cannula in the preauricular crease, targeting the disk condyle assembly at the lateral pole of the condyle (Figures 49-96 to 49-102). The technique exhibits several advantages that call for serious consideration. First, it promotes significantly increased stability, especially long term. Second, it allows immediate aggressive postoperative stage II to IV physiotherapy, significantly reducing the incidence of hemarthrosis,





FIGURE 49-96. Condyle sounding with 20-gauge needle and third puncture.

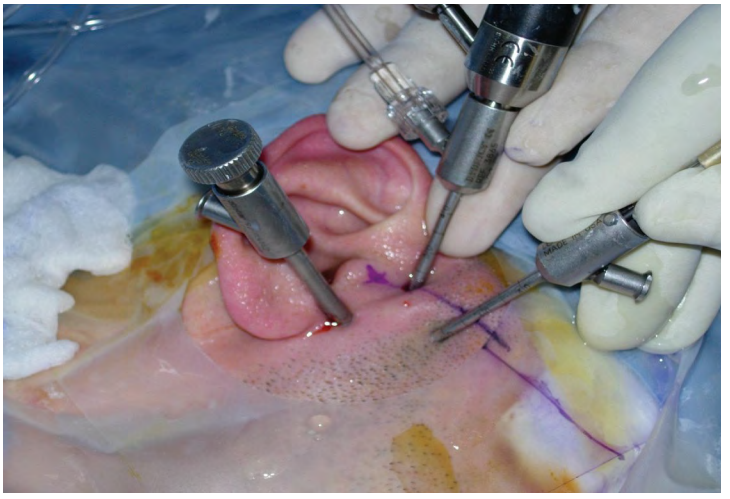
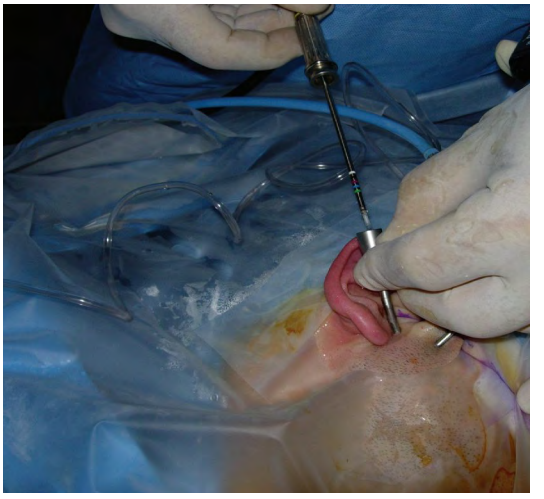



FIGURE 49-97. Third cannula placement and fixation device cannular intromission.

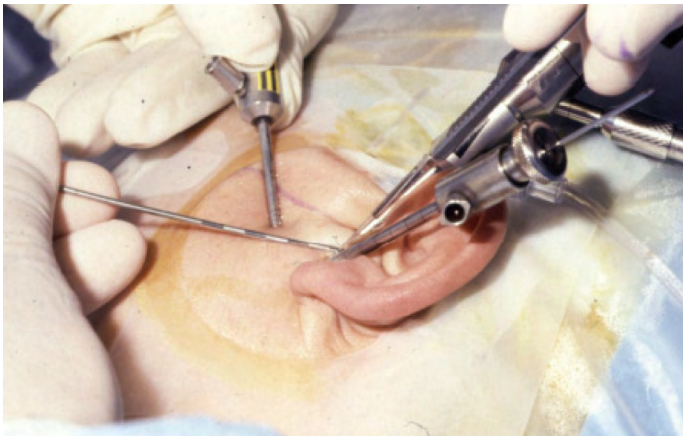



FIGURE 49-98. Fixation device delivery close-up.

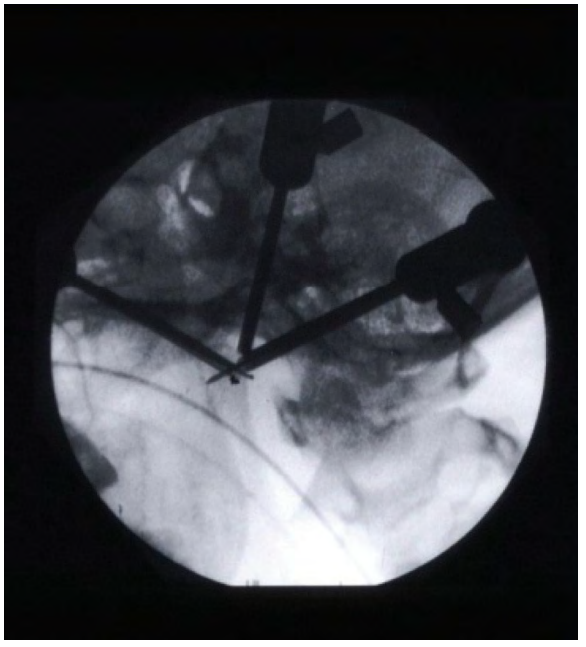



FIGURE 49-99. Fluoroscopic control and confirmation of placement and position of the disk fixation device.

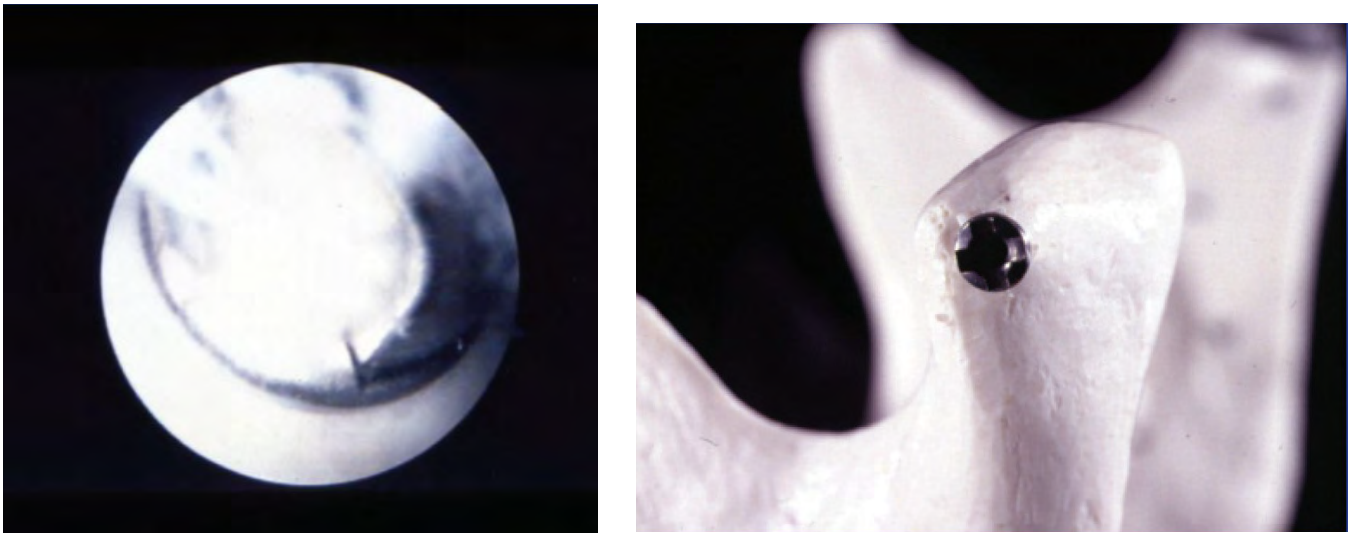


FIGURE 49-100. Target area for placement of the disk fixation device.

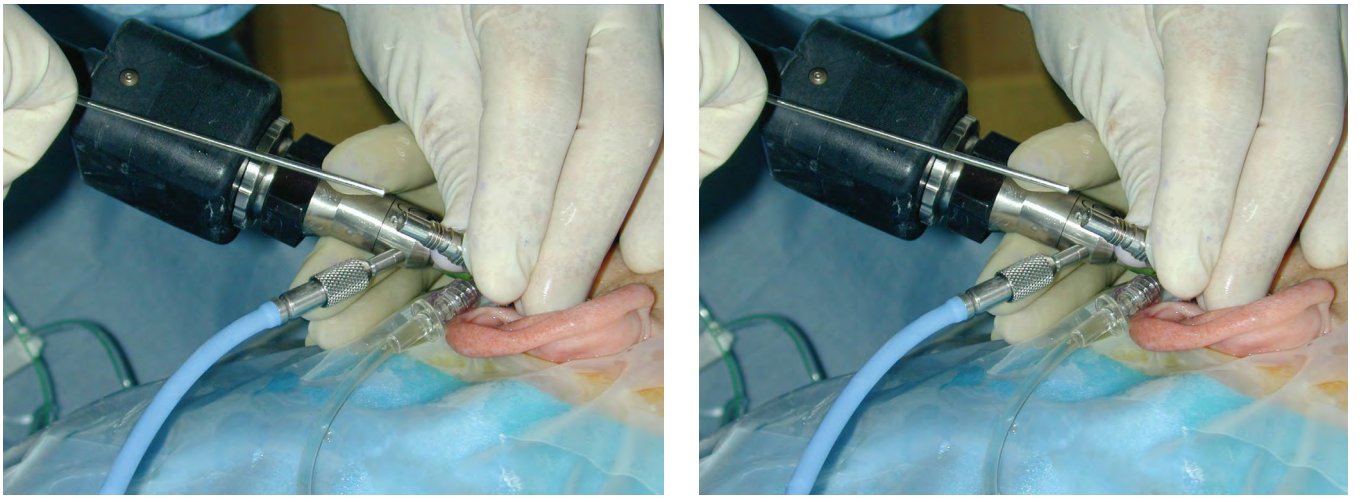


FIGURE 49-101. Delivery of the fixation device.

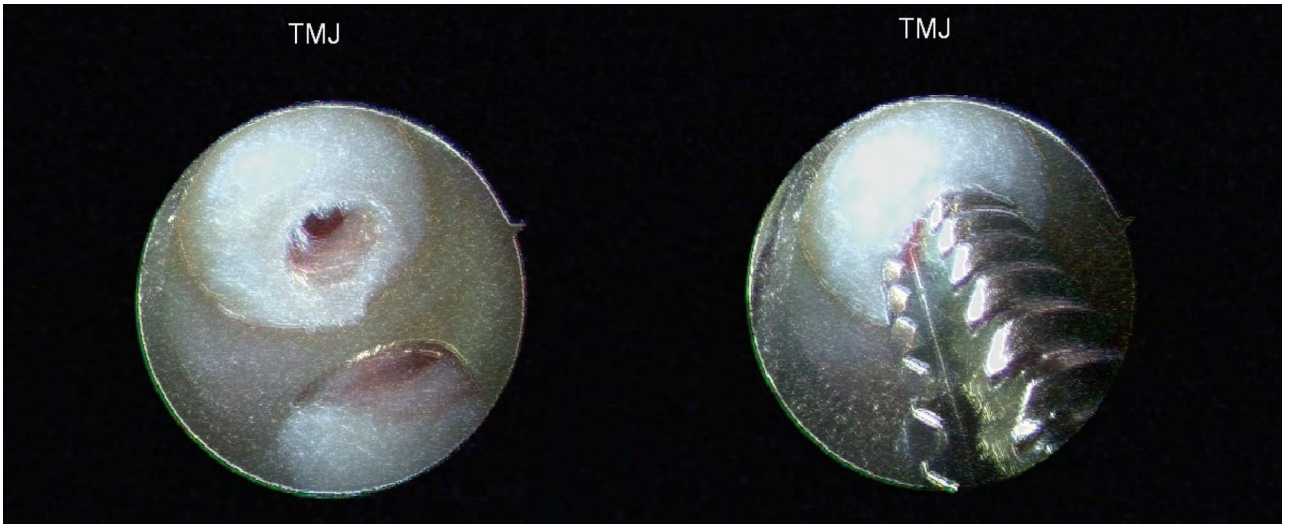


FIGURE 49-102. Arthroscopic view of rigid fixation.

with subsequent arthrofibrosis. Last, but by no means least, this much more user-friendly technique prevents the infamous nerve injury, because there is no suture passing over the nerve. Also, there is considerable decrease in the levels of postoperative pain. The rigid fixation technique resulted almost consistently in postoperative malocclusion. In the majority of situations, resolution was achieved between 2 weeks and 2 months. Unfortunately, a small number of cases with irretractable malocclusion had to undergo open diskectomy. Wilkes IV cases, with insufficient intra-articular space to accommodate the malformed disk, were eventually incapable of returning spontaneously to preoperative occlusion and had to be treated as described earlier. The "smart nail," extrapolated from orthopedic surgical technique, turned out to be rather sizable for our purposes, in many instances, transfixating the condyle. Finally, the Inion screw, on occasion, demonstrated significant cavitation defects in some of the cases associated with severe synovitis. Although not completely insurmountable, these inadequacies have led this author into revisiting and refining the, thought obsolete, semirigid, suture diskopexy technique described previously. The rigid disk fixation, however, remains a solid option for all Wilkes II and III situations, in which adequate disk shape and joint volume are still present.

Osteoarthritis Management

Obviously, the purpose of developing these techniques is to provide avenues of therapy for TMJ cartilage destruction and severe synovitis. In order for the treatment goals to be achieved, a multidisciplinary approach is necessary, to include stress counseling, PT, and orthotic therapy to reduce joint overloading. The advent of TMJ diagnostic and operative arthroscopy provides the surgeon with the means to prevent the advanced degeneration of articular cartilage and osseous resorption secondary to advanced osteoarthritis. The final consensus emphasizes that the definitive surgical treatment of these pathologic entities is ultimately decided and finalized at the time of surgery, using TMJ examination under anesthesia, followed by arthroscopic diagnostic sweep. The latter is the decisive factor in determining the final choice of treatment.

Retrodiskal Perforations

Identification and documentation of the retrodiskal perforation should exist from the previous diagnostic arthroscopy. This time, the surgeon performs a short diagnostic sweep and, without further delay, using the vector technique previously described, establishes a second portal in the anterior recess. A blunt probe will, next, ascertain the disk quality and mobility. The anterior disk-synovial juncture is identified with a hook probe. The blunt probe and scope are then swiveled into the posterior recess. The perforation is measured and, if visible, the condyle is palpated. A whisker shaver may be now employed for removal of any "crab-meat" type of cartilage degeneration on the periphery of the perforation, which will permit better assessment of the size and shape of perforation. This also allows for better visualization of the condyle. Now is the time to make all the MRI and arthroscopic finding correlations. If feasible, the anterior release is performed, followed by disk reduction and diskopexy. The suturing technique in this particular situation is modified from the one presented previously in the incorporation of the perforation into the retrodiskal flexure. Sometimes this is not practically achievable with just one suture. Additional anterolateral releasing is indicated together with placement of additional sutures, up to a total of two or three. If, despite all attempts, the perforation is not completely closed, an assessment of the size and position of the residual perforation has to be performed. In the absence of crepitation and osseous contact in the fully seated condyle position, the goal of an interposed soft tissue pad has been met, much like the case of the autogenous dermal graft, temporalis muscle flap graft, conchal graft, or lyophilized/freeze-dried dura graft.

Central Perforations

The goal of managing the large central perforation does not focus so much on restoring the normal functional anatomy as on rather achieving a balance between the loading forces sustained by the condyle, the remainder of disk, and the glenoid fossa. No diskopexy should be attempted for these cases. Efforts are directed at decreasing crepitation, smoothing bone-to-bone contact while improving joint mobility if indicated. Meniscectomy is reduced only to the diseased portions or the ones impinging on condylar motion. The first step is débridement of all advanced degenerative fibrocartilage by means of bipolar/monopolar cautery or laser instrumentation. The margins of the disk perforation are trimmed while the degenerative changes of grade III and IV chondromalacia are removed from the eminence. The joint is then lavaged and the joint mechanics are observed arthroscopically. Particular consideration is given to the bone-to-bone contact areas. Wherever these are noticed, abrasion arthroplasty is performed. Once the area to be reduced is identified, the condyle is translated well past the apex of the articular eminence while the assistant pivots the mandible to the contralateral side, thus increasing the space between the impinging surfaces to allow access for instrumentation. The area is adjusted and blended into the medial and lateral aspects as well as the posterior slope of the eminence into the normal contour of the fossa. The maneuver is repeated until the crepitation is removed or significantly reduced. Osteophytes on the medial aspect of the apex of the eminence are osteoplasticized. Not all medial osseous prominences are removed, because they sometimes provide useful meniscus loadbearing area. High condylar osteoplasty transperforation may sometimes be necessary. The procedure is terminated once osseous impingement is relieved and crepitation abolished/reduced. Irretractable clicking or clicking developed postarthroplasty is usually a consequence of anterior and/or anteromedial meniscus perforation. This author manages this problem by trimming the margins of the perforation with a suction punch or by means of laser instrumentation. In the rare instances in which the anterolateral aspects of a perforation need to be adjusted an interchanging of the straight- and side-firing laser tips will be necessary.

Arthrofibrosis

With the invaluable information provided by the diagnostic arthroscopy at hand, the surgeon proceeds with a short diagnostic sweep, assessing also any changes in the status of joint stenosis and/or refibrosis of the areas that were previously opened by the lysis and lavage. After assessment of the medial sulcus, the scope is pistoned laterally and swiveled through the intermediate zone into the anterior recess. In most instances, this will not be possible for the same reasons (impingement or "white out"). Using the vector triangulation technique, the second puncture establishes the anterior recess portal. The Ho:YAG laser is used for the débridement of the joint. This maneuver is carefully advanced into the anterior recess by contouring along the eminence. As the débridement proceeds anteriorly and more joint space is opened, a third puncture may be necessary to gain adequate access for the procedure. The scope can be switched into the second-puncture portal. The limiting factor for the débridement is a normal anterior recess volume. Evidence may reveal that there is no residual disk present. Osteoplasty of the condylar head is performed in the same fashion as described earlier. Indiscriminate arthrectomy should be discouraged and surfaces with normal aspect fibrocartilage and/or synovium should not be violated. The arthrectomy procedure is terminated when a notable improvement in excursive motion is reached. The interincisal opening is reassessed and documented. In bilateral cases, the procedure follows the same pattern in the contralateral joint. The interincisal opening is always documented at the end of the procedure

POSTOPERATIVE PATIENT MANAGEMENT

General Anesthesia Considerations

The concept of patient management implemented immediately after termination of the surgical procedure. Irrespective of the type of arthroscopic procedure performed, the postarthroscopic surgeon's guidance of the anesthesia team is a necessity, not a recommendation. Reversal of anesthesia stages (refer to American Society of Anesthesiologists [ASA] classification) to consciousness has to be a much more deliberate and controlled process. The classic low stage II/stage I extubation procedure, with the patient following commands of "eyes open" and "deep breath," followed by extromission of the nasoendotracheal (NET) tube, does not necessarily apply to the TMJ arthroscopic patient. The spastic perimandibular musculature contractions secondary to the gag reflex have to be avoided, especially in patients who have undergone suture diskopexies or posterior scarification procedures. Extubation in a semi-obtunded patient is the ideal in these clinical situations. To ensure uneventful extubation, the surgeon passes a nasogastric tube and suctions all existent gastroesophagopharyngeal contents or secretions at the end of surgical procedure. This author has taken the time to get oral and maxillofacial surgery (OMS) residents very proficient with this elementary procedure by implementing its performance with every single operated case by the OMS team. Unfortunately, open airway general anesthesia is more in the comfort zone of the OMS than most of the medical anesthesiologists. This particular request for the anesthesia team may not have a positive response in most hospitals at this point, for more than the previously mentioned reason. Longer awakening time/minimal alveolar concentration would be necessary, encroaching on operating room turnover times that are already "stretched" in many hospital environments. There are, definitely, a few remarks to be made with regards to our foreign colleagues who are performing TMJ arthroscopic surgery under intravenous sedation and local anesthesia.

Analgesia/Pain Management

This author advocates administration of toradol 30mg both intravenously and intramuscularly before extubation, for the purpose of smooth transition from general anesthesia to intravenous or oral. analgesia. A regimen of oral toradol, if tolerated, is instituted for the next 5 to 7 days, with class III narcotic analgesics for breakthrough pain, in the majority of cases. In the immense majority of cases, we have been very successful in managing postoperative discomfort with this regimen.

Anti-inflammatory Management

Regardless of the positive effects of toradol administered by the previously mentioned protocol, our patients are receiving tapered doses of intravenous/oral steroids for the next 18 to 24 hours postoperatively. On occasion, the patient diagnosed with osteoarthritis may be prescribed a longer (up to 4 wk) regimen of nonsteroidal anti-inflammatory drugs (NSAIDs).

Antibiotics

Notwithstanding minimal invasive character of TMJ arthroscopic procedures and impeccable surgical technique, since the inception of routine postoperative antibiotics, our rate of postoperative infection has been 0%. Every patient is bridged from intravenous to oral cephalexin (Keflex) for no more than 8 to 10 days, in the absence of intolerance or allergic reactions.

Diet

The full liquid diet is advanced to a strictly soft diet in a very gradual fashion. The expected transitional malocclusion, usually with posterior inocclusion, is a period of intra-articular settling that has to occur in an undisturbed environment. This critical postoperative transition to norm-occlusion cannot be encroached upon by an accelerated return to function or parafunction. The importance of an appropriate orthotic for this postoperative period cannot be overemphasized. The description of the occlusal splint falls beyond the scope of this chapter. It is sufficient to emphasize that this author prefers to fabricate soft splints with appropriate anterior guidance to be inserted regularly qhs.

Stage	Goal	Exercises
I	Maintain mobility and strength	1. Limited active vertical opening, right and left excursion and protrusion 2. Isometrics in neutral
II	Increase range of motion	1. Active assisted exercises (guided by hand) to opening 2. Left and/or right side excursion 3. Protrusion
III	Smooth active range of motion without deviation or asymmetry	1. Feel for deviation in rotation, translation, and protrusion 2. Correct deviations in rotation, translation, and protrusion through isotonic resistance 3. Create smooth movement through isometrics 4. Create smooth motion with resistance throughout the range through active correction
IV	Progressively load	1. Opening and closing 2. Side excursion 3. Protrusion

FIGURE 49-103. Rehabilitation stages, goals, and exercises.

POSTOPERATIVE REHABILITATION

Routine referral to a physical therapist is not recommended. There are specific situations in which the patient benefits from the help of the therapist, mainly cases that have a very slow progress, no progress at all, or the opposite. The table in Figure 49-103 addresses the stages of rehabilitation, specific objectives, and exercises.

Stage I Physiotherapy

In most instances, patients start stage I rehabilitation on postoperative day 1. The exceptions are the cases with significant loss of ROM, where stretching must be initiated immediately, hence stage II rehabilitation, and at the opposite end of the spectrum, patients who have undergone posterior contracture as treatment for dislocation, in whom mandibular hypomobility is advisable for the immediate postoperative recovery. Nevertheless, most patients are discouraged in developing hypomobility habits to prevent the inherent cicatricial tissue formation. Stage I rehabilitation focuses on reducing discomfort, pain, spasms, and inflammation (mainly swelling). Stage I exercises consist of limited ROM exercises and gentle isometrics. The entire physiotherapy is performed in a slow and comfortable fashion, short of any sharp pain. Typically, regardless of the results of postoperative manipulation under anesthesia, the patients will only hinge-open 10 to 20 mm (one fingerwidth). The target of stage I PT is for the patient to open two finger widths (30–35 mm). The following exercises are performed in series of 10 each four times a day. With apex lingue contiguous with the anterior palate at all times, the patient will vertically open as permitted by tongue length. Patients are educated to feel the rotation of the joint, avoiding excessive translation. Lateral excursion exercises are performed with a finger on the incisor teeth and moving jaw tooth-to-tooth. Protrusion exercises involve only lining up of the anterior dentition. One way to characterize excursion exercises in stage I is "just-to-border" ROM. Isometrics are accomplished by gentle resistance to the elevators and depressors of the mandible in all excursions, with tongue in the palate and slight inocclusion. Stage I PT is performed for at least 2 weeks postoperatively.

Stage II Physiotherapy

The obvious goal of stage II PT is to increase ROM with stretching exercises, using intraoral manipulation. The joint is stretched into rotation, translation, protrusion, lateral excursion, and longitudinal distraction, in order to prevent development of adhesions and/or muscle rigidity/shortening. PT stage II follows the same basic philosophy and frequency as described for stage I. Patients should feel stretching and some discomfort, short of sharp pain or ripping sensation. The following are time-tested PT exercises allowing the patient to maintain and increase ROM. The "contract-relax" is an opening exercise consisting of attempting maximal intermaxillary opening while using a hand to resist opening and closing isometrically and then opening a little farther. In the "hook-pull," another opening exercise, the patient hooks the index around the menton attempting maximal MIO, and then giving a gentle pull. The most effective opening exercise is the "pry-bar." The patient places each pollicis on the incisal margins of the anterior maxillary dentition and each index or middle finger on the antagonist mandibular dentition. The MIO is gently increased by assisting the end motion (prying the mouth open). To increase the ROM of excursions, the exercises described for stage I are performed in a "past borders" fashion. The unilateral surgery cases require more patient education in performing the PT assignments, because stretching a joint past the point of restriction, especially in the opposite direction dictated by the contralateral working condyle, has a certain learning curve from the patient's perspective.

Stage III Physiotherapy

Stage III PT consists of muscle reeducation. The patient is assessed for asymmetry or interruptions in motion, overstretching, and/or inappropriate movement patterns. Because stage II has already addressed the possible ROM problems, adequate joint mobility should be present. Nevertheless, the patients continue with the entire gamut of earlier stages of PT, while making neuromuscular progress in order to ensure maintenance of ROM. Patients learn to correct vertical opening, protrusion, and lateral excursion movements with the goal of fostering appropriate function. Correction of asymmetries is achieved by increased perception of "internal cues" (correction of restrictions and deviations using fingers

and tongue as guides). Visual input (mirrors) is discouraged. Opening deviations are corrected by opening to the point of deviation/excessive translation and using a hand on the contralateral side to resist the incorrect motion. Once initial correction is achieved, the tongue takes over by inserting the apex into the palate and controlling vertical opening without deviation. Also, isometric and isotonic exercises are performed opposite the deviation, at the particular point in the ROM at which the deviation occurs.

Stage IV Physiotherapy

The goal of stage IV PT is to progressively strengthen the mastication musculature until *restitutio ad integrum* (return of full functional ROM and premorbid diet) is reached. Isotonic and isometric exercises are performed throughout the entire ROM. Close monitoring of the patient will prevent development of synovitis and myositis. Prevention of injury by loading in the extremes of motion is implemented. Advancement of diet is also monitored closely in order to prevent an accelerated detrimental progress.

Diagnosis-oriented PT Intervention

MPD

The end point of PT should consist of regaining full painless ROM and significantly raising the sensitivity threshold of the "trigger-zones," while slowly progressing the patient through stages I to IV. Case-oriented injury prevention, neuromuscular relaxation, corporeal mechanics, and stress management to reduce abnormal mechanics to the myofascial system are implemented. Appropriate function of the entire stomatognathic system is ensured.

Maxillomandibular Deformities

While the patients progress through the four stages of PT, the scope of intervention consists of treating the myositis and TMJ dysfunction at the same time with maintenance of uncomplicated PT.

Direct and Indirect Macrotrauma

Whether involving fractures or not, PT for these patients is progressively taken through all stages, with the mind-set of limiting cicatricial and, most important, fibrotic changes that could potentially result in decreased ROM with or without pain.

Hypomobility/Fibrosis

After manipulation under general anesthesia at the end of the surgical procedure, stage I PT is initiated immediately in order to maintain ROM and mobility. Stage II requires aggressive management. Patients are stretched vigorously, short of eliciting severe pain. During stages III and IV, patients progress to normal function via muscle reeducation and strengthening.

Hypermobility

Patients remain in a conservative stage I for at least 2 to 3 weeks. Overstretching is to be avoided. Stage II is also performed in a conservative fashion with emphasis on avoiding overstretching. The focus of stage III is to maintain the patient within, and not beyond, his or her functional ROM. During stage IV, the emphasis is on strength training and progressive joint loading, advancing diet to the premorbid state.

Myositis

PT is performed in conjunction with anti-inflammatory medications and cryotherapy.

Synovitis, chondromalacia and osteoarthritis

Stages I to IV PT with concomitant anti-inflammatory treatment is implemented.

Articular Disk Disorder

These patients require only a short stage I protocol. If any disk repositioning or diskopexy was performed, stage I is maintained for 2 weeks postoperatively. Then stages II to IV follow in a conventional fashion.

COMPLICATIONS OF TMJ ARTHROSCOPIC SURGERY

Damage to Cranial Nerve VII and Facial Palsy/Atony

When approaching the TMJ for a glenoid fossa portal puncture, the frontotemporal branch of the facial nerve should be anteriorly, whereas for an eminence/working portal puncture, it is located posteriorly. However, the zygomatic branch may be in jeopardy as well. In this author's experience, paresis of the frontotemporal branch occurs in less than 1% of procedures (reaching a high of 0.73% in the early days of arthroscopy). Invariably, these patients had undergone open arthrotomy before the minimally invasive procedure. This is suggestive of scarring of the preauricular tissues, causing an added amount of pressure on the nerve branches from joint distention, resulting in palsy of the frontotemporal and zygomatic facial rami. It becomes manifest as an atony of the orbicularis oculi and the frontal division of the occipitofrontalis, translated in the partial ability to elevate the eyebrow and to completely occlude the superior palpebral, both in the immediate postoperative period. A definite diagnosis is made 24 hours postoperatively. Typically, the duration of signs varies between 1 week and 6 months. An additional mechanism of injury for cranial nerve VII with this procedure is the extravasation phenomenon with/without hematoma formation. Misplacement of the irrigating cannula or overpressure joint lavage can force solutions beyond the confines of the capsule into the adjacent tissues. The immense undisputed majority of incidents involving cranial nerve VII are compressive in nature (equivalent to neurapraxia described by Seddon and Sutherland) in which injuries include both

mechanical deforming forces and ischemic factors. Upon injury, first, there is an increased vascular permeability resulting in subperineural and endoneural edema, leading to connective tissue changes including perineural and epineural thickening, followed by localized nerve fiber changes, in which some patients function normally whereas others exhibit segmental demyelinization. As the magnitude and the duration of compression increases, wallerian degeneration becomes apparent. The peripheral fascicles and nerve fibers are affected primarily, whereas more centrally located fascicles and nerve fibers may be spared. Should axonotmesis and/or neurotmesis occur during puncture, the only factor obstructing nerve regeneration would be cicatricial tissue, which competes with the axonal cone growth to bridge the gap. In this author's experience, cranial nerve VII injuries have a good prognosis for *restitutio ad integrum* of function.

Damage to the Collaterals of Cranial Nerve V (Auriculotemporal, Lingual, or Inferior Alveolar Paresthesia)

The ATN along with the superficial temporal artery and vein are posterior but in proximity to the fossa puncture site. Our present experience has revealed that hemorrhage is uncommon, and if encountered, it is not significant and is stopped with direct pressure. Postoperative anesthesia around the entry sites is a common occurrence that spontaneously resolves within 2 weeks. No hyper- or dysesthesia has been reported. Rotation of the trocar cannula while penetrating the portal sites permits the bypass of important vital structures as well as puncture into the intracapsular space with minimal resistance. This complication stems in a placement too far posterior of the fossa portal as a consequence of operator concern for cranial nerve VII damage. When the lingual nerve is concerned, the most immediate proximity to the mandibular condyle report comes from Johansson. On an extensive cadaver study, the mandibular nerve was dissected running vertically from foramen ovale at approximately 10 mm inferior to the foramen. It then divided into the inferior alveolar and lingual rami, with the later passing 3.5 mm medial to the condyle. The nerve can be injured if the operator is not very cognizant of the intra-articular anatomy and perforates the drape at a distance of more than 35 mm from the tegument. Another possibility is the medial extravasation of irrigation fluid secondary to medial capsular perforation without a correct irrigation system, involving also the inferior alveolar nerve. Hyperesthesia of the infraorbital nerve is associated with long operative time, excessive volumes of irrigation fluid, and extravasation of irrigant into the medial tissues. The condition is self-limiting because the fluid is resorbed into the lymphatic and venous systems. Extravasation is prevented by careful puncture technique, observation of the surgical site, gentle pressure to irrigation, and a patent inflow/outflow system.

Damage to Cranial Nerve VIII and Vestibulocochlear Dysfunction

Tympanic membrane perforation and ossicles disruption in the middle ear has been reported, including otitis media with subsequent hypoacousia. The mechanism of entry into the middle ear is through either the osseous or soft tissue external auditory meatus. If the tympanic membrane is perforated and the ossicles appear in the view field, immediate cessation of procedure and intraoperative ENT/ORL consultation is warranted. The ossicles have a classic melted-wax appearance. If no manipulation is performed, the incidence of permanent disruption of ossicles is low and the complication will be limited to the tympanic membrane. Small perforations in the anterior or inferior portions of the tympanum typically cause minimal hearing loss and heal uneventfully, without sequelae. Posterior tympanum injuries may dislocate the ossicles and potentially result in more significant loss of hearing. All perforations involving more than 30% of the tympanum surface or the posterior portion of it should be managed by an ORL. Postoperative complaints of severe hearing loss and/or vertigo alert the surgeon for a possible middle ear injury and an ORL/ENT referral should not be delayed. Laceration of the external auditory meatus occurs by transfixion of the canal with the arthroscope at the junction of tragus and osseous meatus. Minor hemorrhage is controlled with bipolar cautery, while the external auditory meatus is treated with hydrocortisone suspension drops for up to 2 weeks. If granulation tissue develops at the osseous/cartilaginous junction, bipolar cautery is employed. Baldursson and Blackmer have suggested the existence of a correlation between specific sensorineural hearing loss, with a predictable decrease in hearing levels at the 1000- to 2000-Hz range, and TMJ dysfunction and/or parafunction. In this author's experience, all patients regained normal hearing levels within 2 months postoperatively. This confirms the fact that arthroscopy does not affect hearing levels if no direct trauma to the middle ear has occurred. Close consideration of the detail of not advancing the scope past 20 to 25 mm without accurately checking its position will prevent the occurrence of this complication.

Scuffing of Fibrocartilage

The cartilage covering the eminence and fossa is most prone to iatrogeny, the most common arthroscopic complication. At the time of insufflation, the needle point is directed toward the posterior slope of the eminence, making contact with the fibrocartilage. Also, examination sweeps of the joint cavity, involving translation of arthroscope along with cannula, could also release pieces of cartilage into the superior joint space. If scuffing becomes significant, it impairs visibility during arthroscopic procedures to the point of misdiagnosis of chondromalacia by the inexperienced arthroscopist.

Damage to the Maxillary Artery/ Collaterals with or without Formation of Arteriovenous Fistula

As it courses medial to the condylar neck, the artery was found immediately lateral to the lateral pterygoid in two thirds of reviewed cases. This author has encountered this complication, resulting in one isolated case of left pterygoid arteriovenous fistula (AVF).

Damage to the Superficial Temporal Vessels with or without Formation of AVF

Whether profuse or not, all cases of hemorrhage from the superficial temporal artery and vein intimately related to the posterior aspect of the joint capsule were managed uneventfully by applying controlled pressure. Other authors have reported the formation of an AVF. Typically, the patient complains of a constant "hissing"/"whishing" sound over the operated TMJ. The fistulectomy and subsequent embolization of the superficial temporal artery were uneventful.

Perforation of the Glenoid Fossa

As previously discussed under "Danger Areas," this complication can be consistently avoided by directing the instruments toward the tubercle and away from the fossa. Most violations of the middle cranial fossa will result in cerebrospinal fluid leaks that resolve spontaneously. Should the leak persist in the wound or through the incision, a pressure dressing is applied and the patient is hospitalized with head elevation. Persistence of leak after 48 hours mandates neurosurgical consult and lumbar subarachnoid drain placement. Head CT with bone windows is obtained to document the site. Surgical dural neuroraphy is very uncommon.

Damage to the Disk

Repairs of the meniscus result in a fibrous tissue seal of the surfaces as seen arthroscopically. Core biopsies have shown minimal tissue reaction at the area of repair, consisting of fibrous cell repair with sparse vasculature. There is minimal collagen formation at the repair state and no collagen penetration or angioproliferation into the substance of the repaired meniscus. All needle punctures are filled in and smooth with the meniscus surface 12 weeks postoperatively. Exposed non-resorbable sutures are covered with translucent fibrous tissue. The cellular response to meniscal tears stems in four potential sources: the adjacent synovium, the capsule vascularity, the intra-articular microhemorrhage, and the free synovial cells. The adjacent synovium contributes angioblastic and cellular tissue for peripheral meniscal tears. The synovium proliferates in the area and migrates over the disk in a similar way to the pannus noticed on articular cartilage defects. Tears in the lateral one third of the meniscus have abundant vascularity for hemorrhage and angioblastic proliferation. The avascular portion of disk presents fibrous tissue healing response 4 to 6 weeks postoperatively.

Hemarthrosis

Hemarthrosis is a consequence of laceration of the superficial temporal artery and vein and severely inflamed synovium/retrodiskal tissue upon entry, as well as laceration of the pterygoid artery during myotomy for anterior release procedures. Hemorrhage can be significant enough to cause abortion of procedure. When pressure irrigation cannot tamponade the hemorrhage (despite some clearing of the surgical field), the joint remains congested, prolonging the healing, increasing postoperative discomfort, and extending recovery time. While the intra-articular coagulum organizes, it increases the risk of formation of adhesive bands and scarring, reflected clinically in fibrous ankylosis. This results in limited range of motion of the mandible in all excursions (vertical, protrusive, and lateral). Interincisal opening will be less than 35 mm, while the lateral and protrusive movements are in excess of 6 mm. This author uses the following protocol for stopping excessive hemorrhage:

1. The pressure in the irrigation bag is increased.
2. A small amount of Healon (0.5 mL) is injected intra-articularly.
3. Cautery/laser is applied to the bleeding area.
4. Using a $3^{1}/_{2}$-inch spinal needle on a 3-mL syringe, passed through the cannula into the affected area, a small amount of local anesthetic with vasoconstrictor is administered directly into the bleeding site.
5. De novo insufflation of the joint with local anesthetic, transcannula (2% lidocaine 1/100,000 epinephrine) should tamponade the hemorrhage.
6. The entire joint is insufflated under pressure, with irrigation fluid while all cannulas are obturated for 5 minutes for the hydrostatic pressure to achieve tamponade of the site.
7. Should all previous measures be unsuccessful, all the instruments are removed from the joint and direct palmar or digital external pressure is applied for 5 minutes by the clock. For added pressure, the condyle is seated in the fossa, particularly if the source of bleeding is located in the posterior pouch. If the source is in the anterior pouch, the mandible is manipulated to a protrusive position. The instruments are then reinserted in their original portals and the condition of the joint is reassessed.
8. A no. 4 catheter balloon is inserted through the working portal and inflated with normal saline. It is left in place for 5 minutes, then deflated, and the joint is reassessed.
9. If all measures were rendered unsuccessful, the joint is approached via open technique.

Infection

Infection is quasi-infrequent as a consequence of proper sterilization, sterile operating environment, forgiving high

vascular tissues, antibiotherapy, and high-volume irrigation. The presentation is with erythematous puncture sites with surrounding edematous halos 3 to 5 days postoperatively. Administration of a cephalosporin for 7 days resolves the problem. This author now uses a standard protocol for all arthroscopic cases. Preoperative cephalosporin (with the alternative for erythromycin in penicillin-allergic patients) is administered at recommended doses. The intravenous antibiotics are continued while in-patient status is maintained (usually 24 hr). Oral antibiotics are continued for 7 days.

Noninfectious Postoperative Effusions

Noninfections postoperative effusions present with preauricular edema, with a higher level of tenderness to palpation than normally encountered postoperatively. The patients were managed with soft diet, joint rest, and application of heat over the affected area. NSAIDs were prescribed as needed. Uneventful, complete resolution of symptoms was evident at 4 weeks postoperatively. If an effusion is persistent after 6 weeks, it can be aspirated. If that fails, steroid injection is administered as the last nonsurgical modality. Caution should be exercised to avoid misdiagnosis of this condition, because some effusions develop a few weeks/months after the surgery. Those are an indication of progression of degenerative disease.

Instrument Failure/Loose Bodies

Instrument failure can be attributed to manufacturing defects, misuse, and wear of parts of the instrument itself, all potentially leading to breakage. Backup instruments, including the arthroscope, are mandatory while performing arthroscopy. All instruments with flexible parts should be tested by the surgeon before introduction in the joint (including On Point scope, trocar/cannula, scissors, biopsy forceps). Application of excessive force, extreme bending of the scope, especially when negotiating access to the most anterolateral aspect of the superior joint compartment to determine the site for the second puncture, can cause damage or breakage to the optical system and need for replacement or repair. The "golden rules" include never force/power-move an instrument; use ferromagnetic instruments; always have a "golden retriever" available; avoid force in removing the instruments; and confirm that every movable instrument is closed before removal. In the case of such an inadvertent event occurrence, this author has established a standard protocol. The following steps are to be followed:

1. Stop the procedure, while maintaining the position of the arthroscope and working cannulas.
2. Keep the instrument or foreign body in view.
3. Check the inflow bags to confirm sufficient irrigation fluid remains in order for the joint to be maintained distended at all times.
4. Record and measure the depth of the instrument with a scored cannula.
5. Have adequate removal instruments available, including extras.
6. Adjust inflow to ensure optimal visibility.
7. Take a radiograph of the joint if the instrument cannot be located arthroscopically.
8. Consider fluoroscopic assistance.
9. Remove the fragment/object.
10. When using a grabber, tips may not fit in the working cannula upon removal. It may be desirable to step-up the working cannula to a 3-mm diameter, using a "switch stick," and then perform the retrieval maneuvers. If the instrument cannot be retrieved, the next arthroscopic attempt should be made in the early postoperative phase (10 days to no longer than 6 wk postoperative). Should this second attempt fail, the doctor and the patient must review all possible outcomes associated with leaving the foreign body as is. The possibility/likelihood of future osteoarthritis or foreign body reaction must be very well understood by the patient.

CONCLUSIONS

This chapter contains rather overwhelming information for the reader, whether a resident training in our specialty or a seasoned specialist. One of the points we have, time and time again, made over the years is that training in this particular aspect of our specialty is inadequate. Henceforth, it is not our intention to dissuade the oral surgeon from pursuing training in this particular aspect of our specialty, but the contrary. By writing this chapter, we hope to make the readership aware of how underrated this subspecialty is in reality.

Throughout our formation as maxillofacial surgeons we have self-trained to give particular consideration to minute detail. This author has designed the office arthroscopy procedure banking on this particular trait of character of the OMS. It is one of the products of almost 30 years of experience in the field of arthroscopy. The surgical procedure has been refined and simplified to prevent the surgeon from thinking the arthroscopy is complicated and inefficient. Whereas the advanced arthroscopist does need to possess not only skill but also a certain personality to develop the interest in perfecting her or his knowledge and technique, the office-based arthroscopy is designed in such a manner that makes it feasible and predictable in the hands of all OMSs. As mentioned earlier in the chapter, the lysis and lavage procedure will resolve the acute TMJ-related symptomatology in 65% to 70% of patients. Performed arthroscopically, this procedure provides valuable visual information and makes the surgeon comfortable with basic arthroscopic maneuvers. We feel most surgeons will develop the interest to make the progression to advanced arthroscopy once confident at this level.

The second major point we are making at the end of this chapter is the importance of the arthroscopic cascade. This concept is also a product of this author's vast experience. The results of treating TMJ disorders based on this concept have

shown impressive percentages of success. These results have been published numerous times over the past few years. The predictability of achieving success in the arthroscopic treatment of Wilkes stages I to V disease dramatically decreased the necessity for traditional/"open-joint" surgery. Although many reputed academicians of OMS will retort by showing numbers revealing success with open technique, ultimately culminating with a total joint replacement, we should bear in mind that, as successful as it may be, the final treatment (i.e., total joint) is the end point of TMJ surgical therapy. It is the strong belief of this author that the TMJ surgeon should not indiscriminately stampede to the final treatment. One of the characters that brought Clint Eastwood to stardom, inspector Harry Callahan a.k.a. "Dirty Harry," often used to state that "A man's gotta know his limitations!" As TMJ surgeons, we need to accept the idea that we are managing an existing problem and not curing a disease. Notwithstanding the tremendous results and improvement in the quality of life of the patient after total joint replacement, these devices have major limitations that fall out of the purpose of discussion of this chapter. The arthroscopic cascade stems in these very premises. It will postpone the end point procedure, in most instances, indefinitely.

We close the circle of conclusions for the chapter with a pleonasm, exactly where we started. As difficult as TMJ arthroscopy seems, attention to detail and strict adherence to the steps and stages of arthroscopic technique will make the surgeon not only successful but also comfortable and confident. We are overemphasizing this concept because, as opposed to traditional surgical technique, any minor deviations from the arthroscopic surgical technique will result in less than desirable outcomes or complications.

Ending on a positive note, this very reasonable approach to the orthopedic management of TMJ-related problems has a very high rate of acceptance and satisfaction with patients. Because the progress of the science and technology is virtually unstoppable, the discipline of TMJ arthroscopy will continue to evolve. This author is committed to the advancement of technology by directing a number of manufacturers in specifically decreasing the size of instruments, making them more accurate, more multitasking, and more suited to minimally invasive TMJ arthroscopic surgery.

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CHAPTER

Internal Derangement of the Temporomandibular Joint

Luis G. Vega, DDS, Florencio Monje Gil, MD, and Rajesh Gutta, BDS, MS

With a multifactorial etiology and poorly understood pathophysiology, the surgical management of temporomandibular joint (TMJ) disorders is one of the most controversial and challenging topics in oral and maxillofacial surgery. Without randomized clinical trials comparing treatment options for TMJ disease including surgical treatment, medical management, and no treatment, the true value of surgery in the management of TMJ disorders may never be established conclusively. Albeit, the literature on TMJ surgery is considered suboptimal, the results cannot be ignored, and current recommendations for TMJ surgery must rely on the best available evidence.[1]

The term *TMJ internal derangement* was adopted to classify any pathologic entity that interfered with the smooth function of the TMJ. Movement disturbances in which the articular disc plays a central role are referred to as TMJ disc derangements. Although they are viewed as a separate category of intra-articular conditions, some authors suggest that disc derangements and osteoarthritis are intimately related since much of their clinical courses overlap.[2–4]

Historically, clinicians have recognized that surgery for disc derangements should be reserved for patients with pain or dysfunction that is severe, disabling, and is refractory to nonsurgical management. These conditions still form the basic indications for TMJ surgery. However, the approach to open joint surgery for TMJ disc derangements has undergone a complete metamorphosis as a result of the research and clinical results of TMJ arthrocentesis and arthroscopy. At one time, only a handful of surgeons professed the viability of function with a displaced disc and argued against surgical repositioning of the disc. Today the philosophy is reversed, and the majority of surgeons recognize that a disc derangement does not imply an ipso facto need for open joint surgery. Furthermore, the presence of persistent symptoms in light of disc derangement does not imply that surgical correction is necessary or imminent. Surgery is indicated only if the mechanical obstruction is felt to be the primary etiology of the patient's symptoms.

This chapter discusses the pathophysiology and possible etiologic factors of TMJ disc displacement. The chapter also describes the clinical examination and imaging assessment, as well as the indications and goals for surgical intervention. A brief discussion of surgical anatomic considerations is followed by a description of the classic surgical approaches to the TMJ. Finally, a review of the most common surgical techniques for TMJ disc derangements and their clinical outcomes is also presented.

PATHOPHYSIOLOGY

Clinical course

TMJ disc derangement is a commonly found condition in which the disc is usually displaced from its normal anatomical position. Several stages of progression have been identified wherein not only the disc position, but also its configuration, is altered. The belief that TMJ disc displacements follow a progressive course is based on retrospective and cross-sectional studies.[5,6] For a disc to become displaced, the functional capability of the associated TMJ ligaments must be compromised by deterioration, elongation, or detachment.[7] Once the disc is displaced, the absence of a normal anatomic relationship between the disc, condyle, and fossa has been identified as the most important factor in the initiation of disc deformation.[8] Studies have demonstrated that

reducing disc derangements have a greater tendency to progress to nonreducing disc derangements when the posterior band of the disc shows significant plastic deformation as well as a gradual flattening of the eminence.[9,10] Yet studies have also shown that disc displacements can remain constant over the years[6,7,11,12]. Other studies have demonstrated that in the vast majority of patients with disc displacement, the signs and symptoms gradually resolved regardless of the type of treatment.[13–16] Thus a disc displacement with reduction does not necessarily progress to disc displacement without reduction.

Etiologic factors

Many etiologic factors have been proposed to explain the occurrence of disc derangement including *trauma, joint laxity, bruxism, and changes in the joint lubrication system.*

Trauma

Trauma is probably the most common cause of disc derangement, and this includes acute injury such as blunt trauma to the jaws or whiplash injury, or chronic trauma such as habits bruxism, or occlusal trauma. The traumatic event, or series of events, usually results in soft tissue injury that elongates, tears, or detaches ligaments and disc attachments. Additionally, the traumatic arthritis that may follow produces intra-articular bleeding that can result in fibrosis and adhesions, or hyperplastic tissue leading to pain and dysfunction.[17]

Joint Laxity

Laxity of the joint ligaments, with hypermobility, have been associated with disc derangements.[18] However, the prevalence of joint laxity does not parallel the higher presence of disc derangements. Due to the female predominance of this condition, it has been suggested that hormonal factors may also play a role in the development of disc derangements.[19,20]

Bruxism

Bruxism has been implicated due to the compressive overloading that stimulates production of highly reactive oxidative radicals that destroy hyaluronic acid, collagen, and proteoglycans.[21]

Altered Joint Lubrication System

Although the exact mechanism of joint lubrication is still unknown, it has been suggested that it is primarily dependent upon the synovial fluid. Synovial fluid provides the nutritional and metabolic needs for the articular tissues of the joint. It has been theorized that regardless of the etiologic factor, disc derangement seems to be associated with an increased intra-articular friction preventing the smooth gliding of the joint.[22]

Two major components have been identified as responsible for free joint movement: surface-active phospholipids and hyaluronic acid. Surface-active phospholipids are major boundary lubricants protecting the articular surfaces.[23] Hyaluronic acid, a high molecular weight mucopolysaccharide, forms a film that keeps the articular surfaces separated and prevents friction. It additionally plays an indirect role in joint lubrication by adhering to surface-active phospholipids protecting them against uncontrolled degradation by phospholipase A_2.[24] Joint function remains normal until its adaptive capacity is compromised.[25] Once it is compromised, joint overloading generates a hypoxia-reperfusion cycle that induces the release of free-oxygen radicals that degrade hyaluronic acid, causing a marked decrease in synovial fluid viscosity. Moreover the lack of hyaluronic acid allows the phospholipase A_2 to lyse surface-active phospholipids further jeopardizing the lubrication system. The absence of lubrication will lead to increased adhesiveness, friction, shear and rupture of articular surfaces.[26]

DIAGNOSIS

Clinical Diagnosis

The diagnosis of TMJ disc derangement is achieved predominantly through a thorough clinical examination. A large segment of the general population has only minimal signs and symptoms associated with disc derangements. Careful recording of the chief complaint and history of present illness is imperative, with attention being paid to the details of onset and duration of facial pain and joint noise, timing of symptoms of facial tightness, inability to open or close the mouth, and distribution of headaches. The mere coexistence of muscle dysfunction and a disc derangement does not imply a causal relationship. Concomitant sources of pain need to be identified and consultations are obtained with other medical and dental specialty services including neurology, otolaryngology, psychology, rheumatology, internal medicine, and/or general dentistry, as required. The history of previous surgical and nonsurgical treatment is equally important since the literature suggests that the likelihood of improvement in function and pain management decreases after multiple surgical procedures.[27] Imaging of the joint is performed after a thorough physical examination indicates the need for further information. When a diagnostic dilemma exists, a magnetic resonance imaging (MRI) is usually required to elucidate the clinical presentation. No pain and/or dysfunction are identical to one another, and consequently, cookbook approaches to the diagnosis and surgical management of TMJ disc derangements should not be used. Furthermore, in the surgical decision-making process the surgeon should not take into consideration only the specific diagnosis, but also the patient's perception of the problem, the effect on activities of daily living, and the patient's overall psyche.

TMJ disc derangements can be classified into four clinical settings: disc *incoordination, disc displacement with reduction, disc displacement without reduction, and anchored-disc phenomenon*. Qualifying descriptors are sometimes included, such as the direction of disc displacement, degree of disc

displacement, and presence of a disc perforation. Unfortunately, these large diagnostic rubrics fail to identify the finer stages of the disease process. Disc morphology and severity of displacement are only gross indicators of the disease process. Although more complicated classifications such as the Wilkes classification do exist, the treatments applied to the diagnostic categories have been diverse, rendering specific boilerplate recommendations unadvisable.

Joint Incoordination

Joint incoordination represents the earliest indication of an increase in the frictional properties of the joint. The patients may describe the need to perform a special maneuver with the mandible to achieve a normal opening, or they may describe an annoying terminal jolting sensation associated with mandibular closing. Usually there is no pain, but when present, it is typically not chronic and appears to be related to the instability of the condyle-disc relationship. Most of these cases demonstrate a reducing disc displacement, in which the disc represents a mechanical obstacle and the condyle is not permanently restricted in its range of motion.

Disc Displacement With Reduction

Disc displacement with reduction represents a category in which the articular disc has usually moved in an anteromedial position following the shape of the condyle and anterior slope of the glenoid fossa as well as the influence of the lateral pterygoid muscle. Posterior, medial and lateral displacements can also occur.[28] Reduction refers to the ability of the condyle to negotiate around the disc, with the ability of the disc to assume a normal position in relation to the condyle and glenoid fossa. Normally in these patients, mouth opening is accompanied by a clicking or popping sound that is produced as the condyle passes over the posterior portion of the disc and it returns to a normal position. Clicks are typically reciprocal, occurring both during mouth opening and during mouth closing. The point in the opening cycle where the click occurs (early, middle, late) may correlate with the degree of ligament damage and disc displacement. Patients may be asymptomatic except for the popping or clicking sound, and if the condition is unilateral, the mandible may deviate to the affected side on mouth opening. MRI studies have estimated that the prevalence of disc displacement with reduction in asymptomatic individuals is approximately 30%.[29,30] When the condition is symptomatic, patients complain of localized preauricular pain particularly while joint loading (function). In late stages of the disease, patients could also suffer from intermittent locking, headaches, and myofascial pain that are associated with chronic parafunction of the joint.

Disc Displacement Without Reduction

In disc displacement without reduction, mouth opening becomes limited as the mandibular condyle fails to pass over the posterior band of the articular disc. In symptomatic patients, the overloading and stretching of the highly innervated retrodiscal tissue elicits pain at the affected joint on forced mouth opening and loading. Mandibular deviation to the affected side can be observed in unilateral cases. In some patients, a prior history of clicking or popping that involves the affected joint, or disc displacement with reduction, may precede the condition of a non-reducing disc. Patients often complain of muscle dysfunction secondary to efforts to achieve a normal mouth opening by deviating or deflecting the mandible in order to negotiate over the disc displacement. In the absence of pain, many patients are able to tolerate the restriction in mouth opening, that may gradually improve over several months to years with stretching of the retrodiscal tissues.

Anchored Disc Phenomenon

The anchored disc phenomenon, or acute disc displacement without reduction, is characterized by a sudden, severe and persistent limited mouth opening that is considerably more decreased than disc displacement without reduction (10-30mm). Since the disc is not anatomically displaced, the highly innervated retrodiscal tissue is not compressed, and pain is only experienced when the patient attempts forced mouth opening. Research has suggested that a suction-cup effect occurs, in which the disc is adherent to the articular eminence due to a vacuum effect in the joint space, and well as the presence of increased joint viscosity.[26]

Other Conditions

While establishing a diagnosis of internal derangement of the TMJ, clinicians must be aware of other conditions that could mimic the signs and symptoms to TMJ disc derangement such as joint sounds, limitation of mouth opening, and facial pain. Intra-articular causes include synovial chondromatosis, pigmented villinodular synovitis, various forms of arthritis, and primary and secondary neoplasms involving the TMJ. Extra-articular conditions include pathologies such as myofascial pain, myositis, scleroderma, masticatory muscle fibrosis, hypertrophy of the coronoid process, depressed zygomatic arch fracture, parotid gland pathology, and intraoral and lateral pharyngeal tumors.

Imaging Diagnosis

Although studies have shown that TMJ arthrography has a higher diagnostic value than computer tomography (CT) scans and magnetic resonance imaging (MRI) in the diagnosis of TMJ disc derangements, it has the disadvantage of being an invasive imaging modality that currently is rarely performed.[31]

Magnetic resonance imaging (MRI) has become the study of choice when evaluating TMJ disc derangements. The standard imaging protocol consists of oblique sagittal and coronal images of the TMJ that are obtained perpendicular and parallel to the long axis of the mandibular condyle, obtained in the open- and closed-mouth positions of the mandible. Disc position and morphology as well as bony structures are clearly visualized on closed-mouth and open-mouth MRI images. Open-mouth images are used to evaluate the function of the disc and condyle, in which the disc position

is compared to the closed-mouth images, and to differentiate disc displacement with reduction from disc displacement without reduction. Additionally T1- and T2-weighted MRI studies may be required to delineate intra-articular fluid accumulation, interstitial synovial inflammation, and overall disc morphology.

In the incoordination phase of TMJ disease, the MRI may be normal. Conversely, a closed-mouth sagittal view of disc displacement with reduction will usually demonstrate a disc with varying degrees of displacement and possible deformation. Upon mouth opening, a TMJ click or pop may occur, and the MRI will show the disc back in the normal relationship to the condyle (Figure 50-1). In the case of a disc displacement without reduction, the disc is located in a position more anterior than in patients with disc displacement with reduction. However, the disc remains displaced in the open mouth position (Figure 50-2). With an increasing displacement of the disc, the retrodiscal tissue comes into contact with the condyle and sustains increasing joint loading. The loading results in decreased vascularity of the retrodiscal tissue. With the reduction in retrodiscal tissue vascularity, this tissue transforms into a fibrous band, or a pseudodisc. MRI of chronically displaced retrodiscal tissues demonstrates a signal intensity of the tissue that resembles the disc itself. In fact, radiologists may inaccurately describe a disc fragmentation because only a portion of the displaced disc may display its original signal intensity. The remainder, due to alterations in the glycoprotein distribution and hence the attraction of water, demonstrate a moderate signal intensity. Adhesions or disc perforations associated with disc displacement without reduction cannot be definitively identified on an MRI. However, the evolution of new technologies may allow their visualization in the future.[32] In the case of a MRI of an anchored disc phenomenon, the disc is either in a normal or displaced position, but does not change position from the closed to the open mouth position. Again, on T2-weighted MRI images, inflammatory fluid or increased vascularity appears as a high intensity signal (Figure 50-3).

INDICATIONS, GOALS AND OUTCOMES ASSESSMENT

The American Association of Oral and Maxillofacial Surgeons (AAOMS) initially developed criteria and guidelines

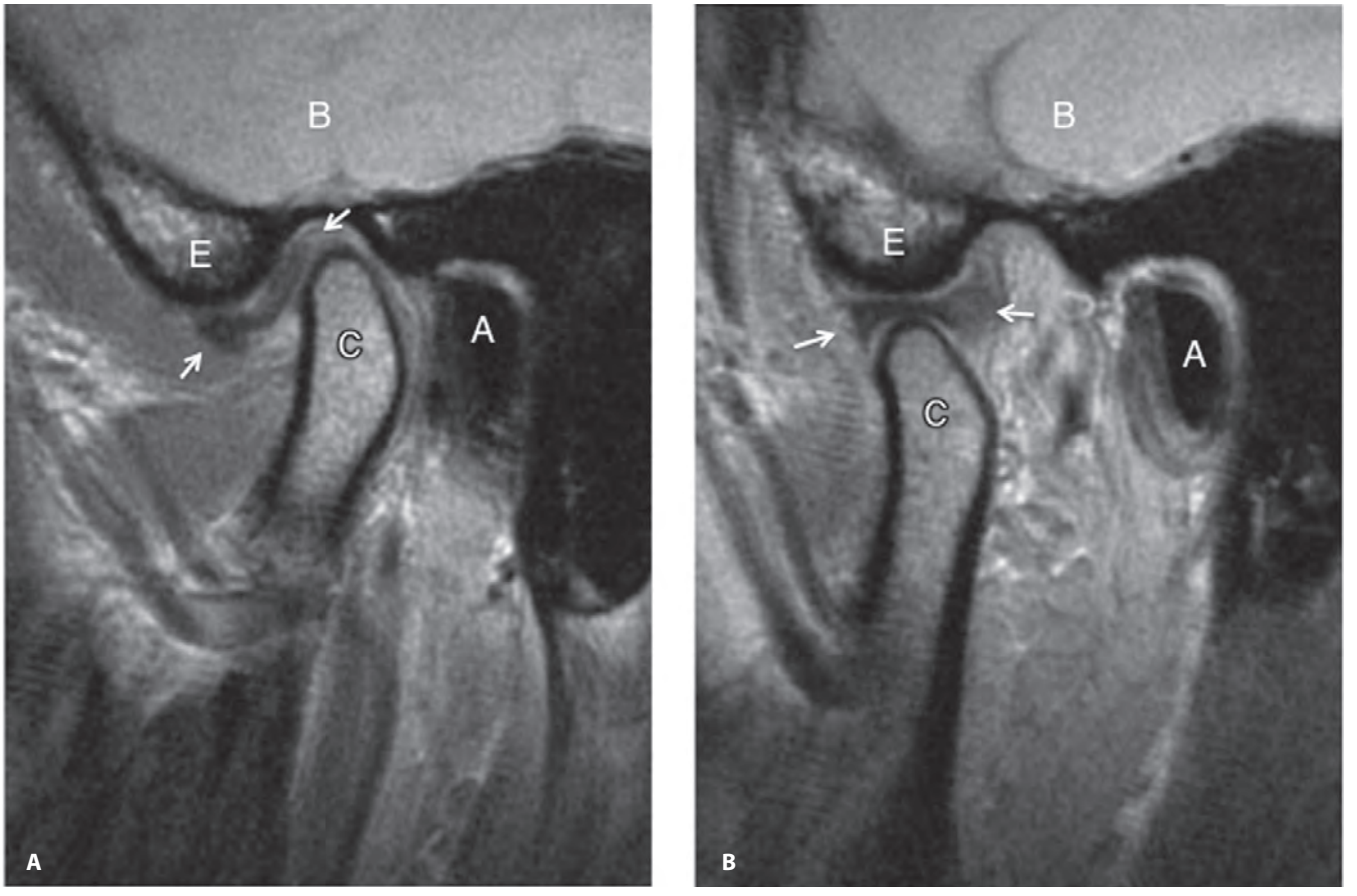


FIGURE 50-1. MRI disc displacement with reduction. **A,** Oblique sagittal view in closed-mouth position showing a partially displaced articular disc (arrows). Note the loss of the normal biconcave (bow-tie) disc morphology. **B,** Oblique sagittal view in open-mouth position showing normalization of the disc morphology and position. (arrows). (A auditory canal, B brain, C condyle, E eminence)

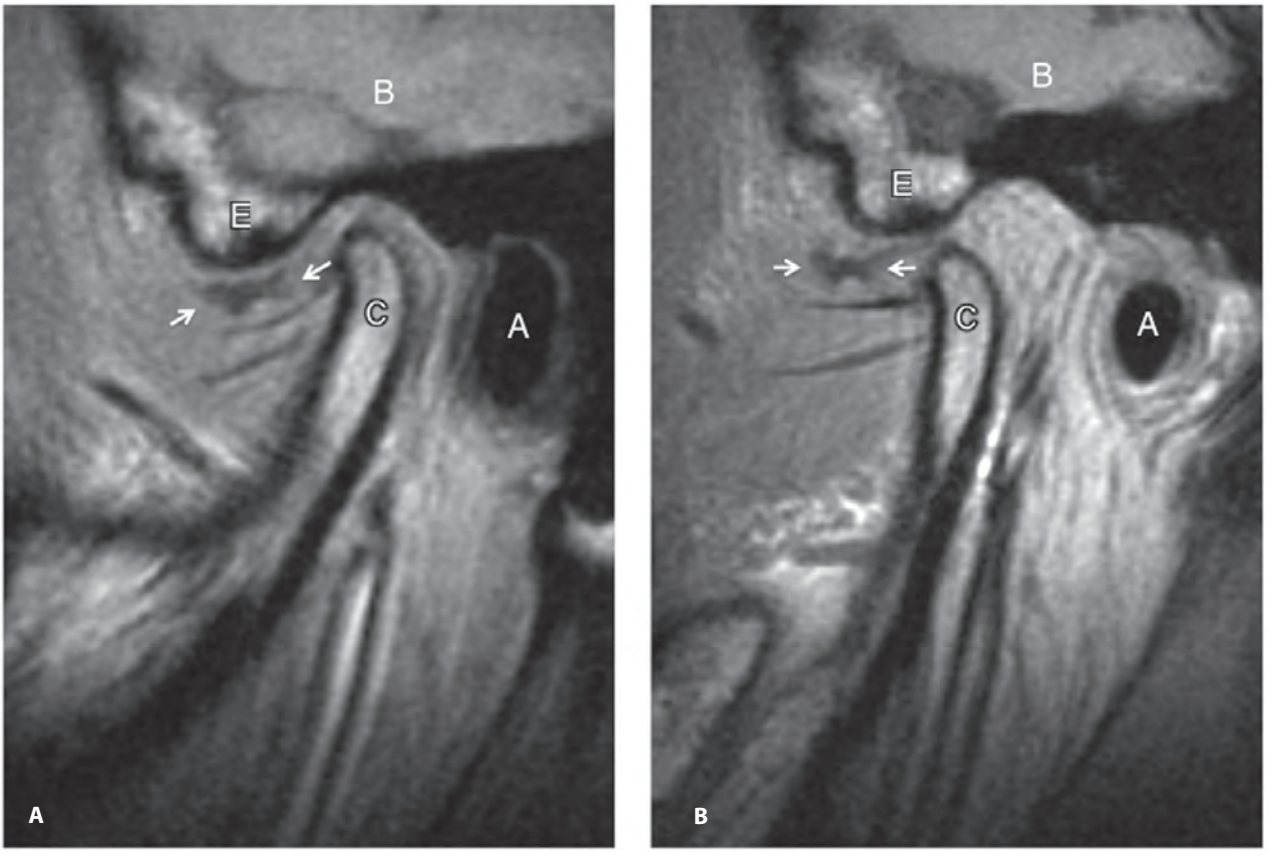


FIGURE 50-2. MRI disc displacement without reduction. **A,** Oblique sagittal view in a closed-mouth position showing a complete displaced articular disc (arrows). **B,** Oblique sagittal view in open-mouth position showing lack of normalization of the disc position. (arrows). (A auditory canal, B brain, C condyle, E eminence)

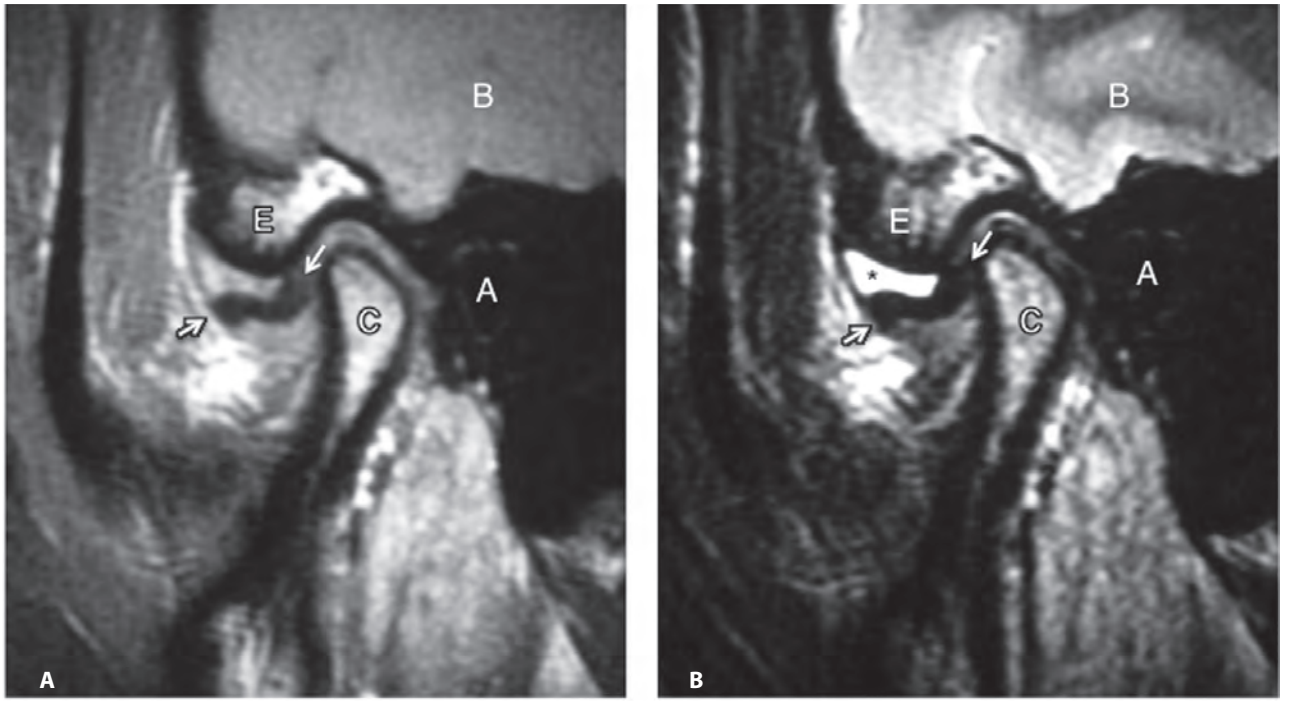


FIGURE 50-3. MRI disc disc placement with joint effusion. **A,** T1 weighted MRI in closed-mouth position showing a displaced articular disc. (arrows) **B,** Same view as a T2 weighted MRI, showing a superior joint space effusion (*). (A auditory canal, B brain, C condyle, E eminence)

for TMJ disc surgery in 1984.[33] The standards were established through a comprehensive literature review and surgeon consensus opinion. Subsequent documents have been updated and further established the indications, goals, and outcome assessment for TMJ surgery. The AAOMS Parameters of Care 2007 (ParCare07) is the latest of such documents.[34]

Indications

Surgical intervention for TMJ disc derangements is indicated only when nonsurgical therapy has been ineffective and pain and/or dysfunction are moderate to severe. Surgery is not indicated for preventive reasons in patients without pain and with satisfactory function.

The AAOMS ParCare 07[34] established the indications for surgical therapy for TMJ disc derangements as a combination of the following factors:

1. Moderate-to-severe pain
   a. Localized TMJ pain
   b. Preauricular pain
   c. Referred pain (e.g. otalgia)
2. Dysfunction that is disabling and characterized by any of the following:
   a. Restricted range of motion
   b. Excessive range of motion
   c. Joint noises
   d. Abnormal masticatory function
3. Imaging evidence of disc derangement
4. Arthroscopic evidence of disc derangement

Goals

The general goals of any surgical intervention are to return the patient to function and/or form. In the case of the surgery of TMJ disc derangements, the goal is to return the patient to a regular diet, with some limitations, and to establish an adequate functional range of motion. However, risk factors and potential complications may preclude the complete restoration of form and function. Each patient complaint must be analyzed individually, and specific outcomes must be determined for each surgical procedure. The goals for TMJ disc derangement surgical procedures should include preservation of articular tissue to permit normalization and regeneration of synovium, and a restoration of the articular relations allowing the joint structures to adapt and function through an adequate range of motion. Joint function may be asymptomatic and satisfactory in the presence of various types of disc derangement. Thus, surgically returning a displaced disc to the ideal position found in a healthy joint may not be appropriate for each individual patient. To illustrate this point, it would not be prudent to reposition a disc in a joint in which the articular tissue is so severely damaged that it is incapable of healing; in this situation, removing the disc is recommended.

Specific therapeutic goals for surgery of TMJ disc displacement established by AAOMS ParCare 07[34] include:

1. Relief or reduction of TMJ pain
2. Limited period of disability
3. Improved range of jaw motion and/or function
4. Elimination or reduction of noise in the joint
5. Appropriate understanding by patient of treatment options and acceptance of treatment plan
6. Appropriate understanding and acceptance by the patient of favorable outcomes and known risk and complications

Outcomes assessment

Folowing any surgical intervention, the surgeon should evaluate the patient response to therapy including whether the patient feels there has been a total eradication, significant reduction, or minimal reduction of the initial complaints, or no change or worsening of the condition. Studies on TMJ surgery contain a wide range of definitions of surgical success criteria, or symptom improvement.[35] It is unreasonable for the surgeon to evaluate the results of an operation on the basis of attainment of a normal mouth opening. Many patients are quite satisfied with a reduction in their mouth opening, as long as their facial pain is relieved. Efforts to standardize clinical outcomes and criteria for success have been reported in the literature.[36]

Favorable therapeutic outcomes established by AAOMS ParCare 07[34] include:

1. A level of pain that is of little or no concern to the patient, preferably measure objectively (e.g. visual analog scale)
2. Improved mandibular function that is compatible with mastication, deglutition, speech, and oral hygiene
3. A stable occlusion
4. Acceptable clinical appearance (e.g., absence of motor deficits, absence of hypertrophic scar formation, etc.)
5. Limited period of disability
6. Patient acceptance of procedure and understanding of outcomes

NONSURGICAL AND MINIMALLY INVASIVE SURGERY

Nonsurgical therapies such as nonsteroidal anti-inflammatory drugs, muscle relaxants, soft diet, moist heat, physical therapy, splint therapy, and behavioral therapy are, in the vast majority of the cases, the first line of nonsurgical treatment in the management of TMJ disc derangements. Surgery should be considered only when the dysfunction or pain could not be corrected to a level of patient satisfaction by nonsurgical modalities. The role of minimally invasive procedures such as TMJ arthrocentesis and arthroscopy in the surgical management of TMJ disc derangements has been well identified in the literature[37]. Both nonsurgical therapies and minimally invasive TMJ surgery are discussed in greater detail in other chapters of this text.

OPEN JOINT SURGERY

According to a meta-analysis on surgical treatment for TMJ disc derangement, open joint surgery treatments appear to provide some benefit to patients refractory to nonsurgical therapies.[37] Multiple open joint surgical procedures have also been described in the literature for the management of TMJ disc derangements. They include *disc repositioning, disc repair, discectomy, and discectomy with disc replacement*. Studies have suggested that success rates with these types of procedures are similar to more conservative procedures such as TMJ arthrocentesis, arthroscopy, and even modified condylotomy.[38–41] Still, some surgeons have advocated open joint procedures as first-line treatment for TMJ disc derangements.[42–44] According to Dimitroulis[1], three main controversies exist in open joint surgery:

1. The role of disc repositioning in light of the results of TMJ arthrocentesis and arthroscopic lysis and lavage.
2. The necessity for disc replacement after discectomy.
3. Use of alloplastic TMJ replacement for end-stage TMJ disease.

Surgical anatomy

Fascial Layers

Anterior to the auricle, the auricularis anterior and superior muscles overlie the superficial temporalis fascia and the temporalis fascia. These muscles are incised in the classic preauricular and endaural approaches. The fascia superficial to the muscles is thin and a dull white color. This layer is confluent with the galea aponeurotica above and the parotideomasseteric fascia below. The temporalis fascia is a tough fibrous connective tissue layer, substantially thicker than the overlying superficial fascia. It is stark white and extends from the superior temporal line of the temporal bone to the zygomatic arch. The deep surface furnishes one of the origins of the temporalis muscle. Inferiorly, at a variable distance, the fascia splits into two well-defined layers (Figure 50-4). The outer layer attaches to the lateral margin of the superior border of the zygomatic arch, and the inner layer to the medial margin. A small quantity of fat, the zygomatico-orbital branch of the temporal artery, and zygomaticotemporal branch of the maxillary nerve are located between the fascial layers in this area. The splitting of the fascial layers is most noticeable at the level of the zygomatic arch. Posteriorly, superior to the glenoid fossa, the separation is not as well defined (Figure 50-5).

Facial Nerve (Main Trunk and Frontal Branch)

Numerous authors have studied the anatomic relations of the facial nerve to determine clinically applicable landmarks for its main trunk, the temporofacial division, and the temporal branches. Al-Kayat and Bramley[45] noted that the facial nerve bifurcated into temporofacial and cervicofacial components within 2.3 cm (range 1.5 - 2.8 cm) inferior to the lowest concavity of the bony external auditory canal and within 3.0 cm

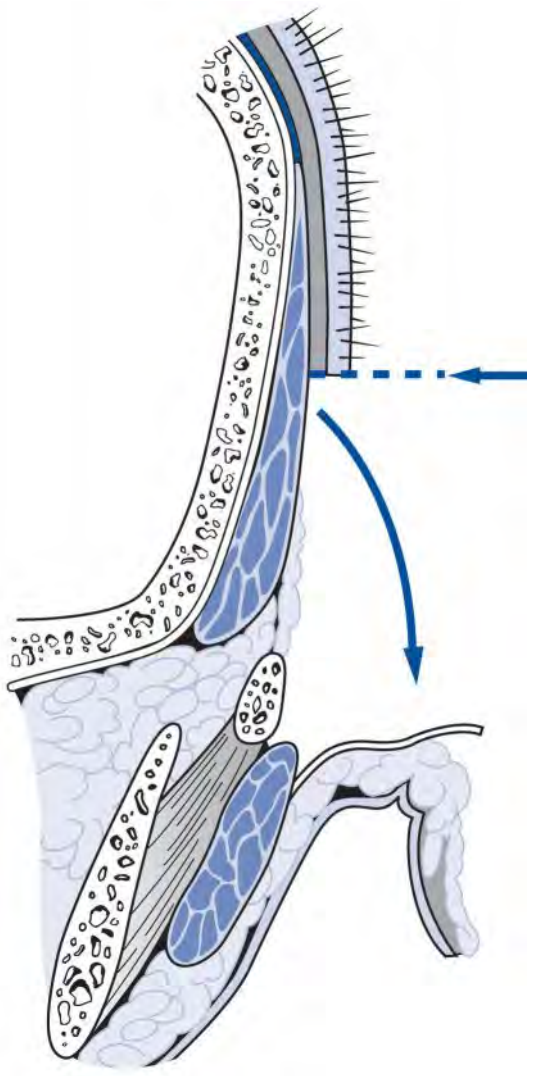


FIGURE 50-4. Coronal section at the level of the zygomatic arch. Two well-defined layers of temporalis fascia are noted (arrows).

(range 2.4 - 3.5 cm) in an inferoposterior direction from the postglenoid tubercle. The temporal nerve branches lie closest to the joint and are the most commonly injured branches during surgery. These nerves are located in a condensation of superficial fascia, temporalis fascia, and periosteum as they cross the zygomatic arch. The most posterior temporal branches lie anteriorly to the postglenoid tubercle. Their location was measured as 2.0 cm (range 0.8 - 3.5 cm) from the anterior margin of the bony external auditory canal (Figure 50-6). Using a high-resolution MRI and the same landmarks as Al-Kayat and Bramley, Miloro and colleagues[46] found that the temporal branch is in a slightly less vulnerable position during a preauricular approach with a mean distance of 2.12 cm (range 1.7 - 2.5cm). Thus, the two potential sources of facial nerve injury are dissection anterior to the posterior glenoid tubercle where the temporal branches cross the arch, and aggressive retraction at the inferior margin of the flap where the main trunk and temporofacial division are located.

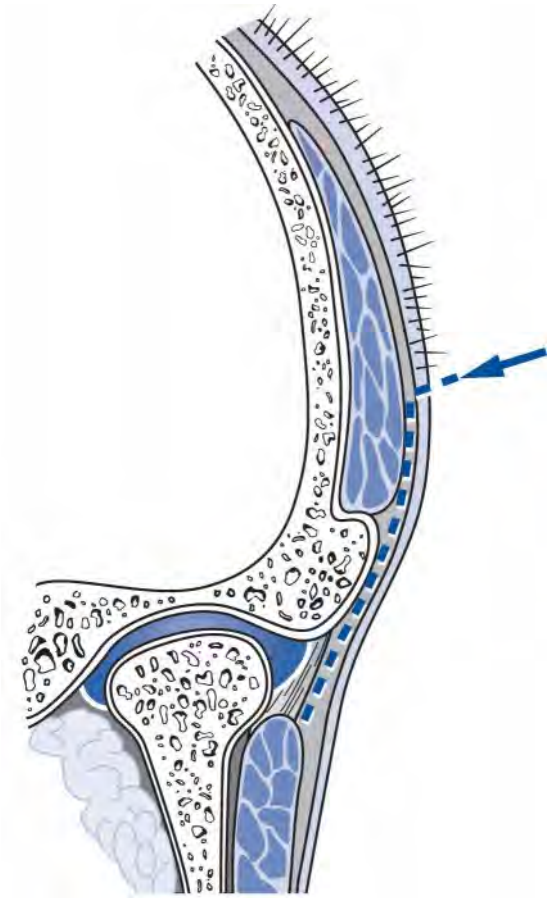


FIGURE 50-5. Coronal section at the level of the glenoid fossa. The splitting of the temporalis fascia is not as well-defined (broken line).

Auriculotemporal Nerve

The auriculotemporal nerve represents the first branch of the third division of the trigeminal nerve (V_3) after exiting the skull through the foramen ovale. It runs medial to lateral behind the neck of the mandibular condyle to give the sensitivity of the skin of the temporal and preauricular region, the external auditory canal and tympanic membrane. The other branch of this nerve descends to innervate the TMJ capsule and it also enters the joint in a posterolateral fashion to innervate the retrodiscal tissues. Damage to this branch is inevitable during a classic open TMJ surgery but rarely gives clinical problems.

Superficial Temporal Vessels

The superficial temporal artery (STA) is one of the terminal branches of the external carotid artery. It normally runs with the superficial temporal vein posterior to the condylar neck approximately 2mm anterior to external auditory canal. Before it crosses the posterior portion of the zygomatic arch, the STA gives rise to the transverse facial artery that runs in an anterior direction. Then it continues all the way to the temporal fossa where it divides into two branches: frontal and parietal. The superficial temporal vessels are typically located in the superficial fascia below the auricularis anterior muscle. The vessels are often visible, invested in the superficial fascia without incising the muscle. The superficial temporal vein lies posterior to the artery and the auriculotemporal nerve immediately behind the vessels.

Internal Maxillary Artery

The internal maxillary artery, or maxillary artery, emerges from the external carotid artery deep and posterior to the

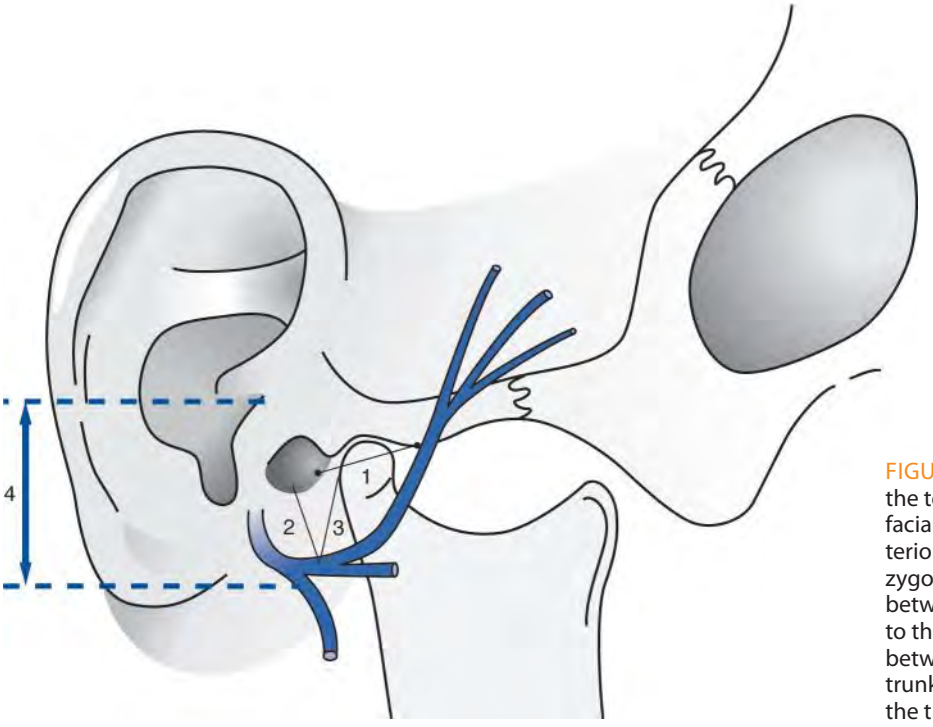


FIGURE 50-6. Landmarks for the location of the temporal branches and main trunk of the facial nerve: (1) the distance between the anterior margin concavity of the meatus to the zygomatic arch (2.0 ± 0.5 cm); (2) the distance between the inferior margin of the meatus to the trunk (2.3 ± 0.28 cm); (3) the distance between the postglenoid tubercle to the main trunk (3.0 ± 0. 31 cm); (4) the distance from the tragus to the facial nerve trunk is variable.

mandible at the level of the sigmoid notch. The internal maxillary artery crosses laterally to the external pterygoid muscle in 50% of the cases and medially to the muscle in the other 50%. Additionally according to Turvey and Fonseca[47] the internal maxillary artery exits the infratemporal fossa 2.5 cm above the pterygomaxillary fissure. Injury to this vessel could create a considerable hemorrhage especially in cases that require osteotomy of the condylar neck.

Surgical approaches

The classical surgical approaches to the TMJ may be classified as preauricular, endaural, and postauricular. The choice of approach is usually a matter of surgeon preference and is based on his or her ability and surgical experience. Cosmetic considerations may also influence the choice of surgical approach.

Preauricular Approach

Historically, a myriad of preauricular incisions have been proposed. Many of the earlier designs afforded good access but increased the risk of facial nerve injury and compromised esthetics. The preauricular incisions used today are essentially modifications of the Blair curvilinear or inverted-L incision.[48] This approach has become the favorite chosen by most oral and maxillofacial surgeons. The technique consists of an incision commencing from within the temporal hairline and extending inferiorly into a preauricular crease immediately anterior to the auricle. The exact length and decision to incorporate an anterior temporal extension are governed largely by the nature of the surgical procedure. For some surgeons, the approach for discectomy requires a smaller incision than that for discoplasty. The incision is approximately 3 to 4 cm in length and consists of two limbs: a small superior curved limb (1–2 cm) and an inferior vertical limb anterior to the tragus (variable distance approximately 1–2 cm) (Figure 50-7). The junction of these limbs is the site of attachment of the superior aspect of the helix to the temporal tissue. The extent of the superior limb of the preauricular incision is dictated by the amount of access required, which may not be determined until the dissection has reached the lateral TMJ ligament and capsule. The incision is usually not extended as inferiorly as the lobule of the ear.

The incision should be placed posteriorly to the superficial temporal vessels and auriculotemporal nerve and within a preauricular crease. The skin and subcutaneous tissues are incised the length of the entire incision. The deeper dissection is begun in the temporal region by sharply dissecting progressively through the auricularis anterior and superficial fascial layers to the stark white temporalis fascia (Figure 50-8). A retractor is placed on the anterior flap, and tension is applied in a forward direction. The dissection over the zygomatic arch is addressed. The anatomic layers in this region are usually not clearly defined. There is a condensation of tissues consisting variably of the auricularis interior, superficial fascia, temporalis fascia, periosteum, and occasionally cartilage.



FIGURE 50-7. The preauricular incision

This tissue is incised to the level of fibrous connective tissue. A retractor is placed in the incision opposite the tragus, and forward traction is applied to the flap. This results in the definition of a cleft between the perichondrium and cartilage



FIGURE 50-8. The preauricular incision has been carried sharply through the skin, subcutaneous tissue, superficial temporal fascia, auricularis anterior and superior, and outer layers of the temporal fascia. The flap is reflected anteroinferiorly, revealing the inner layer of the temporal fascia.

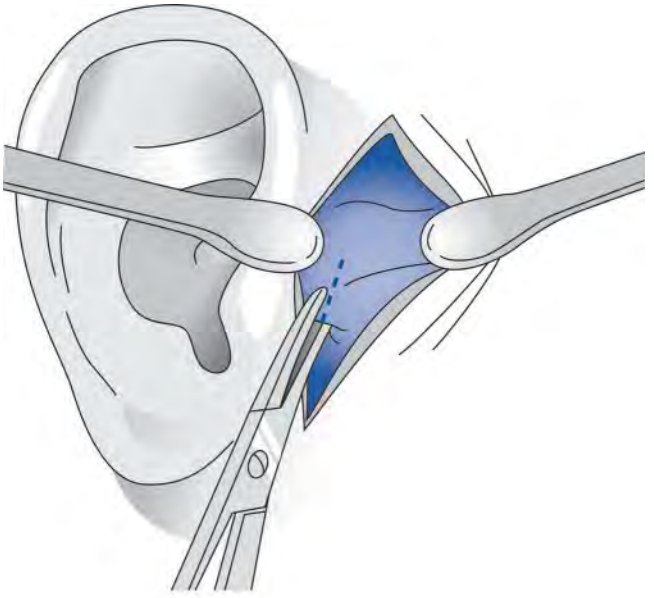


FIGURE 50-9. The parotideomasseteric fascia is sharply dissected from the perichondrium of the external auditory canal (broken line).



FIGURE 50-10. Retraction is accomplished by using a self-retaining retractor positioned between the external auditory canal and flap and a right-angle retractor at the interior portion of the flap. The condyle (dotted line) is noted under the lateral TMJ ligament and/or simply the lateral capsule depending on the depth of the reflection.

of the external auditory canal and the parotideomasseteric fascia. The perichondrium is followed medially with sharp dissection (Figure 50-9). Care should be exercised not to proceed perpendicularly to the skin surface, as the external auditory canal inclines anteromedially at approximately 45° to the surface. The dissection is continued along the outer surface of the external auditory canal until the lateral TMJ ligament is reached.

When the condyle and its overlying temporomandibular ligament are palpated, the flap is reflected inferiorly and anteriorly forward with a combination of sharp and blunt dissections. Scissors may be used to cut some fascial attachments to the lateral TMJ ligament. The blades of the scissors are held parallel to the ligament to ensure that the joint is not violated. The flap is reflected as far forward as the midportion of the anterior tubercle. The surgeon can now see the bulging of the lateral pole of the condyle under cover of the lateral ligament and capsule. Gentle manipulation of the jaw to cause movement of the condyle helps to orient the surgeon to the location of the joint space. The deep surface of the flap and the tissues overlying the zygomatic arch may be touched with a nerve stimulator to ascertain the location of the facial nerve. Retraction is accomplished using a self-retaining retractor (eg, Weitlaner or a Dolwick-Reich) placed between the flap and the perichondrium. A small right-angled retractor may be placed at the inferior portion of the flap (Figure 50-10).

Endaural Approach

Rongetti described a modification of Lempert's endaural approach to the mastoid process for surgical improvement of otosclerosis, for approaching the TMJ.[49,50] The endaural incisions employed today is simply a cosmetic modification of the preauricular approach in which the skin incision is incorporated over the prominence of the tragus itself (Figure 50-11). The access obtained is equal to that achieved through the preauricular approach. Disadvantages include the potential for perichondritis and an esthetic compromise if tragal projection is lost or significant iatrogenic cartilage damage occurs.

Postauricular Approach

In the postauricular approach the incision is made posterior to the ear and involves the sectioning of the external auditory meatus.[51] Excellent posterolateral joint space and condylar exposure is afforded with this technique. The flap, once reflected, contains the entire auricle and superficial lobe of the parotid gland. A perimeatal approach combining the preauricular and postauricular incisions has also been described.[52,53]

Preoperative considerations for this approach were described by Walters and Geist[54] and include:

1. History of normal scar formation
2. Healthy auditory system with no infection
3. Normal width of the external auditory canal
4. No TMJ infection
5. Medically-compromised patient unable to tolerate long operative period

The incision in the postauricular approach begins near the superior aspect of the external pinna and is extended to the tip of the mastoid process. The superior portion may be extended obliquely into the hairline for additional exposure.

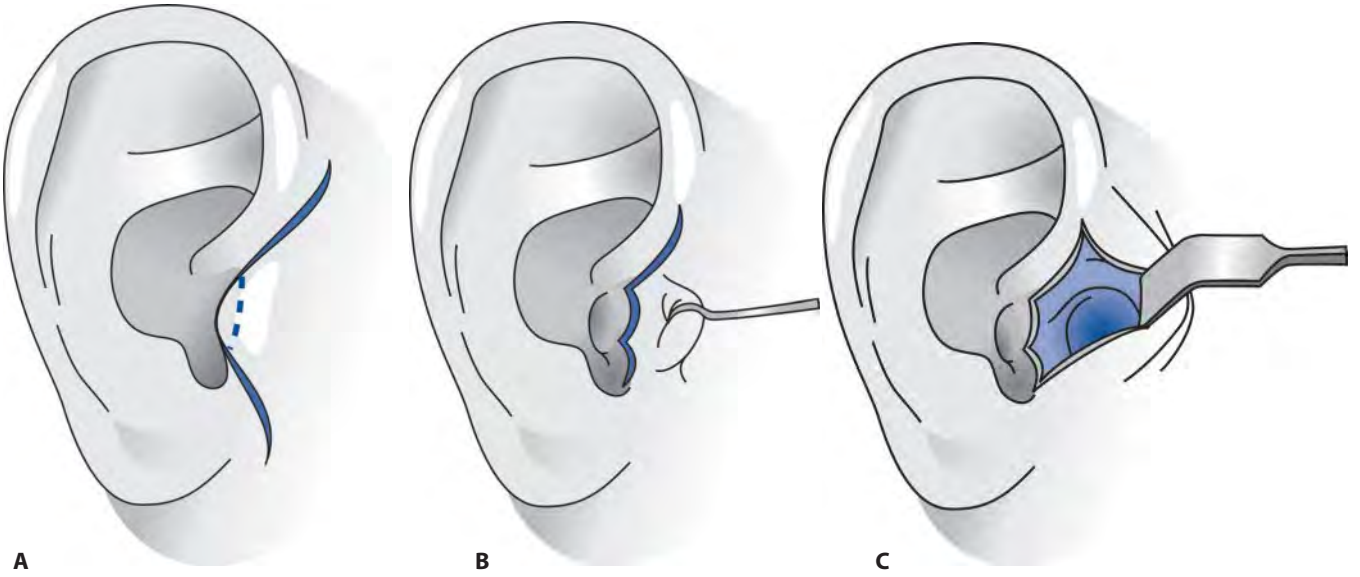


FIGURE 50-11. **A–C,** Endaural approach.

The incision is made 3 to 5 mm parallel and posterior to the postauricular flexure (Figure 50-12). The dissection is performed through the posterior auricular muscle to the level of the mastoid fascia, which is contiguous with the temporalis fascia. A combination of blunt and sharp dissections is used to isolate the cartilaginous portion of the external auditory canal. A blunt instrument is placed in the external auditory canal to assist in the transection of the external auditory canal. The transection may be partial or complete, depending on the need for exposure. The incision should leave 3 to 4 mm of cartilage on the medial aspect to permit adequate reapproximation of the canal (Figure 50-13). This technique helps to prevent meatal, or external auditory canal (EAC), stenosis. The incision is carried through the outer layer of the temporalis fascia, continuing inferiorly, reflecting the parotideomasseteric fascia off the zygomatic arch and lateral TMJ ligament (Figure 50-14). A self-retaining retractor is used to maintain exposure. The advantages of the postauricular approach lie in the predictability of the anatomic exposure as well as the cosmetic superiority and the low risk of facial nerve injury. Dissection to the joint is rapid with minimal bleeding. The approach offers an alternative for a patient who has had previous procedures in this region. This approach



FIGURE 50-12. Postauricular approach. The incision is placed 3 to 5 mm parallel and posterior to the postauricular flexure.

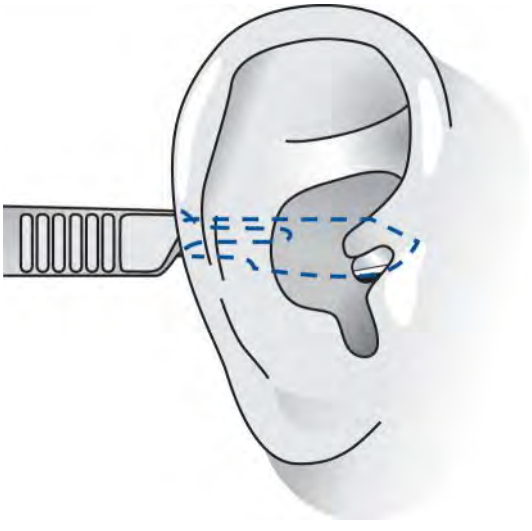


FIGURE 50-13. The external auditory canal is sectioned, leaving 3 to 4 mm of cartilage on the medial aspect to assist reapproximation of the canal.

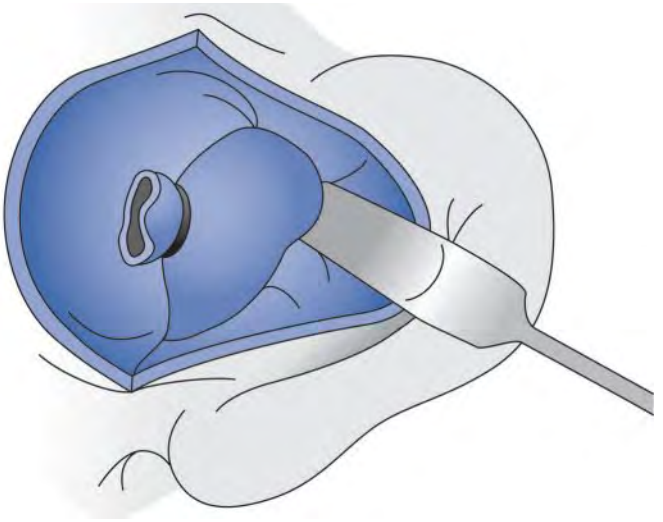


FIGURE 50-14. The external auditory canal has been sectioned and the flap retracted forward.

may not be desirable in the patient susceptible to keloid formation, owing to the potential for a keloid to develop in the meatus. Meatal atresia has been reported with this technique, but can be avoided with the use of moldable ear canal plugs left in place for an extended period of time to prevent EAC scarring and narrowing. The risk of facial nerve injury is reduced, but not eliminated. Paresthesia in the area of the posterior aspect of the auricle usually occurs and may last for 3 to 4 months.

Capsular Incisions

Once the capsule has been identified, access to the articular surfaces (superior and inferior joint spaces) can be obtained by a great variety of incisions. The most commonly used include:

Horizontal incision over the lateral rim of the glenoid fossa

The lateral ligament, capsule, and periosteum are reflected inferiorly en masse. Discal or posterior attachment connections, or both, to the lateral capsule are dissected sharply with scissors to the level of the condylar neck (Figure 50-15A). Insufflation of the superior joint space with local anesthesia can help to identify the entry point. Posterior dissection is performed diligently to avoid severing the retrodiscal tissue. This portion of the dissection exposes the superior joint space (Figure 50-15B). A Freer septum elevator may be used to define and explore the space. The posterior attachment and disc attachments are then severed sharply at the lateral pole of the condyle from within the developed flap. The Freer septum elevator is used to reflect the posterior attachment and disc superiorly off the head of the condyle to expose the inferior joint space. A periosteal elevator may be used to stretch the capsule and lateral ligament flap outward to form a pocket (Figure 50-15C).

There is a risk of reflecting the fibrous connective tissue that lines the glenoid fossa when this approach is used (Figure 50-16A). The surgeon may form the incorrect assumption that he or she is stripping adhesions from the temporal bone while defining the space. The result may be a partial or total synovectomy of the superior joint space. Prearthrotomy arthroscopic examinations have alerted clinicians to this error. The ability of the pathologic joint to regenerate this synovium and fibrous connective tissue layer has not been determined.

Horizontal incision below the lateral rim of the glenoid fossa

A no. 11 blade may be used to puncture into the superior joint

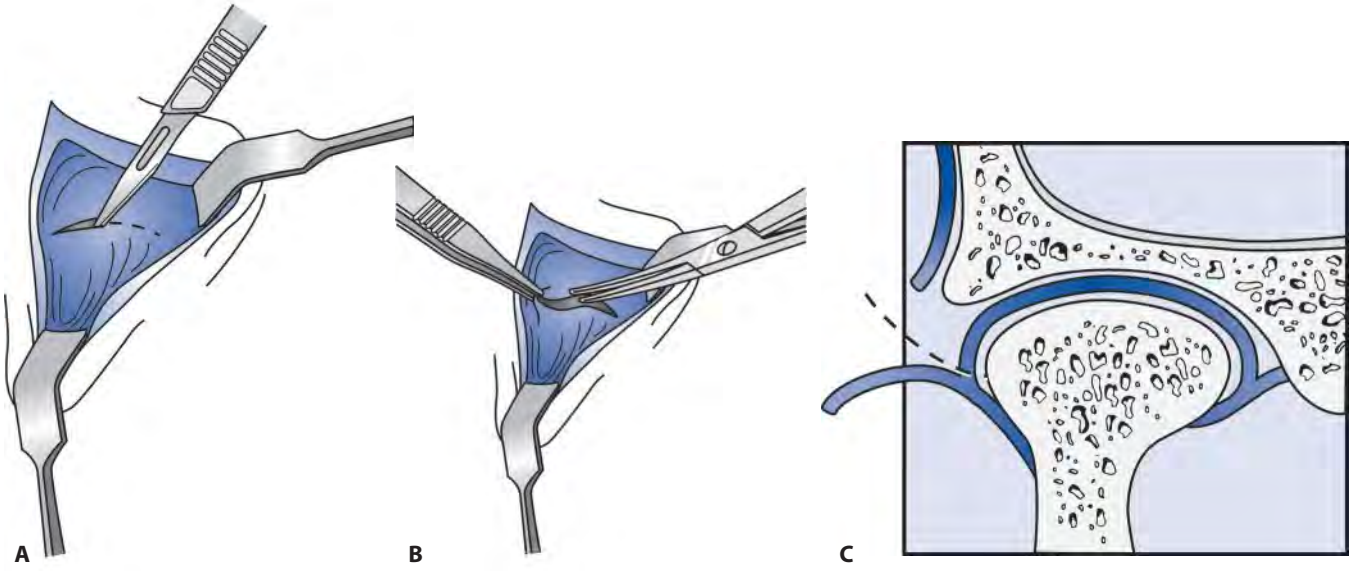


FIGURE 50-15. **A,** Entry into the superior joint space following its distention with fluid. A no. 11 blade incises the lateral capsule and ligament *(broken line)*. Care is maintained not to prolong the incision posteriorly to avoid injuring the retrodiscal tissues. **B,** The incision is prolonged posteriorly using baby metzenbaum scissors. **C,** The horizontal capsular and ligament flap is developed inferiorly and the discal insertions sharply dissected (*broken line*). The inferior joint space is defined by incising along the superolateral aspect of the condyle. A Freer septum elevator is used to define the joint spaces.

space at the level of the lateral discocapsular sulcus (Figure 50-16B). The opening is then lengthened anteriorly and posteriorly using sharp-pointed scissors. A dissection technique, similar to that described in the foregoing approach, is used to define the superior joint space. A dissection is then carried inferiorly removing the attachment of the capsule to the disc and exposing the inferior joint space. There is less risk of injury to the retrodiscal tissue with this approach; the risk to the fibrocartilage is also reduced.

Horizontal incisions above and below the disc

The horizontal approach above and below the disc (Figure 50-16C) leaves some of the capsule and ligament attached to the disc or remodeled retrodiscal tissue.

T-Shaped incision

A horizontal incision may be joined by a vertical incision that extents over the capsule insertion over the lateral condyle to create a T-shaped incision over the midportion of the glenoid fossa (Figure 50-16D).

Wound closure

Closure of the capsule is often difficult to obtain following open surgical joint procedures. Support for the lateral ligament can be obtained by raising a temporalis muscle and fascia flap, about 2 cm in length, pedicled inferiorly, and rotated inferiorly over the lateral rim of the glenoid fossa and sutured to the lateral capsular tissue. The pedicle stabilizes the flap but has not been shown to contain nutrient vessels. Closure of the capsule and ligament after disc repositioning lends stability to the discorrhaphy but may not critical to the success of the discectomy.

Surgical Procedures

Disc repositioning

The goal of disc-repositioning procedures is to relocate the disc to the normal condyle-disc-fossa relationship. The repositioned disc facilitates movement of the condyle previously blocked by the displaced disc, provides joint stabilization, and improves articular cartilage nutrition and lubrication. Moreover, the workload of the masticatory muscles is reduced when the obstructing disc is repositioned. The literature appears to support the successful application of disc repositioning surgery in 80-95% of cases[55,56] (Table 50-1). However the role of disc repositioning surgery has significantly diminished with the success of less invasive procedures, as well as the reported

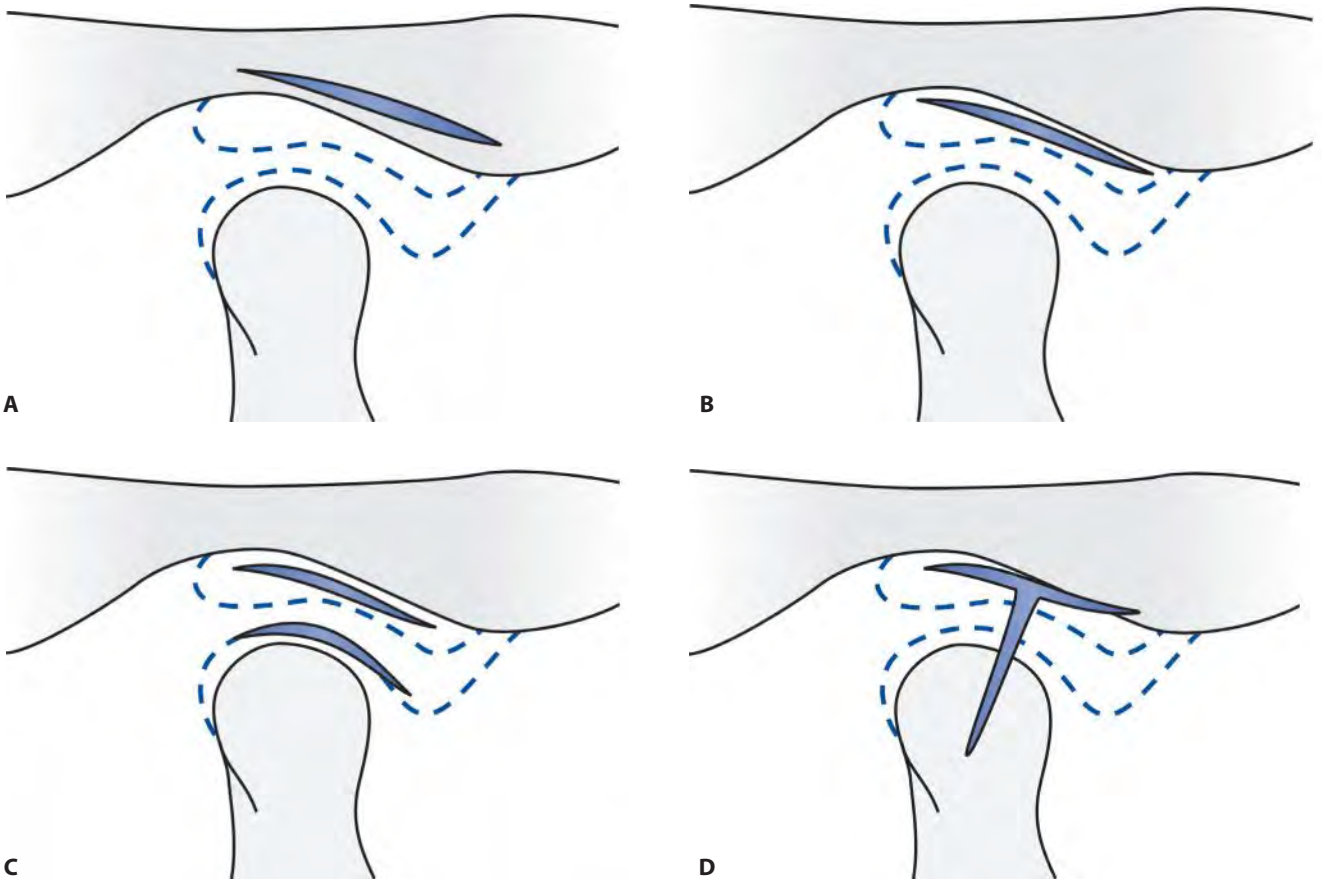


FIGURE 50-16. Capsular incision designs: **A,** horizontal incision over the lateral rim of the glenoid fossa; **B,** horizontal incision below the lateral rim of the glenoid fossa; **C,** horizontal incisions above and below the disc; **D,** T-shaped incision. The lateral pole of the condyle and lateral aspect of the remodeled posterior attachment (*broken lines*) are illustrated.

TABLE 50-1. **Selective Studies In TMJ Disc Repositioning Surgery**

Author	Year	Type of study	Procedure	Patients/Joints	Follow-up	Results
Diskoplasty						
Dolwick[1]	1983	Retrospective Case Series	Diskoplasty	50 pts /54jts	12-60m	94% success
Dolwick et al[2]	1990	Retrospective Case Series	Diskoplasty	152pts/155jts	6m-8yrs	70-80% improvement
Anderson et al[3]	1991	Retrospective Case Series	Diskoplasty	33pts/39jts	8-35m	77% pain free
Montgomery et al[4]	1992	Retrospective Case Series	Diskoplasty	51pts/74jts	6m-6yrs	65% pain-free 78% improved jaw motion
Zeitler et al[5]	1993	Retrospective Comparative Study	Diskoplasty	24pts	25m	83%
Kuwahara et al[6]	1994	Retrospective Comparative Study	Diskoplasty	90pts	12m	92% improvement
Abramowicz et al[7]	2010	Retrospective Case Series	Diskoplasty	18pts/36jts	20yrs	94% improvement quality of life
Diskoplasty & eminectomy						
Hall[8]	1984	Retrospective Case Series	Diskoplasty & eminectomy	20jts	18m	65% pain –free
Weinberg[9]	1984	Retrospective Case Series	Diskoplasty & eminectomy	33pt/40jts	44m	89% better
Dolwick et al[10]	1985	Retrospective Case Series	Diskoplasty & eminectomy	68pts/78jts	18-60m	55% excellent 35% good
Kertens et al[11]	1989	Retrospective Case Series	Diskoplasty & eminectomy	25pts	14-29m	87% felt better
Trumpy et al[12]	1995	Retrospective comparative study	Diskoplasty & eminectomy	13pts	74-91m	77% improvement
Baldwin et al[13]	2004	Retrospective Case Series	Diskoplasty & eminectomy	92pts/119jts	44m	49% asymptomatic 19% improved
Meniscocondylar plication						
Weinberg et al[14]	1987	Retrospective Case Series	Meniscocondylar plication	84pt/89jts	6-24m	91% improvement
Vazquez-Delgado et al[15]	2004	Retrospective Case Series	Meniscocondylar plication	20pts/29jts	51.2m	75% success
Diskoplasty & high condylotomy						
Benson et al[16]	1985	Retrospective comparative study	Diskoplasty & high condylotomy	60pts	6-48m	88% improved
Walker et al[17]	1987	Retrospective Case Series	Diskoplasty & high condylotomy	50pts/65jts	4-24monts	100% success
Griffits et al[18]	2007	Retrospective Case Series	Diskoplasty & high condylotomy	117pts/154jts	1-16yrs	86% success
Others						
McCarty et al.[19]	1979	Retrospective Case Series	Diskoplasty & arthroplasty	327jts	6yrs	94% success
Mercuri et al.[20]	1982	Retrospective Case Series	Diskoplasty, arthroplasty & eminectomy	13pts	45m	87% success
Wolford[21]	1997	Retrospective Case Series	Disk reposition & mini anchor	43pts/78jts	2yrs	91% success
Kondoh et al[22]	2003	Retrospective Case Series	Disk reshaping	11pts/11jts	5yrs	91% sucess

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21. Wolford LM. Temporomandibular joint devices: treatment factors and outcomes. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1997;83:143–9.
22. Kondoh T, Hamada Y, Kamei K, Seto K. Simple disc reshaping surgery for internal derangement of the temporomandibular joint: 5-year follow-up results. J Oral Maxillofac Surg. 2003;6:41–8

success of discectomy without replacement.

There are two main indications for disc-repositioning procedures:

1. Patients with painful anterior disc displacement with reduction that has not responded to nonsurgical and minimally invasive procedures
2. Patients with anterior disc displacement without reduction with persistent pain and limited mouth opening that has not responded to nonsurgical and minimally invasive procedures

Absolute contraindications of the procedure include: infection in the surgical site and uncontrolled psychogenic disease. Relative contraindications involve medically compromised patients, lack of signs and symptoms of intra-articular disease, psychiatric disorders, and the possibility of solving the problem using minimally invasive techniques.[57]

Disc repositioning can be accomplished by either of the following techniques: a partial-thickness excision in which the superior lamina of the retrodiscal tissue and posterior attachment are removed, without violation of the inferior joint space, and the lateroposterior tissues are reapproximated[58] (Figure 50-17); or full-thickness excision in which a wedge-shaped portion of the posterior attachment is removed and the lateroposterior tissues are approximated[59] (Figure 50-18); or full-thickness excision of a wedge-shaped portion of the posterior attachment with stabilization of the disc with mini-anchors[60].

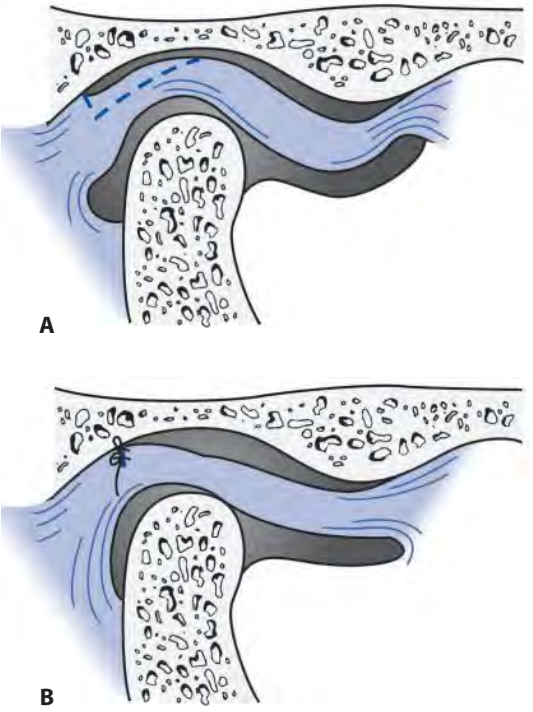


FIGURE 50-17. Disc repositioning achieved through a partial-thickness excision of the superior lamina of the retrodiscal tissue. The inferior joint space is not violated. **A,** Outline of a partial-thickness excision of the superior lamina. **B,** Excision is closed, resulting in posterior repositioning of the disc.

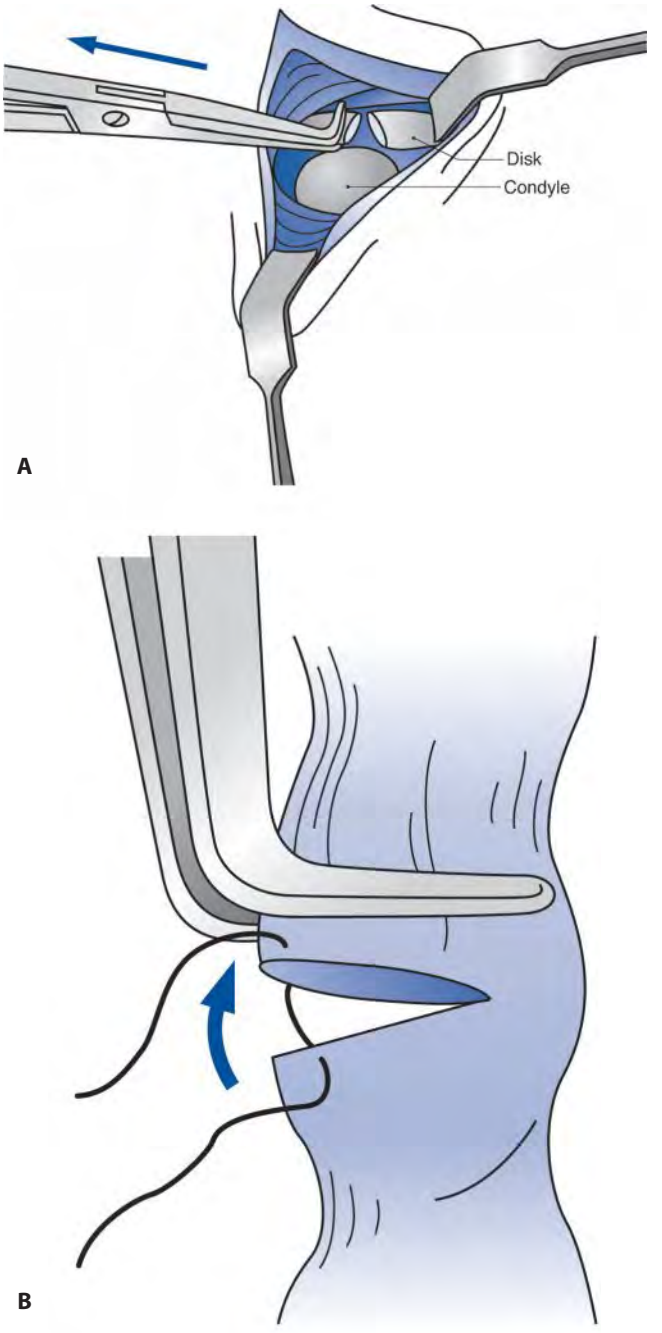


FIGURE 50-18. Disc repositioning achieved through a full-thickness excision of the posterior attachment. Retention of disc position is through sutures placed on posterior and lateral margins. **A,** A clamp has been placed over the posterior attachment. The arrow represents the direction of pull of the clamp to complete the incision and reveal the condylar surface. **B,** View from above demonstrating the wedge-shaped resection (arrow indicates the direction of closure).

The chances of a successful disc repositioning procedure increase if the disc has: 1) minimal displacement, 2) near-normal length, 3) near-normal anatomic structure. When the disc displaces, the pathologic changes are not seen uniformly throughout the entire lateromedial extent of the joint. It is believed that a variable decrease in the vascularity of

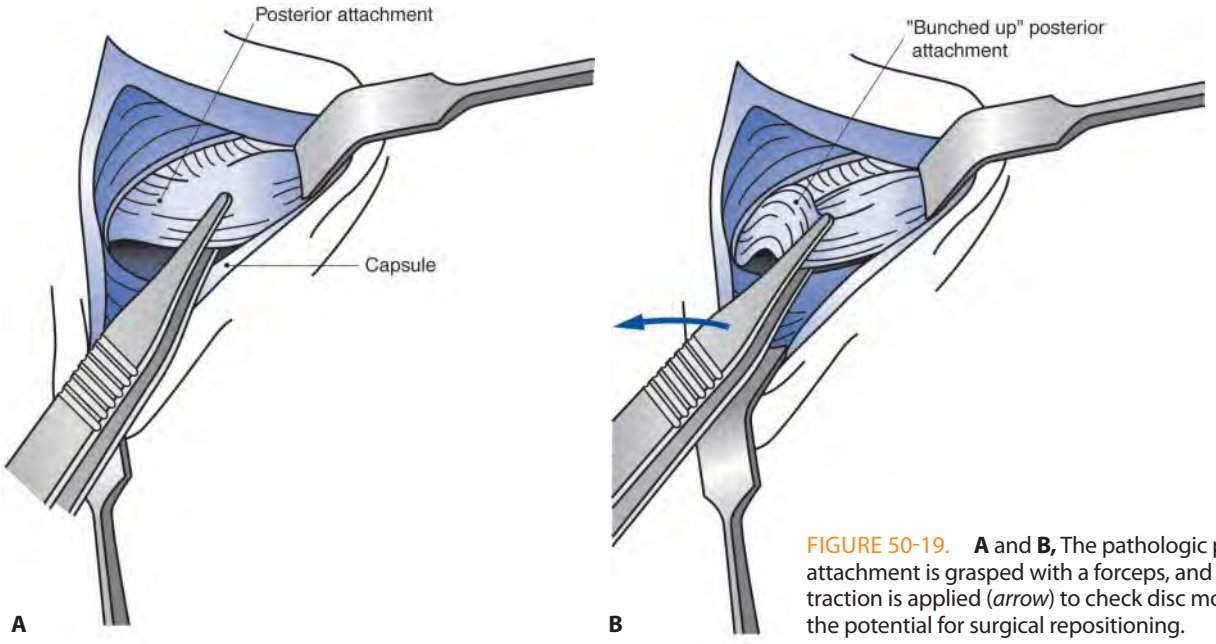


FIGURE 50-19. **A** and **B,** The pathologic posterior attachment is grasped with a forceps, and posterior traction is applied (*arrow*) to check disc mobility and the potential for surgical repositioning.

the remodeled posterior attachment occurs with an increasing duration of disc displacement and load. The disc-repositioning techniques thus involve a repair in the pathologic retrodiscal tissue with a variable degree of vascularity. The primary source of nourishment to the repositioned disc appears to be through the synovium on the medial aspect of the disc and posterior recesses of the joint spaces. Thus, a critical aspect of the successful surgical repair in the retrodiscal tissue appears to be the rapid migration of synoviocytes to the area of the surgical repair. In general, the limiting factor to disc repositioning is the degree of lateral disc atrophy or resorption. Despite severe lateral atrophy, the most medial aspect of the disc may have a normal length and shape.

Disc repositioning and discoplasty

Deformation of the disc in all planes is an important feature to recognize when planning a repositioning procedure. When a bulge-shaped disc is of appropriate length and can be repositioned, a discoplasty may be performed to minimize the change in the occlusion. In addition, removal of the posterior attachment overlying the condyle is intended to remove a source of localized inflammation.

The surgical technique for disc repositioning with discoplasty includes the exposure of the articular surfaces with any of the previously discussed surgical approaches. Once the capsule has been incised, joint distention for better visualization can be achieved by manual manipulation or placement of Wilkes retractor. A Freer elevator can be used to sweep gently across the top of the disc. Access to the medial aspect of the joint is greatly improved when the anterior attachment is released, permitting the surgeon to draw the disc outward posterolaterally while it remains pedicled to the medial attachment. The disc is then inspected for deformities, perforations. Disc mobility is evaluated by applying traction with a forceps (Figure 50-19). Typically, the medially displaced disc must be rotated posterolaterally to achieve a correct condyle-disc-fossa relation; therefore, a greater amount of tissue is plicated or excised laterally rather than medially. A DeBakey bulldog vascular clamp is inserted to the medial limit of the posterior attachment and guided posteriorly as far as possible in the glenoid fossa (Figure 50-20). The clamp greatly assists in the control of hemorrhage from the retrodiscal tissue, stabilization of the posterior attachment during tissue excision, and stabilization of the posterior attachment during suturing.[61] The design of the instrument minimizes tissue damage. A wedge of remodeled posterior attachment is excised, leaving a 1 mm margin anterior to the beaks of the clamp. This permits suturing of the disc to the retrodiscal tissue without removal of the clamp. Range of

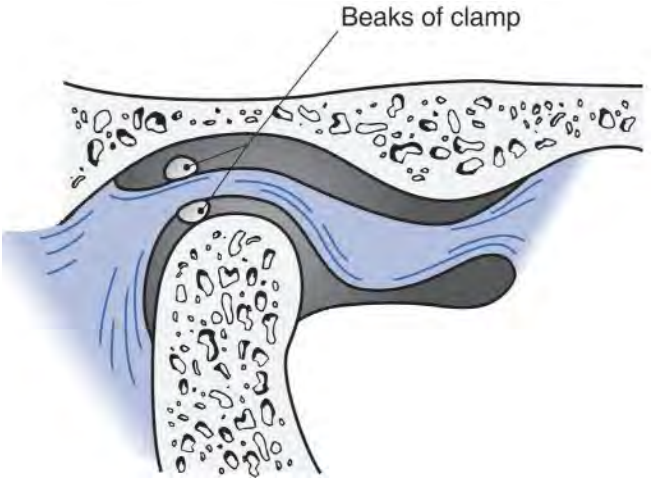


FIGURE 50-20. Positioning of the beaks of the DeBakey clamp on retrodiscal tissue

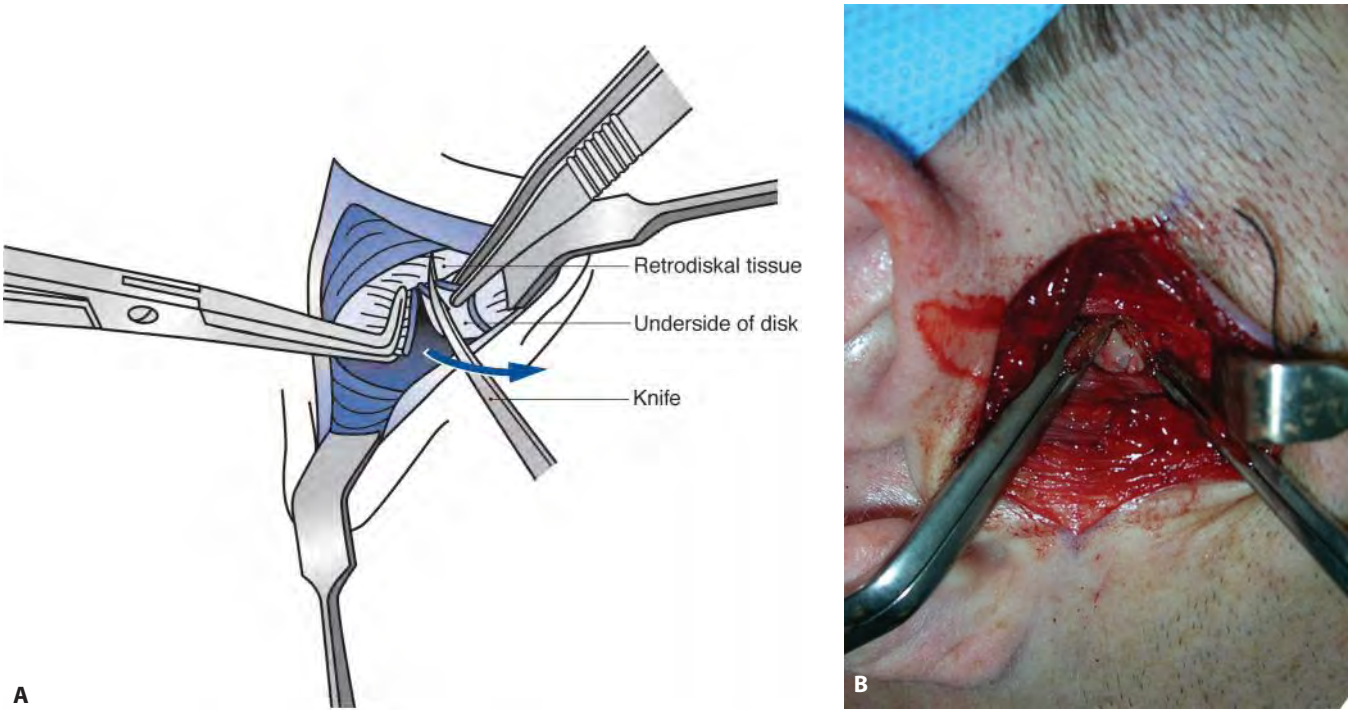


FIGURE 50-21. **A,** Discoplasty is performed following wedge resection of the pathologic posterior attachment. The disc is slightly evened, and an arthroscopic orthopedic knife is used to sculpt the inferior surface of the bulge-shaped disc. **B,** The DeBakey vascular clamp is in place. Note the protruding edge of the posterior attachment used for reapproximation to the disc and lateral capsule.

motion is then verified. Tissue forceps are used to stabilize and slightly evert the disc so that the inferior surface may be sculpted (Figure 50-21). The tissue is closed with nonresorbable suture (Figure 50-22). Once the disc has been sutured into its new position, its lateral rim is sutured to the lateral capsule ligament.

In a study by Montgomery and colleagues, postoperative MRIs demonstrated an unchanged disc position in 86% of patients 2 years after TMJ disc repositioning surgery.[62] Some authors have advocated the use of mini-anchors (Mitek) for better stabilization of the disc in a more physiologic position with success rates reported between 82-91%.[63–65] Long-term studies are required to further determine the stability of the technique as well as the best moment to be applied. The surgical procedure for placement of mini-anchors establishes that once the disc is properly reduced, the mini-anchor, which is a metal insert with a suture attached to it, is placed in the posterolateral portion of condylar head. The suture is then used to secure the lateral and posterior of the disc to the condylar head.

Disc repositioning and arthroplasty

Several authors have advocated combining a disc repositioning procedure with an arthroplasty of the condyle or the articular eminence.[66–70] Arthroplasty reduces the amount of posterolateral repositioning required and therefore permits an easier repositioning of an atrophic disc (Figure 50-23).

A 2 to 4 mm condylar/eminence arthroplasty procedure can be performed with rotary or hand instruments. Hand instruments such as fine chisels are preferable to avoid heat generation (Figure 50-24). Bone files should be used judiciously because, once the compact bony layer is interrupted, the trabeculae of bone can be easily and rapidly removed. A periosteal elevator may be used to burnish sharp edges. Care should be exercised not to exaggerate the arthroplasty in the lateral condylar regions while accessing the medial condylar region. In some cases an arthroplasty of the eminence is essentially a lateral tuberculectomy for access and decompression of the anterior recess of the superior joint space (Figure 50-25). Disc repositioning is then performed through the plication or excision technique. The capsule is closed in the customary fashion. Intermaxillary fixation or training elastics are used for 1 to 3 weeks to allow muscular adaptation and dental compensations to occur. The current trend, however, is to avoid removal of any normal articular bone since the postoperative healing phase already involves some loss of bone substance, which may be additive and result in occlusal disturbances. In addition, postoperative bleeding from cut bone surfaces into the joint can result in fibrous adhesions of the disc or fibrous/bony ankylosis of the joint.

Disc Repair

Perforations rarely occur within the disc proper but rather within the lateral third of the remodeled posterior attachment.[71]. When the disc is perforated, it may be secondary to a developmental rather than a pathologic process. Small perforations (1-3mm) can be treated with primary closure. The atrophic displaced disc is repositioned posteriorly to only a

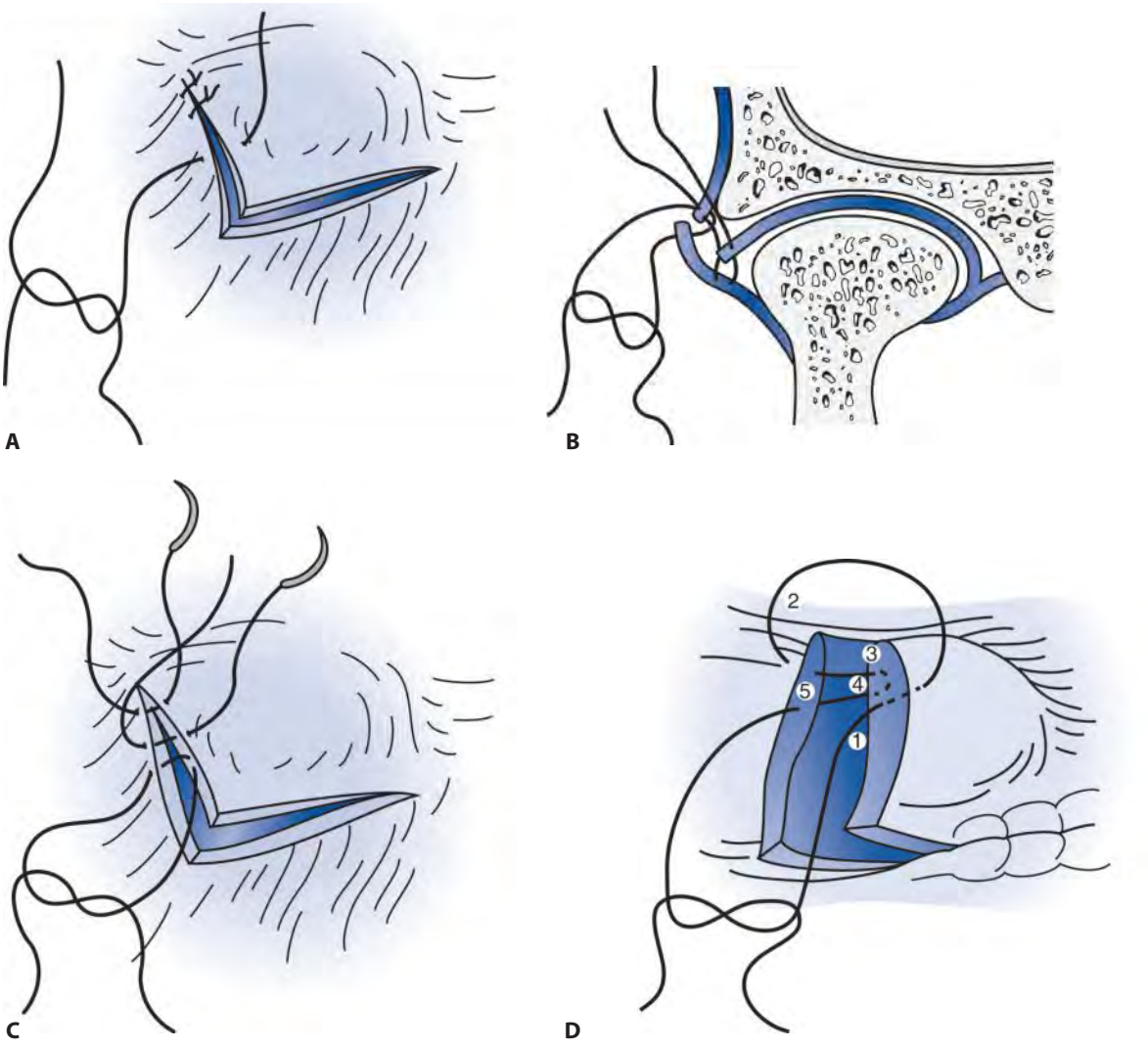


FIGURE 50-22. Disc reapproximation: **A,** simple posterior and lateral sutures; B, layered closure of the superior and inferior lamina; **C,** figure-of-8 closure; **D,** the order of passage of the figure-of-8 suture labeled 1 to 5.

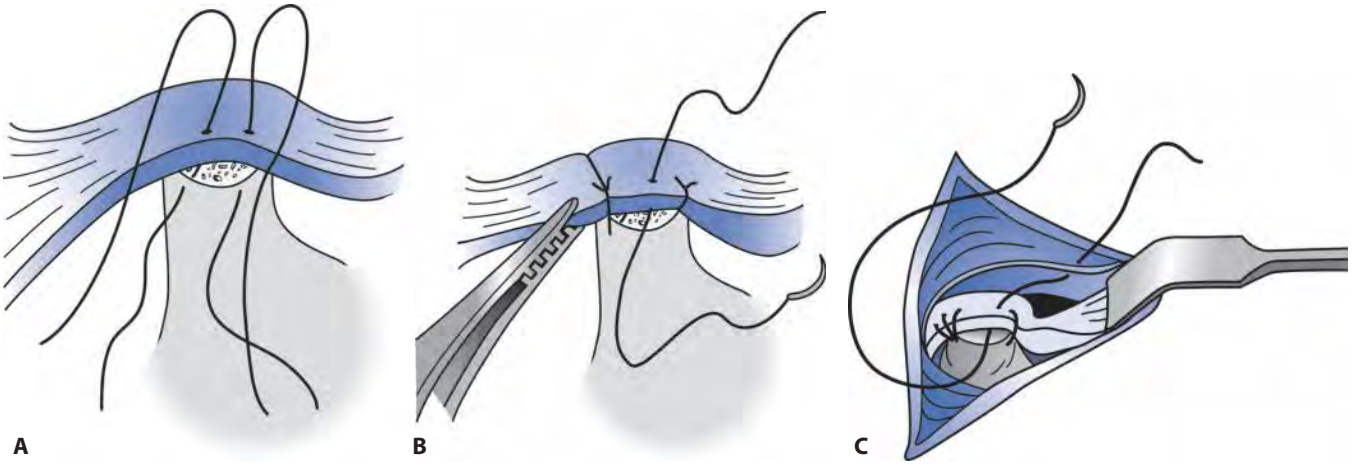


FIGURE 50-23. **A–C,** Disc repositioning with arthroplasty according to Walker and Kalamchi. The disc is sutured to the condyle stump. Adapted from Walker RV and Kalamchi S[67]

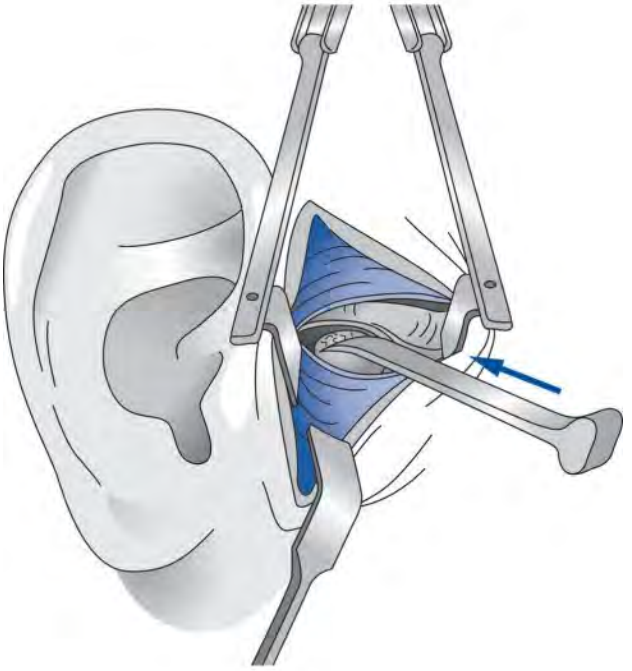


FIGURE 50-24. Condylar arthroplasty using an osteotome. An osteophyte has already been excised. The direction of the osteotome (*arrow*) is indicated in order to skim the condylar surface. Self-retaining and right-angle retractors are in place.

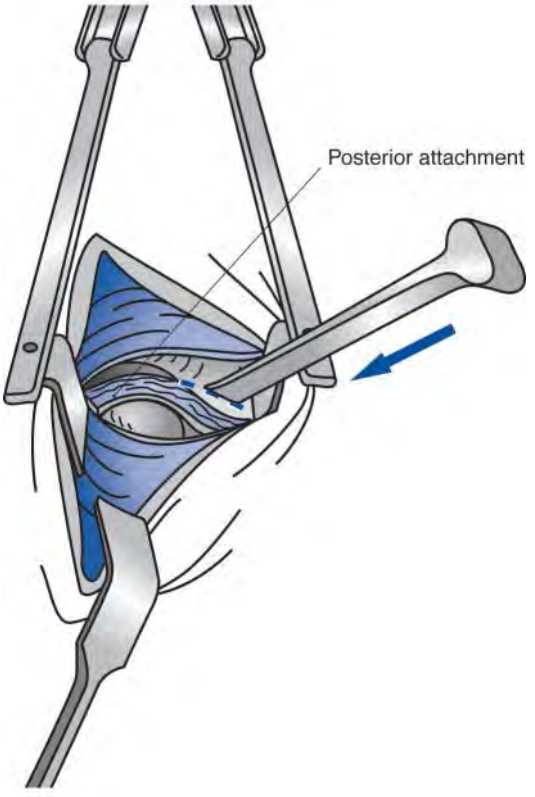


FIGURE 50-25. Lateral tuberculectomy may be performed to acquire access to the anterior glenoid and eminence regions (*broken line* indicates bone to be excised and *arrow* indicates direction of osteotome).

minor degree. If the disc is to be fully repositioned, the margins of the perforation should be excised and the posterior attachment on the posterior edge of the disc approximated to the tympanic portion of the retrodiscal tissue. Anterolateral release of the discal attachments is usually necessary to mobilize the disc posteriorly. The margins of the perforation are oversewn in a straight-line fashion with a nonresorbable material. The repaired retrodiscal tissue is intended to maintain the shape of the articular surface and to prevent ankylosis. Repair of a perforation without repositioning the disc is successful only if the disc is atrophied and is not an obstruction to condylar movement. This procedure is performed rarely and only in those patients refractory to nonsurgical management, arthrocentesis, or arthroscopy. Condylar overgrowth often occurs in the areas of the perforations; therefore, an arthroplasty is frequently performed in conjunction with the procedure. Large perforations will require more extensive repair because the disc usually cannot be repositioned. If the disc cannot be adequately repaired a decision must be made to replace the disc or performed a discectomy (Figures 50-26, 50-27).

Discectomy

Ideally all surgical efforts should be made to retain the articular disc, but this is not always possible. Discectomy should be considered in cases in which the disc is determined to be unsalvageable due to deformation, perforation, calcification and/or severe displacement. Partial and total discectomies have been described in the literature.[72,73] The goal of surgery is to assist the patient to adapt to the pathology at hand by removing the physical impediment to movement. With discectomy, the surgeon transforms a joint into what more appropriately would be described as two bones in close apposition. Adult cartilage derives its nutrients solely from synovial fluid. The prolonged contact of bony surfaces following discectomy may interfere with diffusion of nutrients from the synovial fluid. The decreased diffusion of nutrients

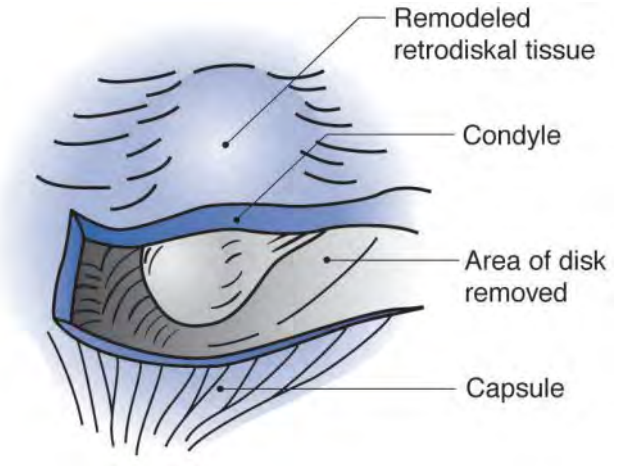


FIGURE 50-26. For repair of a large perforation, a partial discectomy is performed first. A portion of the disc and the retrodiscal tissue may be retained.

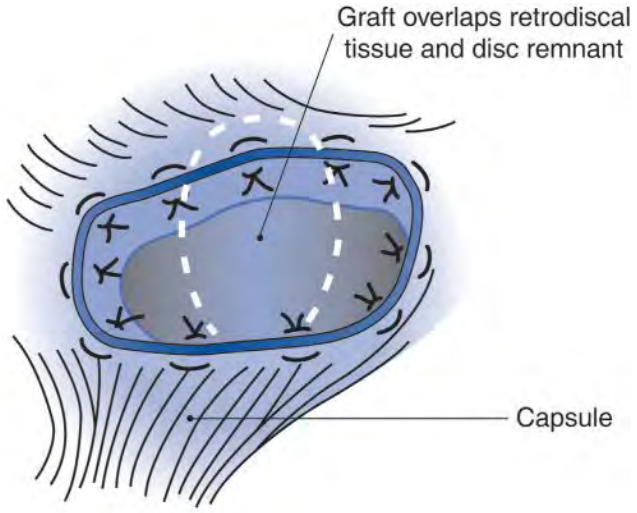


FIGURE 50-27. A dermal graft covers the surgically created perforation. The edges of the graft overlay the disc, retrodiscal tissue, and lateral capsule to assist in suturing.

to cartilage may result in the eventual resorption of noncalcified cartilage. As a result, several adaptive changes rapidly occur. Agerberg and Lundberg[74] described radiographical erosion of the articular surfaces of the operated and nonoperated joints. They also concluded that the remodeling process is due to altered joint loading after discectomy but stabilization occurred after 2 years. They used the term *remodeling* and not osteoarthrosis to describe the radiographic changes because the osseous changes occurred in the absence of symptoms. The bony changes appear similar to those that are observed longitudinally with chronic disc displacement, suggestive of the same mechanism. The rate of remodeling, however, is accelerated in the post-discectomy state.

After a discectomy some masticatory muscle and joint tenderness can be expected for a variable period, extending from several weeks to months. The patient at first favors mastication on the operated side. Later, when healing is advanced, mastication is performed on the nonoperated side. An opening deviation of as much as 8 to 9 mm may occur toward the operated side. Hypermobility of the nonoperated joint may develop or increase after discectomy. Limitation of mandibular movement on the operated side appears to be responsible for the hypermobility. Physical therapy greatly assists the control of the ipsilateral deviation and hence contralateral hypermobility. Patients often report an alteration in their bite, although rarely as a major complaint. The thicker the retrodiscal tissue removed, the greater is the anticipated change in occlusion. The sensation of an altered bite usually resolves within a week to several months and occlusal equilibration is rarely indicated.

There is considerable variation in the ability of each patient and joint to adapt to the postdiscectomy state. Individual factors, such as inclination of the eminence, state of preoperative symptoms, loss of molar support, and amount of postoperative remodeling, seem to play a substantial role.

The mechanism of pain relief and improvement in function over the long-term following discectomy is still unknown. However long-term follow up studies on TMJ discectomies have shown complete resolution in pain and dysfunction in almost all the patients reviewed[75–79]. (Table 50-2)

TABLE 50-2. **Selective Studies in TMJ Discectomy**

Author	Year	Type of study	Patients/Joints	Follow-up	Results
Brown et al[1]	1980	Retrospective Case Series	214pts	15yrs	85% improvement
Takaku et al[2]	1994	Retrospective Case Series	39pts	20yrs	95% improvement
Kuwahara et al[3]	1994	Retrospective Comparative Study	74pts	1yr	95% improvement
Trumpy et al[4]	1995	Retrospective Comparative Study	17pts	6–7.5yrs	94% improvement
Windmark et al[5]	1997	Retrospective Case Series	16ts	2yrs	88% improvement
Holmlund et al[6]	2001	Prospective Randomized Comparative Study	72pts	1yr	83% improvement
Eriksson et al[7]	2001	Retrospective Case Series	64pts	5yrs	85% good results
Bjørnland et al[8]	2003	Retrospective Case Series	24pts	10yrs	79% no pain
Nyberg et al[9]	2004	Retrospective Case Series	15pts	5yrs	87% successful
Miloro et al[10]	2010	Retrospective Case Series	24pts	2–60m	83% sucessful

1. Brown WA. Internal derangement of the temporomandibular joint: review of 214 patients following meniscectomy. Can J Surg 1980;23:30–2.
2. Takaku S, Toyoda T. Long term evaluation of discectomy of the temporomandibular joint. J Oral Maxillofac Surg 1994; 52:722–726.
3. Kuwahara T, Bessette RW, Maruyama T. A retrospective study on the clinical results of temporomandibular joint surgery. Cranio 1994; 12:179–83
4. Trumpy IG, Lyberg T. Surgical treatment of internal derangement of the temporomandibular joint: long-term evaluation of three techniques. J Oral Maxillofac Surg. 1995;53:740–6.
5. Widmark G, Dahlström L, Kahnberg KE, Lindvall AM. Diskectomy in temporomandibular joints with internal derangement: a follow-up study. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1997;83(3):314–320.
6. Holmlund AB, Axelsson S, Gynther GW. A comparison of discectomy and arthroscopic lysis and lavage for the treatment of chronic closed lock of the temporomandibular joint: a randomized outcome study. J Oral Maxillofac Surg 2001;59:972–7.
7. Eriksson L, Westesson PL. Discectomy as an effective treatment for painful temporomandibular joint internal derangement: a 5-year clinical and radiographic follow-up. J Oral Maxillofac Surg. 2001;59:750–8.
8. Bjørnland T, Larheim TA. Discectomy of the temporomandibular joint: 3-year follow-up as a predictor of the 10-year outcome. J Oral Maxillofac Surg. 2003;61:55–60.
9. Nyberg J, Adell R, Svensson B. Temporomandibular joint discectomy for treatment of unilateral internal derangements—a 5 year follow-up evaluation. Int J Oral Maxillofac Surg. 2004;33:8–12.
10. Miloro M, Henriksen B. Discectomy as the primary surgical option for internal derangement of the temporomandibular joint. J Oral Maxillofac Surg 2010;68:782–9.

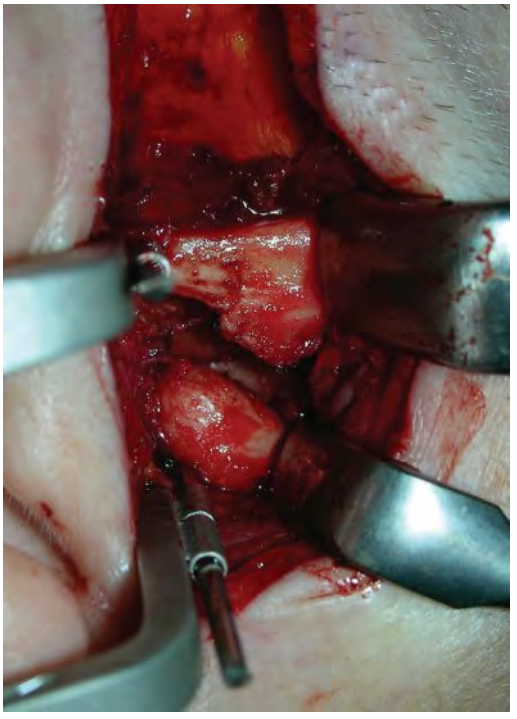


FIGURE 50-28. The Wilkes retractor allows an increased area of maneuverability within the joint space. As it retracts the condyle moves in an anterior and inferior direction. Note the better visualization of medial remanents of the disc.

During discectomy without replacement, once the capsule has been incised, joint distention for better visualization can be achieved by manual manipulation or placement of Wilkes retractor (Figure 50-28). Disc extirpation is facilitated when the disc is severed from its anterior and lateral attachments and then retracted laterally and posteriorly to complete the incisions. This approach permits the surgeon to verify the ability of the disc to be repositioned posteriorly before excision. With severe atrophy of the disc, substantial resistance to posterolateral traction is noted. A hemostatic clamp is positioned across the anterior attachment to serve as a guide plane for the knife, which is used to sever the attachment lateromedially. As the posterior attachment demonstrates a variable degree of vascularity changes, the DeBakey bulldog vascular clamp or straight mosquito clamp may be applied here before severing the posterior attachment. Next, a hemostat is used to apply outward traction to the tissue to be extirpated. A meniscus knife is used to sever the medial attachments. When the remodeled posterior attachment and disc are extirpated, the retrodiscal tissue is electrocauterized to control bleeding. Care is taken not to disrupt the fibrous connective tissue lining of the fossa and condyle. The morphology of the condyle and glenoid fossa often prevents excision in one piece. Incomplete excision of the posterior attachment over the lateral pole of the condyle may account for some cases of failure with discectomy. With the disc and posterior attachment removed, mandibular range of motion is checked. Joint noises, characterized as snappings, may indicate a disc remnant. Disc remnants are usually located on the medial aspect of the joint cavity. (Figure 50-29) The surgeon should remove all disc remnants that appear to impede movement.

Disc Replacement

Despite the reported success of long-term studies of TMJ discectomy, there is a perception among surgeons that disc



FIGURE 50-29. Discectomy without disk replacement. **A,** Large perforation through the articular disc. The top of the condylar head can be seen through the disc perforation. **B,** Postdiscectomy view of the joint with the condyle free of mechanical interferences. **C,** Surgical specimen.

replacement is required to help prevent or reduce the significant osseous remodeling, intra-articular adhesions, and recurrent pain that is seen following discectomy alone. An interpositional material was believed to decrease joint noises by dissipating loading forces on the osseous surfaces. The effectiveness of routine interpositional materials and grafts to reduce adhesions, protect the articular surfaces, and diminish pain and postdiscectomy joint noise has not been substantiated by the literature, as long-term results of discectomy alone are far superior to short-term discectomy with grafts and disc preservation procedures.[80–82]

The popularity of alloplastic for TMJ disc displacement materials suffered considerably after the experience with silastic[83] and teflon-proplast.[84] Both materials while enduring continuous loading by the mandibular condyle suffered fragmentation, migration, foreign body giant cell reactions, and recurrent joint symptoms.[85] The recognition of these limitations led to the abandonment of the permanent placement of both materials.[86] The use of silastic as a temporary disc replacement material is not supported by the literature.[87–88] However, it is currently used as a temporary spacer to prevent TMJ ankylosis, for a 6 to 12-week period of waiting for the fabrication of a CAD-CAM generated custom permanent alloplastic TMJ prosthesis. Additionally the vitallium alloy (cobalt, chromium, molybdenum) fossa-eminence prosthesis is another alloplastic material used after a discectomy that has reported in the literature with moderate success.[89–93] (Figure 50-30)



FIGURE 50-30. Alloplastic disc replacement. Vitallium alloy (cobalt, chromium, molybdenum) fossa-eminence prosthesis.

The debacle of silastic and teflon-proplast created an increased use of autogenous grafts.[94] Most autogenous grafts have been used to replace failed alloplastic implants, which means there are few reports of autogenous grafts placed at the time of the discectomy. In a critical review of interpositional grafts, Dimitroulis[95] suggests that the criteria for the ideal interpositional material used to replace the articular disc after discectomy should include:

1. Long term safety
2. Adequate bulk
3. Good handling properties
4. Easy to procure
5. Abundantly available
6. Survives the intrajoint environment
7. Facilitates normal joint function
8. Prevents bone formation and joint ankylosis
9. Protects condyle from severe remodeling

Temporalis muscle/fascia flap

The temporalis muscle/fascia is the most popular interpositional material for reasons of close proximity and ease of use. The flap may be pedicled in a variety of ways. Advantages of this technique over a free graft include its stability, owing to its connection at the base, its availability at the same surgical site, and its lack of cosmetic, and functional morbidity. The disadvantages include flap necrosis, fibrosis and similar to other TMJ surgical procedures is the risk of postoperative decreased interincisal opening.

Feinberg and Larsen described a technique which a temporalis muscle flap is develop that includes the temporalis fascia and the underlying pericranium from the most inferior horizontally oriented fibers of the temporalis muscle. The flap is based anteriorly off the coronoid process and passed anteriorly around the zygomatic arch in the TMJ that pedicled the posterior temporalis muscle fibers anteriorly. The muscle is then sutured to the retrodiscal tissues.[96,97] (Figure 50-31). Studies have reported that the muscle maintains its viability with some fibrosis.[96,98]

A procedure in which the muscle is placed over the zygomatic arch has also been described.[99] (Figure 50-32), The shape and size of the flap is outlined by incising posteriorly near the postglenoid spine of the joint through temporalis fascia muscle and periosteum. This incision is extended superiorly near the temporal line. Subperiosteal dissection elevates the amount of flap needed from the temporal bone. A transverse incision is made at the superior portion anteriorly to create a 3 cm–wide flap. The width should be greater than the anteroposterior coverage desired in the joint to allow flap contraction. An anterior incision is made parallel to the posterior incision. The superior aspect of the anterior incision is carried to bone in this thin area of the temporalis. Inferiorly, as the arch is approached, the muscle thickens; therefore, the dissections are not carried completely through muscle to bone. Blunt dissection is carried inferiorly to a point just medial to the arch to permit adequate mobility of the flap. Branches of the temporal artery found in this area are pre-served if possible. The

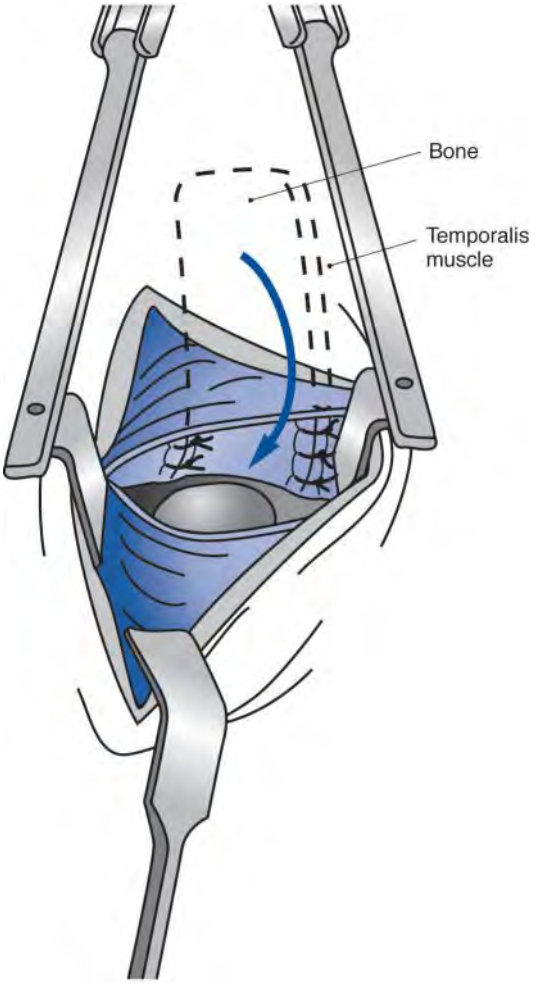


FIGURE 50-31. Disc replacement using temporalis mucle/fascial graft (*broken line*) pedicled from above the glenoid fossa and rotated inward (*arrow*). Lateral, anterior, and posterior sutures hold the graft in place.

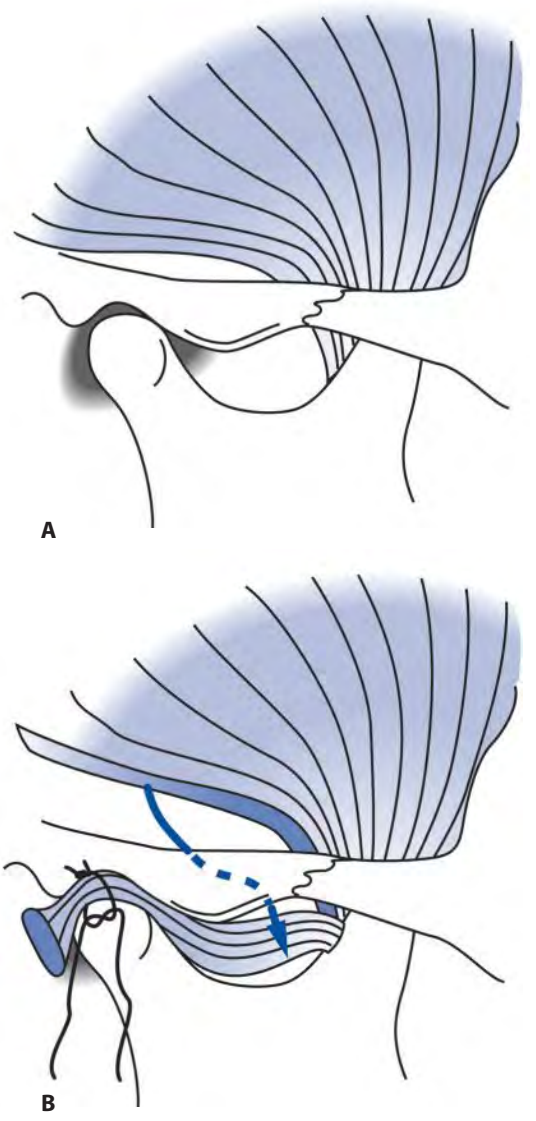


FIGURE 50-32. **A** and **B,** Disc replacement using temporalis muscle/fascial graft pedicled anteriorly and rotated anteriorly and inferiorly around (beneath) the posterior root of the zygomatic arch (*arrow*). The graft is sutured to the retrodiscal tissues.

length of the flap is usually 5 cm. The flap is fully reflected off the bone, and resorbable interrupted sutures are placed in several areas on the edge of the flap through fascia, muscle, and the periosteum to keep the layers from separating. Holes are drilled in the bone of the lateral lip of the glenoid fossa posteriorly and anteriorly before placement of the flap into the joint. One suture is placed through bone anteriorly near the eminence, and a second posterior suture is placed near the postglenoid spine. Two additional sutures hold the medial edge to anterior and posterior medial tissues. These medial sutures are sometimes difficult or impossible to secure, and the sutures through lateral bone are usually adequate to hold the flap in place. A cosmetic temporal defect may result depending on the thickness of tissue harvested.

The results from numerous clinical studies show a high success rate with the use of a variety of techniques involving temporalis muscle flaps of varying sizes and shapes, based either inferiorly or anteriorly.[96,99–102] A meta-analysis of the literature from 1965 to 2001 calculated mean success rate of 91.4% for the temporalis muscle flap.[82]

Ear Cartilage

Autogenous conchal cartilage has been described and praised as TMJ disc replacement by several authors.[103–107] Cartilage harvested from the cavum conchae results in minimal esthetic compromise and the graft can be tailored to fit the condyle or glenoid fossa. Notably, the quality and thickness of the aural cartilage is variable. In some cases an iatrogenic tear in the cartilage may occur during the harvesting process.

The procedure to obtain chondral cartilage as interpositional material for TMJ reconstruction has been described by Hall and Link.[105] A 3 to 4 cm postauricular incision is made on the ear a few millimeters lateral to the auriculocephalic sulcus and is carried through to the perichondrium. The middle division of the posterior auricular artery may be encountered and ligated or cauterized. A careful supraperichondral

dissection with a fine dissecting scissors exposes the surface of the cartilage. A scalpel is used to cut through the cartilage in the shape of the desired amount of graft, usually 1.5 by 2.5 cm. It is important not to extend to the rim of the antihelix to avoid permanent deformity of the ear. Subperichondral dissection between the skin of the bowl and cartilage permits the cartilage to be removed without tearing it or perforating the skin. The ear is packed with gauze or other material to maintain the shape of the bowl and to apply pressure to the skin. The pressure pack is maintained for 48 hours.

Although mean success rates of 82.4% have been published with auricular graft as disc replacement[82], other studies have shown their tendency to fragment, proliferate and result in fibrous ankylosis with progressive mouth opening limitation.[108–110]

Dermal Fat Graft

Georgiade originally reported the use of dermal fat graft for surgical correction of TMJ dysfunction.[111] Only a few clinical studies published on the use of dermis grafts for disc repair[112] and disc replacement material in the TMJ.[113] Dimitroulis showed 35 joints in 29 patients that demonstrated dermis grafts were effective in reducing joint sounds, but the author conceded that dermis grafts were difficult to anchor and failed to prevent regressive remodeling of the condyle.[114]

The dermal graft may be harvested from the buttock, upper lateral thigh, groin, or the inner aspect of the upper extremity.[112,115] An elliptic wedge of epidermis and underlying dermis is harvested. The donor site wound is closed primarily and occlusive dressing placed. The underlying surface of the dermis must be defatted before being the graft is implanted. The defatted graft is trimmed and sutured to the retrodiscal tissue and the anterior and lateral capsular attachments. Unfortunately, in some cases, anchoring is difficult and the graft is simply inserted as space filler.

Abdominal dermis-fat graft

Dimitroulis published the first description of abdominal dermis-fat graft as disc replacement after TMJ discectomy.[116] The greatest advantage of this type of graft is its ability to be easily sculptured to fit precisely into any size cavity. Several studies by Dimitroulis and collegues have demonstrated that abdominal dermis-fat is a promising graft material that satisfies most, but not all the criteria for an ideal interpositional graft following discectomy.[95,116–120] They showed, in a quality of life study, that although discectomy was largely responsible for the resolution of joint pain and dysfunction, abdominal dermis-fat was a safe procedure and helps promote smooth, pain-free joint function.[118] While their studies have been largely positive, there are still concerns as dermis-fat graft is not always successful, as it fails to protect the condyle from further degeneration in one-third of cases and the ultimate fate of a joint replacement in 7% of cases.[119–120]

Tissue engineered TMJ disc

The rapidly growing technologies of tissue engineering will provide in the future alternatives for TMJ disc replacement.[121–123] Tissue engineered articular discs are at the nascent phase of development but the real challenge will come when researchers try to anchor the newly developed disc to the surrounding articular structures without compromising the normal function of the joint.

Modified mandibular condylotomy

Modified mandibular condylotomy is an extra-articular surgical procedure that differs from other TMJ disc derangement procedures, as it does not directly involve the articular surfaces. Essentially, the procedure consists of an intraoral vertical ramus osteotmy in which the medial pterygoid muscle is partially detached to allow for 3 to 4 mm of condylar sag. Ideally, the downward position of the condylar segment increases the joint space, reducing the load on the retrodiscal tissue and relieving the pain and dysfunction[124] The extra-articular nature of the procedure explains the observation that a joint will not be made worse by a modified condylotomy[125] The procedure carries low morbidity and significant complications are rare. It has the disadvantage that requires a brief period of maxillomandibular fixation (MMF) followed by several weeks of training elastics. The principal indication for modified mandibular condylotomy is the management of pain and mechanical symptoms in patients who have disc displacement with reduction (79-94% successful)[126–128] or patients who recently progressed to disc displacement without reduction (82% successful)[129]. Still, regardless of the stage of disc derangement, the increase of the joint space unloads of the retrodiscal tissues and articular surfaces, as well as relieves mechanical interferences. Thus, contrasting intra-articular procedures, no surgical joint pain occurs and joint pain is relief nearly immediate.[130] Absolute contraindication of the procedure includes uncontrolled bruxism and relative contraindications involve risk factors for occlusal disturbances after surgery. Two interesting facts about modified mandibular condylotomy are: 1) it is claimed that it can result in a more normal disc-condyle relationship[128] and 2) it seems to positively affect the progression of the disease.[131]

The surgical technique to perform a modified condylotomy includes the standard instrumentation for an intraoral vertical ramus osteotomy. Once arch bars are placed and local anesthesia is given, a standard incision is made over the external oblique ridge. Periosteum is elevated from the whole lateral ramus to identify important landmarks for the osteotomy that include the sigmoid notch, the mandibular angle and the posterior border of the mandibular ramus. Visualization is greatly facilitated with the use of endoscopy. Although the classical description of this procedure calls for the identification of the ante-lingula to avoid damage to the inferior alveolar nerve, Aziz and colleagues demonstrated complete concordance of the position of the lingula and antelingula in only 11.1% of the specimens studied.[132] In an anatomic study of the position of the mandibular foramen in relation to the LeVasseur-Merrill retractor, da Fontoura and colleagues[133] showed that the mandibular foramen was located within 7 mm of the posterior edge of the mandible in only 3.3% of the rami. When properly placed, the LeVasseur-Merrill

retractor was effective in avoiding the inferior alveolar nerve in 98.9% of the analyzed rami. The osteotomy is carried out in the central portion of the ramus using a 7mm angled oscillating saw against the LaVasseur-Merrill retractor. The inferior limb of the osteotomy is done through the inferior border of the mandible to allow for the desired condylar sag and better control of the condylar segment. The superior portion of the osteotomy is directed towards the sigmoid notch, with care to avoid excesive soft tissue trauma. Once the osteotomy is completed MMF is established and condylar sag is estimated mobilizing the segment and evaluating the step between the tip of the condylar segment and the inferior border. Medial pterygoid muscle is detached from the distal end of the condylar segment using a periosteal elevator to achieve 3-4 mm of condylar sag. Wire or plate fixation of the osteotomized segments is not necessary except in the cases of excessive condylar after excessive stripping of the medial pterygoid muscle or in cases in which condylar segment mobility is inadequate after previous TMJ surgical procedures. Finally if necessary, the tip of the condylar segment can be trimmed to avoid palpation. Immediate postoperative imaging is obtained to corroborate the correct position and the sag of the condylar segment. (Figure 50-33) Postoperatively after unilateral modified condylotomy, MMF is maintained for approximately 1 week, followed by 5 weeks of training elastics (single class II canine region on the operated side and single vertical on the nonoperated side). Bilateral cases will require 2 to 3 weeks of MMF follow by 3 to 4 weeks of training elastics (bilateral class II canine region).

Complications of this procedure include: bleeding, excessive condylar sag, fracture of the condylar segment, injury to the inferior alveolar nerve (1-8%), infection (1-2%), and malocclusion (22%). Three potential complications of modified condylotomy that may require reoperation are: excessive condylar sag with condylar dislocation, malocclusion, and fracture of the condylar segment.[134]

Postoperative Management

After surgery a combination of physical, pharmacological, and splint therapies are normally employed postoperatively to achieve an acceptable postoperative pain and range of motion. The positive role of physical therapy in recovery after TMJ surgery has been reported in the literature.[135–136] It is important to weigh the contribution of masticatory muscle myalgia to the patient's chief complaint. Although joint surgery can relieve the joint pain, in many cases it may be ineffectual in controlling muscle discomfort. Nonsurgical management of the muscle disorders must continue in many patients after surgery for TMJ disc derangements. Joint surgery does not restore the joint to its prepathologic state. The patient should understand that biting force may be reduced and jaw fatigue may become apparent with heavy meals or long conversations. After primary surgery (i.e., no previous TMJ surgery), one should strive for the following passive range-of-motion parameters: maximum interincisal opening of 35 to 40 mm, lateral excursive movements of 4 to 6 mm, and protrusive excursive movements of 4 to 6 mm. However, success should not be measured by the attainment of a finite measurement. A patient's overall success should be measured by the eradication or diminution of the preoperative complaints. Surgery is rarely performed to correct purely functional complaints. Elimination of pain during function is usually the predominant concern for the patient, who is willing to accept some compromise in degree of opening and lateral excursions.

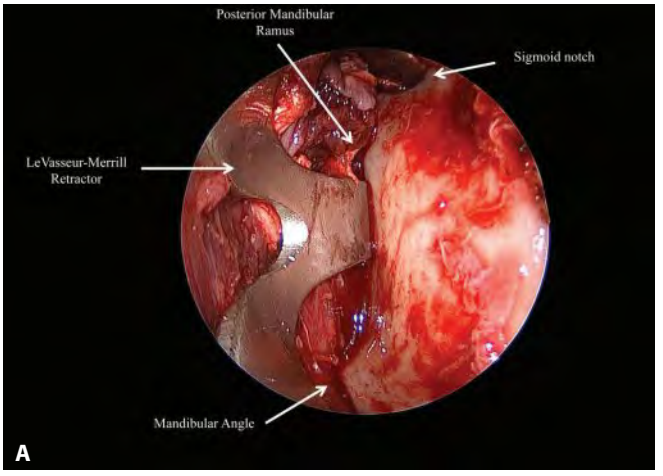
Postoperative outcomes may be influenced by several factors, including concomitant facial pain from other sources, degenerative bony changes, advanced morphologic changes in the disc, perforation of the posterior attachment, poorly controlled parafunctional habits, malocclusion, psychological overlay, previous TMJ surgery, history of facial nerve paralysis or orofacial numbness, history of infection, or systemic diseases affecting the muscles, ligaments, or bone[34].

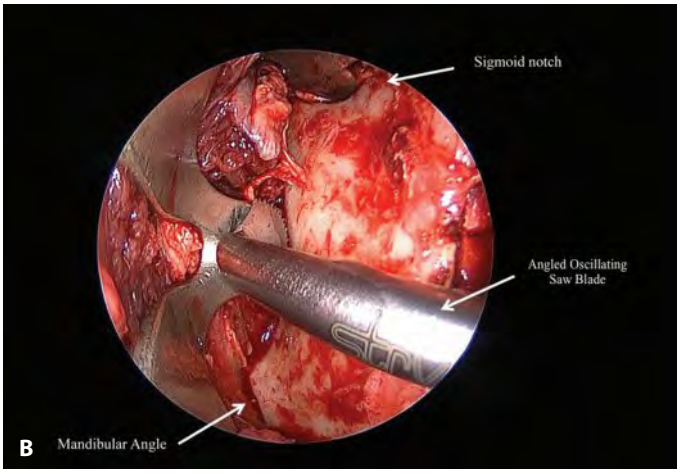
Splint therapy should be used to maintain a stable occlusal relation in the immediate postoperative phase. This is particularly important after disc repositioning. The appliance is frequently adjusted as the edema resolves and disc tissues heal. Splint therapy is routinely used as well when a large parafunctional component is present. The patient should be able to return to a normal mechanical diet with minimal dietary restrictions. Joint sounds may develop or persist, but the asymptomatic sounds should be of minimal concern to the patient.

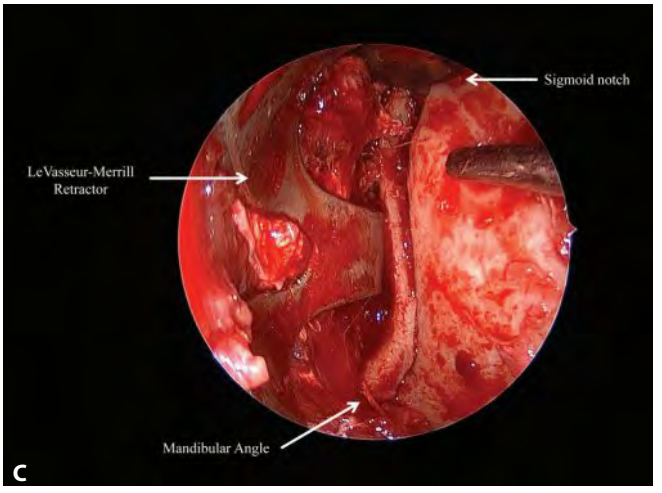
The clinician must balance the desire to rapidly and actively restore a normal range of motion with the capacity of the joint and facial muscles to adapt. Some latitude must be maintained on the part of the clinician in dealing with a patient's rehabilitation schedule. Care should be exercised in the rehabilitative process of the patient with bilateral joint disease whose operation was unilateral. Diet restrictions are important. Excessive lateral excursive movements to the ipsilateral side may contribute to the exacerbation of contralateral symptoms. There is no cookbook recipe to postoperative management of these patients. Some patients, regardless of the procedure, achieve an acceptable range of motion within 7 to 14 days, with minimal effort on their part. Others need to follow a strict physical therapy regimen. The devices such as Therabyte (Actos Medical) or Dynasplint (Dynasplint) are routinely used as part of the patient's physical therapy. Additionally the help of a physical therapist may sometimes be enlisted to regain joint mobility, especially when patient cooperation with a home exercise program is questionable. In general, some light passive opening and protrusion stretching exercises are prescribed four times a day beginning 5 days postoperatively. With disc repair procedures the physical therapy exercises should be more gradual. Patients should be maintained on a full liquid to soft diet for the first 2 post-operative weeks. Heat may be applied before and after exercises to improve comfort.

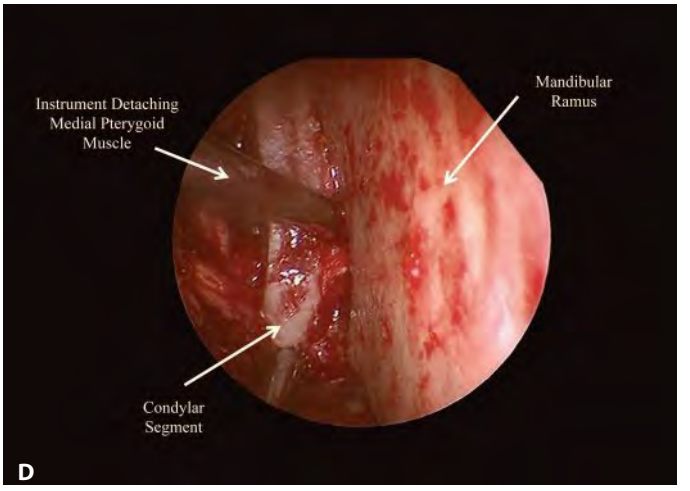
Complications

As with all surgical procedures, TMJ disc surgery is not exempt from potential complications. Complications after









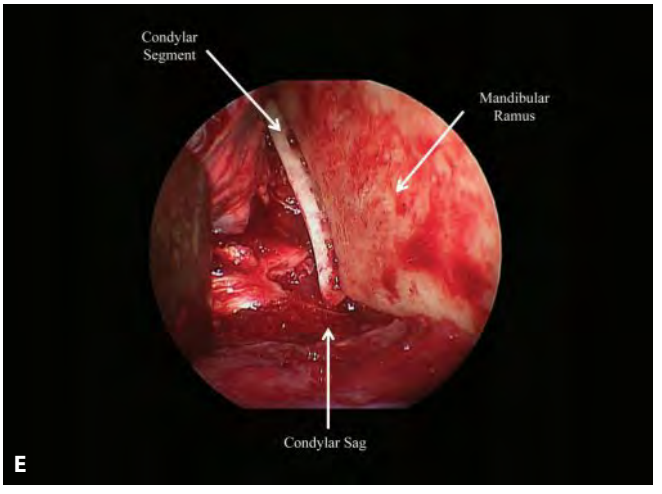




FIGURE 50-33. Endoscopically assisted modified condylotomy. **A,** Complete exposure of the lateral ramus and placement of the LeVasseur-Merrill retractor. Note the identification the landmarks: sigmoid notch, posterior ramus and mandibular angle. **B,** Angled oscillating saw blade in proper position against the LeVassuer-Merrill retractor. **C,** Osteotomy completed. **D,** Detachment of medial pterygoid muscle. **E,** Condylar sag with the amount of downward displacement of the condylar segment. **F,** Postoperative x-ray

TMJ disc surgery can be divided in: *perioperative and long-term surgical complications*.[137–138]

Perioperative complications

Vascular injury and hemorrhage

Hemorrhage from the retrodiscal tissue may interfere with performance of the disc repair. Temporary control may be obtained with seating of the condyle in the glenoid fossa. Electrocautery, injection of epinephrine, or application of hemostatic agents while maintaining the mandible in the closed position may be necessary.

Hemorrhage from the internal maxillary artery can be problematic and difficult to control. Usually the bleeding can be controlled with pressure and vascular clips. Control via selective embolization has also been described in the literature.[139]

Neurologic Injury

The incidence of facial nerve transient neuropraxia after open joint surgery ranges from 1-25%; it is generally limited to the temporal branch and usually resolves in 3 to 6 months.[137] It is also more frequent on patients with previous open joint surgery. Rarely, the zygomatic branches and, even more rarely, the entire temporofacial division may be injured. Injury to the chorda tympani from aggressive condylar retraction in the medial aspect of the fossa may occur rarely as well. Neuropraxia of the inferior alveolar and, less commonly, the lingual nerves may result from clamp placement for joint manipulation.

Auriculotemporal nerve syndrome (Frey's syndrome or gustatory sweating) has been reported after the dissection of the joint.[140] It is the result of auriculotemporal nerve damage with reinervation of the eccrine sweat glands by parasympathetic salivary fibers.

Infection

Infections rarely occur. Normally they are superficial wound infections in which microorganisms cultured may originate from the skin or external auditory meatus flora. To avoid contamination ear packing is avoided as it frequently becomes dislodged during surgery. In addition, the ear is not suctioned during surgery.

Otologic complications

The proximity of the TMJ to the ear increases the risk for potential damage. Lacerations of the external auditory canal (EAC) usually occur at the junction with the bony portion of the canal due to the anteromedial inclination of the canal. If a laceration occurs packing the EAC with iodoform gauze for 5-7 days will be enough to prevent stenosis. Alternatively antibiotic-hydrocortisone otic drops can be used to prevent infection and aid healing. Although rare, an aural-TMJ fistula may develop as long-term complication of EAC laceration.

Perforation of the tympanic membrane occurs when an instrument is inadvertently passed through the membrane. Small perforations in the anterior or inferior portion of the membrane result in minimal hearing loss. Larger and injuries to the posterior region may cause hearing loss due to possible ossicle disruption. If perforation is noted, otolaryngologist consultation is warranted. Auriculitis and external otitis are more likely to occur with the postauricular and endaural approaches. Treatment consists of pain control, cleaning of the EAC, and antibiotic-hydrocortisone otic drops.

Long-term complications

Malocclusion

Significant occlusal changes can be found in some cases after aggressive open joint surgery was performed.

Correction may require the services of orthodontist and/or prosthodontist and even further surgery like orthognathic surgery or total TMJ replacements.

Ankylosis

Fibrous ankylosis can form between the disc and the articular eminence if hemoarthrosis develops. Lack of patient compliance with postoperative physical therapy can also develop fibrous ankylosis. Bony ankylosis is more common after discectomy with poor postoperative physical therapy; with heterotopic bone formation and it can also be seen in the multiple-operated patient.

Reoperations

Unfortunately, TMJ surgeries are not always successful, and the patient's preoperative symptoms persist or may even increase after surgery. There are many potential pitfalls that can occur during any phase of the treatment that can lead to complications, less than desirable results, and short- or long-term failures. Establishing the possible causes of the unsatisfactory outcome is paramount in the reoperation planning process. Multiple causative factors have been described in the literature[134] and include:

1. Misdiagnosis of the original pathology
2. Patient selection
3. Inappropriate procedure
4. Surgeon's experience
5. Complications
6. Systemic disease
7. Improper postoperative care

When contemplating surgical failure and the necessity for reoperation, the surgeon must take into account all the different variables of the disease process to select the best surgical solution with the highest probability of success.

SUMMARY

Remarkably good success has been reported with several TMJ disc procedures, which differ in their fundamental approach to the problem and their aggressiveness. Open surgical approaches to TMJ disc derangements are now relegated to a tertiary line of care following nonsurgical therapy and arthrocentesis/arthroscopy for most conditions. They do,

however, have a clear indication for certain mechanical conditions directly attributed to a disc obstruction.

As we increase our understanding of the pathology, open joint surgical procedures are being performed for specific well-defined conditions. However, the new TMJ surgeon will never quite appreciate the experience that comes with performance of arthrotomy procedures. Arthroscopy developed as a consequence of this experience. Now, as we regress with progress, arthrocentesis with and without steroid injection, a procedure performed by many surgeons, years before the pathology of the joint was even elucidated, has become a mainstay for treatment. This treatment alone has significantly reduced the need to intervene via arthrotomy.

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CHAPTER

Hypomobility and Hypermobility Disorders of the Temporomandibular Joint

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HYPOMOBILITY

Etiology

The etiology of mandibular hypomobility is varied, and successful treatment requires an understanding of the underlying disorder. Trauma is the most commonly identified cause, followed by infection (odontogenic, otitis media, and mastoiditis). Various systemic disease states have been associated with hypomobility, including ankylosing spondylitis, rheumatoid arthritis, and other collagen vascular diseases such as scleroderma. Iatrogenic causes have also been identified and include the sequelae of high-dose radiation involving the muscles of mastication, craniotomy procedures, and uncommonly, orthognathic surgery. Internal temporomandibular joint (TMJ) derangements may also lead to chronic hypomobility problems. Traumatic perinatal events and neuromuscular conditions can result in hypomobility in infancy. In general terms, *congenital ankylosis* is defined as limited interincisal opening noted at birth with no known causative factor. Table 51-1 lists the etiologic factors associated with mandibular hypomobility.

Classification

Various classification schemes have been proposed to describe hypomobility.[1–3]

Trismus is most commonly found in conjunction with spasm of the muscles of mastication. It can be secondary to myofascial pain dysfunction, infection, trauma, tumors, and various medications as well as psychiatric and neurologic factors. Ankylosis may be classified according to location (intra-articular vs. extra-articular), type of tissue involved (bony, fibrous, or mixed), and extent of fusion (complete vs. incomplete). True ankylosis is caused by either fibrous or bony fusion of the structures contained within the TMJ capsule and, in its most severe state, is characterized by a bony union of the condyle to the glenoid fossa. True ankylosis has been further classified into subtypes depending on the anatomic positioning of the condyle and the extent of bridging bone. Topazian[4] proposed a three-stage classification to grade complete ankylosis as follows: stage I, ankylotic bone limited to the condylar process; stage II, ankylotic bone extending to the sigmoid notch; and stage III, ankylotic bone extending to the coronoid process. Other classification schemes have also been proposed.[5] However, the utility of these designations in terms of treatment planning is questionable. So-called false ankylosis (pseudoankylosis), in contrast, describes limited mobility based on extra-articular factors such as fibrosis, mechanical obstruction (e.g., zygomatic arch fracture), muscle spasm, or other pathologies.

Clinical Presentation

Patients with fibrous or bony ankylosis present with restricted mandibular motion and, depending on the patient's age and the condition's etiology, may have an abnormality in mandibular size and shape. Unilateral pathology in children may result in significant problems with lower facial symmetry. A shortened ramus on the affected side is usually accompanied

TABLE 51-1. **Etiologic Factors Associated with Hypomobility of the Mandible**

Trismus
Odontogenic: myofascial pain, malocclusion, erupting teeth
Infection: pterygomandibular, lateral pharyngeal, temporal
Trauma: fracture of the mandible, muscle contusion
Tumors: nasopharyngeal tumors, tumors that invade jaw muscles
Psychologic: hysteric trismus
Pharmacologic: phenothiazines
Neurologic: tetanus
Pseudoankylosis
Depressed zygomatic arch fracture
Fracture dislocation of the condyle
Adhesions of the coronoid process
Hypertrophy of the coronoid process
Fibrosis of the temporalis muscle
Myositis ossificans
Scar contracture after thermal injury
Tumor of the condyle or coronoid process
True Ankylosis
Trauma: intracapsular fracture (child), medial displaced condylar fracture (adult), obstetric trauma, intracapsular fibrosis
Infection: otitis media, suppurative arthritis
Inflammation: rheumatoid arthritis, Still's disease, ankylosing spondylitis, Marie-Strümpell disease, psoriatic arthritis
Surgical: postoperative complications of temporomandibular joint or orthognathic surgery

by a prominent antegonial notch noted on radiographs. Such unilateral mandibular growth disturbances have secondary effects on the maxillary occlusal plane and midfacial structures (pyriform rims and bony orbits).

Ankylosis in adults is characterized by limited jaw opening and decreased translation, but the morphologic characteristics found in the growing patient are frequently absent. Loss of condylar structure and mandibular angle prominence is seen in cases caused by rheumatologic disease, specifically scleroderma. An associated anterior open bite is frequently noted with the loss of ramus/condyle height (Figure 51-1).



FIGURE 51-1. Patient with systemic sclerosis (scleroderma) demonstrating a limitation in jaw opening and skin changes characterized by perioral furrows and telangiectasia.

Unilateral cases with a traumatic etiology may result in malocclusion and ipsilateral dental prematurities. A physical examination is helpful in identifying whether the process is bilateral or unilateral and may be suggestive of the etiology.

Imaging Assessment

In addition to the clinical examination, radiographic assessment is critical in evaluating and treating patients with hypomobility disorders. Plain radiographs are limited in delineating the true extent of the deformity. What can be identified with these studies are the presence or absence of a TMJ space, obvious bony abnormalities in the region of the joint, and coronoid hyperplasia. Sanders and colleagues[6] have reported that conventional radiographs underestimate the extent of bony ankylosis and give little information about the anatomy medial to the condyle. The use of computed tomography (CT) scans (including axial, coronal, and sagittal views with three-dimensional reconstruction) is helpful in fully defining the extent of ankylosis as well as the relationship of the ankylotic mass to important anatomic structures, especially at the skull base (pterygoid plates, carotid canal, jugular foramen, and foramen spinosum) (Figure 51-2).[7,8] Often in post-traumatic cases. the distance between the maxillary artery and the medial pole of the condyle is reduced—a contrast CT helps to determine this distance. Fusion of the ankylotic mass to the base of the skull can also be appreciated on CT scans. Because adequate treatment requires the removal of the mass in toto, knowledge of this anatomy preoperatively is critical to surgical planning and long-term success.

Magnetic resonance imaging (MRI) has had a great impact on TMJ evaluation, especially regarding the delineation of meniscal position. Diagnosis of fibrous ankylosis is possible with the use of MRI, but the CT scan is superior in demonstrating bony pathology.[9]

Post-traumatic Hypomobility

Trauma is the most common cause of bony and fibrous ankylosis as reported by multiple authors.[10–12] It is hypothesized that the formation of an intra-articular hematoma with subsequent scarring and new bone formation is the common precipitant. Most often, a medially displaced fracture dislocation of the condyle is found. Subsequent hypomobility is of particular concern in growing children in whom the development of hypomobility can have significant impact on facial growth (Figure 51-3). In addition, resultant hypomobility can lead to speech impairment, difficulty with chewing, poor oral hygiene, limited access to dental care, and possible airway compromise. In large reviews of pediatric facial fractures, the condylar and subcondylar regions were involved in more than 40% of cases.[13,14] In many cases, a direct blow to the chin with transmission of the impact force to the condyles resulted in the fracture. Prolonged immobilization, secondary to treatment with maxillomandibular fixation, splinting, or mechanical obstruction, can lead to subsequent ankylosis.

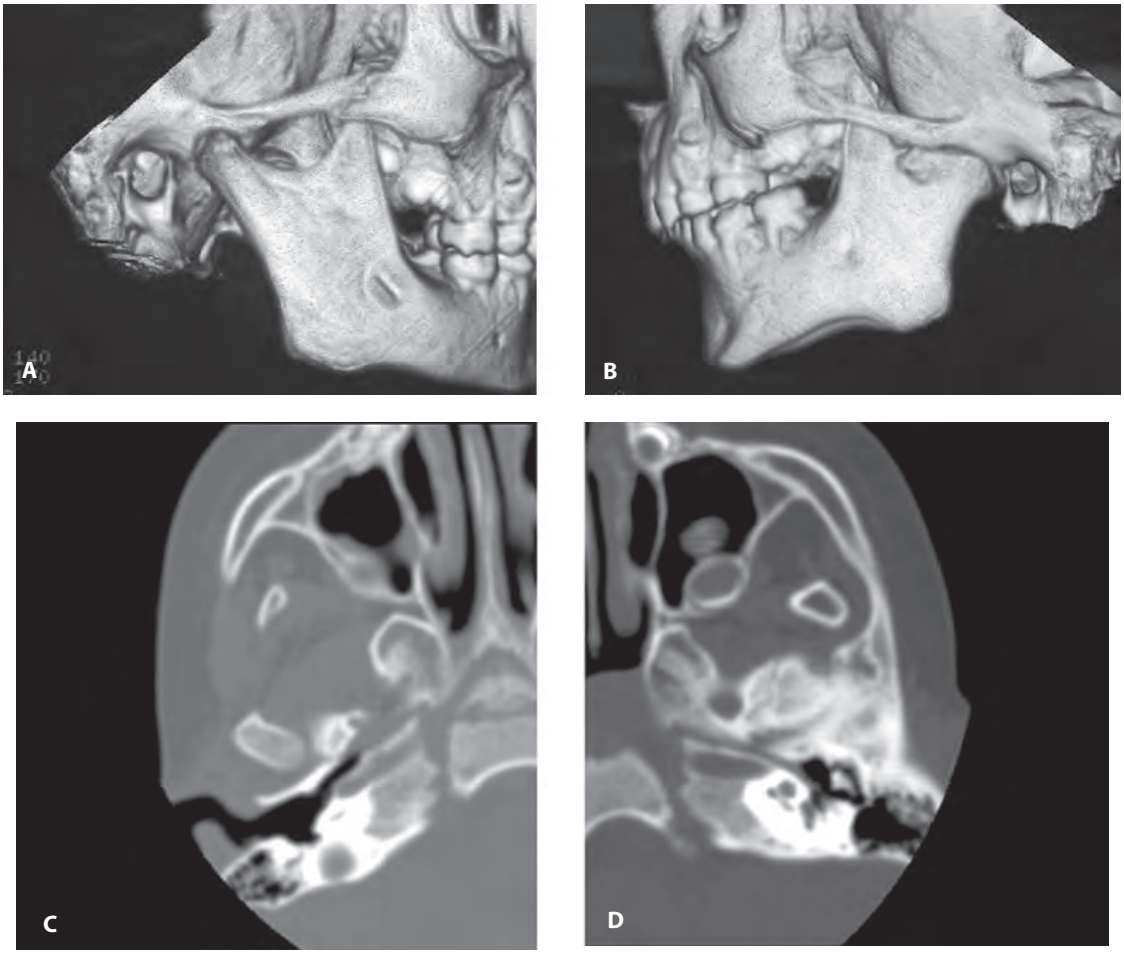


FIGURE 51-2. Three-dimensional (**A** and **B**) and axial (**C** and **D**) computed tomography (CT) scans of a patient with extensive bony ankylosis of the left temporomandibular joint. **A** and **C,** Note the comparison with the unaffected right side. **B** and **D,** Coronoid hyperplasia is also seen on the affected left side.

Extra-articular ankylosis can also occur with coronoid fractures and fractures of the zygomatic arch. In both cases, the resultant hematoma may calcify, resulting in a fusion of the coronoid process to the zygomatic arch.

Myositis ossificans traumatica (MOT), or fibrodysplasia ossificans circumscripta, is generally associated with a traumatic event or repeated episodes of minor trauma and can result in mandibular hypomobility.[15,16] The precise mechanism remains to be elucidated but appears to involve fibrous metaplasia and subsequent ossification of both soft tissues and muscle after bleeding and myonecrosis. Histologically, both mature and woven bone can be noted (sometimes in distinct zones), and both osteoblasts and osteocytes are abundant. MOT is characterized by soft tissue ectopic ossifications and is relatively uncommon in the head and neck regions. Of all reported cases involving the muscles of mastication, the masseter is most commonly affected. MOT involving the medial pterygoid muscle and secondary to local anesthesia injections has also been reported.[17] Diagnosis is confirmed by identification of calcifications within the muscles of mastication on CT scans (Figure 51-4). Minimal response is found with physical therapy and stretching exercises; consequently, surgical treatment is often undertaken to remove the ectopic bone. Other treatment modalities include acetic acid iontophoresis, magnesium therapy, and the use of etidronate sodium.[18,19] Because repeated relapses and refractory cases are common, the use of multiple treatment modalities may be associated with the best outcome.

Postinfectious Hypomobility

A TMJ infection resulting in hypomobility is most commonly the result of contiguous spread from an odontogenic infection, otitis media, or mastoiditis.[20,21] In the era of aggressive antibiotic treatment of infection, such reported cases are now relatively uncommon. Hematogenous spread of infection has also been reported in association with disease states such as tuberculosis, gonorrhea, and scarlet fever.

Various case series describe deep fascial space infections manifesting themselves as hypomobility and often being misdiagnosed at initial presentation.[22,23] Odontogenic infection is commonly associated with trismus. In such cases, associated symptoms (fever, dysphagia) are likely present, and CT scanning is invaluable in determining a diagnosis and



FIGURE 51-3. **A** and **B,** Evident mandibular growth disturbance is noted in this child who has had bilateral condylar fractures. Note the submental scar secondary to a laceration sustained at the time of the bony injury.

in treatment planning. Medial pterygoid abscess formation or fibrosis secondary to hematoma organization can be precipitated by an inferior alveolar nerve block or posterior superior alveolar block. A history of recent dental treatment should suggest this possibility; the use of CT imaging can help delineate the anatomy of the masticator and pharyngeal spaces.

Mass lesions (both benign and malignant) can also result in mandibular hypomobility. Squamous cell carcinoma of the tongue base or tonsillar pillar is often accompanied by trismus. Masses involving the mandibular condyle invariably affect range of motion and need to be included in the differential diagnosis of hypomobility.

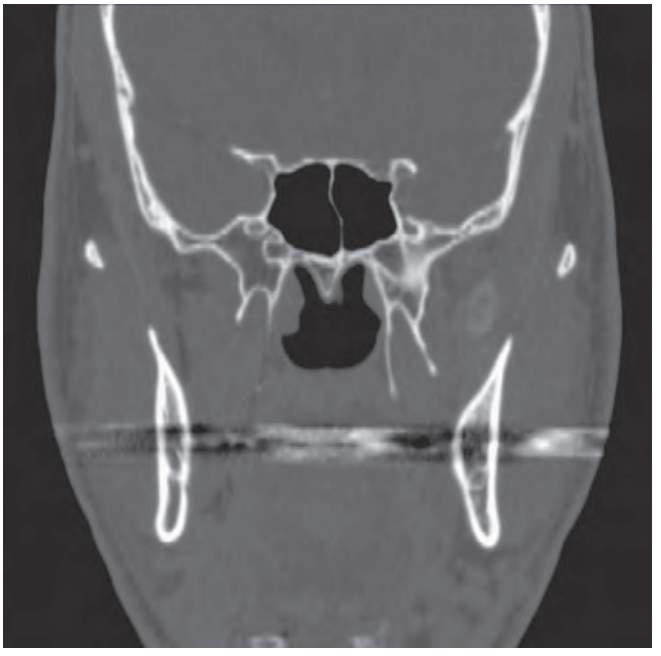


FIGURE 51-4. CT scan of patient with myositis ossificans traumatica demonstrates a focus of calcification within the medial pterygoid muscle on the left side.

Hypomobility after Radiation Therapy

Mandibular hypomobility is a common sequela of the treatment of head and neck malignancies (Figure 51-5). The resultant fibromyositis caused by radiation therapy may exacerbate the postsurgical problems caused by large ablative procedures.[24] Goldstein and coworkers[25] reviewed the effects of tumoricidal radiation therapy on restricted mandibular opening and found a linear dose-related effect. Mandibular dysfunction increased as the dose to the pterygoid muscles increased. The authors reported diminution in opening with doses as low as 15 Gy. Pow and associates reported that 30% of patients treated for nasopharyngeal carcinoma with high-dose radiation therapy had significant trismus compared with age-matched nonradiated control subjects.[26] Radiation therapy for primary tumors of the retromolar trigone was associated with a 12% incidence of long-term trismus. This association, compounded by resultant xerostomia, severely compromises the ability of these patients to maintain oral health.

The efficacy of early interventional physical therapy has been described. Buchbinder and colleagues[27] compared the outcome of unassisted exercise, mechanically assisted exercise with the use of tongue blades, and use of the Therabite System in radiated patients. All patients presented with an interincisal opening of less than 30 mm. The response to each therapy was recorded every 2 weeks over a 10-week period.



FIGURE 51-5. Patient with a history of high-dose radiation therapy and subsequent reirradiation for recurrence of nasopharyngeal carcinoma. Note the severe temporal atrophy and the limitation in opening.

All groups showed improvement over the first 4 weeks, but the group using a mechanical exercising device (i.e., Therabite System) continued to demonstrate an improvement of maximal interincisal opening (MIO) over the full 10-week period that was significantly greater than that of the other two groups.

Postcraniotomy Hypomobility

Mandibular hypomobility after intracranial surgical procedures is an uncommon yet reported phenomenon.[28,29] Mechanistically, this problem is secondary to neurosurgical procedures performed through the temporal bone requiring an incision of the temporalis muscle. Subsequent fibrosis of the muscle may then result in limited opening, which is best treated with coronoid resection followed by vigorous physical therapy. The incidence of this problem is not known, but a review by Kawaguchi and coworkers[30] reported limited mouth opening in as many as 33% of patients undergoing frontotemporal craniotomy procedures. Although most are self-limiting, persistent hypomobility can severely compromise subsequent airway and anesthesia management in these patients and needs to be recognized. The maximal opening is not improved with the use of muscle relaxants or local or general anesthesia. Patients who have undergone skull base surgery may also manifest severe hypomobility postoperatively. If such surgery requires the dissection of the temporalis muscle inferior to the zygoma, pseudoankylosis of the mandible may be encountered.

Inflammatory and Rheumatologic Causes

Ankylosing spondylitis (Bekhterev's disease) is a chronic and progressive inflammatory condition most commonly affecting the sacroiliac joints and the spine. The male-to-female ratio of incidence is reported to be 2.4:1, and the severity and extension of the disease in male patients is found to be more severe. TMJ involvement in ankylosing spondylitis has been reported in between 1% and 22% of affected individuals and can include severe bony deformation and ankylosis.[31,32] The most commonly reported radiographic findings in the condyle and glenoid fossa region include flattening, erosions, sclerosis, osteophytes, subcortical cysts, and bony erosion at the insertion of the masseter (angle of the jaw) and temporalis muscles (coronoid process). One large prospective study evaluating 50 patients with ankylosing spondylitis did not show any correlation between the bony severity noted in the cervical spine and TMJ abnormalities.[33] These authors reported a 22% incidence of TMJ involvement, either clinical or radiographic. Because the majority of patients reported no pain or limitation in function, the radiographic findings included in this study may well have represented early changes in the disease process.

TMJ involvement in rheumatoid arthritis follows the same destructive path as do other joints. Generally, the severity of joint dysfunction is correlated with the stage of rheumatoid arthritis. Radiographically, the most common findings in the condylar region are sclerosis (75%), erosion (50%), and flattening (30%).[34] These bony changes commonly result in progressive malocclusion secondary to the loss of ramus/condyle height and subsequent apertognathia. Juvenile rheumatoid arthritis is chronic arthritis diagnosed in childhood before the age of 16 years. It is estimated that greater than 60% of patients with juvenile rheumatoid arthritis manifest TMJ involvement.[35] However, multiple authors point out that despite radiographic and morphologic changes in the joint, a minority of affected children (generally < 25%) report pain with function.[36,37] Svensson and associates[38] report that restricted mouth opening was a more common finding. The duration of active disease and a history of pain with function correlate positively with progressive TMJ dysfunction. With active disease in growing children, abnormalities in facial growth, mandibular hypoplasia, and hypomobility are common problems (Figure 51-6).

Scleroderma (progressive systemic sclerosis) is a disorder of unknown etiology affecting multiple organ systems and characterized by abundant fibrosis of the skin, blood vessels, and visceral organs. It is believed that abnormalities in small blood vessels result in the progressive thickening and fibrotic changes noted in affected tissues, particularly those of the gastrointestinal tract, heart, lung, and kidney as well as diffuse skin involvement. Mandibular movement can become severely limited in affected individuals secondary to facial



FIGURE 51-6. Adult patient with a history of juvenile rheumatoid arthritis affecting the temporomandibular joints and resulting in a mandibular growth disturbance and hypomobility.

skin fibrosis and atrophy of the muscles of mastication (particularly the masseter and medial pterygoid muscles).[39] Bony changes in the mandible are also reported and include severe resorption of the angles, condyles, and coronoid processes (osteolysis).[40] The bony lesions are believed to be of ischemic origin but may be exacerbated by the tightness of the tissue in the region of the mandibular angles causing pressure resorption as well. In addition to the severe limitation in jaw movement, the small mouth orifice and progressive malocclusion make oral function and access to dental care problematic for these patients.

Hypomobility after Orthognathic Surgery

Hypomobility after orthognathic surgery has been reported by multiple authors and appears to be most commonly associated with the bilateral sagittal split osteotomy.[41,42] This postoperative limited opening has been commonly attributed to muscle atrophy and soft tissue scar formation. Atrophic muscular changes seem to be exacerbated by prolonged use of maxillomandibular fixation, and the advent of rigid internal fixation appears to have limited this problem. Intra-articular pathology (edema, hemorrhage) as well as condylar torque may also result in hypomobility. In such cases, rigid internal fixation may predispose to this problem. Van Sickels and colleagues[43] have hypothesized that condylar torque at the time of the bilateral sagittal split osteotomy may cause impingement of the condyle against the disk, causing a mechanical impediment to opening.

Management of hypomobility after orthognathic surgery depends on the underlying cause. Trauma to the muscles of mastication is best managed postoperatively by vigorous physical therapy protocols. Those patients who fail to improve within the first 3 months need to be carefully evaluated for an intra-articular source of the problem. Edema, bleeding, and fibrosis within the joint space can frequently be managed by arthrocentesis procedures, especially when recognized early. If a mechanical obstruction to opening is suspected, CT is a helpful diagnostic aid. Condylar torque is best treated by reoperation with appropriate positioning of the proximal segment.[44]

General Treatment Considerations

The treatment goal for all hypomobility states is the restoration of normal and comfortable jaw motion and prevention of disease progression. Reversible causes such as muscular hyperactivity or spasm, infectious and inflammatory causes, and medication-induced limitations must be identified and treated. Restoration of function in cases of ankylosis can be difficult. Proper treatment requires excision of the involved structures and immediate reconstruction. Many operative techniques have been described in the literature, with varying and often less than satisfactory results. As mentioned previously, understanding the etiology and anatomy of the problem is critical and can be greatly aided with CT.

The gap arthroplasty is a procedure that creates a new area of articulation distal to the fused TMJ and ankylotic segment.[45,46] Advocates of this procedure describe its simplicity. However, the creation of a pseudoarticulation significantly shortens the ramus height, and the procedure is associated with a high degree of reported reankylosis. Development of postoperative malocclusion and a decreased range of motion are the most common problems associated with this procedure as reported by Rajgopal and coworkers.[47] Because of these limitations, the use of the gap arthroplasty to treat ankylosis has been largely abandoned.

TMJ ankylosis is more commonly treated with complete excision of the ankylotic mass and, if required, by subsequent joint reconstruction. Our protocol for the treatment of ankylosis follows that documented by Kaban and associates,[48] a sequential protocol for the treatment of TMJ ankylosis that is based on aggressive resection of the ankylotic mass. Wide intraoperative exposure is required, and special attention is directed to the medial aspect of the joint to ensure that bony, fibrous, and granulation tissue are completely removed. In addition to this aggressive resection of the bony and fibrous mass, dissection and stripping of the temporalis, masseter, and medial pterygoid muscles followed by ipsilateral coronoidectomy are performed in all cases through the same incision. Longstanding ankylosis frequently results in muscle fibrosis and coronoid hyperplasia. After this resection is completed, the MIO is measured. If it is found to be less than 35 mm, a contralateral coronoidectomy is performed via an intraoral approach to attain the desired level of opening. Because complete resection of the ankylotic mass frequently results in substantial loss of ramus height, subsequent reconstruction must address this fact and attempt to restore occlusion as well as function. Commonly, a temporalis fascia flap and costochondral graft are employed to both line the glenoid fossa and create ramus height. The patient is placed into maxillomandibular fixation after the reconstruction, and the teeth are placed into a prefabricated occlusal splint. Fixation is maintained for approximately 10 days, and after release, a strict protocol of physiotherapy is employed. Overall results have been excellent with this approach. After 1 year, MIO was maintained at greater than 35 mm in all 18 patients included in the report. Furthermore, absence of pain with function was reported in all but 2 patients (Figure 51-7).[48]

Recently, the Kaban protocol[49] has been modified to substitute ramus/condyle reconstruction using distraction osteogenesis, when possible, instead of costochondral grafting. This protocol has the major advantage of eliminating the donor site operation and allowing for immediate vigorous TMJ mobilization. The surgical procedure for the release of the ankylosis is identical to that described previously. After the release, the jaw is mobilized and the glenoid fossa lined with a temporalis myofascial flap if the native disk is unavailable. The remaining mandibular stump is reshaped to create a narrow and rounded top. A corticotomy is created distally, leaving sufficient bone to serve as a transport disk. The distraction device is secured, the corticotomy completed, and



FIGURE 51-7. Three-year-old boy with bilateral bony ankylosis after a motor vehicle accident that also produced bilateral lacerations of the commissures. Frontal photograph **(A),** frontal maximal incisal opening (MIO; **B),** and lateral photograph **(C).** Note the limited opening. Right **(D)** and left **(E)** panoramic views of the ankylotic masses of the temporomandibular joints (TMJs). Right **(F)** and left **(G)** TMJs exposed after the dissection was completed. **H,** Harvested costochondral grafts with 1- to 2-mm cartilaginous caps. *(continued)*

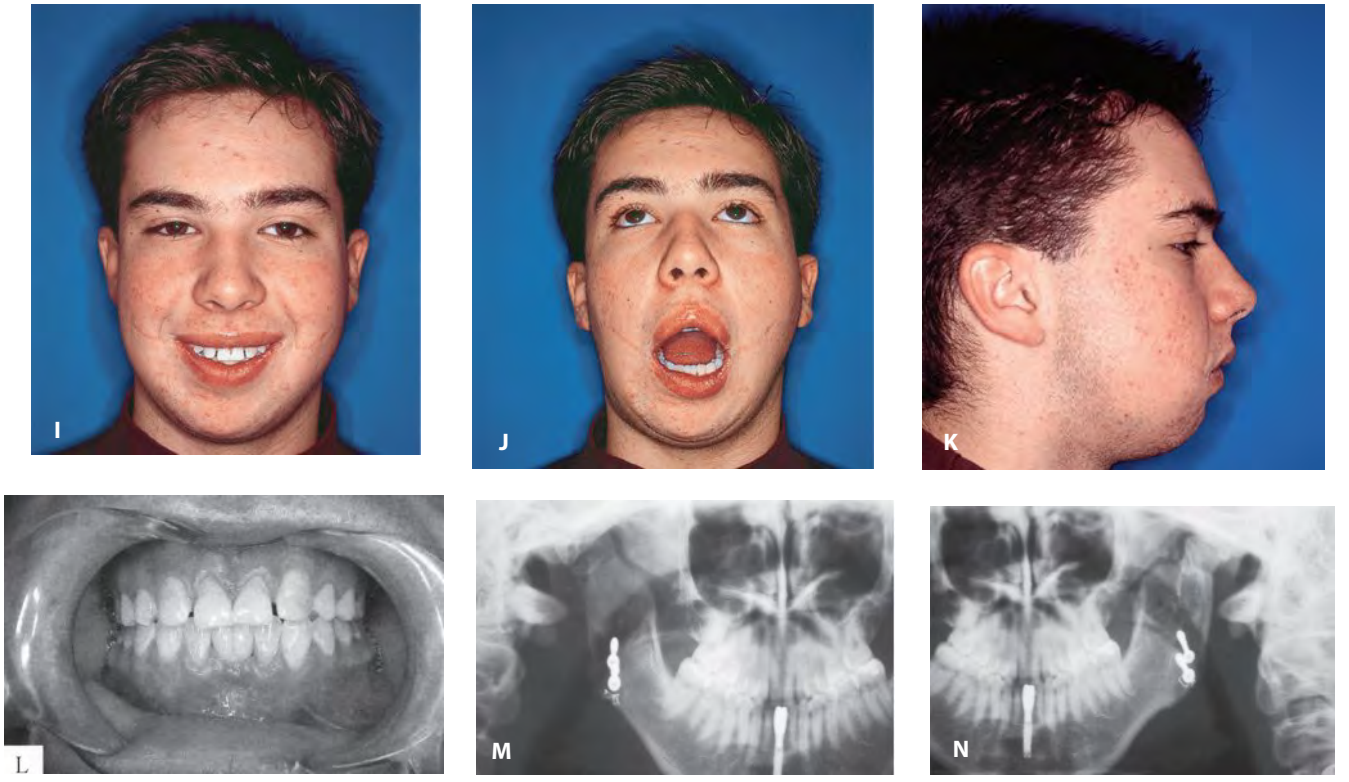


FIGURE 51-7. *(continued)* Frontal **(I),** frontal opening **(J),** and lateral **(K)** facial views of the patient 11 years postoperatively. Note maintenance of the normal MIO. **L,** Intraoral views. Right **(M)** and left **(N)** panoramic radiographs show remodeling of the costochondral grafts. (**A–M,** Reproduced with permission from Kaban LB. Acquired temporomandibular deformities. In Kaban LB, Troulis MJ, editors. Pediatric Oral and Maxillofacial Surgery. St. Louis: Elsevier; 2004; pp. 361–365.)

the mobility of the segments tested. Distraction then proceeds at 1 mm/day until the desired length is achieved. The patient begins a program of active jaw motion exercises immediately postoperatively (Figure 51-8).

The use of total joint prostheses has an interesting history in the TMJ. Advocates describe two major advantages over autogenous reconstruction: (1) the absence of a donor site and (2) the ability of the patient to return to function more quickly. However, multiple complications have been reported—some with devastating consequences for patients.[50–52] Foreign body reaction to any alloplast may occur. In its most severe form, extensive bony erosion in the area of the glenoid fossa has been found. Fragmentation of alloplastic material secondary to function with a migration of particles into contiguous tissue and regional lymph nodes has also been reported. Progressive wear may result in a loosening and fracture of the prosthesis. In addition, the lack of growth potential precludes the use of these joint replacement systems in young children. Recurrent ankylosis after prosthesis placement has also been reported, with periprosthetic calcifications most commonly seen in younger patients.

TMJ reconstruction with a variety of autogenous tissues has been described. When the extent of bony resection does not severely shorten ramus height, autogenous interpositional grafts may be employed. These include skin, temporal muscle, cartilage, and fascia. A review by Chossegros and colleagues[53] demonstrated superior results (defined by the authors as an interincisal opening of ≥ 30 mm over a follow-up period of 3 yr) using full-thickness skin grafts and temporalis muscle. Various bone grafts (costochondral, sternoclavicular, iliac crest, and metatarsal head) have been used to reconstruct ramus height after the resection of ankylosis. First described in the 1920s, the costochondral graft for TMJ reconstruction was popularized in later years by Poswillo[54] and MacIntosh and Henny.[55] Autogenous tissue (particularly the costochondral graft) has the advantage of being biologically acceptable and possessing growth and remodeling potentials that make it a particularly attractive reconstructive choice in the growing child. Potential problems with its use include fracture, resorption, donor site morbidity, recurrence of ankylosis, and a variable growth behavior of the graft in situ.

Complications Associated with Treatment

Various complications have been reported secondary to the treatment of ankylosis. Dolwick and Armstrong[56] caution that a severe limitation of opening can make the palpation of landmarks difficult and increases the surgical risks. The aggressive bony removal and recontouring that is often required can increase the risk of development of an aural-TMJ fistula if the tympanic plate is displaced posteriorly. In



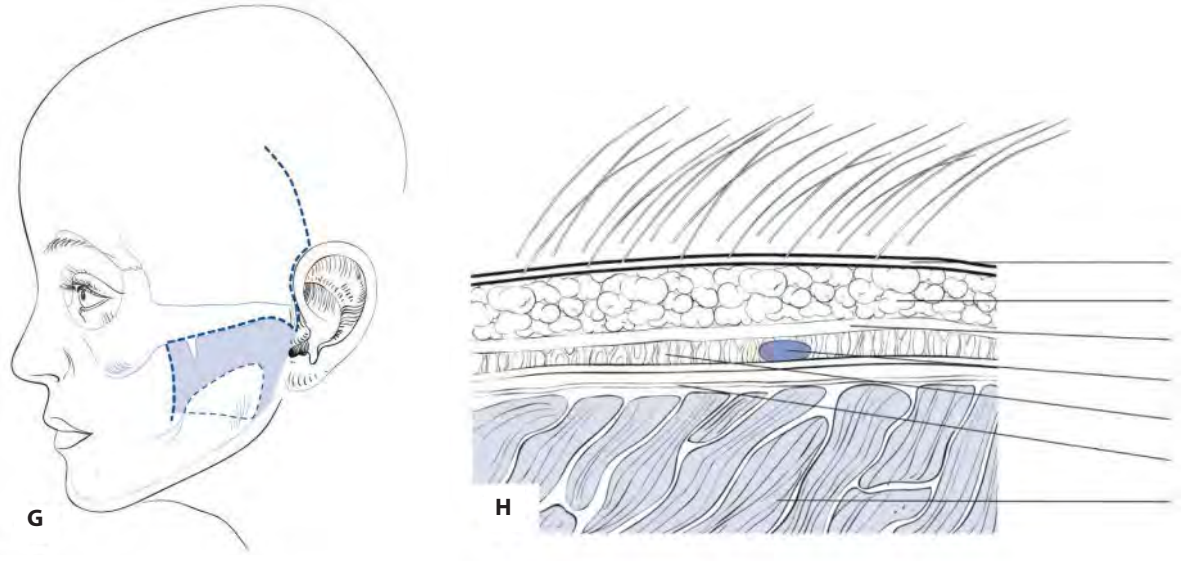
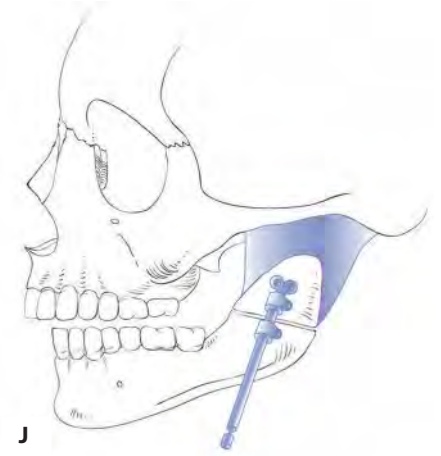
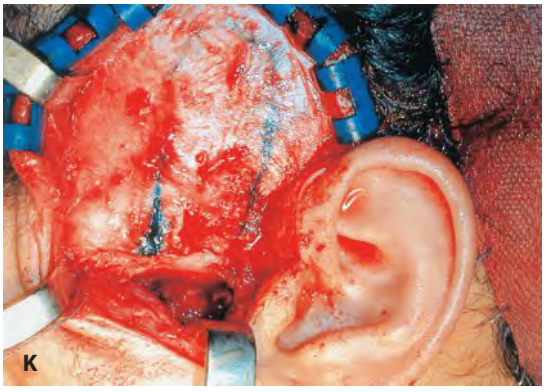
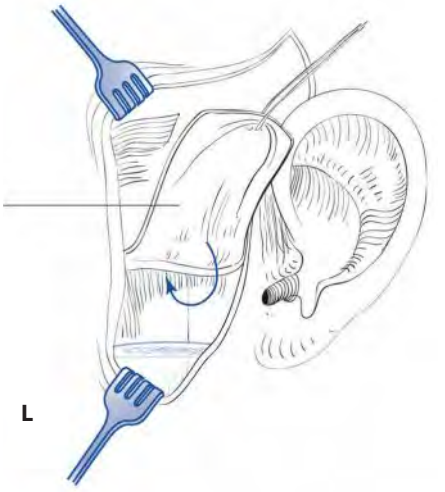


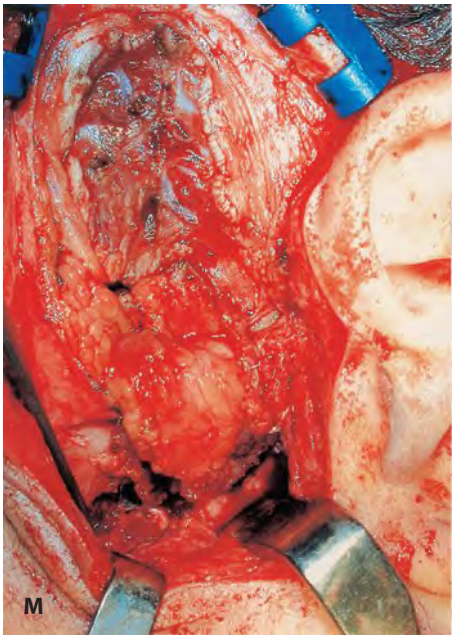
FIGURE 51-8. Thirteen-year-old female with recurrent ankylosis of the left TMJ secondary to trauma sustained in a motor vehicle accident. Frontal **(A),** frontal at MIO **(B),** and lateral facial **(C)** photographs of a teenage female with recurrent ankylosis of the left TMJ. **D,** Panoramic radiograph before the first operation demonstrates bony ankylosis of the left TMJ. **E,** Panoramic radiograph after the patient developed reankylosis. She had had a condylectomy and coronoidotomy at another institution. The TMJ was reconstructed with a costochondral graft. There was no soft tissue lining in the joint. **F,** Lateral cephalogram documents the mandibular retrognathism. **G,** Diagram of the operative plan; the ankylosis release is carried out via a preauricular incision (outlined in *dashed blue line*). Excision of the ankylotic mass and coronoidectomy is shown by the *shaded area*. **H,** Diagram of the layers of the scalp. *(continued)*











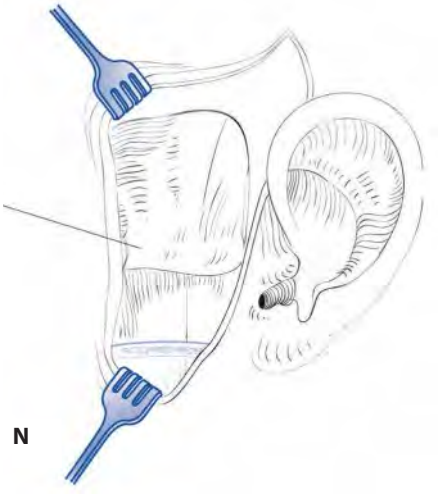
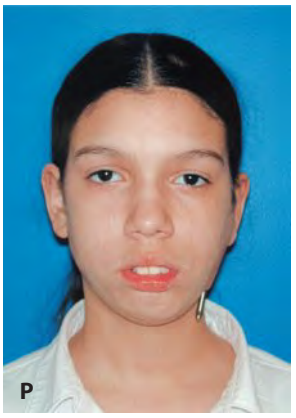
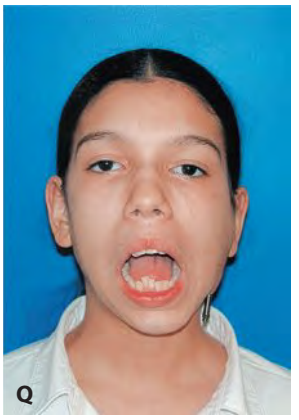
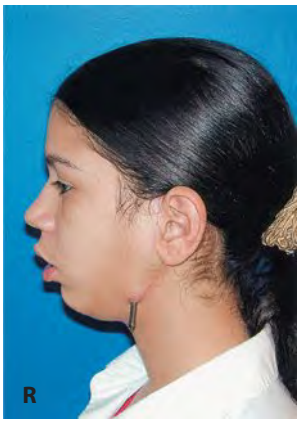




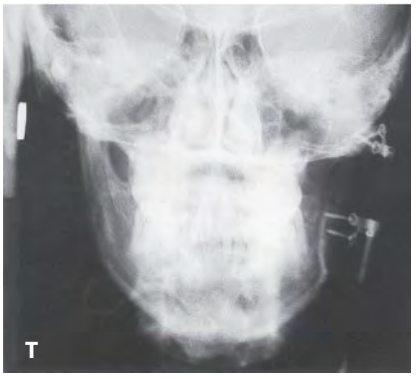
FIGURE 51-8. *(continued)* **I,** Intraoperative view after dissection was completed. Note the bony ankylotic mass and the coronoid process with obliteration of the sigmoid notch. **J,** Diagram of the bone removed *(shaded area)* and the proposed reconstruction using a distraction device (Synthes Maxillofacial, Paoli, PA) instead of a costochondral graft. **K,** Temporalis flap is outlined with malachite green. The flap is dissected and rotated over the arch **(L)** and sutured in place (**M** and **N**). **O,** Specimen: ankylotic mass and coronoid process. *(continued)*

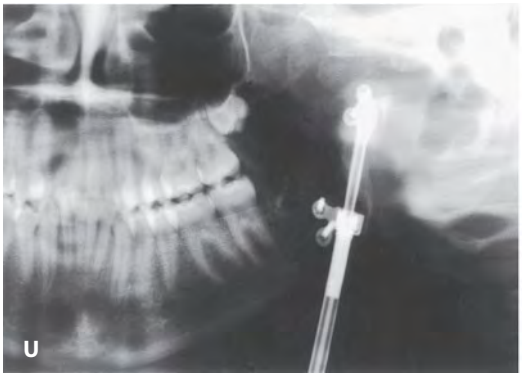














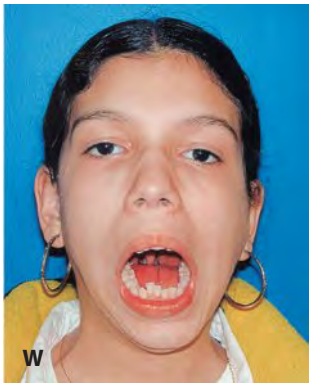








FIGURE 51-8. *(continued)* Frontal **(P),** frontal opening **(Q),** and lateral **(R)** photographs at end distraction. The patient was mobilized and started on physical therapy immediately postoperatively. She was comfortable because there was no donor site operation and no period of maxillomandibular fixation. Lateral **(S)** and anteroposterior (AP; **T)** designated as cephalogram and panoramic radiograph **(U)** at the end of distraction osteogenesis demonstrate the lengthened mandibular ramus. Frontal **(V),** frontal opening **(W),** and lateral **(X)** photographs 1 year after completion of treatment. The patient maintained her TMJ motion and will be beginning presurgical orthodontic treatment to correct her preexisting malocclusion. Open **(Y)** and closed **(Z)** intraoral views with the patient opening 39 mm at 1 year. *(continued)*

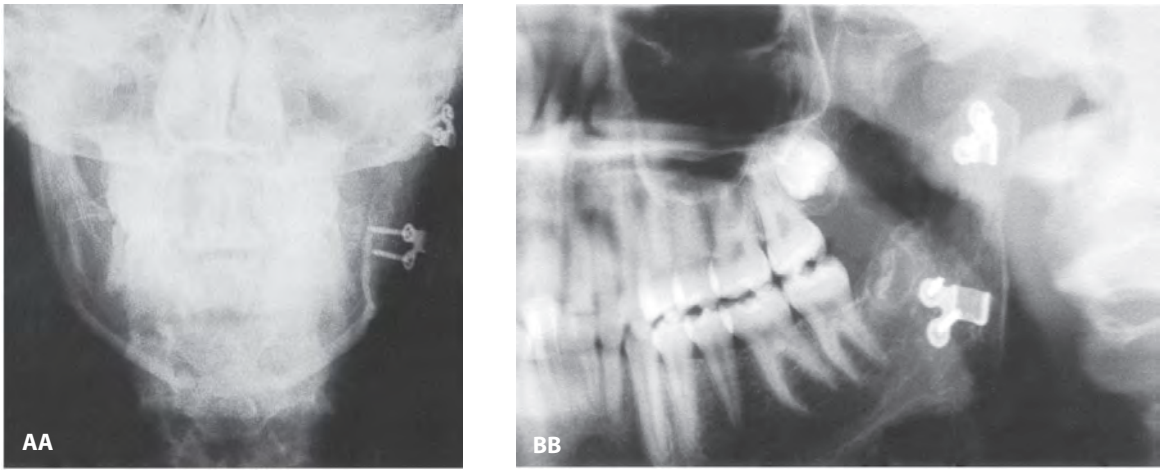


FIGURE 51-8. *(continued)* AP cephalogram **(AA)** and panoramic radiograph **(BB)** at 1 year. The ramus lengthening is demonstrated by the space between the retained footplates. (**A–F, I, K, M,** and **O–BB,** Reproduced with permission from Kaban LB. Acquired temporomandibular deformities. In Kaban LB, Troulis MJ, editors. Pediatric Oral and Maxillofacial Surgery. St. Louis: Elsevier; 2004; pp. 354–357; **G, H, J, L,** and **N,** adapted from Kaban LB. Acquired temporomandibular deformities. In Kaban LB, Troulis MJ, editors. Pediatric Oral and Maxillofacial Surgery. St. Louis: Elsevier; 2004; pp. 354–357.)

addition, stenosis of the external auditory meatus and subsequent hearing impairment may follow tympanic plate displacement.

Recurrent ankylosis may result from inadequate initial treatment. It most commonly occurs on the medial aspect of the condyle where surgical access is most difficult. Such maneuvers as the postoperative use of nonsteroidal anti-inflammatory drugs and vigorous physical therapy limit problems with recurrent hypomobility.[57,58]

In pediatric patients treated for ankylosis, the expected outcome may be less sanguine.[59] The improvement in interincisal opening, despite strict adherence to the previoously discussed treatment protocol and compliance with physical therapy regimens, is often significantly less than 35 mm. Posnik and Goldstein[60] reviewed the outcome of nine children and demonstrated a mean MIO of 24.8 mm in unilateral cases and 17.5 mm in bilateral cases measured an average of 2 years postoperatively. The authors caution that improvement in bilateral congenital cases is particularly problematic and may be confounded by the associated neuromuscular and atrophic changes found in these patients.

Peripheral nerve injuries are possible sequelae of all TMJ operations, with the upper branches of the facial nerve being the most vulnerable. Parotid gland injury with subsequent sialocele and fistula formation has also been reported.

As previously described, the costochondral graft is the most commonly used autogenous material for TMJ reconstruction. However, its growth pattern can be unpredictable. Linear overgrowth with the subsequent development of asymmetry and malocclusion has been reported by multiple authors.[61,62] The frequency is more common in the growing patient. Munro and coworkers[61] reported 2 of 22 cases of considerable linear overgrowth with resultant chin deviation and development of a class III malocclusion. Perrott and associates[62] reported 3 of 26 cases of lateral bony overgrowth (tumor-like overgrowth), with an evident preauricular fullness and subsequent limitation of opening. However, no cases of linear overgrowth were found in that series of patients.

Postoperative Physical Therapy

Patients with hypomobility disorders require aggressive physical therapy programs, often in conjunction with surgical treatment, to maintain a functional MIO. Various rehabilitation programs have been described in the literature, and approaches include unassisted exercise, tongueblade and finger-stretch exercises, manual exercisers, and mechanically assisted mandibular motion devices (Figure 51-9). Manipulation under general anesthesia may also be required in refractory or recurrent cases. Most authors agree that the duration of physical therapy should be prolonged well after a desired MIO is achieved to prevent subsequent relapse.[63]

HYPERMOBILITY

Classification

Mandibular subluxation occurs when there is a momentary inability to close the mouth from a maximally open position. It is defined as a self-reducing partial dislocation of the TMJ, during which the condyle passes anterior to the articular eminence. In distinction, dislocation may be considered a long-lasting inability to close the mouth. Subluxation of the condyle may be an early feature of TMJ pathology in a subset of patients. It is often associated with an abnormally wide opening while eating or yawning. Extended periods of mouth opening (e.g., during dental treatment or endotracheal anesthesia) may also precipitate subluxation. Subluxation

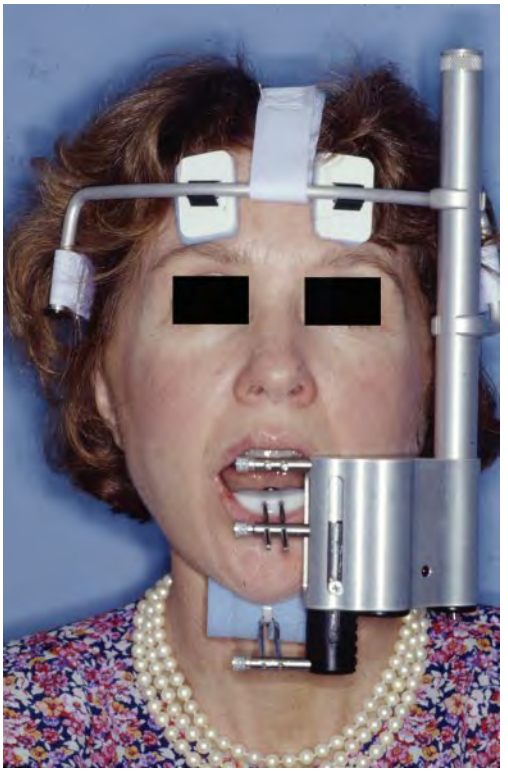


FIGURE 51-9. A continuous passive motion device used in the postoperative management of hypomobility.

may occur secondary to acute trauma or after a seizure and is also associated with systemic diseases such as Ehlers-Danlos syndrome and Parkinson's disease.

Etiology

TMJ dislocation is defined as an internal derangement characterized by a condylar position anterior and superior to the articular eminence that is not self-reducing. Recurrent dislocation is a relatively unusual problem. Much like subluxation, the etiology is varied. It is observed most frequently in patients with neurologic and connective tissue disorders, those with TMJ dysfunction, and those being treated with phenothiazines and other neuroleptic agents (Table 51-2).

Extrinsic trauma, especially that sustained while the mouth is open, may result in dislocation. Wide opening of any type as well as capsular laxity may be etiologic. Muscular problems secondary to medication use or neurologic disorders may be associated. The problem may be unilateral or bilateral, and patients generally present with associated muscle spasm and pain.

Treatment Considerations

In the absence of pain, subluxation requires no specific treatment because it is self-reduced by the patient. When associated with wide mouth opening, conscious efforts to avoid this are usually successful at preventing recurrent subluxation. Patients are advised to modify their diets, and

TABLE 51-2. **Causes of Hypermobility**

Intrinsic Trauma: Overextension Iinjury
Yawning
Vomiting
Wide biting
Seizure disorder
Extrinsic Trauma
Trauma: flexion-extension injury to the mandible, intubation with general anesthesia, endoscopy, dental extractions, forceful hyperextension
Connective Tissue Disorders
Hypermobility syndromes, Ehlers-Danlos syndrome, Marfan syndrome
Miscellaneous Causes
Internal derangement, dyssynchronous muscle function, contralateral intra-articular obstruction, lost vertical dimension, occlusal discrepancies
Psychogenic
Habitual dislocation, tardive dyskinesia
Drug-Induced
Phenothiazines

dental treatment is done over multiple shorter appointments. The use of bite blocks during procedures can also be helpful. In cases in which extreme laxity in the joint results in continued problems, surgical intervention may be warranted.

Reduction of mandibular dislocation should be done precipitously before muscle spasm becomes severe and makes the procedure more difficult. Reduction is accomplished by pressing the mandible downward and then backward to relocate the condyle within the glenoid fossa (Figure 51-10).

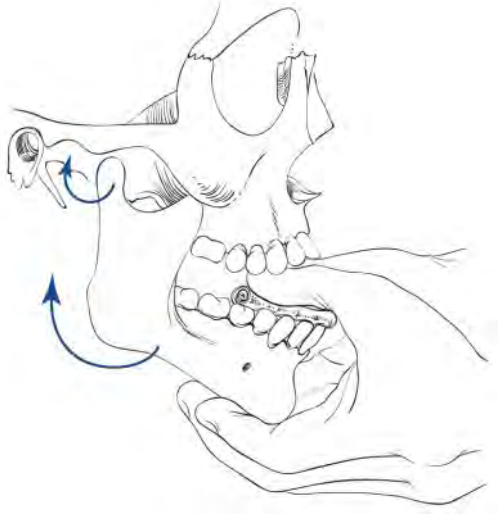


FIGURE 51-10. Bimanual mandibular manipulation in a downward-posterior direction to disengage the condyle from its open-locked position posterior to the articular eminence. (Adapted from Rotskoff KS. Management of hypomobility and hypermobility disorders of the temporomandibular joint. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 2009.)

In acute cases, this can generally be accomplished without the use of anesthesia. In cases of prolonged or chronic dislocation, the use of muscle relaxants and analgesics may be required. If reduction cannot be thus achieved, general anesthesia may be required. After reduction, the mandible should be immobilized for several days to allow for capsular repair, muscle rest, and prevention of recurrence.[64,65]

Chronic dislocation usually requires a more interventional approach. The use of various sclerosing agents has been described in the past. However, caustic agents can result in progressive damage to other joint structures, and multiple reports of misapplications and complications have resulted in the abandonment of this technique. Surgical treatments of various types are reported. Identification of etiology is important when considering surgical correction. In cases of extreme joint laxity, mechanical tightening may be indicated. Plication procedures involve fastening the condyle to a fixed structure to maintain its position within the glenoid fossa. Certain authors advocate the creation of a mechanical impediment to translation by altering the conformation of the articular eminence. Procedures targeting a decrease in muscle pull can also be effective.

Plication procedures are aimed at limiting mandibular motion and may be accomplished in various ways. Removal of redundant capsular tissue (Figure 51-11) is a relatively simple method for addressing laxity, and a review by MacFarlane[66] reported excellent long-term results. Plication of the condyle to the temporal bone and of the coronoid process to the zygomatic arch have also been described. Multiple materials have been used for plication procedures, including both resorbable and nonresorbable sutures and wire. Miniplates and surgical anchors have also been used in both the lateral pole of the condyle and the posterior roof of the zygomatic arch. Wolford and colleagues[67] have described the threading of heavy suture material between the eyelets of the surgical anchors, thereby preventing condylar dislocation.

Mechanical impediments to condylar translation effectively deepen the glenoid fossa. Bone and cartilage grafts (cranial, iliac crest, rib, tibial) have been used for this purpose. Nonautogenous material has also been onlayed to the articular eminence. In 1943, LeClerc and Girald[68] described a procedure for inferior displacement of the zygomatic arch to prevent translation (Figure 51-12). Access was gained through an extended preauricular incision, and dissection of the zygomatic arch was performed. An oblique osteotomy downward and forward then allowed the arch to be moved inferiorly. Chossegros and colleagues[53] reported excellent success using this technique in 36 patients with chronic and recurrent dislocation.

The eminectomy procedure was first introduced by Myrhaug in 1951[69] as a treatment for chronic and habitual dislocation of the condyle. In addition to the standard open eminectomy, reports describing the use of the arthroscope for this purpose have more recently appeared in the literature.[70] Both procedures involve the removal of a portion of the articular tubercle and eminence to allow the condyle to move freely.

Concerns regarding the use of the eminectomy procedure include hypermobility of the joint with further damage to contiguous tissues; significant and often bothersome TMJ noise (clicking and crepitation) with function; and the potential for facial nerve injury, recurrent dislocation, and inadvertent temporal lobe exposure (anatomic variant).[71]

Reported success rates of surgery to treat dislocation vary considerably. Recurrent dislocation after standard eminectomy procedures ranges from 7% to 33%.[72–74] Patients with significant ligamentous laxity or predisposing conditions (e.g., seizure disorders) are prone to recurrent problems.

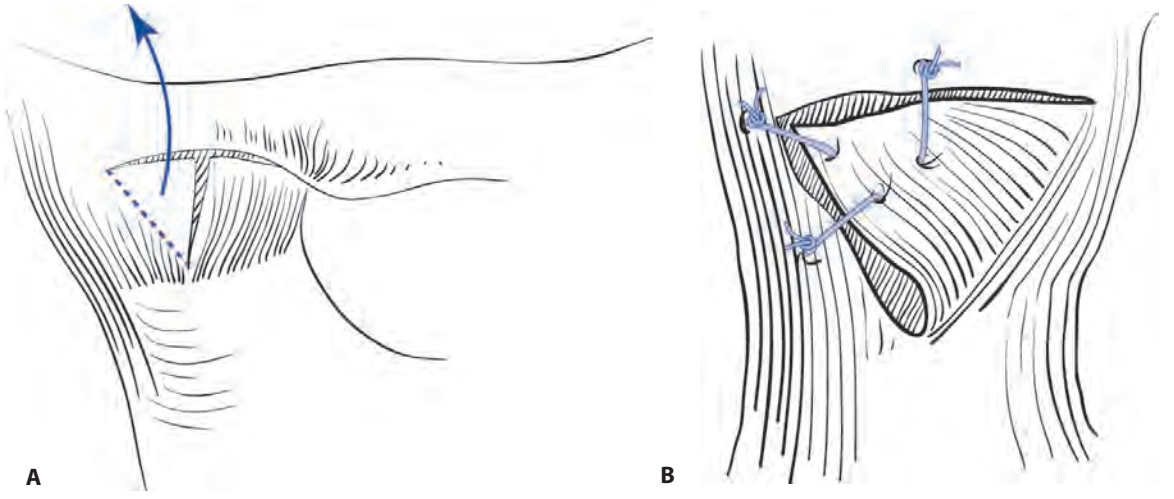


FIGURE 51-11. Capsular plication. The exposed lateral capsule is incised **(A)** and sutured back on itself **(B)** to tighten and limit capsular laxity. (**A** and **B,** Adapted from Rotskoff KS. Management of hypomobility and hypermobility disorders of the temporomandibular joint. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 2010.)

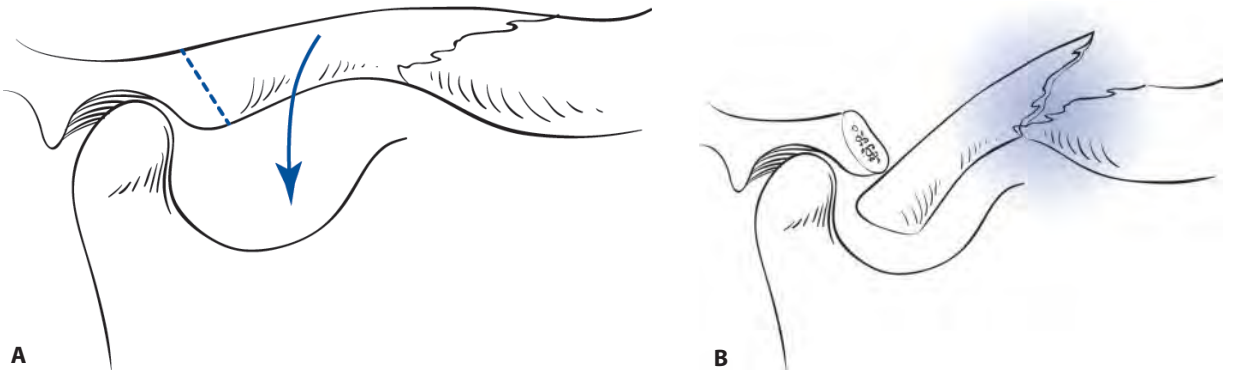


FIGURE 51-12. LeClerc procedure. **A,** An oblique cut using a fissure bur is created anterior to the articular eminence to decrease the frequency of condylar dislocation by obstructing the path of condylar movement. **B,** The osteotomized segments of the articular eminence are made to overlap one another. (**A** and **B,** Adapted from Rotskoff KS. Management of hypomobility and hypermobility disorders of the temporomandibular joint. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 2010.)

Arthroscopic eminectomy, owing to technical limitations, prevents the complete removal of the medial aspect of the eminence. The consequence of this in terms of recurrence remains to be elucidated.[72,73]

If muscular hyperactivity is associated with chronic recurrent dislocation, removal of the insertion of the lateral pterygoid muscle (lateral pterygoid myotomy) may be an effective treatment. Bowman has reported good success with this procedure,[74] but subsequent animal studies have shown lateral pterygoid electromyographic activity returning to baseline several months after the procedure.[75] However, the long-term efficacy often attributed to this procedure may be secondary to scarring anterior to the joint capsule, thereby limiting condylar excursion.[76]

The injection of botulinum toxin type A into the lateral pterygoid muscles has also been proposed as a treatment for chronic and recurrent dislocation of the mandible. Ziegler and coworkers[77] reviewed 21 patients treated in this fashion. Injections were given on a 3-month basis with only 2 of 21 patients suffering further dislocations. No adverse side effects were reported in this series. Botulinum toxin type A has an associated latency of 1 week, and its duration of action is between 2 and 3 months. Injections should not be done more often than every 12 weeks to avoid the development of antibodies. An injection dose of between 10 and 50 U into the targeted muscle is usually sufficient.

Clark[78] reviewed the use of botulinum toxin for the treatment of mandibular motor disorders, as well as for the treatment of facial spasm, and expanded on the potential side effects of such treatment. Although local side effects are unusual, the two most common problems encountered were alterations in salivary consistency and an inadvertent weakness of swallowing, speech, and facial muscles. These complications were more commonly reported with lateral pterygoid, soft palate, and tongue injections and were found to be dose-dependent.

SUMMARY

This chapter summarizes the spectrum of mobility problems that can affect the TMJ and contiguous structures. The varied etiologic factors associated with hypo- and hypermobility have been reviewed; an understanding of the etiology in each particular case is imperative for appropriate treatment to be rendered. Fortunately, improved imaging techniques, including three-dimensional CT, can be invaluable adjuncts to the history and physical examination. In cases of ankylosis, the extent and nature of the problem is best appreciated with these CT images. Altered anatomy and the extent of bony bridging can be assessed preoperatively. In addition to operative intervention, long-term success in the management of ankylosis requires aggressive physical therapy programs and longitudinal follow-up.

Hypermobility (both subluxation and dislocation) is similarly reviewed. Again, understanding the causative factors (ligamentous laxity, shallow eminentia, muscular hyperactivity) helps one to focus the treatment planning and to minimize problems with recurrence.

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52

CHAPTER

End-Stage Temporomandibular Joint Disease

Louis G. Mercuri, DDS, MS

INTRODUCTION

End-stage disease represents the worst condition or disease state of an organ system, at which point in time, the organ is functioning minimally or not at all. Systemic examples include end-stage renal disease, in which the kidneys have essentially shut down and the patient requires dialysis or renal transplantation in order to accomplish the essential roles of the kidney; and end-stage cardiac disease, in which the heart is functioning very poorly with minimal cardiac output and a compromised ejection fraction, and may need mechanical support (e.g., left ventricular assist device) or cardiac transplantation in order for the patient to survive, considering the vital role of the cardiovascular system. When applying the term *end-stage* to the functional joints of the human body, *end-stage joint disease* indicates a joint that is so negatively affected architecturally by disease or injury that severe functional impairment is the final result for the patient. As with all other joints in the body, the temporomandibular joint (TMJ) is affected by any and all of the end-stage joint diseases (Table 52-1). These disease processes may result from developmental disorders (Figure 52-1), neoplasia (Figure 52-2), trauma, arthritic disease (Figures 52-3 and 52-4), failed prior joint surgery (Figure 52-5), or fibrous or bony ankylosis (Figure 52-6). Regardless of the etiology of the end-stage disorder, it should be noted that the goals of any reconstructive joint surgery include improvement of mandibular function and form; reduction of further suffering and disability; containment of excessive treatment and cost; and prevention of further morbidity (Table 52-2).[1]

Reconstruction of the TMJ presents many unique problems as opposed to other joints because of the integral and complex roles this specific joint plays in establishing and maintaining proper form and function within the stomatognathic and upper respiratory and digestive systems. The TMJ not only provides a secondary growth center for the mandible in the prepubertal phases but also is of primary importance to the functions of mastication, speech, airway support, and deglutition in both child and adulthood. At present, there are three accepted management modalities available to

TABLE 52-1. **Indications for Total Joint Replacement**

Developmental Disorders
Condylar agenesis
Condylar hyperplasia
Condylar hypoplasia
Neoplasia
Benign
Osteoma
Chondroma
Osteochondroma
Malignant
Osteosarcoma
Chondrosarcoma
Arthritic Disease
High inflammatory
Low inflammatory
Trauma
Fracture
Intracapsular
Extracapsular
Multiple Prior Surgical Procedures
Ankylosis
Fibrous ankylosis
Bony ankylosis



FIGURE 52-1. Developmental anomaly (cystic hygroma treated in a child with left facial anomalies with temporomandibular joint [TMJ] ankylosis). (Courtesy of Michael Bowler, Newcastle, New South Wales, Australia.)



FIGURE 52-2. Pathology stereolithography (SLA model) with osteoma of the left condyle.

reconstructive TMJ surgeons for total temporomandibular joint reconstruction (TJR) including autogenous bone grafting, alloplastic replacement, and transport distraction osteogenesis techniques. Bioengineered TMJ devices are currently being investigated and may provide a viable option for the future. This chapter addresses the management of end-stage TMJ disease considering all four of these treatment modalities, presents an evidence-based literature review for each, and provides a suggested algorithm based on those reviews from which the reconstructive surgeon may determine which alternative provides the most appropriate management option as a TMJ salvage procedure in order to provide acceptable facial form and joint function in each individual clinical case scenario.

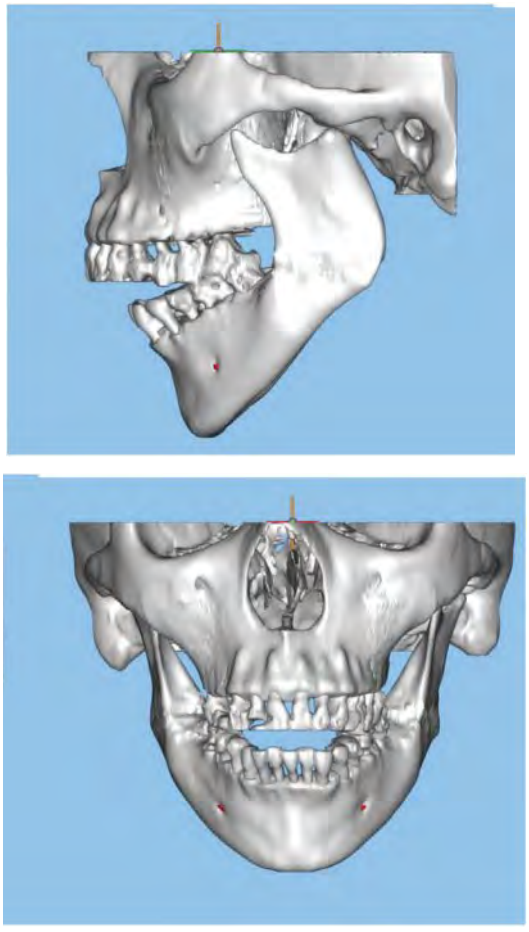


FIGURE 52-3. High inflammatory arthritic disease (rheumatoid arthritis).

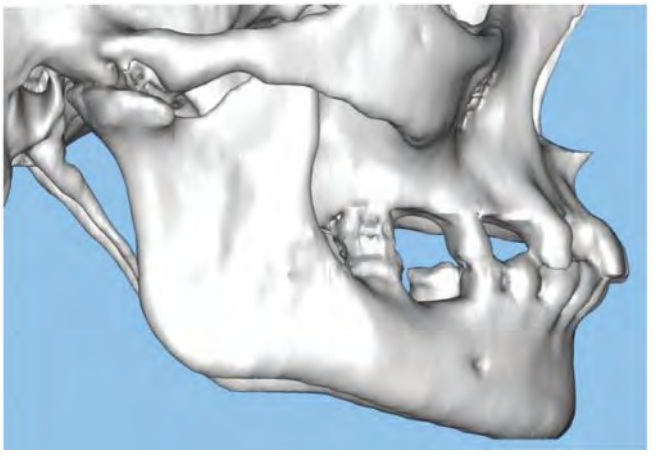


FIGURE 52-4. Low inflammatory arthritic disease (degenerative joint disease, osteoarthritis) with flattening of the condylar head, loss of joint space, and osteophyte formation.

AUTOGENOUS BONE GRAFTING

Classically, end-stage TMJ disorders have been managed with the use of autogenous bone and soft tissue grafting (Tables 52-3 and 52-4). The costochondral graft has been the



FIGURE 52-5. Progressive loss of condylar architecture after placement of a fossa-eminence device in a patient with degenerative joint disease.

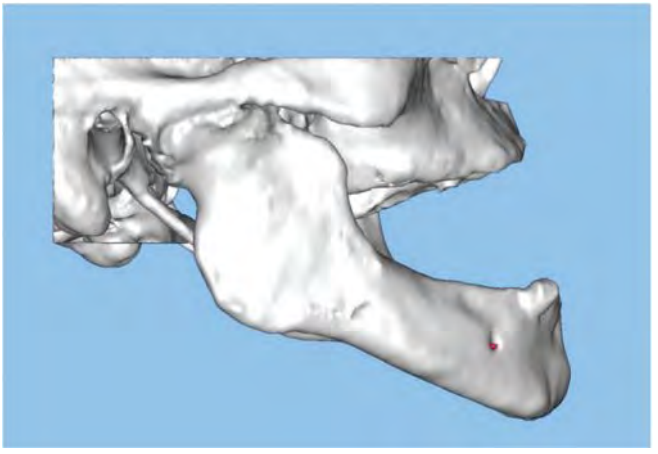


FIGURE 52-6. Ankylosis after trauma and failed gap arthroplasty with a temporalis myofascial flap.

TABLE 52-2. **Goals of Temporomandibular Joint Reconstruction**

Improvement in mandibular function and form
Reduction of further suffering and disability
Containment of excessive treatment and cost
Prevention of further patient morbidity

most frequently recommended autogenous bone for the reconstruction of the TMJ owing to its ease of adaptation to the recipient site, its gross anatomic similarity to the mandibular condyle, the reported low morbidity rate at the donor site, and its "growth potential"[2–7] (Figure 52-7). In theory, costochondral autogenous grafting will "grow with the patient"; however, often this so-called growth potential has

TABLE 52-3. **Autogenous Bone Grafting Total Joint Replacement Options**

Costochondral (rib) graft
Sternoclavicular joint
Calvarium
Iliac crest
Fibula
Second, fourth, or fifth metatarsophalangeal joint

TABLE 52-4. **History of Autogenous Total Joint Replacement**

1860—Temporalis flap (Verneuil)
1914—Autogenous fat (Murphy)
1953—Sternoclavicular joint (Steinhardt)
1957—Dermis (Georgiade)
1967—Metatarsophalangeal joint (Glahn)
1973—Auricular cartilage (Perko)
1975—Fascia lata (Narang)
1981—Costochondral graft (Ware & Brown)

been shown to be unpredictable or may result in ankylosis due to graft and/or fixation failure; or the poor results may be related to the expected noncompliance of the young patient with essential postreconstruction physical therapy.[2,8–21] Several studies have questioned the necessity for using a cartilaginous graft to restore and maintain mandibular growth.[22,23] Long-term reports of mandibular growth in children whose TMJs were reconstructed with costochondral grafts revealed that excessive growth on the treated side occurred in 54% of the 72 cases examined, and equal growth with the opposite side occurred in only 38% of the cases.[20,24–28] Further, Peltomäki and coworkers,[29–31] in an investigation of mandibular growth after costochondral grafting, supported previous

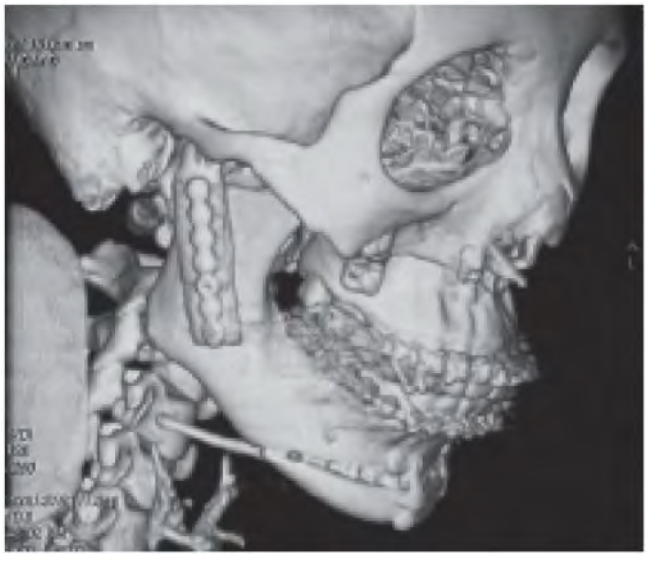


FIGURE 52-7. Costochondral graft fixated to the right mandible with a bone plate and screws.

experiments with regards to the inability of the graft to adapt to the growth velocity of the new environment. Cole and colleagues[32] recommend the use of a composite costochondral–iliac crest technique to resolve the issue of the flexibility of growing patient ribs. They states that "although graft instability and resorption seems to affect adults much more frequently, we believe the limited, sometimes overly malleable costochondral material harvested in pediatric cases leads to an unacceptably high rate of failure." The authors conclude, "in the adult population where costochondral grafts for the TMJ are decidedly unpredictable, this modification may also be advantageous." Peltomäki and coworkers[29] studied the growth of the mandible in the marmoset animal model after costochondral grafting with emphasis on the required amount of cartilage to be included in the graft. These investigators found that depending on the amount of cartilage in the unilateral costochondral graft, the mandible is shifted toward the unoperated side. This occurs during the entire period of growth, which indicated to them a strong hormonal influence. These authors concluded that these results supported previous experiments with regard to adaptability[2,3,30,31] in that costochondral grafts do not seem to adapt to the growth velocity of the new environment. Poswillo[3] had reported this adaptability in young monkeys; however, the animals used in his experiments had essentially ceased their craniofacial growth. Ellis and associates[22] compared the use of sternoclavicular grafts with costochondral grafts in the monkey and concluded that these grafts behaved similarly with regard to growth/overgrowth, suggesting that in growing individuals, maintenance of biomechanical architecture and reasonably normal function may be more important than the nature of the autogenous graft used for condylar replacement. Further, these investigators recommended retaining as little cartilage as possible to prevent overgrowth when replacing the mandibular condyle with a costochondral graft in growing individuals. Although contour overgrowth with deviation of the chin to the contralateral side was reported as a significant aesthetic problem, Perrott and coworkers[25] state that they documented contour overgrowth of the articulating surface of only "a small group" of growing and nongrowing patients. Despite the fact that these patients did not develop malocclusions, they developed decreased mandibular range of motion, as has been reported by other investigators.[20,21] MacIntosh[7] states that the chief disadvantages of costochondral grafting are their unpredictable growth, particularly in childhood and adolescent application, and the potential for reankylosis when used to treat cases of ankylosis. Saeed and Kent[33] retrospectively reviewed 76 costochondral grafts (57 patients) after a mean of 53 months (range 24–161 mo). They reported that in patients with no previous TMJ surgery, "arthritic disease" or congenital deformity, the costochondral graft performed well. However, they concluded that a preoperative diagnosis of ankylosis was associated with a high complication rate, suggesting caution in such patients using the autogenous costochondral graft.

With increasing patient age, adult ribs become mainly cortical bone and, therefore, contain very little cancellous bone. Consequently, when transplanted to the mandible, ribs are free grafts that rely on the ability of the local tissues to provide the optimal conditions for revascularization and, ultimately, the graft's survival and function. As with any bone grafting technique, it is important not only that the receptor site support revascularization and promote osteogenesis but also that stable fixation is provided over this period to allow incorporation of the donor bone into the host site. The vascularity of the recipient site will be adversely affected by the scar tissue that invariably develops from previous surgery, thus compromising the vascularity and ultimate success of the autogenous costochondral free graft. It has been reported that capillaries can penetrate a maximum thickness of 180 to 220 μm of tissue, whereas scar tissue surrounding previously operated bone averages 440 μm in thickness. This may account for the clinical observation that free autogenous tissue grafts, such as cartilage, costochondral, and sternoclavicular grafts, often fail in cases of multiply operated patients or those with extreme anatomic architectural discrepancies resulting from pathology.[34] Reitzik[35] reported that in an analogous situation to autogenous costochondral grafting, cortex-to-cortex healing after vertical ramus osteotomy in monkeys requires 20 weeks and probably 25 weeks in humans. Typically, in patients reconstructed with costochondral grafts, maxillomandibular fixation is maintained for only 4 to 6 weeks in order to return the mandible to function and prevent ankylosis. Despite screw and plate fixation, micromotion at the donor-host interface in such cases will invariably occur with early mandibular function resulting in shear movements of the graft, leading to poor vascularization, nonunion, and/or potential failure.[36] Matsuura and colleagues[37] reported a high incidence of failure and ankylosis of autogenous costochondral grafts in sheep after condylectomy if the jaws were only partially immobilized. Further, Whitaker and associates[38–40] reported the incidence of pneumothorax in 149 rib graft harvest patients as 5.3% and in the next 151 such patients as 18%. They reported pneumothorax in 20% to 30% of the 793 patients they studied.[40] James and Irvine[41] reported an incidence of postoperative donor site chest wound infection after rib harvest of 14.6%.

TRANSPORT DISTRACTION OSTEOGENESIS

The use of transport distraction osteogenesis, which induces new bone formation along the vector of movement without the use of a bone graft, has been employed as a reconstruction option for a variety of mandibular defects, including condylar reconstruction.[42–44] The techniques described include a vertical ramus-type osteotomy of the mandible after correction of the indication for TJR (e.g., gap arthroplasty for ankylosis). After the ramus osteotomy (or corticotomy), a distraction device is placed with a vertical vector to move the proximal segment of the mandible toward the glenoid fossa (Figure 52-8). This form of reconstruction

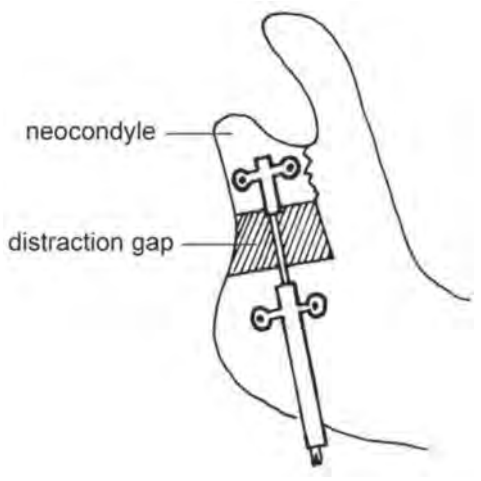


FIGURE 52-8. Transport distraction osteogenesis in an animal study. (Adapted from Zhu S, et al. Biomechanical properties of the condyle created by osteodistraction. J Dent Res 2008;87:490–494).

takes advantage of the fibrocartilagenous cap that forms on the advancing front of the distracted bone heading toward the fossa, thereby creating a "cartilage cap" on the newly formed condyle, which may function more normally than a pseudoarthrosis (Figure 52-9). Stucki-McCormick and coworkers[45,46] reported the application of transport distraction osteogenesis to reconstruct the mandibular condyle in two patients as well as in the animal model. Cavaliere and Buchman[47] reported using similar methods to manage hemifacial microsomia patients with 1- and 2-year follow-up, respectively. The long-term growth potential for the use of this modality in the reconstruction of prepubescent cases also has yet to be determined. In 1992, McCarthy and colleagues[42] developed the technique for mandibular elongation in patients with hemifacial microsomia and Nager's syndrome. Since then, distraction osteogenesis has been used for craniofacial lengthening,[48–50] bifocal distraction for repair of mandibular defects,[51,52] mandibular widening,[53] and increasing alveolar height.[54] Pensler and associates in 1995[55] reported a case of a

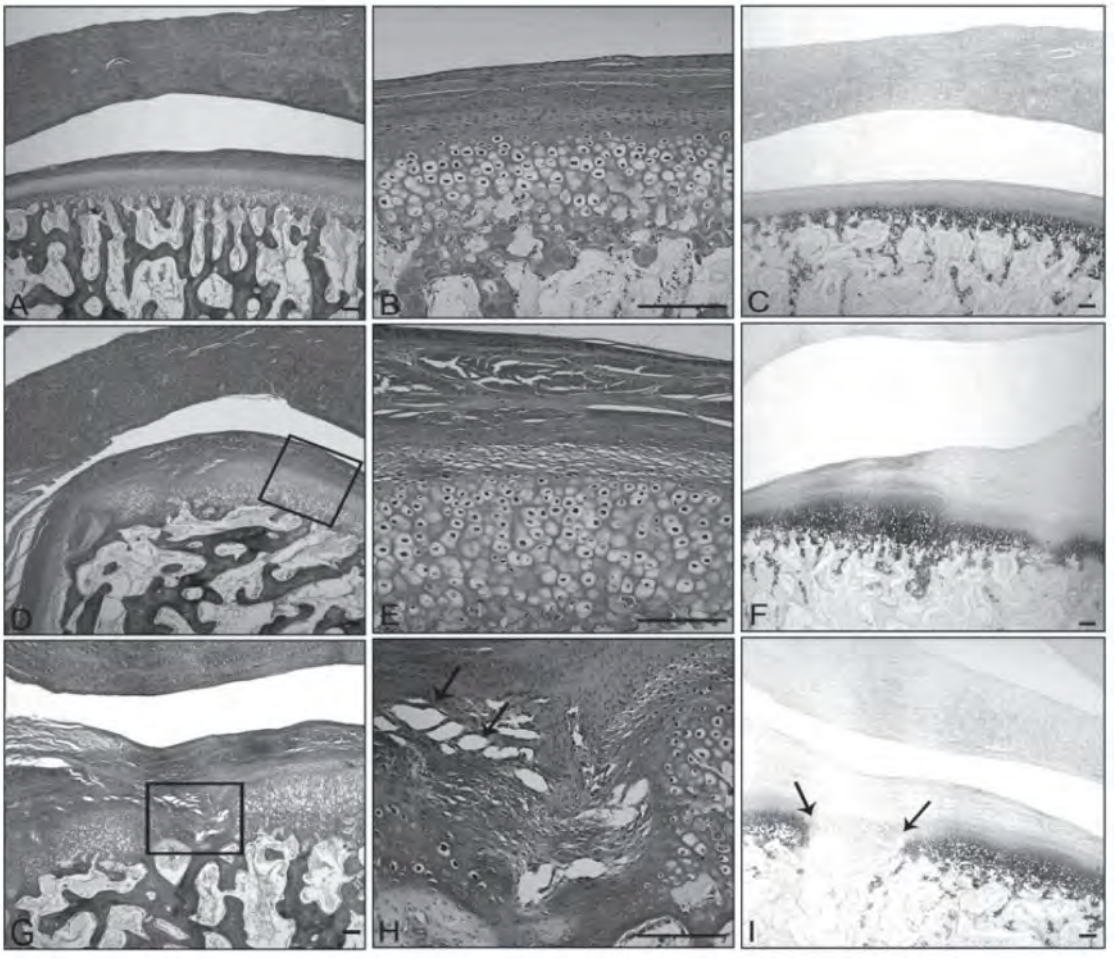


FIGURE 52-9. Histologic features of normal condyles and the neocondyles (**left** and **middle,** Masson trichrome staining; **right,** toluidine blue staining). **A–C,** A normal condyle showing the normal structures. **D** and **G,** The newly formed fibrocartilage covering the upper surface of the neocondyle. **E** and **H,** High-power views of the boxed areas in **D** and **G.** Lacunae can be observed *(arrow).* **F** and **I,** Uneven distribution and complete discontinuity *(between two arrows)* of the cartilaginous matrix were detected by toluidine blue staining. *Scale bar* represents 200 μm. (**A–I,** Adapted from Zhu S, et al. Biomechanical properties of the condyle created by osteodistraction. J Dent Res 2008;87:490–494.)

5½-year-old with micrognathia and TMJ ankylosis treated with distraction osteogenesis. Diner and coworkers in 1997[56] reported on a series of nine children with mandibular hypoplasia caused by TMJ ankylosis treated with distraction osteogenesis. Also in 1997, Acero-Sanz[57] and colleagues reported the use of distraction osteogenesis in the management of a 2-year-old with bilateral TMJ hypoplasia. In 1999, Dean and Alamillos[58] and Papageorge and Apostolidis[59] reported the management of TMJ ankylosis in growing patients utilizing simultaneous distraction osteogenesis and TMJ arthroplasty. These studies stress that experience with this technique in the management of childhood ankylosis and long-term follow-up are important, but according to Moss and Rankow's functional matrix theory,[60] with the association of distraction osteogenesis and TMJ arthroplasty, the conditions for normal mandibular growth should develop properly. In 2000, Yonehara and associates[61] reported a case of an 11-year-old boy with bilateral TMJ ankylosis and resultant severe micrognathia managed with bilateral distraction osteogenesis. They describe the greatest advantage of distraction osteogenesis as the gradual distraction of the soft tissues as well as the hard tissues associated with the mandible. Yoon and Kim in 2002[62] reported good results after more than 2 years follow-up after the use of intraoral distraction osteogenesis in a growing and an adult patient with mandibular asymmetry and unilateral TMJ bony ankylosis. Molina and Ortiz-Monteserio[63] and Ortiz Monasterio and coworkers[64] state that many cases of hemifacial microsomia treated in infancy will require a second course of distraction osteogenesis or formal orthognathic surgery later in life to maintain the position of the mandible. However, in cases associated with mandibular hypoplasia as the result of TMJ ankylosis, the etiology is traumatic, rather than developmental, in nature; therefore, correction of function of the associated bone and soft tissues should result in normal mandibular growth potential based on the functional matrix theory.[60] A further demonstration of Moss and Rankow's theory may be inferred from a report by Nitzan and colleagues.[65] They report on the management of four patients (three female, one male; ages 9–48 yr) with type III TMJ ankylosis in whom they freed the ankylosis and retained the displaced disk and condyle complex only, followed by aggressive active physiotherapy to restore mandibular function. After 15- to 60-month follow-up, all patients had significantly improved mandibular function and the 9-year-old with preoperative facial asymmetry demonstrated complete symmetrical growth 5 years after surgery. In the other growing patients, 15 months postoperative, improved facial symmetry was reported. Despite the short-term promising clinical results, the histomorphologic changes in the associated joint structures (i.e., cartilage, disk, and masticatory musculature) have not been elucidated completely. Experimental studies with rabbits and monkeys addressing this issue have appeared in the literature.[66,67] Based on their animal experiments, Zhu and associates[68] list several potential advantages of mandibular condyle reconstruction using transport distraction over conventional autogenous bone grafting: (1) avoidance of donor site morbidity; (2) the rigid distraction device allows for immediate postoperative physical therapy, which allows for the formation of a pseudoarthrosis and prevents ankylosis; and (3) the risk of bone graft necrosis and resorption is greatly reduced compared with nonvascularized free-bone grafts (i.e., costochondral) because the transported bone in this technique retains its internal muscle and periosteal blood supply. However, these experimental studies were carried out in animals with normal TMJs and extrapolation of these data to patients with compromised TMJ anatomy due to pathology or multiple prior surgeries remains a question and concern when utilizing this technique.

BIOENGINEERED TISSUE FOR TJR

Mandibular condyle tissue engineering has been proposed recently to address the current limitations of both autogenous and alloplastic reconstruction options.[69] A tissue engineering strategy would incorporate a biodegradable scaffold delivering biologic substances such as cells, genes, and/or proteins to regenerate the bone/cartilage tissues of the mandibular condyle. Tissue engineering could be applied to regenerate localized defects or, theoretically, the entire articulating condylar surface. Implementation could take one of three approaches: (1) ex vivo growth of tissue on the scaffold/biologic composite in a bioreactor before implantation on the mandible; (2) implantation of the scaffold/biologic composite at secondary surgical site before implantation on the mandible; and (3) direct implantation at the mandible site.[69] Whereas this modality holds the potential to revolutionize the TMJ reconstruction in the future, many biologic, physiologic, mechanical, regulatory, and ethical issues must be tested and addressed before it becomes a clinical reality.

ALLOPLASTIC TJR

In order to avoid the donor site morbidity of autogenous bone grafting, alloplastic TJR has become an acceptable alternative to autogenous grafting (Table 52-5 and Table 52-6). In 1960, Sir John Charnley, the father of orthopedic low-friction alloplastic joint replacement, reported the use of a total alloplastic prosthetic hip reconstruction system utilizing a titanium-backed ultrahigh-molecular-weight polyethylene acetabular cup articulating with a chromium-cobalt femoral head attached to a titanium femoral shaft component that was cemented into place with polymethylmethacrylate (PMMA).[70] Modifications of this device with the same materials, now without the cement, have become the gold standard not only for hip but also knee and shoulder replacement devices in orthopedic surgery.[71] The modern practice of reconstructive orthopedic surgery would be unthinkable without the use of alloplastic joint replacements. All implanted alloplastic devices (dental implants, orthopedic, and TMJ prostheses) depend on the principle of osseointegration of

TABLE 52-5. **Comparison of Autogenous and Alloplastic Total Joint Replacement**

Autogenous	Alloplastic
Biocompatible	Biocompatible
Donor site	No donor site
Ankylosis potential	Low ankylosis (heterotopic bone)
Longevity	May require revision/replacement
Young patient with growth potential	Adult patient with growth completion
Lack of growth or overgrowth	Mechanical wear

the components for their ultimate stability and longevity. *Osseointegration* implies the direct incorporation of the implant by the bone without the preliminary phase of fibrous tissue ingrowth. The requirements for osseointegration are essentially the same as for primary fracture healing—the transmission of forces from the implant to the bone and vice versa must occur without relative motion or without intermittent loading.[72] Therefore, just as with a dental implant, the most important principle in total joint alloplastic reconstruction is primary stability of the device components from the moment of their implantation. In orthopedic surgery, total joint alloplastic devices can be initially stabilized by press fitting or cementation into the cancellous shaft of the host long bone. Unfortunately, in the case of the TMJ, the anatomy of the mandible and the nonlinear architecture of temporal glenoid fossa do not provide such options. Therefore, screw fixation of both the fossa and the ramus/condyle components of all TMJ alloplastic devices must be utilized for initial fixation and stabilization. Compounding the stability problems in the TMJ region is the fact that most patients presenting with indications for total TMJ alloplastic reconstruction have fossa and/or ramus anatomy distortion as a result of end-stage disease. There may be local and/or systemic relative contraindications to alloplastic TJR (Table 52-7).

TABLE 52-6. **Advantages and Disadvantages of Alloplastic Total Joint Replacement**

Advantages
Rigid fixation allows immediate postoperative physical therapy.
No donor site morbidity.
Decreased surgical time (vs. autogenous bone).
Mimics normal anatomy (CAD/CAM or patient-fitted TJR).
Maintenance of stable occlusion (no remodeling).
Can correct apertognathia, retrognathia without potential for relapse.
Disadvantages
Device cost.
Material wear and/or failure (screw loosening, fracture).
Possible heterotopic bone formation (ankylosis).
Possible need for revision and/or replacement.
Size and design limitations (stock devices).
No growth potential.

CAD/CAM = computer-aided_design/computer-aided_manufacturing; TJR = total joint replacement.

TABLE 52-7. **Relative Contraindications to Alloplastic Total Joint Replacement**

Systemic
Incomplete facial bone growth (child)
Advanced medical-surgical risk
Uncontrolled systemic disease
Psychological instability
Local, Regional
Insufficient hard and soft tissues to support the implants
Active or recent infection (local or systemic)
Allergy to prosthetic materials (rare)
Uncontrolled parafunctional oral habit

History of Alloplastic TMJ Reconstruction

Alfred Stille[73] said "Medicine, like all knowledge, has a past as well as a present and a future, and . . . in that past is the soil out of which improvement must grow." Therefore, a look back is important to the understanding of the use of alloplastic materials in joint reconstruction. The citations made in this section are intended to show that many surgeons in the past found a need for alloplastic TMJ reconstruction in particularly difficult clinical situations. Further, it provides an archive of the materials and methods used in these cases (Table 52-8). Typically, the materials used in these devices mirrored their introduction into industry and medicine. It must be noted that in many instances, the reports of the use of these materials were of single cases and their follow-up was typically 1 year or less with the only criterion for success being that the patient could open the mouth.

Before 1980, alloplastic joint replacement or resurfacing (hemiarthroplasty/partial joint replacement) was performed mainly in cases of ankylosis, after ablative surgery, trauma, or in severe joint disease. After the mid- to late 1980s, partial and total TMJ devices were also being used to manage failed nonsurgical and surgical TMJ patients. In 1840, John Murray Carnochan[74] was credited with the idea of interposing material between the surfaces of a diseased joint. He reported an attempt to mobilize a patient's ankylosed TMJ by placing a small block of wood between the raw bony surfaces of the residual mandible after creating a gap at the neck of the condyle. In 1890, a German surgeon, Gluck[75] reported total joint arthroplasties using ivory prosthetic TMJ and hip joints that he stabilized with cement made of colophony, pumice, and gypsum. Risdon in 1933[76] reported the treatment of an ankylosis of the TMJ by interposing gold foil between the bony surfaces after a gap was created. Eggers in 1946[77] and Goodsell in 1947[78] reported the use of tantalum foil in cases of TMJ ankylosis. In 1951, Castigliano and Gross[79] and Kleitsch[80] resurfaced the bone in TMJ ankylosis cases with vitallium. In 1952, Smith and Robinson[81] reported hemiarthroplasty for ankylosis using stainless steel. In 1955, Ueno and coworkers[82] reported experimental and clinical results with zirconium in TMJ ankylosis. In 1960, Henry[83] described replacement of an ankylosed TMJ with prosthesis

TABLE 52-8. **History of Alloplastic Total Joint Replacement**

1840—Wood (Carnochan)
1890—Ivory (Gluck)
1934—Gold foil (Risdon)
1946—Tantalum foil over condyle (Eggers)
1947—Tantalum foil (Goodsell)
1951—Vitallium (Castigliano & Kleitsch)
1952—Stainless steel (Smith)
1955—Zirconium (Ueno)
1957—Stainless steel fossa (Robinson)
1963—Vitallium fossa (Christensen)
1964—Tantalum foil/PT (Hellinger)
1968—Silastic sponge fossa (Robinson)
1970—Mesh/PMMA (Corgill & Hahn)
1971—Vitallium/Silastic fossa (Morgan)
1972—Gold custom (Taurus)
1974—Proplast-coated ticonium (Hinds)
1974—PMMA (Wallace)
1974—Cr-Co fossa and condyle with PMMA head (Kiehn)
1974—Vitallium condyle, Proplast I fossa (Hinds & Kent)
1976—Stainless steel ramus and condyle reconstruction plate (Spiessl)
1976—Vitallium-acrylic condylar prosthesis (Robinson)
1977—Corgill-Hahn prosthesis (Seymour)
1977—Charnley intramedullary orthopedic pin, PMMA cement (Silver)
1978—Cr-Co fossa and condyle with PMMA head (Kummona)
1982—Three-dimensional THORP design (Raveh)
1983—Vitek, Kent I fossa, Cr-Co ramus and condyle (Kent)
1984—Hip joint prosthetic design (Flot & Chassagne)
1984—Delrin condyle with Cr-Co ramus, Cr-Co/Silastic fossa (House & Morgan)
1985—Cr-Co ramus and fossa, PMMA condyle, PMMA cement (Sonnenburg)
1986—VK I (20% success) and VK II (80% success) (Kent)
1987—Delrin (Boyne)
1988/95—CAD/CAM (Techmedica/TMJ Concepts), CP-Ti backed UHMWPE custom fossa, Cr-Co-Mb condyle, 6Al/4V Ti custom ramus (Mercuri/Wolford)
1990—Ceramics (Szabo)
1990—CP-Ti backed UHMWPE fossa, Cr-Co condyle, 6Al/4V Ti ramus (Sonnenburg)
1993—Titanium/Cr-Co-Mo (Falkenstrom)
1994—Osteomed, UHMWPE fossa, PMMA cement, Cr-Co condyle, 6Al/4V Ti ramus (McBride)
1995—Cr-Co fossa and ramus, PMMA condyle (stock) (Chase)
1999—TMJ Concepts (Mercuri)
2000—Groningen device (van Loon & DeBont)
2006—Biomet/Lorenz device (Quinn)

CAD/CAM = computer-aided_design/computer-aided_manufacturing; PMMA = polymethylmethacrylate; PT = ; THORP = titanium-coated hollow screw and reconstruction plate; TMJ = temporomandibular joint; UHMWPE = ultrahigh-molecular-weight polyethylene.

and Robinson[84] reported correction of a TMJ ankylosis by creating an artificial stainless steel fossa. In 1963,[85] 1964,[86] and 1970,[87] Christensen reported resurfacing of the glenoid fossa with a thin vitallium fossa-eminence prosthesis in cases of TMJ ankylosis. He added a ramus-condyle component with a PMMA head and a vitallium ramal component to create a total TMJ prosthesis. Chase and colleagues in 1995[88] reported on the results of the use of both devices. In 1996, a chromium-cobalt condylar head was offered as an option to the PMMA. Garrett and associates[89] reported the use of a custom modification of the standard Christensen total alloplastic TMJ prosthesis for use in multiply operated patients. Small and coworkers[90] experimented with the use of Teflon and Silastic in mandibular reconstruction and in 1964 reported that Teflon seemed more adaptable to restoration of large mandibular resections whereas Silastic seemed better suited for replacement of the condyle. In 1968, Robinson[91] reported on the use of Silastic in a case of TMJ ankylosis as an interpositional material; other reports of the use of Silastic for partial TMJ reconstruction soon followed.[92–98] Hellinger, also in 1964,[99] reported the use of tantalum foil in such cases and was the first to stress the importance of physical therapy in the rehabilitation of these patients. Hahn and Corgill in 1970[100] first reported the use of ramus-condyle hemiarthroplasty prosthesis for the treatment of ankylosis. The condylar component was fashioned from dental PMMA. The ramus component was stainless steel wire mesh. In 1971,[101] 1975,[102] and 1977,[103] Morgan and colleagues presented another form of fossa resurfacing device that consisted of a vitallium eminence prosthesis and a Silastic fossa component. They added a variation of the Hahn and Corgill ramus-condyle component with a polyoxymethylene (Delrin) condyle to make a total joint prosthesis. In 1984, House and associates[104] reported the results of the use of the Morgan devices. In 1992, Morgan[105] reviewed the development of alloplastic materials for TMJ prostheses with an emphasis on his prostheses described previously. In 1972, Taurus and coworkers[106] first reported the use of a custom-made cast gold ramus-condyle hemiarticulation in reconstruction of a TMJ.

Hinds and colleagues in 1972[107] reported the use of a Proplast-Teflon–coated vitallium ramus-condyle component for hemiarticulation reconstruction of the TMJ. Kent and associates in 1983[108–110] reported the use of a Dacron/Proplast-Teflon (VK-I) and later still a Dacron/Proplast-Teflon/ultrahigh-molecular-weight polyethylene (UHMWPE) (VK-II) fossa. The ramus-condyle and fossa components were then reported as used as a total alloplastic TMJ reconstruction prosthetic device. Kent and associates in 1993[111] long-term follow-ups.

In 1974, Kiehn and coworkers[112] applied the principles used in total hip reconstruction to the TMJ, utilizing a vitallium mandibular fossa plate reinforced on its temporal side with PMMA and a vitallium-modified Cargill-Hahn ramus/condyle prosthesis. In 1978, the same authors[113] presented a follow-up of 27 patients who had undergone total TMJ reconstruction using the same prostheses previously described for the treatment of ankylosis, arthritis, neoplasm, infection, or refractory pain. They reported 23 successful cases over a 1- to 3-year period of follow-up, success being defined by the authors as the ability to open the mouth to eat without pain.[113–117] In 1978, Kammoona[118] essentially reproduced

the work of Kiehn and coworkers in 6 monkeys. After 9 to 10 months of function, half failed due to condylar component failure. Microscopically, there was a minimum of inflammatory cells, no evidence of infection, and well-organized granulation tissue and collagen fibers with fibrous tissue beneath the cement and the condylar component. Reaction to the bone cement appeared good. Collagen fibers ran parallel to the implant. The bone in the surrounding area was vital and healthy, and in some areas, the fibrous tissue had turned to bone. Microradiographs demonstrated tolerance of the metallic joint and bone cement, with incorporation by healthy granulation tissue, collagen fibers, and new bone to such an extent as to justify complete biologic acceptance of the implant by the natural tissue. This was the second report of animal studies with alloplastic TMJ devices after Ueno and coworkers.[82] In 1985[119] and again in 1990,[120] Sonnenburg and Sonnenburg reported the use of a total TMJ device that consisted of a chromium-cobalt-molybdenum ramus-condyle component that articulated against an UHMWPE fossa. This mimicked the materials and geometry used in the design of alloplastic joint prostheses by orthopedic surgeons.

From 1990 to 1995, others reported on the development and use of alloplastic partial and total TMJ reconstruction prostheses. The indications for their use ranged from ankylosis to reconstruction after ablative surgery for disease and/or trauma to the multiply operated failed TMJ patient.[121–131] Mercuri and colleagues in 1995[132] reported on preliminary results with the use of a patient-fitted computer-aided_design/computer-aided_manufacturing (CAD/CAM) total alloplastic TMJ reconstruction prosthesis in a prospective limited clinical study. The ramus component of this prosthesis was titanium, and the condylar component chromium-cobalt-molybdenum and the fossa component titanium were backed with UHMWPE. This was followed by further long-term results indicating the safety and effectiveness of TJR for the TMJ using this device.[133,134]

At present, there are three U.S. Food and Drug administration (FDA)–sanctioned TMJ prosthetic systems available for implantation, the pre-1976 Amendment Christensen stock as well as the Christensen/Garrett custom devices (TMJ Implant, Inc., Golden, CO) (Figure 52-10); the 1999 FDA-approved



FIGURE 52-10. TMJ, Inc. (Golden, CO) stock and custom TMJ total joint replacement (TJR) devices.



FIGURE 52-11. TMJ Concepts (Ventura, CA) patient-fitted TMJ TJR device.



FIGURE 52-12. Biomet-Microfixation (Jacksonville, FL) stock TMJ TJR device.

TMJ Concepts patient-fitted device (TMJ Concepts [previously Techmedica], Ventura, CA) (Figure 52-11); and the 2006 FDA-approved Biomet Microfixation total joint stock device (Jacksonville, FL) (Figure 52-12).

TMJ Implants, Inc., also manufactures a hemiarthroplasty fossa-eminence device, originally designed to manage TMJ ankylosis, that has been utilized as a disk substitute in the recent past. Hemiarthroplasty, a metallic-bearing surface articulating with normal articular cartilage, is frequently utilized in orthopedic surgery for fractures of the hip and shoulder in geriatric patients. The surgery can be quite successful in such cases in which functional demands are low; however, over time, the metallic component against the articular cartilage causes cartilage wear and may cause pain, requiring total joint replacement. For this reason, hemiarthroplasty is generally not performed in young patients or in patients with preexisting degenerative joint disease[135,136] (see Figure 52-5).

Although some surgeons have also advocated partial joint replacement utilizing only the condylar component, it is best to

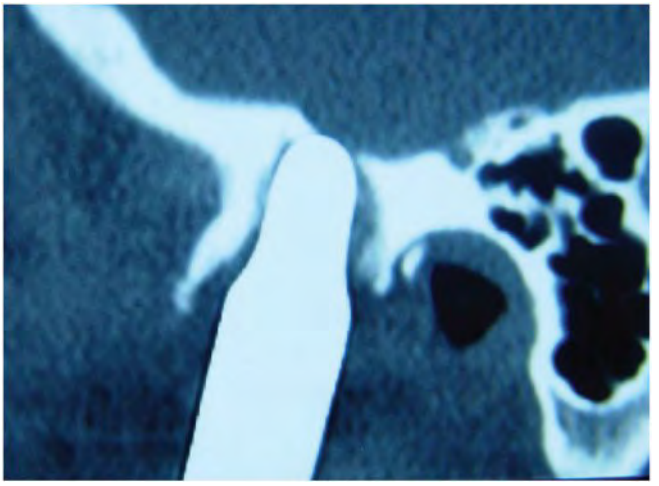


FIGURE 52-13. Erosion of the condyle from a hemiarthroplasty procedure into the middle cranial fossa and development of heterotopic bone.



FIGURE 52-14. Postoperative home physical therapy using a commercially available jaw exercising device.

perform a total joint replacement. Lindqvist and associates in 1992[127] and Westermark and coworkers in 2006[137] found that in cases of condylar element replacement alone, there was severe erosion of the fossa with significant heterotopic bone formation. (Figure 52-13). Stock or "off-the-shelf" joint replacement devices must be bent to fit or shimmed with bone and/or PMMA cement or the precious host bone must be recontoured to develop some reasonable host-to-bone interface. All such maneuvers can lead to component material fatigue or overload, promoting early failure with functional loading. More significantly, these manipulations can lead to "stock" component micromotion that will interfere with osseointegration. Micromotion leads to the formation of a fibrous connective tissue interface between the altered component and the host bone, resulting in early loosening of that component and potential early catastrophic or certain premature device failure in the future. In addition, there are patient-fitted or custom-made devices that are designed and manufactured for each specific anatomic situation, can conform to any unique anatomic situation, and require no alteration or supplementation to achieve initial implantation stability. Because the components interface precisely with the host bone, the screw fixation secures the components to the host bone, mitigating micromotion and maximizing the opportunity for osseointegration of the components and fixation screws.

In 2003, Wolford and colleagues[138] compared two of the available TMJ TJR systems in 45 patients, including TMJ Implants (23 patients) and TMJ Concepts (22 patients). The average number of previous surgeries was 3.9 for the TMJ Implants patients and 2.6 for the TMJ Concepts patients, and the average follow-up period was 2 to 3 years. It was determined that TMJ Concepts had statistically significant improved outcomes compared with the TMJ Implants group in terms of postsurgical maximum interincisal opening, pain, jaw function, and diet. It was noted that both groups showed good skeletal and occlusal stability over the follow-up period.

Returning the joint(s) and muscles of mastication to function as soon as possible postoperatively enhances healing and decreases the development of intra-articular scar tissue that will compromise mandibular range of motion.[139] One of the biggest advantages of alloplastic over autogenous TMJ replacement is the ability to start active physical therapy immediately postoperatively utilizing any of the commercially available jaw exercising devices (Figure 52-14).

When calculating cost of a procedure or device, it is important that not only is the cost of the device considered but also all factors associated with its placement, including overall treatment time and patient morbidity. The major advantages of alloplastic over autogenous TMJ reconstruction is that there is no need for a second surgical site with the potential of increasing morbidity and the expensive intraoperative and postoperative hospital time is greatly decreased.

Finally, the concern often raised that any alloplastic devices when utilized in the TMJ will result in the same scenario as Proplast-Teflon/silicone rubber are unfounded and unsubstantiated in a growing body of literature.[132–134,140–147]

In conclusion, this chapter discussed the management of end-stage TMJ disease employing the management options of autogenous tissue, alloplastic materials, osseodistraction, and bioengineered tissue. It also presented an evidence-based literature review for each of these modalities from which the reconstructive surgeon may determine which alternative provides the best management option for regaining and maintaining savaged TMJ function and facial form in the face of end-stage disease for each individual case. Table 52-9 provides a useful resource when considering clinical options for TJR based upon the specific indication for surgery. It should be kept in mind that, in general, TJR may be considered a "salvage" procedure for end-stage TMJ disease and that "success" may need to be qualified especially in the multiply operated patient with chronic facial pain. The quality of life in these patients remains poor and possible revision procedures may be necessary in the life-long management of this patient group.

TABLE 52-9. **Options for Total Joint Replacement Based upon End-Stage Disease**

	Bone Grafting	Distraction	Bioengineered	Alloplastic
Developmental	+++	++	+++	+*
Neoplastic	+++	+	++	+++
Traumatic	+++	++	+++	+++
Arthritic	+†	+	+	+++
Multiple Failed Surgery	+	+	+	+++
Ankylosis	+	++	+	+++

*Mercuri LG, Swift JQ. Considerations for the use of alloplastic temporomandibular joint replacement in the growing patient. J Oral Maxillofac Surg 2009;67:1979–1990.
†Juvenile rheumatoid arthritis: Svensson B. On costochondral grafts replacing mandibular condyles in juvenile chronic arthritis: a clinical, histologic and experimental study. Goteborg, Sweden: Orebro, 2000.

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SECTION 7

Orthognathic Surgery

53

CHAPTER

Craniofacial Growth and Development

Peter M. Spalding, DDS, MS, MS

The goal of this chapter is to provide a review of the current understanding of prenatal and postnatal craniofacial growth and its relevance for clinical orthognathic and craniofacial treatment. Although there clearly is awareness of the importance of genetic and environmental influences on craniofacial growth and development, the control and precise biologic mechanisms are not well understood and continue to be fertile areas of investigation. This chapter reviews human morphogenesis, prenatal and postnatal growth and development, the factors that influence these phases of growth and development, and the orthopedic and orthodontic clinical considerations that will determine whether surgical intervention will be necessary to achieve optimum cosmetic and functional craniofacial treatment outcomes.

PRENATAL CRANIOFACIAL DEVELOPMENT

Human prenatal development can be divided simply into the embryonic period, from fertilization through the 8th week of development, and the fetal period, continuing from the 9th to the 40th week at birth. The embryonic period is characterized by new tissue differentiation and organogenesis, whereas the fetal period is distinguished by growth and expansion of the basic structures already formed.

During the first few days after the formation of the single-cell zygote at conception, four mitotic divisions occur to form the 16-cell morula. After entering the uterus, the morula develops into a 100-cell blastocyst consisting of an outer (trophoblast) and inner (embryoblast) cell mass. The trophoblast further differentiates to form the placenta and other peripheral embryonic structures, whereas the embryoblast differentiates into the future embryo. At the end of the first week, the blastocyst adheres to the uterine endometrium to begin implantation. During the second week, the embryoblast forms a bilaminar disk composed of two germ layers: the ectoderm, forming the amniotic cavity floor; and the endoderm, lying beneath and forming the yolk sac floor. Later, the ectoderm will form a variety of epidermal structures including dental enamel, oral mucosa, and nasal epithelia. The endoderm will later form the pharyngeal epithelium. By the end of the second week, the endoderm develops a thickened area called the *prechordal plate*, located at the cranial end of the bilaminar disk, that prefaces the development of the head (Figure 53-1).

Embryonic Period

Germ Layer Formation

Craniofacial embryogenesis begins during the third week of gestation, when gastrulation and neurulation occur. *Gastrulation* is the process whereby the bilaminar disk is converted into a trilaminar one with the appearance of the third germ layer, the mesoderm, forming between the other two from ectodermal cell proliferation and differentiation in the caudal area of the disk. The prominence created from this proliferation forms a craniocaudal midline furrow termed the *primitive streak*. Cell proliferation and differentiation of the cranial end of the primitive streak form the notochord around which the axial skeleton will form.

Neural Tube Formation

Neurulation, occurring at the same time as gastrulation during the third week and continuing through the fourth week, is a process that results in the formation of the neural tube, the primordium of the central nervous system (CNS). Neurulation is characterized by development of the neural

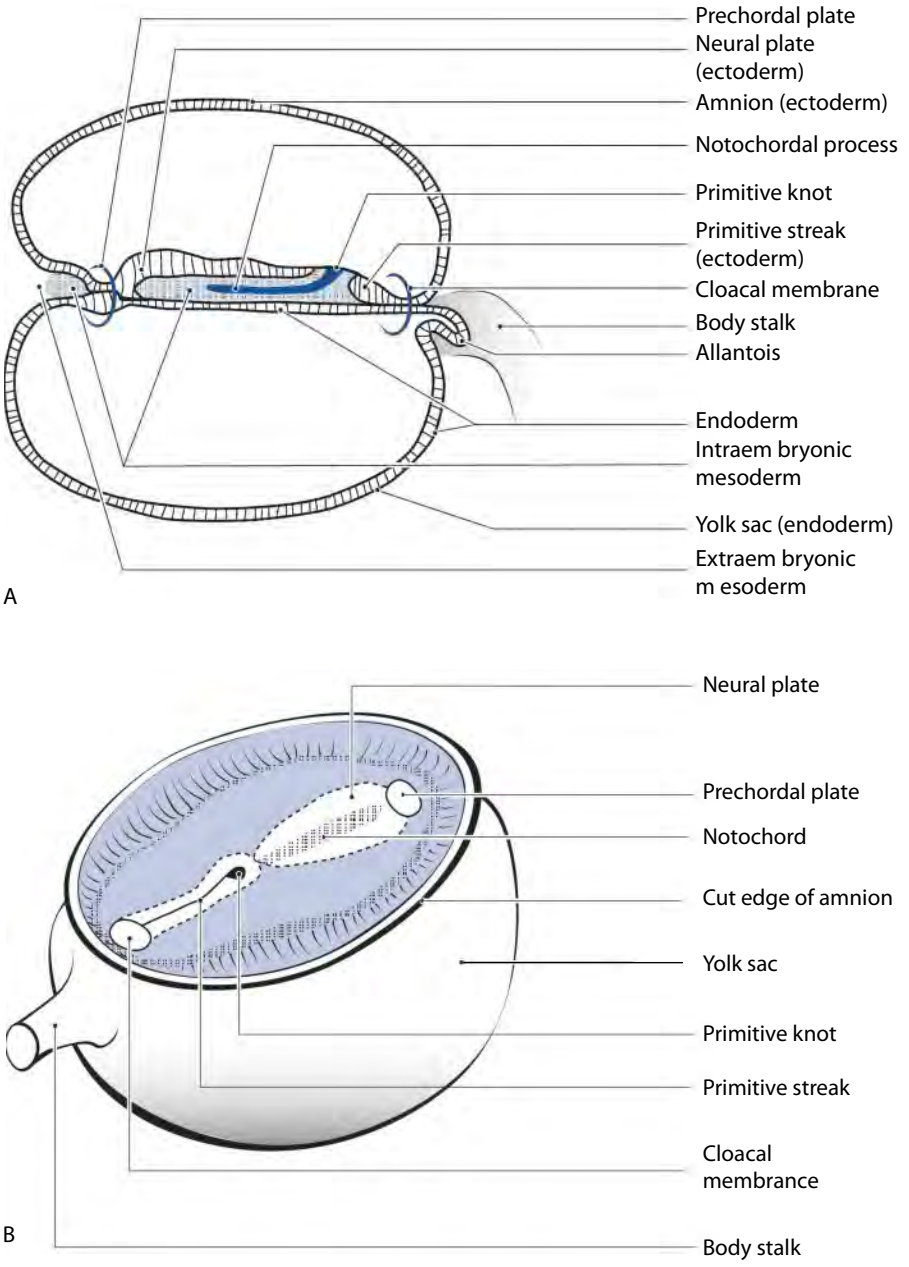


FIGURE 53-1. Embryo, 14 days old. **A,** Longitudinal section shows amnion *(top)* and yolk sac *(bottom)*. **B,** Dorsal surface view with the amnion cover removed, shows embryonic disk. Adapted from Sperber G. Craniofacial development. Hamilton (ON): BC Decker Inc; 2001. pp. 19 and 20.

plate from the ectoderm overlying the notochord. As the neural plate grows caudally toward the primitive streak, the lateral edges of the neural plate rise up to create neural folds, forming the neural groove between them. Mesoderm on either side of the groove develops into paired blocks of tissue called *somites* (ultimately 48 somite pairs will develop). In the fourth week, the neural folds begin to fuse at the midline in the central part of the embryo, at the level of the fourth to fifth somite, to form the neural tube (Figure 53-2). The neural tube continues to form toward the cranial and caudal ends, completing caudal formation by the time about 20 somite pairs are present. The anterior portion of the neural tube develops into the forebrain, midbrain, and hindbrain. After neural tube closure is complete on day 28, the two hemispheres of the brain begin development, increasing in size to eventually cover the roof of the brainstem. The otic, optic, and olfactory placodes develop in association with the forebrain neuroectoderm.

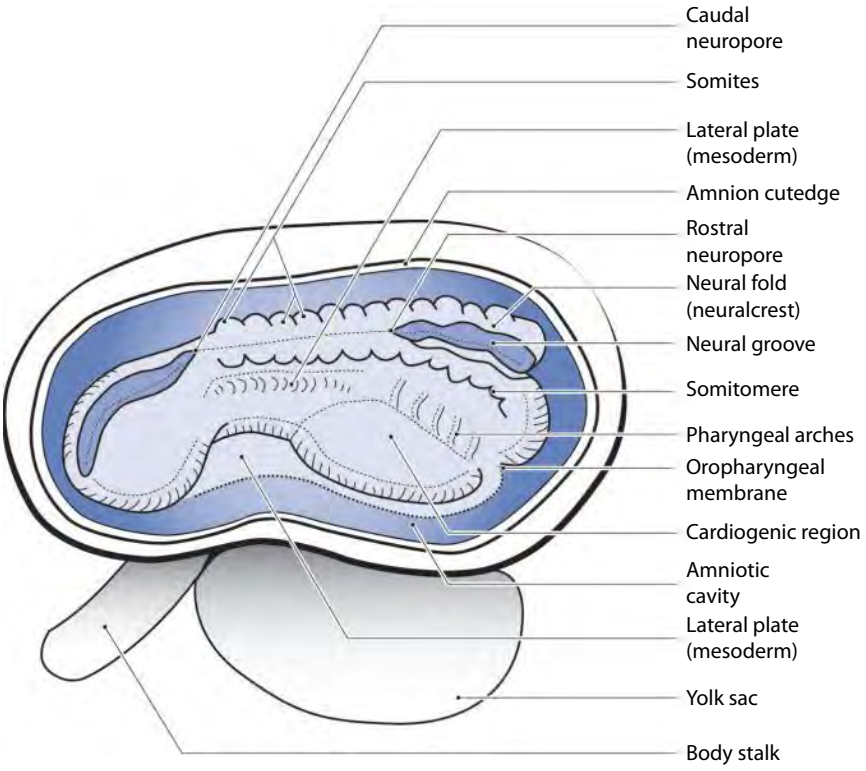


FIGURE 53-2. Longitudinal section of amnion in a 23-day-old embryo shows fusion of the neural folds and initial formation of somites. Adapted from Sperber G. Craniofacial development. Hamilton (ON): BC Decker Inc; 2001. p. 21.

The Cranial Neural Crest

The neural crest is a multipotential cell population derived from the lateral edges of the neural plate during the fourth week of embryonic life. It was first described in 1868 as a band of cells between the surface epithelium and the neural tube in chicken embryos that appears at the dorsolateral edge of the closing neural folds along nearly the entire length of vertebrate neuraxis.[1] This is the line usually termed the *neural plate border* where the surface ectodermal epithelium functionally splits into two areas, the neuroectoderm or neural plate, which forms the neural tube and completely invaginates into the embryonic body, and the body surface epithelium that becomes the epidermis. Cell fate tracing experiments have revealed that neural crest cells originate from the dorsal surface of the neural tube by delamination from the epithelium and migrate extensively in a proper sequence over extensive distances through the embryo, developing into a wide variety of differentiated cell types at multiple distant sites (Figures 53-3 and 53-4).[2]

Neural crest cell differentiation occurs along multiple distinctive pathways and its contribution to the formation of a specific tissue or organ has generated considerable interest in developmental biology.[3,4] Progress has been made toward our understanding of how this important population of pluripotent cells is initially established in the early embryo and how genetic and epigenetic mechanisms mediate their subsequent lineage segregation, differentiation, and final contribution to a particular phenotype.[5–7]

Neural crest cells migrate ventrolaterally as they populate the pharyngeal (or branchial) arches. Each pharyngeal arch is delineated by discrete swellings that are produced by the proliferative activity of these crest cells. These ectodermally derived cells contribute extensively to the formation of mesenchymal structures in the head and neck as they migrate. All of the skeletal and connective tissue of the face, with the exception of dental enamel, is derived from neural crest cells, whereas skeletal and connective tissue of the trunk is mesodermal in origin. Cell labeling studies have demonstrated that neuroectoderm cells of rhombomeres 1–3 in the forming posterior midbrain and anterior hindbrain transform into cranial neural crest cells that migrate into the first pharyngeal arch and thereafter reside within the maxillary and mandibular prominences.[3,8,9] The migration of these rhombencephalic crest cells may be regulated by growth factor signaling pathways and their downstream transcription factors before they become committed to several different phenotypes, including progenitor tooth mesenchmal cells, osteoblasts, chondroblasts, and cranial nerve ganglia of the first pharyngeal arch.[4,6,10–12] Several studies provide strong supportive evidence that the cranial neural crest cells are developmentally

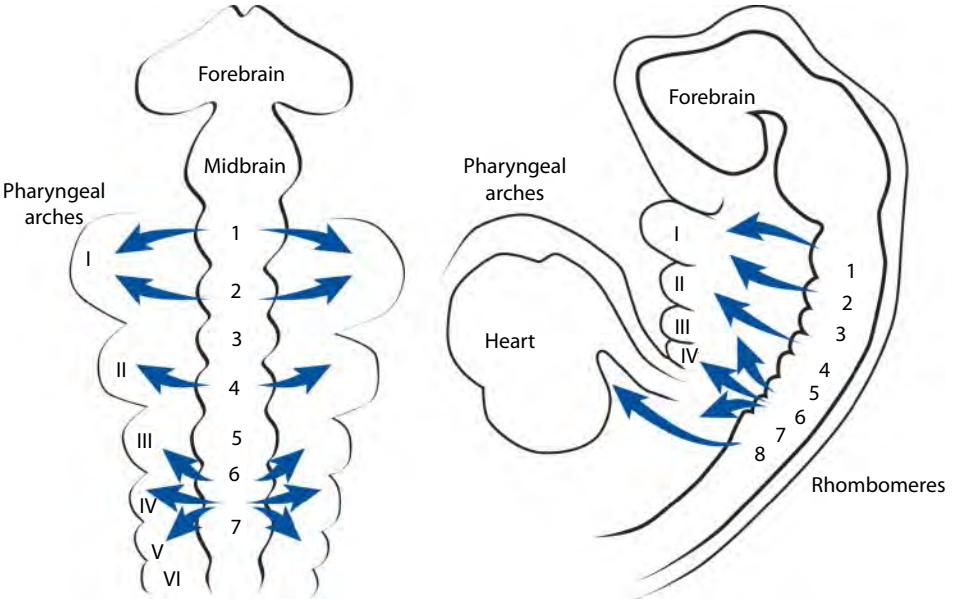


FIGURE 53-3. Illustration of neural crest migration pathways from rhombomeres 1 through 7 to pharyngeal arches I through VI, from dorsal **(left)** and lateral **(right)** views. Adapted from Sperber GH. Pathogenesis and morphogenesis of craniofacial developmental anomalies. Ann Acad Med Singapore 1999; 28:708–13.

modifiable with their fate not being predetermined before they reach their final destination. Rather, these progenitor cells must be instructed by signals from other tissues to generate craniofacial skeletal elements of appropriate shape and size. Tissues that provide the instructive signaling for cranial neural crest fate specification include, but are not limited to, the isthmic organizer behind the midbrain region, cranial placode, and the pharyngeal arch ectoderm and endoderm.[13–15] Transforming growth factor-beta (TGF-β) signaling has been shown to be critical in specifying neural crest cell differentiation.[16]

Our understanding of the migration and differentiation pathways of neural crest cells during embryogenesis have been signifcantly advanced by cell lineage studies using chick-quail chimeras,[4] cell labeling with a vital dye such as DiI,[9] neural crest cell–specific antibodies,[17] and retroviral-mediated gene transfer.[18] However, a comprehensive cell lineage analysis of mammalian neural crest cells that follow their complete differentiation ultimately into a particular phenotype has been historically precluded by the lack of a genetic marker permitting these cells to be indefinitely followed. Fortunately, a two-component genetic system for indelibly marking the progeny of neural crest cells has significantly improved our ability to analyze the fate of the cranial neural crest during normal as well as abnormal craniofacial embryogenesis.[19–21] This genetic system utilizes expression of the proto-oncogene *Wnt1* as a marker, following *cre* expression restricted to the precursors of the neural crest.[22] An additional benefit of this genetic system has been its ability to integrate the analysis of the fate and function of mammalian neural crest with mouse molecular genetics, which has helped to clarify the molecular mechanism of neural crest–related congenital malformations.[23]

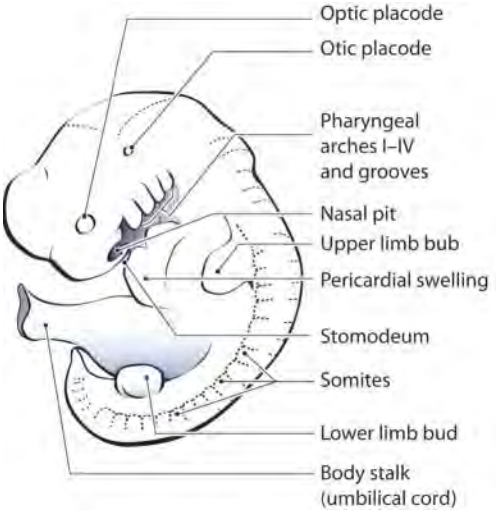


FIGURE 53-4. Lateral view of a 31-day-old embryo shows somites along the back and development of the pharyngeal arches and limb buds. Adapted from Sperber G. Craniofacial development. Hamilton (ON): BC Decker Inc; 2001. p. 24.

Facial Prominences and Formation of the Face

Facial formation occurs between the fourth and the eighth week of gestation. Early facial development is remarkably similar between different vertebrate species. In mice, cranial neural crest cells start to migrate soon after gastrulation, and the entire delamination takes place between the stages of 3 to 16 somites.[24] During the fourth week of gestation, neural crest cells selectively migrate to form four bilaterally paired pharyngeal arches on the ventral external surface of the embryo that will give rise to most of the head and neck structures. Pharyngeal arch identity, similar to other repetitive structures (somites, vertebrae, limb parts), is determined by expression

of a unique combination of homeobox genes that determine when particular groups of genes are expressed during embryologic development.[12,25,26] The subepithelial mass of the first pharyngeal arch is populated by neural crest cells delaminating from the midbrain to the rhombomere 2 that do not show homeobox expression.[24,27] This represents an exception from other pharyngeal arches expressing *Hox* (a subgroup of homeobox) genes and may be a result of distinct regulation of the corresponding neural crest by the isthmus.[4,15,28] Following immigration of neural crest cells, the first pharyngeal arch consists of the ectodermal epithelium of the stomodeum (future oral cavity), the primitive gut endoderm beyond the pharyngeal membrane, surface ectodermal epithelium (future skin) and ectomesenchyme,[19,29] and a small portion of central mesenchymal core, probably derived from the original mesodermal cells of the first pharyngeal arch.[24]

Five facial prominences (primordia or processes) form from the first pair of pharyngeal arches that include the single median frontonasal prominence and the paired maxillary and the paired mandibular prominences that form the superior, inferior, and lateral boundaries of the stomodeum or primitive oral cavity. These facial prominences, each of whose outgrowth is composed of different neural crest cells and regulated by different genes, are covered by a thin epithelium derived from the ectoderm.[30] The mesenchcyme is composed primarily of the neural crest whereas the core of the facial prominences contains some mesodermal cells.[24] The final shape of the embryonic face is believed to be defined by the differential proliferation of the facial prominences.[31] Following the development of appropriate form and size, the facial prominences merge with adjacent prominences when the epithelium between them breaks down, followed by invasion of the mesenchyme and coalescence of the adjoining prominences (Figure 53-5).[32] Shortly after neural crest cell migration, the paired mandibular prominences merge in the midline to form the chin and lower lip. At the same time, the ectoderm of the frontonasal prominences forms two thickened epithelial regions, the nasal placodes, which

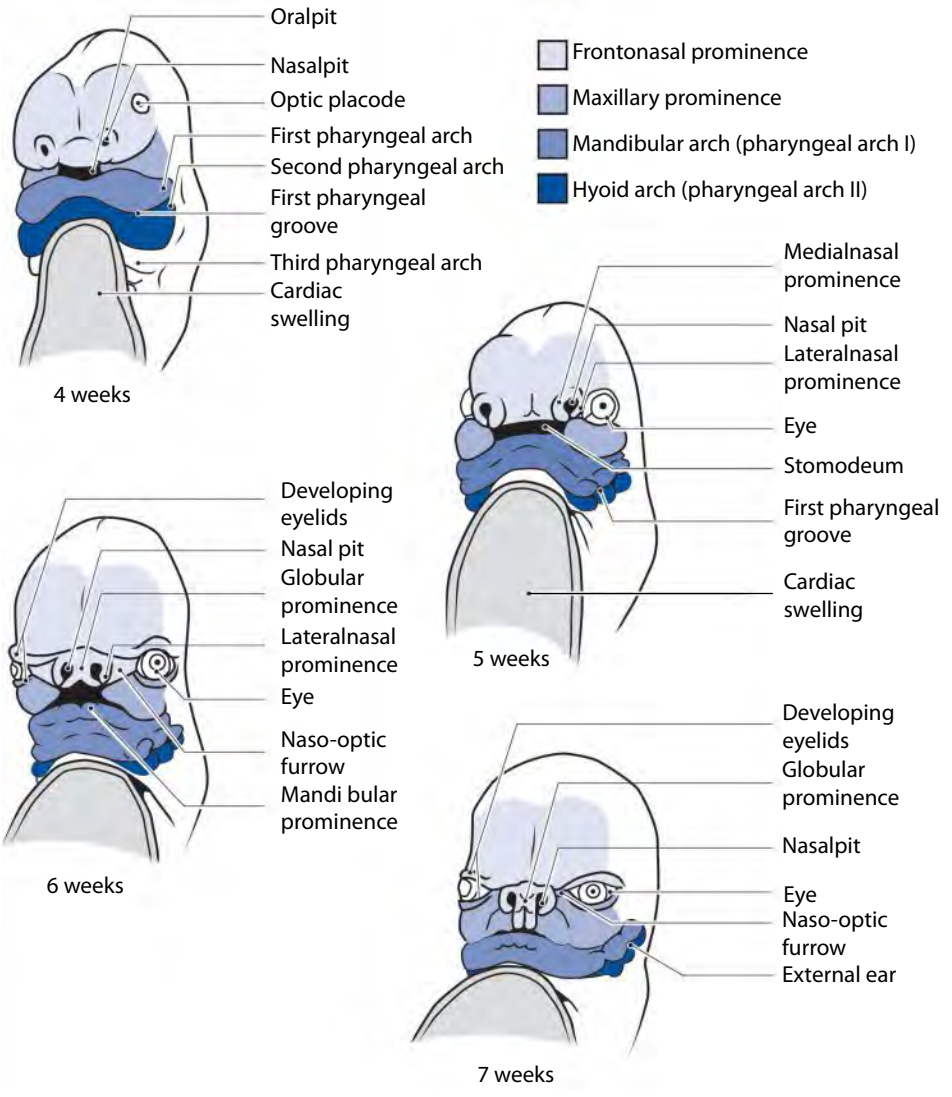


FIGURE 53-5. Frontal view of the developing face in 4-, 5-, 6-, and 7-week-old embryos shows merging of the facial prominences (primordia or processes). Adapted from Sperber G. Craniofacial development, Hamilton (ON): BC Decker Inc; 2001. p. 32.

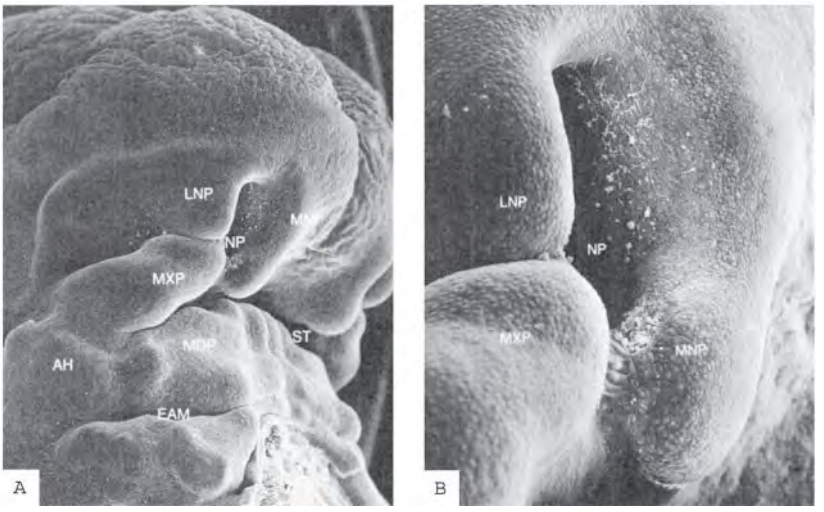


FIGURE 53-6. Scanning electron micrograph of a 41-day-old human embryo. **A,** Craniofacial region. AH = right auricular hillock; EAM = right external acoustic meatus; LNP = right lateral nasal prominence; MDP = mandibular prominence; MNP = right medial nasal prominence; MXP = right maxillary prominence; NP = right nasal pit; ST = left side of stomodeum. **B,** Enlarged view shows epithelial bridges between the merging MXP) and the MNP). Failure of the prominences to merge together with the LNP results in cleft of the lip. (**A** and **B,** Reproduced with permission from Hinrichsen K. The early development of morphology and patterns of the face in the human embryo. In: Advances in anatomy, embryology and cell biology. Vol. 98. New York: Springer-Verlag; 1985.)

subsequently curl outward to give rise to the medial and lateral nasal prominences. The medial nasal prominences grow further, moving toward each other and eventually merge in the midline (globular prominence) early in the sixth week to form the central part of the upper lip and the primary palate. There still is some controversy regarding the origin of the central part of the upper lip, which some believe is of frontonasal prominence origin.[21] The maxillary prominences move medially as well, merging with the lateral and medial nasal prominences during the sixth week, to complete formation of the upper lip. At this same time, the maxillary and mandibular prominences merge laterally, determining the width of the mouth. Merging of facial prominences requires disintegration of surface epithelia in order to permit the underlying mesenchymal cells to unite (Figure 53-6). The groove between the prominences is gradually filled out by proliferation of the mesenchyme so that the prominences appear to merge. Facial clefting is a result of failure of epithelial disintegration and lack of merging. Facial prominence growth and merging is dependent on ectodermal-mesenchymal interactions that appear to be regulated by the secreted protein sonic hedgehog (*SHH*).[30] Mutations in *SHH* that prevent its signaling during early neural plate patterning cause midline defects that range from hypotelorism and cleft lip/palate to holoprosencephaly and cyclopia.[33] There also is evidence that adequate epidermal growth factor receptor signaling is necessary for sufficient secretion of matrix metalloproteinases for normal facial development.[34] From 5 weeks' gestation to the early part of the fetal period at 9 weeks, there is medial migration of the eyes, assisted by frontal and temporal lobe expansion and greater proliferation of the lateral facial regions relative to the central face, resulting in facial expansion and interocular reduction.

The nasal placodes that formed at about 5 weeks each are separated inferiorly by a nasal groove. With continued proliferation of mesenchyme, the placodes submerge to form the nasal pits, the precursors to the anterior nares. As the nasal pits continue to submerge with the proliferating mesenchyme, they are eventually separated from the stomatodeum by only a thin oronasal membrane. This membrane will rupture at the beginning of the seventh week, forming a continuous nasal and oral cavity.

Genetic Analysis of Craniofacial Development

Our fundamental understanding of normal and abnormal human craniofacial development has been enormously advanced by gene targeting in mice in which more than 90 knockout mutants have been created to date. Although this large number reflects both the genetic and the morphogenetic complexity of head development, many of these malformations cluster into a few groups that can be characterized by the age of onset and the pattern of malformations observed in humans. The following is a summary of what is known at present of the genetics of the major human craniofacial malformations gained from homozygous knockout mouse studies.[35]

Midline Defects or Cranial Truncations

These defects initiate at mouse embryonic days 7 to 8 and are occasionally accompanied by laterality defects, with mutants frequently being resorbed before birth. Some examples include mice that are null for *Hesx1* (homeobox gene expressed in embryonic stem cells), *Lhx1* (LIM homeobox protein 1), *Otx2* (orthodenticle homologue 2), *Pcsk6* (proprotein convertase subtilisin/kexin type 6), *SHH*, and *Sil* (Tal1 interrupting locus). A number of these genes are expressed in the anterior visceral endoderm, which may act as an independent head organizer during early mammalian development.

Neural Tube Defects

These defects initiate at mouse embryonic day 8 and are designated as *exencephaly* when they are present in the cranial region. Examples of this rapidly increasing number of defects being identified include mice that are null for *Cart1* (cartilage homeoprotein 1), *Pax3* (paired box gene 3), *Tcfap2a* (transcription factor AP-2, α), and *Twist*, *Gli3* (GLI-Kruppel family member 3).

Neural Crest Defects

These defects influence neural crest specification, migration, proliferation, and epithelial-mesenchymal induction. This is

a notably large group that includes many members of the *Dlx* (distal-less), *Msx* (Msh-like) and *Prrx* (paired-related) homeodomain families, as well as *Gsc* (goosecoid), *Pax3*, and elements of the endothelial pathway. An unusual case is a homozygous knockout of *Hoxa2* (normally expressed in the second pharyngeal arch), which produces a homeotic respecification into first archlike structures. Clefts of the secondary palate frequently occur with this group, as an outcome of disturbed facial structural anatomic relationships.

Sensory Organ Defects

These defects include mice that are null for *Pax2*, *Pax6*, *Chx10* (ceh-10 homeodomain homologue), *Chrd* (chordin), *Rax* (retina and anterior neural fold homeobox), and *Bmp7* (bone morphogenetic protein 7). They exhibit sensory organ defects typically affecting the ear or eye.

Isolated Clefts of the Secondary Palate

Although these defects are rare, they occur in the absence of other abnormalities in the *Jag2* (Jagged 2), *Lhx8*, and *Tgf*β*3* (TGF-β3) knockout mice. These palatal clefts occur by different mechanisms, with the palatal shelves failing to elevate in *Jag2–/–* mice, whereas correctly positioned palatal shelves fail to fuse in *Tgf*β*3–/–* mice.

Skeletal Differentiation Defects

These defects of skeletal differentiation include mice that are null for *Runx2* (runt-related transcription factor 2), which is a master regulator of osteoblast and extracellular matrix differentiation such as *Col2a1* and *Col11al* (collagens), *Crtl1* (cartilage link protein 1), and *Hspg2* (perlecan).

Some notable examples of orthologous gene mutations associated with craniofacial malformations in mice or humans include *SHH*, a signalling molecule that produces cyclopia and absent limbs in mice and holoprosencephaly in humans; *DHCR7*, an enzyme that produces growth and motor retardation in mice and Smith-Lemli-Opitz syndrome in humans; *TCOF1*, a nucleolar protein that produces multiple abnormalities in mice and Treacher Collins syndrome in humans; *EYA1*, a nuclear protein that produces a middle ear abnormality in mice and branchio-otorenal syndrome in humans; *TP63*, a transcription factor that produces a generalized epithelial defect in mice and ectrodactyly, ectodermal dysplasia, and cleft lip/palate in humans; *COL11A1*, a structural protein that produces a generalized cartilage defect in mice and Stickler and Marshall syndromes in humans; fibroblast growth factor receptor 1 *(FGFR1)*, a signal transduction that produces craniosynostosis in mice and Pfeiffer syndrome in humans; and *ALX4*, a transcription factor that produces polydactyly in mice and parietal foramina in humans.[35]

There is a progressively rapid increase in the identification of a known genetic map location or molecular basis for many human craniofacial malformations. Some selected examples include achondroplasia, from *FGFR3* gene at chromosome location 4p16.3; Apert syndrome, from *FGFR2* gene at 10q26; cleft lip/palate-ectodermal dysplasia syndrome from gene *PVRL1* at 11q23-q24; isolated cleft palate at chromosome location 2q32; X-linked cleft palate at Xq21.3; cleidocranial dysplasia from gene *RUNX2* at 6p21; Crouzon syndrome with acanthosis nigricans from gene *FGFR3* at 4p16.3; Crouzon syndrome and Jackson-Weiss syndrome from gene *FGFR2* at 10q26; isolated dental anomalies from gene *RUNX2* at 6p21; dentogenesis imperfecta from gene *DSPP* at 4q21; DiGeorge syndrome from gene *TBX1* at 22q11; anhydrotic ectodermal dysplasia from gene *ED1* at Xq12-q13.1; hemifacial microsomia at 14q32; holoprosencephaly-1 at 21q22.3; holoprosencephaly-2 from gene *SIX3* at 2p21; holoprosencephaly-3 from gene *SHH* at 7q36; holoprosencephaly-4 from gene *TGIF* at 18p11.3; holoprosencephaly-5 from gene *ZIC2* at 13q32; oligodontia from gene *PAX9* at 14q12-q13; oral-facial-digital syndrome 1 from gene *OFD1* at Xp22.3-p22.2; orofacial cleft-1 at 6p24.3; orofacial cleft-2 at 2p13; otopalatodigital sydrome, type I at Xq28; Pfeiffer syndrome from gene *FGFR1* at 8p11.2-p11.1; Pfeiffer syndrome from gene *FGFR2* at 10q26; Stickler syndrome, type I from gene *COL2A1* at 12q13.11-q13.2; Stickler syndrome, type II from gene *COL11A1* at 1p21; Stickler syndrome, type III from gene *COL11A2* at 6p21.3; Treacher Collins syndrome from gene *TCOF1* at 5q32-q33.1; Van der Woude syndrome at 1q32; and velocardiofacial syndrome at 22q11.[35] This is only a partial list of a constantly growing number of craniofacial malformations in which the genetic or molecular basis is being identified.

Formation of Neurocranium and Viscerocranium

Formation of the craniofacial bones begins with development of the cartilaginous and membranous precursors to the neurocranium and viscerocranium during the latter part of the fifth week of gestation (Figure 53-7). Major signaling pathways control skeletal patterning and are mediated by *Wnt* signaling proteins (Wnts), hedgehogs (SHH), bone morphogenetic proteins (BMPs), fibroblast growth factors (FGFs), and Notch/Delt.[36] The membranous neurocranium (desmocranium) that will give rise to the flat bones of the calvaria is connective tissue derived from the paraxial mesoderm and neural crest. The cartilaginous neurocranium (chondrocranium) that will form the cranial base is cartilage from neural crest origin. Cartilage maturation occurs in a caudal-rostral sequence. The membranous viscerocranium that will give rise to the maxilla, zygomatic bone, squamous temporal bone, and mandible is derived from the neural crest. The cartilaginous viscerocranium that will form the middle ear ossicles, styloid process of the temporal bone, hyoid bone, and laryngeal cartilages is from neural crest ectoderm. Endochondral ossification centers occur in the cartilaginous components and intramembranous ossification sites form in the membranous components of the neurocranium and viscerocranium. Osteoblast differentiation with the onset of mineralization results from a rapid angiogenic process with vascular ingrowth closely surrounding the centers and sites of ossification. The earliest ossification of the craniofacial bones begins in the seventh and eighth weeks of gestation. There are eventually 110 ossification centers, nearly all of

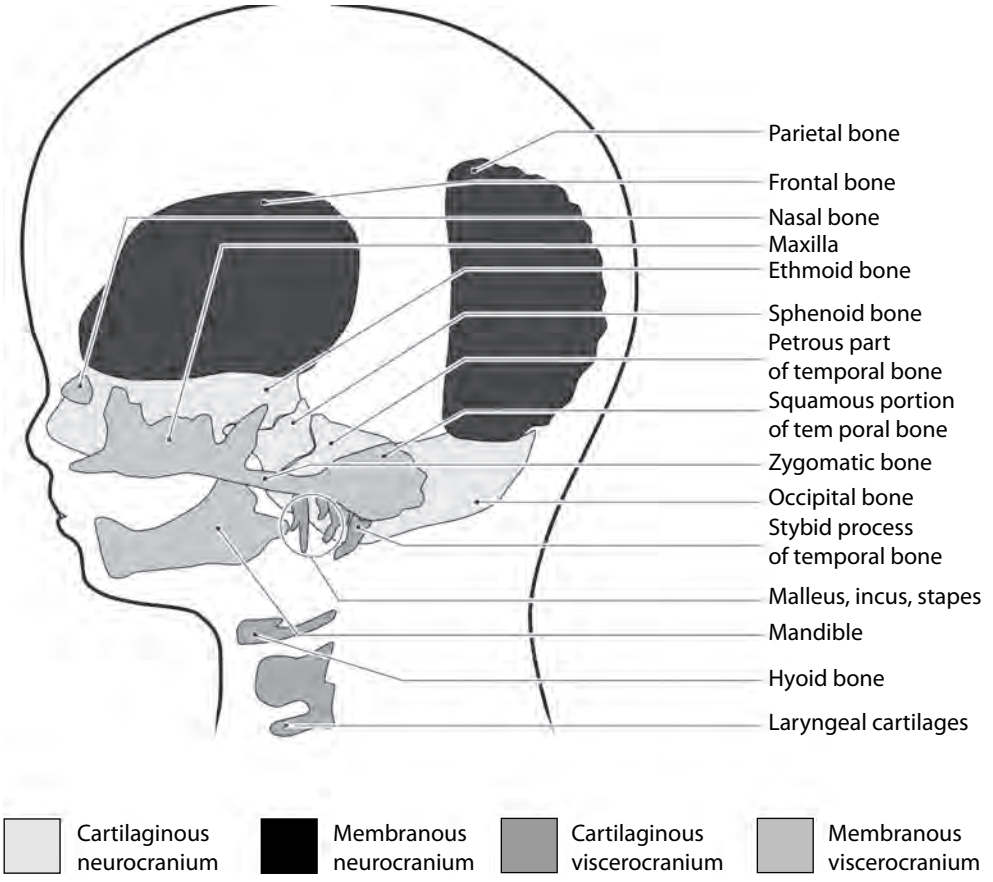


FIGURE 53-7. Lateral view of a 20-week-old embryo illustrates initial development of the cartilaginous and membranous neurocranium and viscerocranium. Adapted from Moore KL, Persaud TVN. The developing human: clinically oriented embryology. 5th ed. Philadelphia (PA): W.B. Saunders; 1993. p. 361.

which appear between 6 and 12 weeks' gestation, that develop in the embryo to form 45 bones at birth, which ultimately form 22 bones in the adult.

Ossification

The onset of ossification generally follows the chronologic sequence of mandible, maxilla, palatine, cranial base, and cranium, with intramembranous sites usually preceding endochondral centers.[37] Intramembranous ossification of the mandible begins in the mental foramen region at 6 weeks of gestation lateral to Meckel's cartilage. The secondary mandibular condylar cartilage forms at 10 weeks' gestation as a separate endochondral condensation, adjacent to but independent of Meckel's cartilage and separated from the intramembranous the body of the mandible. Endochondral ossification of the mandibular condylar cartilage does not begin until 15 weeks. Maxillary intramembranous ossification begins in the area of the infraorbital foramen lateral to the nasal capsule. This ossification progresses anteriorly and posteriorly from this region. The vertical portion of the palatine bone then begins intramembranous ossification in the region of the palatine nerve, followed by ossification of the anterior, then the posterior borders of the incisive foramen, spreading through the hard palate from the canine area. Following ossification of the main portion of the mandible and maxilla, during the sixth week of gestation, endochondral ossification of the cranial base occurs in the midline from the foramen magnum to the nasal bone, and intramembranous ossification occurs laterally. Finally, intramembranous ossification of the cranial bones follows.

Final Tissue Differentiation

Interaction between pharyngeal endoderm and neural crest tissue, followed by oral ectoderm proliferation, produces identifiable odontogenic tissue by the end of 4 weeks' gestation. There are four origin sites of odontogenic epithelium for both the maxillary and the mandibular arches, appearing at the end of 5 weeks' gestation. The primary anterior and first molar tooth germs appear at 6 weeks' gestation, followed by development of the primary second molar germs at 7 weeks. Apposition of bone on the alveolar margins of the maxilla and mandible in the presence of developing tooth germs forms the initial alveolar processes.

One of the last orofacial structures to reach completion at the end of the embryonic period is the secondary palate,

developing from the paired lateral palatine shelves of the maxilla. These shelves are oriented vertically with the tongue interposed, but the tongue and floor of the oral cavity descend as the nasal chambers expand laterally and inferiorly (Figure 53-8). As this occurs, the palatal shelves become elongated and elevate medially toward each other, beginning fusion at the end of the eighth week and completing in the ninth week of gestation. There is evidence that TGF-β3 is intimately involved in regulating secondary palatal fusion by mediating the breakdown of the midline epithelial seam before fusion.[38]

Fetal Period

The fetal period begins during the ninth week, at 60 days' gestation, lasting until birth at 40 weeks, and overall

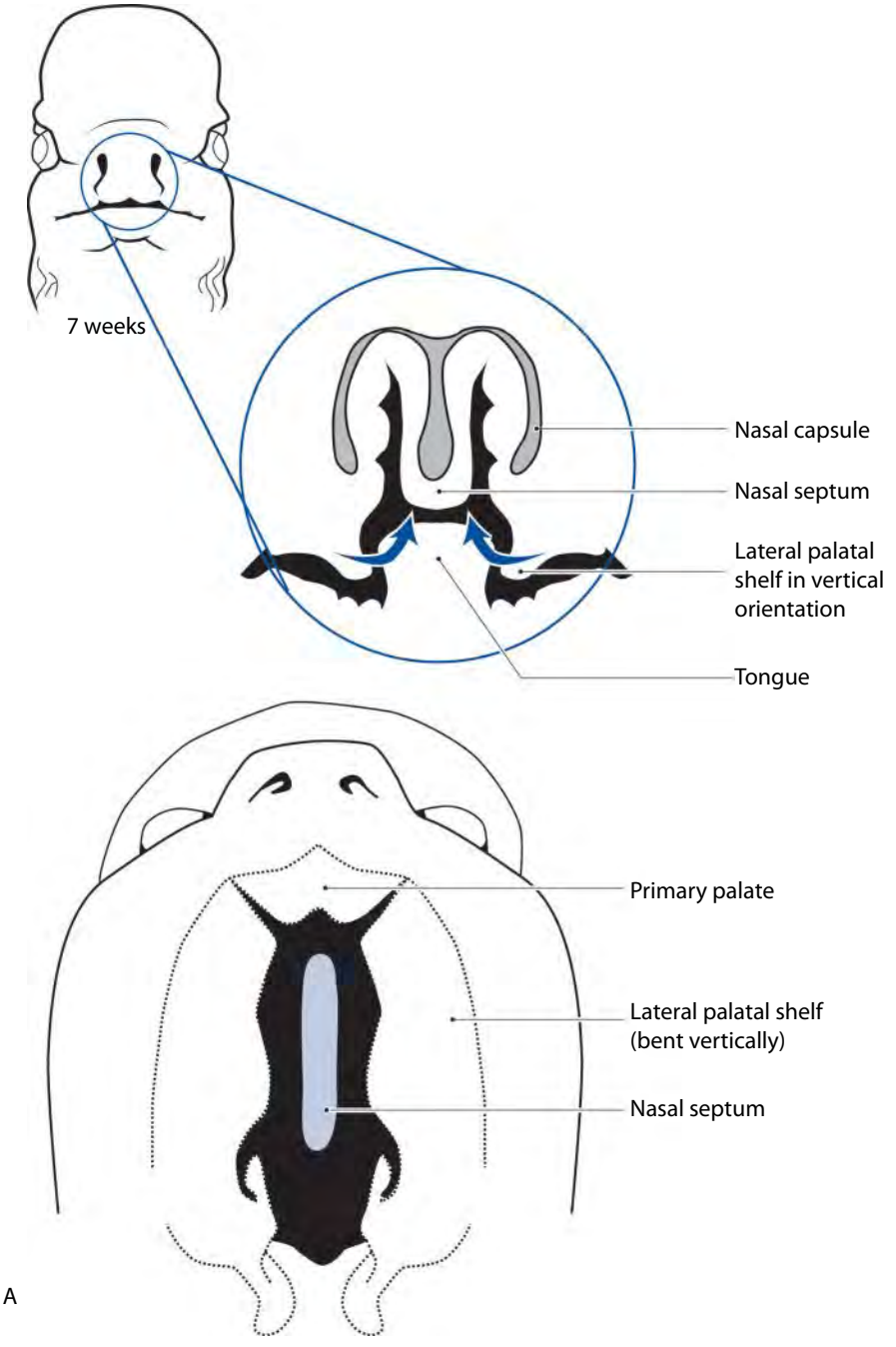


FIGURE 53-8. Frontal view of face, coronal section of stomodeum, and inferior view of palate in 7- and 12-week-old embryos. **A,** Embryo at 7 weeks shows the palatal shelves oriented vertically. *(Continued)*

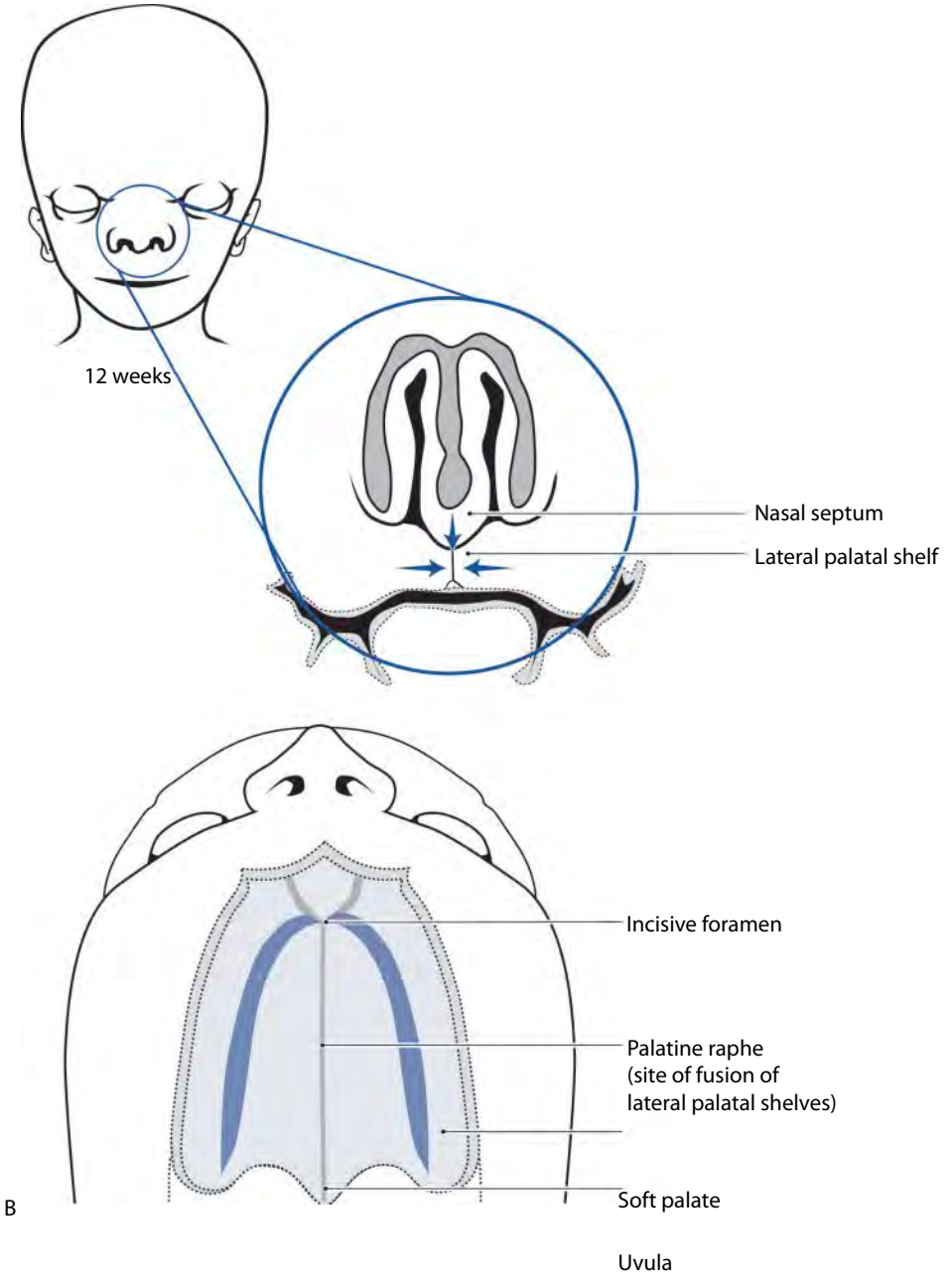


FIGURE 53-8. *(Continued)* **B,** Embryo at 12 weeks shows fusion after elevation of the palatal shelves. Adapted from Sperber G. Craniofacial development. Hamilton (ON): BC Decker Inc; 2001. pp. 41, 114, 116.

somatic growth follows a cephalocaudal growth gradient (Figure 53-9). There is a prenatal growth spurt between 20 and 30 weeks' gestation with the peak growth velocity at 27 to 28 weeks being approximately 2.5 cm per week. The prenatal spurt in weight is slightly later at 30 to 40 weeks' gestation with a peak at 34 to 36 weeks.[39] The rate steadily decreases during the last trimester and continues to decline after birth until adulthood, with two exceptions. The first is a small "midgrowth" spurt that occurs in many children at 6 to 8 years old that has been attributed to increased adrenal secretion of androgenic hormones. The second is a dramatic endocrine-mediated "pubertal growth" spurt during adolescence. Growth of the craniofacial complex during the fetal period is characterized by a constant rate during the second trimester. The craniofacial skeletal components increase more in the anteroposterior dimension than in the vertical or

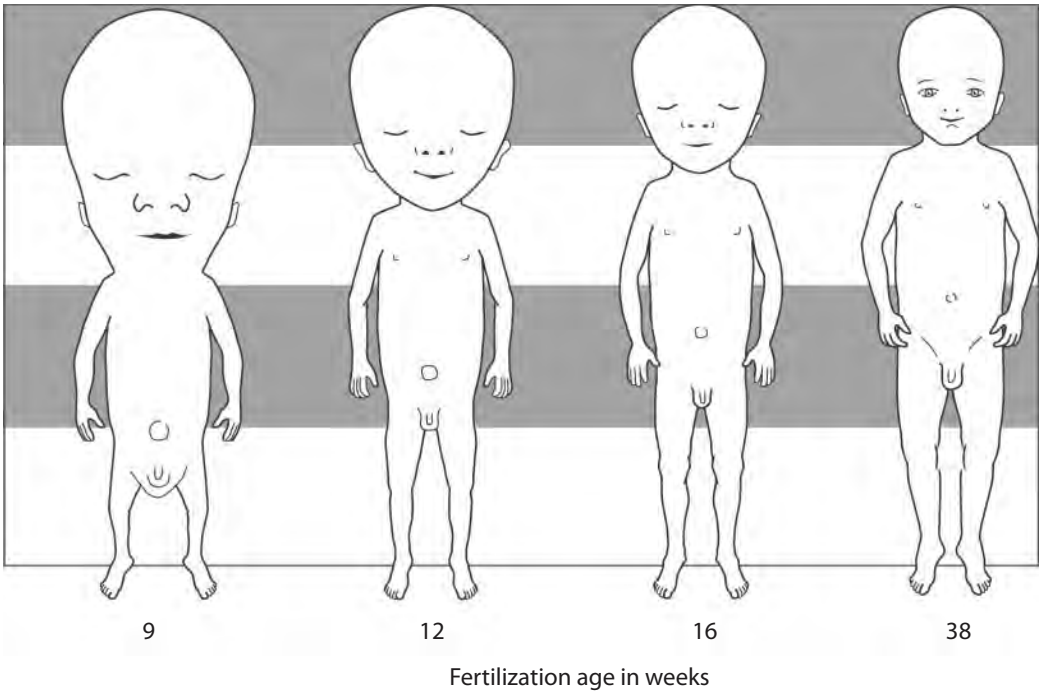


FIGURE 53-9. Changes in fetal body proportions with all stages drawn to same total height. At the start of the fetal period, the head is half the length of the fetus, and by birth, it is one quarter the length. Adapted from Moore KL, Persaud TVN. The developing human: clinically oriented embryology. 5th ed. Philadelphia (PA): W.B. Saunders; 1993. p. 97.

transverse, with the exception of the mandible, which increases more in the transverse dimension in order to maintain appropriate articulation.[40]

During the fetal period, the neurocranium undergoes precocious development relative to the viscerocranium with earlier brain and neurocranial bone vault growth than facial and masticatory portions of the skull. This results in an early proportional predominance of the neurocranium over the face that only reduces to an 8:1 proportion by birth. The brain nearly doubles in size from 4 months' gestation to birth, achieving about 25% of its adult dimension. The formation and maintenance of cranial sutures are regulated by tissue interactions with the underlying dura mater as the brain develops.[41] A number of growth factors have been identified that regulate cranial bone growth and suture fusion, including TGF-β1, TGF-β2, TGF-β3, BMP-2, BMP-7, FGF-4, insulin-like growth factor-I (IGF-I), and SHH.[42,43] Transcription factors MSX2 and TWIST also play a role in suture development, binding to target effector genes to determine their expression.[44] The cranial vault elements are developed at the boundary between cranial neural crest– and mesoderm-derived tissue. The frontal bones are derived from neural crest, whereas the parietal and occipital bones are of mesoderm origin.[21] This results in the coronal, sagittal, and lambdoid sutures occurring at the neural crest–mesoderm interface. The metopic suture is the only human cranial suture not formed between neural crest and mesoderm, being entirely of neural crest origin. Mandibular morphogenesis is tightly controlled by BMP and FGF signaling in the ectoderm.[45] The eyeballs grow concurrently with the early brain growth, increasing facial expansion and separating the neural and facial skeletons to increase skull height. The cranial base growth parallels the rapid growth of the cranial vault during the fetal period. The anterior cranial base grows sevenfold whereas the posterior cranial base increases fivefold. The intraethmoidal and intrasphenoidal synchondroses close before birth.

The ossification centers that begin the formation of the facial bones late in the embryonic period at 6 to 8 weeks enlarge during the early fetal period until most of the bones have developed into a definitive shape by 14 weeks. At this time, they begin to remodel as they continue to grow by intramembranous and/or endochondral ossification. Mandibular condylar cartilage appears relatively late at 10 weeks, well after the embryonic period when a rudimentary mandible already is present.[46] This secondary cartilage may serve as an intrinsic center of growth at this stage, in contrast to its later growth that is predominantly extrinsically determined. The anterior aspects of the maxilla, mandible, and zygoma of the fetal and early postnatal face undergo osseous deposition at this time. This early anterior deposition is necessary to permit adequate osseous mass for the developing tooth buds of the primary and permanent dentitions. Although the tooth germs start to develop as early as 6 weeks' gestation, the onset of dental mineralization does not begin until ossification has occurred. The maxilla demonstrates a rapid height increase associated with dental development.[47] Once the primary teeth have erupted, the anterior aspect of the maxilla undergoes resorption rather than deposition to produce the descent of the maxilla with continued growth. Meanwhile,

the posterior, infraorbital, and lingual surfaces of the maxilla are depository in both fetal and postnatal development. The fetal temporal bone grows faster in height than width whereas the lateral and inferior margins of the zygomatic bone grow faster than its orbital margin.[47]

The paranasal sinuses, including the maxillary, sphenoidal, frontal, and ethmoidal, start developing at the beginning of the fetal period. Pneumatization begins first with the maxillary sinus, starting at 5 months' gestation. It is proposed that a septomaxillary ligament, attached to the sides and anteroinferior border of the nasal septum and inserted in the nasal spine, transmits septal growth, pulling the maxilla downward. Between the 10th week of gestation and birth, the nasal septum increases its vertical height sevenfold. The nasal septum growth, together with neural growth and facial sutural growth, transposes the maxilla inferiorly and anteriorly. The frontomaxillary, frontonasal, frontozygomatic, frontoethmoidal, and ethmoidomaxillary sutures grow predominantly in a vertical direction. The temporozygomatic and nasomaxillary sutures contribute most of the anteroposterior change. The intermaxillary and zygomaticomaxillary sutures provide most of the transverse expansion of the face. Overall, the middle and lower thirds of the face develop primarily in a downward and slightly forward direction away from the cranial base owing to brain development, maxillary and palatine sutural growth, and possibly nasal septum growth.

Although the midsagittal part of the middle face consists entirely of nasal septal cartilage during the fetal period, ossification leaves only a small anterior part of this cartilage remaining postnatally. Currently, there is controversy regarding the role of the nasal septum in postnatal facial growth. Some believe it is limited to a compensatory and biomechanical role, and others believe it serves a more extensive role, particularly in promoting vertical maxillary growth.

Although the mandible is larger than the maxilla during the embryonic period, the mandible approximates the size of the maxilla within the first month of the fetal period. The three secondary cartilages of the mandible do not appear until the 10th and 14th weeks of gestation, forming on the lateral and superior aspects of the condylar processes. This secondary type of cartilage differs morphologically from epiphyseal and synchondrosal cartilage.[48] Two of these secondary cartilages forming at the mental protuberance and the coronoid process ossify before birth, leaving only the cartilage on the condylar head as a site of postnatal mandibular endochondral growth. This cartilage never undergoes complete ossification, providing a means for absorbing functional forces and retaining growth potential throughout life. Between the 13th and the 20th weeks of gestation, the mandible lags behind the maxilla again while there is a transition from Meckel's cartilage to condylar cartilage as the primary growth site. During the third trimester, there is a significant deepening of the corpus in association with the developing dentition. The mandibular ramus growth rate is greater than the growth rate of the mandibular body during this time.[49] At the time of birth, the mandible usually is equal in size again to the maxilla, although it often is in a retrognathic position relative to the maxilla.

Development of the permanent tooth germs begins at 16 weeks' gestation, with the first permanent molar germs developing posteriorly from the dental lamina followed by the permanent anterior tooth germs emerging from the lingual side of the primary enamel organs. At birth, the primary tooth crowns are still not completely calcified, as the first permanent molars begin to calcify.

POSTNATAL CRANIOFACIAL DEVELOPMENT

Skeletal Development

An area of craniofacial growth can be differentiated as a growth center or growth site. A *growth center* is where there is primarily intrinsic genetic growth control with a minimal environmental or functional role. Although a *growth site* also is controlled to some extent by genetic programming, it is more vulnerable to extrinsic growth control, being dependent more on the functional influence of the surrounding tissues. Cranial base synchondroses, where endochondral ossification of primary cartilage occurs, represent growth centers. The role of the cartilaginous nasal septum as a growth center or site remains controversial. There is a clearer understanding that the endochondral growth of the secondary cartilage of the mandibular condyles acts as a growth site, being greatly influenced by mandibular and soft tissue function. Areas of membranous bone growth resulting from sutural or periosteal ossification are primarily growth sites and represent the bulk of the remaining craniofacial complex. There are exceptions, such as craniosynostosis, that can be due to an underlying genetic cause. Membranous ossification by sutural and periosteal remodeling is essentially the only type of craniofacial bone growth that occurs after adolescence and throughout adulthood.

Development and completion of craniofacial growth follow the overall somatic cephalocaudal growth gradient throughout prenatal and postnatal growth, with cranial vault growth completing before the cranial base, followed by the nasomaxilla and finishing with the mandible. During postnatal growth, the neurocranium continues to develop ahead of the viscerocranium (Figure 53-10). It increases from about 30% of its ultimate adult size at the time of birth to 50% by 6 months of age, 75% by 2 years of age, and nearly 90% by 3 years of age. By 5 years of age, the orbits have reached nearly 80% of their adult size.[50] This is why a child of this age appears to have a disproportionately large cranium and eyes. After birth, the neurocranium increases about five times in size, whereas the viscerocranium increases about 10 times. There is also a difference in the amount of postnatal increase in the three dimensions, with the vertical increasing by about 200%, the anteroposterior by somewhat less, and the transverse by the least at approximately 75%. By 10 years of age, the neurocranial growth is nearly 95% complete, whereas facial growth is only about 60% complete.

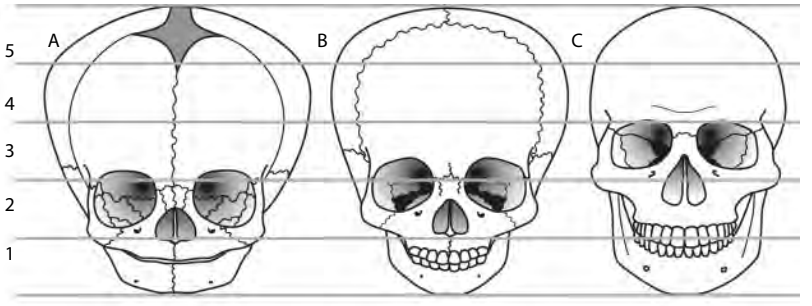


FIGURE 53-10. Changes in postnatal skull proportions with all stages enlarged to the same skull height and oriented in Frankfort horizontal plane with skull height divided into fifths. **A,** Neonate, showing the viscerocranium representing one fifth of the total height. Three-year-old **(B)** and adult **(C)** show the proportional increase in height of the viscerocranium relative to the neurocranium. Adapted from Sarnat BG. Normal and abnormal craniofacial growth. Some experimental and clinical considerations. Angle Orthod 1983;53:263.

The craniofacial complex can be divided conveniently into four primary units: the cranial vault, the cranial base, the nasomaxilla, and the mandible. Each of these units has its growth regulated to some extent by both intrinsic and extrinsic controls. Our understanding of postnatal craniofacial growth has developed in part from cross-sectional anatomic and histologic studies of human cadavers and skeletal material. What has been particularly helpful in supplementing this material is a number of North American longitudinal craniofacial growth records and longitudinal implant (used for stable reference points) studies that were gathered from the 1940s to the mid-1960s before radiation hygiene and human subject research standards became more stringent.[51]

Cranial Vault

At birth, the cranial bones are separated by sutures with fontanelles where the corners of the bones meet, permitting compression of the skull during the birthing process (Figure 53-11). Postnatal bone growth results in narrowing of the sutures with all of the fontanelles closing within the first 2 years. In the absence of any intrinsic growth potential in the sutures, the pressures exerted by the developing brain determine the size and shape of the cranium. As the brain expands, the internal pressure creates tension across the connective tissue of the sutures and compression against the cranial bones, resulting in intramembranous bone growth by suture and surface apposition. Modeling of the cranial bones to a flatter shape is necessary to adapt to the expanding surface of the brain. This occurs primarily from endocranial resorption and ectocranial apposition. Although suture apposition plays a larger role than surface apposition in overall cranial vault capacity, the postnatal shape primarily is determined by extrinsic factors.

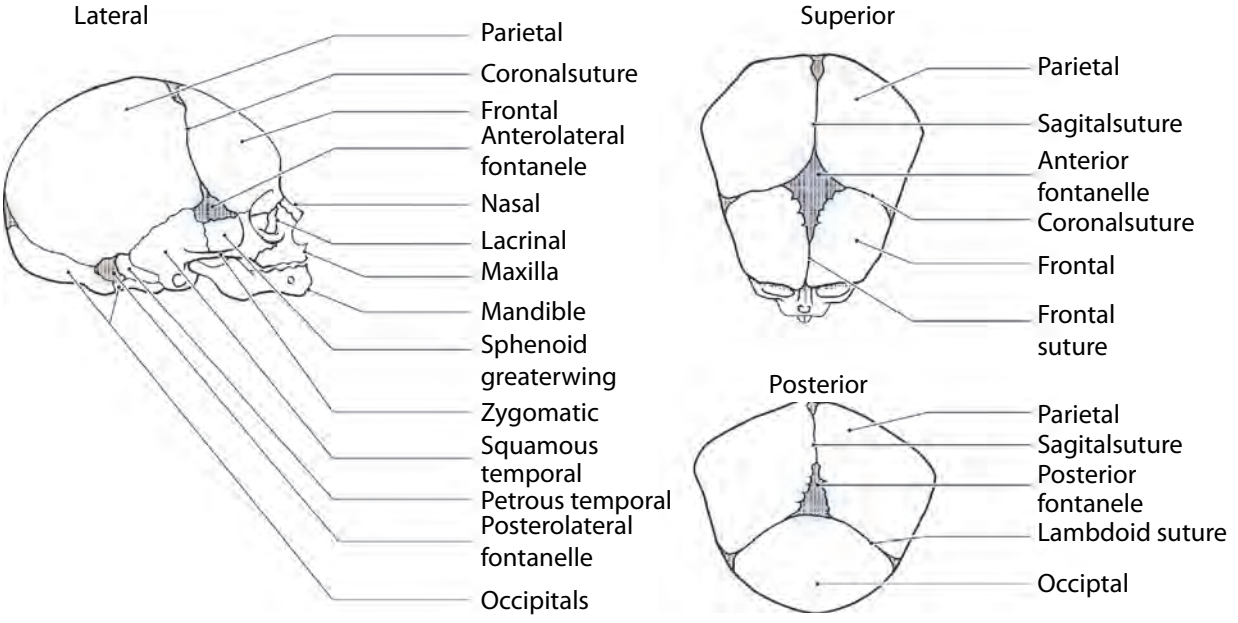


FIGURE 53-11. Fontanelles and sutures of the neonatal skull. Adapted from Sperber G. Craniofacial development. Hamilton (ON): BC Decker Inc; 2001. p. 84.

The premature fusion (craniosynostosis) of one cranial suture may not cause a significant functional effect but, if left untreated, will distort the shape of the cranium. Craniosynostosis is often caused by dominant mutations in one of three fibroblast growth factor receptor genes (*FGFR*1, *FGFR2*, and *FGFR3*) or in the transcription factor *TWIST*.[52] Untreated premature fusion of multiple cranial sutures may adversely affect neurologic development as well. Early surgical release during the first 2 postnatal years is required to prevent or minimize cranial deformation or neurologic deficit.

By 6 to 7 years of age, the inner table of the cranial bones becomes stable due to the cessation of cerebral growth. However, the outer table continues to model in response to extracranial muscular forces. The temporal muscles tend to laterally compress the cranium, forming temporal sulci and zygomatic arches. The lateral and posterior cervical muscles insert primarily on the squamous part of the temporal and occipital bones, influencing their shape. Even after attainment of the adult form, the cranial bones continue to thicken during adulthood.

Cranial Base

Compared with the other craniofacial units, the shape of the cranial base is relatively stable during growth, due likely to its greater intrinsic growth potential. Perhaps more than any other craniofacial area, growth of the cranial base is genetically predetermined in the midline by endochondral bone growth at the synchodroses and influenced the least by functional matrices.[53,54] However, prenatal brain growth may provide a minor extrinsic influence, causing some flattening of the cranial base, because this does not occur with anencephaly. In addition, there is evidence that chondral growth of the cranial base can be altered with mechanical forces.[55] Lateral to the cranial base midline, there is intramembranous bone growth from sutures and surface remodeling that are affected by extrinsic factors.

The cranial base is formed from primary cartilage that appears underneath the brain in the sixth week of embryonic life. Following initial osseous calcification during the eighth week, synchodroses are created by the residual cartilage and continue the endochondral bone formation, pushing the cranial base bones apart centrally while sutures more laterally produce intramembranous bone growth. The anterior cranial base matures earlier than the posterior cranial base, with the posterior intraoccipital synchondroses closing during the second and third years postnatally and the anterior intraoccipital synchondroses closing at 3 to 4 years of age (Figure 53-12). The sphenoethmoidal synchondrosis closes at about 6 years of age. Although the spheno-occipital synchondrosis is not a main growth site before birth, it provides the greatest contribution to cranial base growth postnatally, delaying fusion until adolescence. The prolonged postnatal growth period of the spheno-occipital synchondrosis permits posterior growth of the maxilla to provide adequate bone for the developing posterior permanent teeth and adequate space for the nasopharynx. In addition to endochondral bone growth in the midline, intramembranous remodeling of the cranial base occurs, including apposition on the basioccipital bone and anterior margin of the foramen magnum, resulting in continued lengthening of the posterior cranial base even

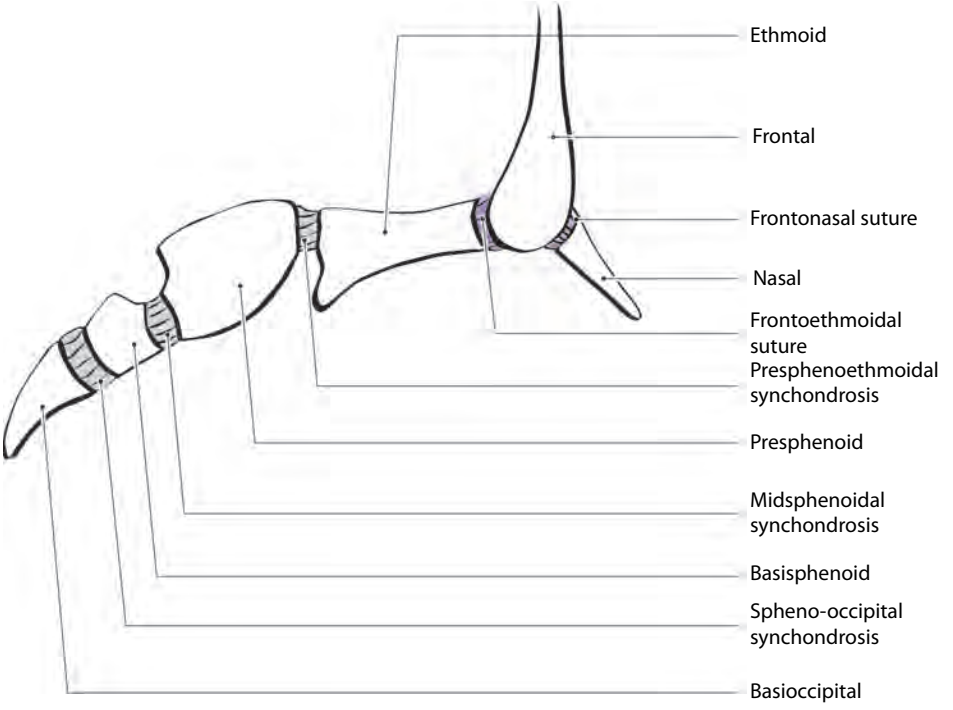


FIGURE 53-12. Midsagittal neonatal cranial base shows sutures and synchondroses. Adapted from Mooney MP, Siegel MI. Understanding craniofacial anomalies: the etiopathogenesis of craniosynostoses and facial clefting. New York (NY): Wiley-Liss, Inc.; 2002.

after adolescence. Enlargement of the sella turcica continues postnatally, with the anterior wall stabilizing at about 6 years of age and the posterior wall continuing to resorb until late adolescence.

Failure of normal endochondral bone growth in the cranial base is manifested in achondroplasia, caused from a mutation in FGFR3 gene, that usually results in midface deficiency associated with a class III malocclusion (Figure 53-13). Premature fusion of multiple sutures lateral to the cranial base midline may occur with some congenital malformations that also result in midface deficiency, such as craniofacial dysostosis (Crouzon syndrome) or acrocephalosyndactyly (Apert syndrome) both of which are caused from a mutation in the *FGFR2* gene (Figure 53-14).

Nasomaxilla

The prenatal precocity of neurocranial growth relative to the face becomes less predominant postnatally. Nevertheless, considerable postnatal displacement of the nasomaxilla downward and forward occurs owing to continued growth of the brain and cranial base. This inferior and anterior maxillary transposition is augmented by the sutural growth between the cranial base and the maxilla and growth of the nasal septum. After birth, the vertical growth of the maxilla continues with contributions from the frontomaxillary, frontonasal, frontozygomatic, frontoethmoidal, ethmoidomaxillary sutures, and possibly, the nasal septum (Figure 53-15). The vertical descent of the maxilla is further increased by modeling with resorption on the nasal surfaces and the simultaneous apposition on the oral surfaces. The zygomas are repositioned down and forward with resorption on their internal surfaces and apposition on their external surfaces. Anteroposterior growth continues with temporozygomatic and nasomaxillary sutural growth and transverse growth from intermaxillary and zygomaticomaxillary sutures. The resulting downward and forward translation displaces



FIGURE 53-13. A 10-year and 3-month-old female with achondroplasia, characterized by short stature and midface deficiency from failure of normal primary cartilage growth. The severity of the skeletal discrepancy from the deficient cranial base growth usually results in class III malocclusion but is not present here. **A,** Frontal view. **B,** Three-quarter view. **C,** Profile view. **D,** Lateral cephalometric radiograph.



FIGURE 53-14. A 7-year and 1-month-old male with craniofacial dysostosis (Crouzon syndrome), characterized by midface deficiency from multiple premature suture synostoses and abnormal cranial base growth. This can result in significant optic and nasal airway functional issues as well as intracranial pressure and a class III malocclusion. This patient had a frontal bone advancement at 14 months old to relieve intracranial pressure. **A,** Frontal view. **B,** Three-quarter view. **C,** Profile view. **D,** Lateral cephalometric radiograph.

adjacent bones and permits adequate space for the developing nasopharnyx and appositional growth at the posterior aspect of the maxilla and maxillary tuberosities to provide additional posterior alveolar space for the development and eruption of the maxillary molars. Following the postnatal growth of the facial sutures, they serve as sites of fibrous union where some remodeling still can take place. In fact, a number of cranial and facial sutures are interdigitated but still not fused even beyond 50 years of age.

Growth determinants for postnatal nasomaxillary growth are not well understood and include a variety of intrinsic and extrinsic factors. Passive displacement secondary to genetic intrinsic brain and cranial base growth and, perhaps, nasal septal growth guidance are the most significant influences on the downward and forward movements of the maxilla after birth until about the seventh postnatal year when most neural growth is complete. From that age through adolescence, these influences dramatically decrease, accounting for about

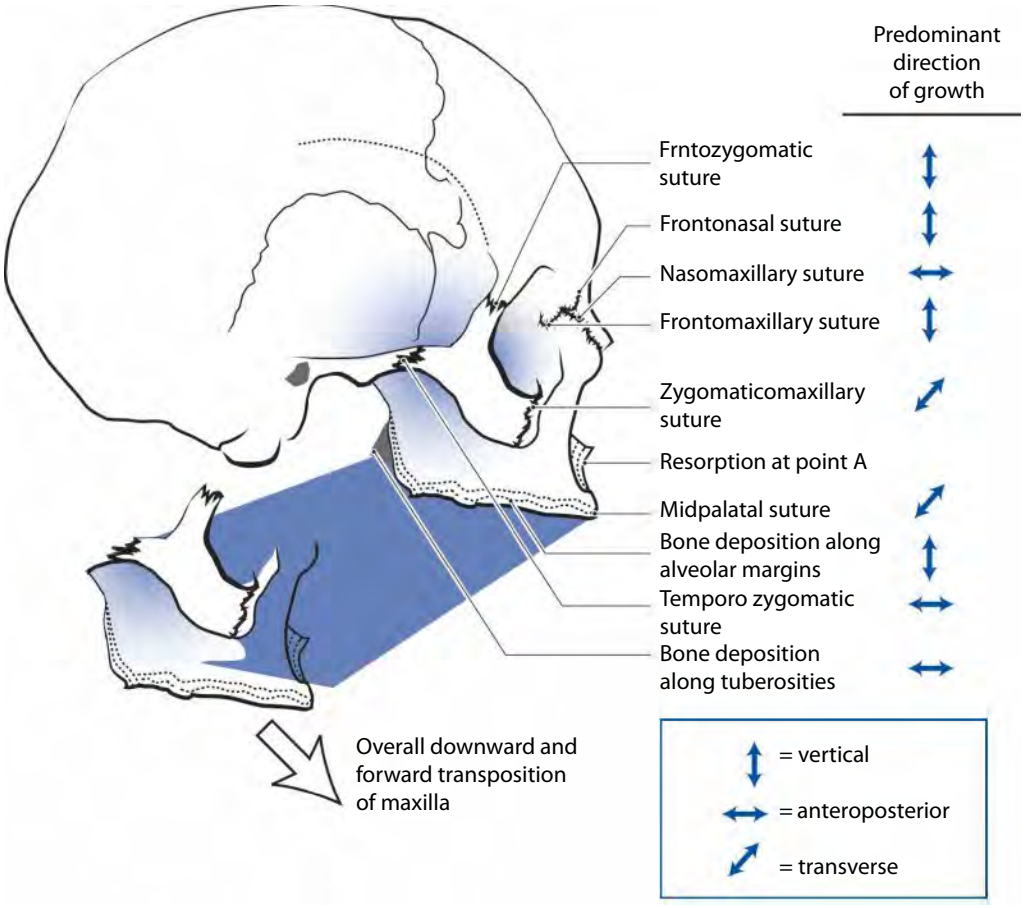


FIGURE 53-15. Nasomaxillary intramembranous growth at various sites, resulting in an overall downward and forward transposition of the maxilla relative to the cranial base. Adapted from Sperber G. Craniofacial development. Hamilton (ON): BC Decker Inc; 2001. p. 107.

one third of forward maxillary movement, as intramembranous epigenetic extrinsic sutural growth and surface modeling predominate. Maxillary growth also depends to a great extent on various functional matrices. In addition to the effect of brain growth on the maxillary position, there is the influence of eyeball growth and their functional movement on the size and shape of the orbits, the influence of respiration on the nasal cavity, the influence of oral function in determining tuberosity, palatal and alveolar development, and the surrounding facial soft tissues, all of which contribute functional roles in determining the extent of sutural growth and modeling of the nasomaxilla.[56]

Although the mode of nasomaxillary growth is predominantly intramembranous from sutural growth and surface remodeling, some endochondral bone growth takes place in the nasal septum, made up of secondary cartilage. As mentioned previously, the growth mechanism is mostly the nasomaxilla being pushed from behind by the brain and cranial base growth until about age 7, when a different mechanism begins to predominate whereby the nasomaxilla is pulled forward by soft tissue and probably to a small extent from nasal cartilage growth with concomitant growth of circumaxillary sutures. Significant modeling must occur in order to maintain the general shape of the maxilla as it is displaced downward. As mentioned previously, resorption of the nasal side of the maxilla, providing nasal cavity enlargement, occurs concomitantly with apposition on the oral side, resulting in descent of the maxilla. This descent, together with variable amounts of dental eruption, is reflected as external rotation of the maxilla. The maxilla undergoes a modest and variable amount of internal rotation as it remodels that can result in slight forward or backward rotation but usually is equal to the external rotation, resulting in a zero net rotational change. However, the cumulative rotation may be significant in individuals with short or long face types.[57]

Although secondary pneumatization of the maxillary sinus begins prenatally, it does not occur for the other paranasal sinuses until after birth (first 2 postnatal years for ethmoidal and frontal sinuses and the 6–7 postnatal years for sphenoidal sinuses). The vertical growth of the maxillary alveolar process is rapid during dental eruption, surpassing the vertical descent of the palate threefold. This alveolar development provides a large contribution to the depth and width of the palate and vertical height of the face. Considerable resorption of the anterior surface of the maxilla minimizes the overall forward displacement of the maxilla by about 25% and creates a deeper supra-alveolar concavity while increasing the relative prominence of the anterior nasal

spine. Transverse growth occurs by lateral displacement of the maxillary bodies by means of the midpalatal suture and bone resorption on the lateral borders of the nasal cavity. Transverse development of the maxillary alveolar process continues with buccal eruption of the posterior teeth. Growth of the midpalatal suture ends after the first 2 postnatal years, but the suture remains patent until late adolescence, with fusion usually not being complete until the third decade.

Preadolescent and adolescent nasal growth increases on average at a rate about 25% greater than maxillary growth. The mechanism of nasal growth is a combination of an endochondral component from the nasal cartilage and an intramembranous component from sutural growth behind the nose. Although the nasal bone grows up until about age 10 with frontonasal sutural apposition and modeling, interstitial nasal cartilage growth continues increasing the overall size beyond adolescence and throughout adulthood.

Mandible

The mandible has the most delayed growth and the most postnatal growth of all the facial bones. Although usually in a retrognathic position relative to the maxilla at birth, rapid postnatal growth corrects this discrepancy. The right and left bodies of the mandible are still separate at birth, uniting at the midline mental symphysis during the first year of life. There are no intrinsic centers of mandibular growth. The primary sites of mandibular postnatal growth are the endochondral apposition occurring at the condylar cartilages and extensive intramembranous surface modeling. This modeling includes, but is not limited to, apposition on the posterior and lateral aspects of the rami and the posterior alveolar ridges as well as resorption of the anterior surface of the condyles, the anterior contours of the rami, and the inner surface of the mandibular bodies (Figure 53-16). The accentuation of chin prominence that typically occurs results primarily from facial resorption between the chin and the base of the mandibular alveolus rather than apposition on the chin itself. The growth of the condylar cartilage contributes most of the total ramus height, whereas growth of alveolar bone contributes aproximately 60% to the mandibular body height.[58] Proliferation of condylar cartilage, in response to growth and function of muscles and adjacent soft tissue, results in superior and posterior growth of the condylar heads, displacing the mandible downward and forward in concert with the maxilla. Condylar growth appears to involve the sequential involvement of transcription factor SOX9, expressed by chondrocytes, which regulates the synthesis of type II collagen, type X collagen secreted as matrix, and vascular endothelial growth factor secreted to regulate the neovascularization of the cartilage.[59]

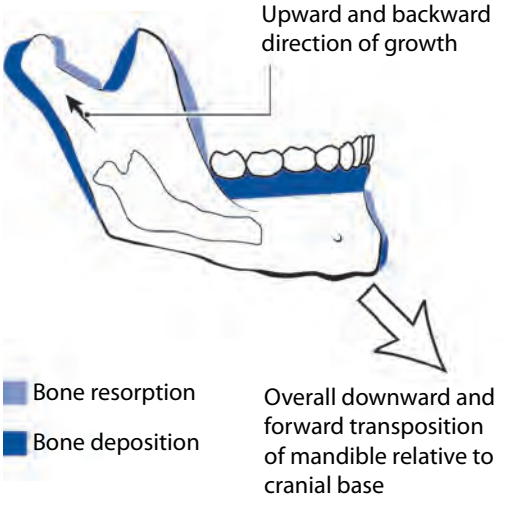


FIGURE 53-16. Mandibular intramembranous and endochondral growth, resulting in an overall downward and forward transposition of mandible relative to cranial base. An outline of the fetal mandible is superimposed on the adult mandible for size and shape comparison. Adapted from Sperber G. Craniofacial development. Hamilton (ON): BC Decker Inc; 2001. p. 130.

At birth, the inclination of the mandibular condyles is more horizontal, resulting in a greater increase in length than height. During childhood, the inclination becomes more vertical so that condylar growth results in a greater increase in height than length. However, there is great variability in this inclination within the general population, influencing the degree to which the mandibular growth is expressed in a forward anteriorly rotating, as opposed to downward posteriorly rotating, direction. Simultaneous modeling of the inferior mandibular border with resorption at the gonial angle and minimal apposition of the anterior aspect of the lower border tends to reduce the effect of this rotation on facial morphology. The final rotational position of the mandible with growth is a result of the combined influences of external mandibular rotation from downward nasomaxillary growth and dental eruption and the internal mandibular rotation from compensatory modeling. Internal mandibular rotation is variable but is typically 15 degrees whereas external mandibular rotation averages 11 to 12 degrees, producing an overall forward rotation of 3 to 4 degrees.[57,60] This overall rotation tends to decrease with greater internal rotation in individuals with short faces, resulting in a deep anterior overbite. With the long face individual, the internal rotation is decreased or even backward internal rotation may occur, often resulting in an anterior open bite and mandibular deficiency.

The mandible has its greatest postnatal growth velocity in the first 6 months after birth with the greatest change in mandibular length, followed by ramus height, then corpus length.[61] Although minimal maxillary growth occurs after approximately 10 years of age, mandibular growth continues longer, to the end of adolescent growth. This differential growth, typically characterized by a peak in the rate of mandibular growth at puberty, usually results in the final correction of the mandibular position relative to the maxilla. Longitudinal growth studies have demonstrated that there are a substantial number of individuals, more commonly females, who demonstrate an early acceleration in mandibular growth that can occur during preadolescence.[62] This "juvenile" acceleration may equal or even exceed the later acceleration during puberty.

If the cranial base is the craniofacial skeletal unit whose growth is least determined by extrinsic epigenetic or environmental factors, the mandible is the opposite extreme with its growth highly dependent on the postnatal functional demands placed on it. It has a great capacity to adapt to mandibular displacement and accommodate to lingual and labial soft tissue, resting postural and active muscular function, and mastication. The condylar inclination, dictating the type of mandibular growth rotation, is determined by these pulling secondary functional influences. The relevant functional matrices include the lateral pterygoid attached to the condylar neck, the growth and function of the tongue, the masticatory muscles attached to the buccal and lingual aspects and to the coronoid process, and the facial soft tissue and musculature, all influencing the ultimate size and shape of the mandible. For this reason, the mandible, more than any other part of the craniofacial complex, may have a low intrinsic growth potential that is significantly increased and regulated in response to extrinsic functional demand. This mandibular growth dependence on function is clearly reflected in clinical conditions in which mandibular growth has been restricted from persistent unilateral or bilateral limited mandibular function owing to a congenital disorder, postnatal trauma, or pathology. It is just as evident in clinical conditions in which mandibular growth is enhanced in the presence of a postnatal pathology in which persistant lingual soft tissue affects the size or resting position of the tongue or a pituitary adenoma increases skeletal and soft tissue growth (Figures 53-17 and 53-18).

Dental Development

The alveolar processes contribute a great portion of the vertical height of the lower face. Their development is entirely dependent on the presence and eruption of the primary and



FIGURE 53-17. A 10-year and 4-month-old female with a lymphatic malformation of the floor of the mouth and tongue, with an elevated and enlarged tongue, creating a class III malocclusion with mandibular prognathism, anterior and posterior open bites, and an expanded mandibular arch from the lingual soft tissue mass and additional mandibular condylar growth. **A,** Frontal view. **B,** Three-quarter view. **C,** Profile view. **D,** Lateral cephalometric radiograph.



FIGURE 53-18. A 33-year and 3-month-old male with acromegaly (adult hyperpituitarism), with mandibular prognathism and coarse facial features from additional intramembranous and mandibular condylar endochondral skeletal growth as well as soft tissue growth. This condition results in a class III malocclusion and an expanded mandibular arch from tongue and mandibular condylar growth. **A,** Frontal view. **B,** Three-quarter view. **C,** Profile view. **D,** Lateral cephalometric radiograph.

permanent dentition. Just as vertical appositional alveolar growth accompanies vertical dental eruption, transverse apposition complements transverse dental eruption. This minor contribution to the transverse dimension of the alveolar processes continues until about 7 years of age, with eruption of the permanent incisors. Further transverse dentoalveolar growth is minimal, occurring with eruption of the premolars and canines. Facial growth and the concomitant increase in the size of the jaws occur posteriorly, creating additional space for the dentition only in the molar region.

Eruption of the maxillary teeth enhances the vertical dimension of the maxilla with posterior development of the maxillary tuberosities to accommodate the development and eruption of the maxillary posterior teeth. In the mandible, progressive resorption of the anterior ramal surfaces and apposition of the posterior ramal surfaces provide alveolar space for the sequential development and eruption of the mandibular posterior teeth. Eruption of the mandibular teeth enhances the vertical growth of the mandible and also contributes to the height of the face. However, compensatory condylar growth and internal mandibular rotational modeling must occur to prevent the mandible from rotating posteriorly as the maxilla grows downward and the dentition erupts. Dental emergence into the oral cavity begins at approximately the sixth postnatal month, and the primary dentition is established by 2.5 years of age. The primary incisors begin to exfoliate at 6 to 7 years of age, and the permanent dentition begins to emerge with eruption of the mandibular incisors and first molars. The permanent dentition is established by 12 to 14 years of age except for the eruption of the third molars, contributing to the vertical dimension of the lower face during adolescent growth.

The anteroposterior eruption of the permanent incisors is affected by the facial growth pattern.[63a] Individuals with class III facial growth tend to have minimal anterior and some distal mandibular dental eruption, whereas class II facial growth

results in greater anterior mandibular dental eruption to compensate for the underlying anteroposterior skeletal discrepancies. Short facial growth pattern tends to promote anterior mandibular rotation with increased incisor uprighting and deepening of the anterior overbite, whereas long facial growth pattern results in posterior mandibular rotation with increased incisor protrusion and opening of the anterior overbite.[63]

Facial Development

The growth of the facial soft tissue follows the underlying facial bones but is not directly correlated with bone growth. Facial soft tissue is thicker relative to the underlying skeletal tissue in the young child owing to subcutaneous fat. This is one of the reasons it is more challenging to assess potential underlying skeletal discrepancies in the young child based only on a clinical appraisal. The thicker soft tissue envelope, together with the relative retrognathic position of the mandible, creates a more convex profile in infancy and early childhood. Lip thickness increases until it reaches a maximum at the end of the pubertal growth spurt, then decreases in late teens and adulthood.[64,65] These later changes, combined with continued forward nasal growth as well as anterior mandibular and chin projection, leave the lips with a more retrusive appearance and the nose and chin with a more prominent appearance. These changes usually create a flatter facial profile in older adolescents and adults. This tendency is even greater on average in males than females, owing to less subcutaneous fat, combined with more nasal cartilage growth and anterior mandibular and chin projection in males.

The facial soft tissue also follows the cephalocaudal growth gradient, with the soft tissue of the lower face growing more in magnitude and duration than the upper face. Before adolescence, vertical lip growth lags behind skeletal jaw growth, resulting in lack of resting lip apposition. During and after pubertal growth, vertical lip growth increases proportional to the underlying vertical skeletal growth, creating a more likely chance of resting lip apposition in adults.[66]

There is significant growth in the length and prominence of the nose during adolescence, influencing the facial balance between the nose, the lips, and the chin.[67] In fact, the vertical nasal growth is much greater than anteroposterior or transverse nasal growth. Nasal growth during adolescence is primarily limited to cartilage and soft tissue because the nasal bone usually has completed growth before puberty. The nasal shape often changes before adolescence, with the upper nasal dorsum developing superiorly and anteriorly, and the lower nasal dorsum more often following the lower facial growth pattern. In other words, individuals who have a more anterior and superior rotational pattern of lower face growth will exhibit a similar rotation of the lower nasal dorsum. There is some evidence that skeletal class II jaw relationships usually demonstrate a more prominent nasal bridge and convex dorsum than balanced jaw relationships.[68]

The upper third of the face grows the most rapidly early in life owing to brain growth and achieves its ultimate size earliest, finishing most growth by 12 years of age. Orbital height already reaches 55% of its adult height at birth and 94% by 7 years of age.[69] The middle and lower thirds of the face are less affected by brain growth, growing more slowly and for a longer time. Most of the middle third growth is completed later during puberty, with the lower third of the face continuing to grow beyond puberty into adulthood.

In addition to this vertical sequential growth gradient, craniofacial growth does not take place at an equal rate in the three planes of space. Facial growth follows a sequence in which transverse growth finishes first, followed by anteroposterior and finally vertical growth, although all three continue to some extent during adulthood.[70] The face reflects the early transverse neural expansion of the cranium, the early fusion of the mandibular symphysis, and the early growth cessation of the midpalatal suture during the first few years of life. The width of both jaws, including the dental arches, is largely completed before adolescent growth, with the exception of dental arches achieving greater width posteriorly owing to continued increase in jaw length. This presents clinically as a disproportionately wide face relative to the height in the infant and young child. Both jaws continue to grow in length and height throughout pubertal growth. As the maxilla and mandible displace and grow downward and forward, the anteroposterior and vertical growth begin to take proportionately greater roles. The growth rate of the maxilla slows down after approximately 10 years of age and, together with anterior maxillary resorption, reduces the relative anterior projection of the midface. The maxillary length reaches maturity before the upper facial height, which is followed by mandibular length and finally ramus height.[71] The somewhat retrognathic position of the mandible at birth usually is corrected early in postnatal life. The mandible grows for a longer duration than the maxilla, typically undergoing a growth spurt at puberty. Anteroposterior growth is accompanied and then followed by vertical facial growth, primarily in the mandible, often continuing well beyond puberty, even into the third and fourth decades.

There are gender differences in facial growth, with males characteristically having volume changes of greater magnitude than females. Females have much less nasal growth on average, with many not even exhibiting a pubertal nasal growth spurt, in contrast to males who characteristically have a nasal growth spurt throughout puberty. Females have earlier soft tissue growth that follows their earlier puberty and they have greater lip thickness at all ages. The flattening of the facial profile during adolescence is less dramatic in females, owing in part to their fuller lips and less prominent noses but also owing to females having less forward mandibular growth projection and chin growth. Females have on average more late vertical maxillary growth than males. If mandibular growth is not matching these late maxillary changes, the mandible translates downward and backward, resulting in a more convex profile. Not only are male facial volume changes on average greater in magnitude, but the duration of the changes is longer and there is more predomi-

nance of volume increase in the lower third of the face.[72] Males are on average more likely to have late mandibular growth that may be beneficial in improving a maxillary protrusion or mandibular retrusion but is disadvantageous when a mandibular prognathism or maxillary retrusion is present before late growth.

Growth and Facial Changes during Adulthood

There has been awareness since the late 19th century that human growth continues beyond adolescence, at least until the fourth or fifth decade of life.[73] Nevertheless, investigators in the mid-20th century were surprised to find that facial growth continues into the sixth decade of life.[74] Later, it was found that the craniofacial complex models throughout adulthood, with thickening of the frontal region of the cranium and a symmetrical modest increase in the size of the cranium, cranial base, maxilla, and mandible.[75,76]

In the late 20th century and early 21st century, a number of longitudinal craniofacial growth studies have examined changes during adulthood.[77–84] Evaluation of the serial cephalometric radiographs in these studies revealed that craniofacial growth continues in all three dimensions, similar to the growth pattern and changes seen during adolescence, but of a much lesser magnitude and rate. Vertical increases are more prominent than anteroposterior changes with the least changes in width. The cephalocaudal gradient continues with more mandibular than maxillary changes. There is evidence of continued jaw rotation and dental eruption with the vertical changes. Females grow less and their craniofacial growth is expressed more vertically with posterior mandibular rotation and an increased mandibular plane angle, whereas males tend to grow with anterior mandibular rotation during adulthood, thereby straightening their profile and decreasing the mandibular plane angle (Figure 53-19).

It appears that there is a subtle growth acceleration in females during their 20s that may be attributable to the endocrine influence of their first pregnancy. Late mandibular incisor crowding usually develops as these incisors are positioned distally relative to the mandible owing to late mandibular growth. Accompanying the skeletal changes throughout adulthood, dental arches continue to change as well with decreasing arch width and depth, further increasing mandibular incisor irregularity.[85]

Facial soft tissue changes during adulthood are much larger in magnitude than changes in hard tissue. Typical lip changes during adulthood include less prominence with decreased thickness, thinning of the vermilion and flattening of its contour (more so in the upper than the lower lip), increased upper lip vertical length, and less prominence of the philtral columns. Male lips continue to appear more retrusive with age, whereas female lips generally do not become as retrusive and their lower lip thickness tends to slightly increase. The lips become positioned more inferiorly, resulting in less vertical display of maxillary incisors, more vertical display of mandibular incisors, and less lip separation with age.[86] Glabella moves forward and downward. The nose and ears continue to increase in size in all dimensions, with the nasal tip and columella dropping inferiorly to create a more acute nasolabial angle, with all these features occurring to a greater extent in males. There is deepening of the nasolabial folds with jowling developing and the oral commissures tend to sag inferiorly with the loss of elasticity.[87] Males tend to exhibit more chin prominence owing to continued soft tissue increase. The cervicomental angle usually becomes more obtuse from sagging soft tissue that is accentuated with submental fat deposition.[88]

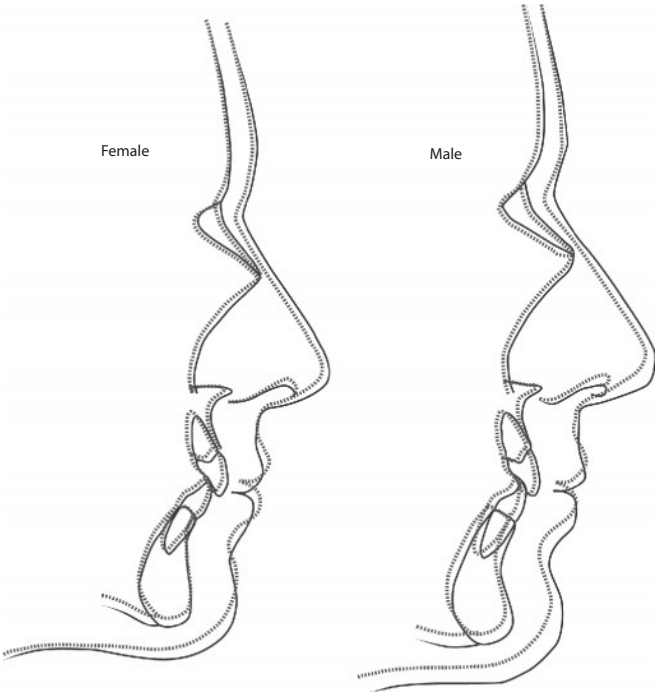


FIGURE 53-19. Composite lateral cephalometric views of female and male show mean longitudinal growth changes from young adult (*broken line* at mean age 17) to middle age adult (*solid line* at mean age 47–51). Note continued downward and forward skeletal and soft tissue growth with relative flattening of the lips. Adapted from Behrents RG Growth in the aging craniofacial skeleton. Craniofacial growth monograph series. Ann Arbor (MI): Center for Human Growth and Development, University of Michigan; 1985.

It is clear that while the great preponderance of craniofacial growth subsides after the completion of sexual maturity at the end of adolescence, growth continues on a small scale in all three planes throughout adulthood, following the adolescent pattern. Essentially, craniofacial growth must be viewed as an ongoing process throughout life with postadolescent transverse growth being minimal, followed by anteroposterior growth, with vertical growth being the greatest during adulthood.[70] Our improved understanding of the aging face should compel orthodontists and surgeons to carefully consider their treatment procedures in terms of their future cosmetic effects on the aging face. It is wise to attempt to increase dental and skeletal volume to tighten the facial soft tissue when possible. Any treatment that deepens nasolabial folds or decreases lower face height, lip projection, or

vertical maxillary incisor display should be avoided. If any of these adverse changes are unavoidable in order to achieve an acceptable dental occlusion, secondary soft tissue cosmetic procedures may ultimately be considered.

The biologic regulator mechanism for initiating and directing craniofacial growth and dental eruption timing, pattern, and rate remains a poorly understood phenomenon. It is clear that it is a complex mechanism, influenced by an intricate interaction of genetic, epigenetic, and local environmental factors.

FACTORS INFLUENCING CRANIOFACIAL GROWTH

Craniofacial growth is a complex process influenced by both prenatal and postnatal genetic, epigenetic, and environmental factors. The principal influence on craniofacial growth and morphogenesis is one of multifactorial genetic control. However, the interaction of this genetic control with environmental factors is a complex one, and it is usually impossible to accurately differentiate between these influences.

Prenatal Factors

Prenatal defects of craniofacial development can be classified conveniently into three categories: (1) malformation—a morphologic defect of an organ, part of an organ, or larger region of the body resulting from an intrinsically abnormal developmental process, which is intrinsically determined owing to the genome or a teratogen, and occurs during the embryonic period; (2) deformation—an abnormal form, shape, or position of a part of the body caused by mechanical forces, which is influenced directly by the fetal environment; and (3) disruption—a morphologic defect of an organ, part of an organ, or a larger region of the body resulting from the extrinsic breakdown of, or an interference with, an originally normal developmental process, which also occurs during the fetal period and may result from intrauterine pressure as well, but can be of metabolic, vascular, and/or teratogenic origin.[89]

Genetic

Craniofacial malformations arise from disturbance in morphogenesis as early as the germ layer formation to the final formation of organ systems at the end of the embryonic period. Four to 8 weeks' gestation is a particularly critical time because this is the period when neural crest migration is at its most active, the facial prominences and dental laminae are forming, and neurovascular bundles are being generated before facial bone ossification. Malformations are caused from chromosome abnormalities or single-gene mutations or are multifactorial (genetic and/or teratogenic) in origin. Growth retardation, premature death, and mental retardation seem to be more frequent in autosomal recessive or X-linked syndromes.[90] Craniofacial malformations range from acephaly to mild facial defects such as a microform cleft or notching of the lip. Cranial malformations include premature or delayed fusion of the cranial sutures due to mutations in FGFR1, FGFR2 and FGFR3 and the transcription factor MSX2 associated with syndromes such as trisomy 21 and cleidocraniodysostosis or with simply a deformation craniosynostosis.[52,91] Cranial base malformations usually are related to malformations that affect primary cartilage growth such as achondroplasia (see Figure 53-13) or in combination with premature fusion of facial and cranial sutures as with Apert, Crouzon, and Pfeiffer syndromes (see Figure 53-14).[92] Many facial malformations originate from a deficiency, incomplete migration, or failure in cytodifferentiation of neural crest tissue during embryogenesis.[93] The result is a failure in normal formation of the skeletal and connective tissue portions of the facial prominences. Nasomaxillary malformations include deficiencies and/or absence of facial bones that occur in ectodermal dysplasia or mandibulofacial dysostosis, as well as facial clefts that are associated with over 250 syndromes. The most common craniofacial malformation is unilateral cleft lip, affecting 1 in 700 to 800 births. Malformations that affect the mandible range from the rare absence (agnathia) to various forms of micrognathia, associated with a number of syndromes, such as mandibulofacial dysostosis (Treacher Collins syndrome) or Turner syndrome, to macrognathia, associated with hyperpituitarism or hemifacial hypertrophy.

Two more common chromosomal disorders that result in growth retardation are Down syndrome and Turner syndrome, both of which are characterized by short stature and brachycephaly. The protruding resting tongue typical of Down syndrome usually results in an anterior open bite, whereas a narrow, high-arched palate often is seen with Turner syndrome. Triple X syndrome is characterized by overall small craniofacial dimensions.[94] The Russell-Silver syndrome is a chromosomal disorder characterized by poor fetal and postnatal growth and small triangular facies. Other syndromes associated with prenatal growth retardation include Bloom syndrome, de Lange syndrome, leprechaunism (mutations of the insulin receptor gene), Ellis-van Creveld syndrome, Aarskog syndrome, Rubenstein-Taybi syndrome, Perheentupa syndrome, Dubowitz syndrome, and Johanson Blizzard syndrome.[95]

Single-gene disorders that result in fetal overgrowth include Sotos syndrome, Weaver syndrome, and Beckwith-Wiedemann syndrome. Sotos syndrome includes craniofacial features of macrocephaly, dolichocephaly, a prominent forehead, hypertelorism, prominent ears, high-arched palate, and mandibular prognathism. The Beckwith-Wiedemann syndrome, an example of uniparental disomy, is associated with excessive somatic and specific organ growth (e.g., macroglossia) apparently caused by excess IGF-II. In spite of the overgrowth with these disorders that extends from the fetal period into early childhood, both lead to early epiphyseal fusion, resulting in adult short stature. Klinefelter syndrome (XXY) is a chromosomal disorder that leads to postnatal extended growth from pubertal failure, resulting in tall adult stature.

Another example of a single-gene growth disorder is achondroplasia (see Figure 53-13), the most common form of human dwarfism, which is autosomal dominant with complete penetrance, involving mutations in the *FGFR3* gene. Because the primary cartilage of the cranial base synchondroses is affected, and not the secondary cartilage of the mandibular condyles, midfacial hypoplasia resulting in a class III skeletal discrepancy is the usual facial outcome. A single-gene disorder that leads to postnatal overgrowth resulting in tall adult stature is Marfan syndrome.

It is fortunate that embryologic defects are relatively rare in spite of the extensive number of possibilities. It is estimated that the number of children who have an embryologic disturbance as the major contributing factor to their malocclusion is less than 1%.[96]

There appears to be more evolutionary gene conservation for mammalian organ systems, including humans. The coding genomes vary little between mammals with humans differing by less than 2% with chimpanzees and only 25% with mice.[97,98] There only are subtle differences in the shape and size of human organ systems because appreciable differences can compromise optimal organ function. In contrast, facial characteristics seem to be characterized by more diversity owing to genetic, epigenetic, and environmental factors. Facial structures certainly need to be present and in proper location for proper function, but there can be significant facial morphology variability without impairing function. No two faces are alike, and this variability may be more necessary in humans than other animals owing to the human dependence on vision, more than other senses, to identify one another.

Environmental

Prenatal environmental growth factors are those not directly determined by the genome, including cytoplasmic and extracellular contents in the embryo or fetus and the placenta, influenced by the mother and her interaction with the external environment. Some of these environmental factors may be internal (such as focal embryonic hemorrhages) or external (from maternal malnutrition, metabolic factors, and disease, or exposure to pollutants, chemicals, drugs, infectious agents, or radiation) and may impair normal growth or act as teratogens during either the embryonic or the fetal period if the maternal exposure is large or frequent enough (Figure 53-20).

Cytomegalovirus and rubella are examples of pathogens that can cause microcephaly, hydrocephaly, and microphthalmia. Glucocorticoids, phenytoin, ethyl alcohol, tobacco

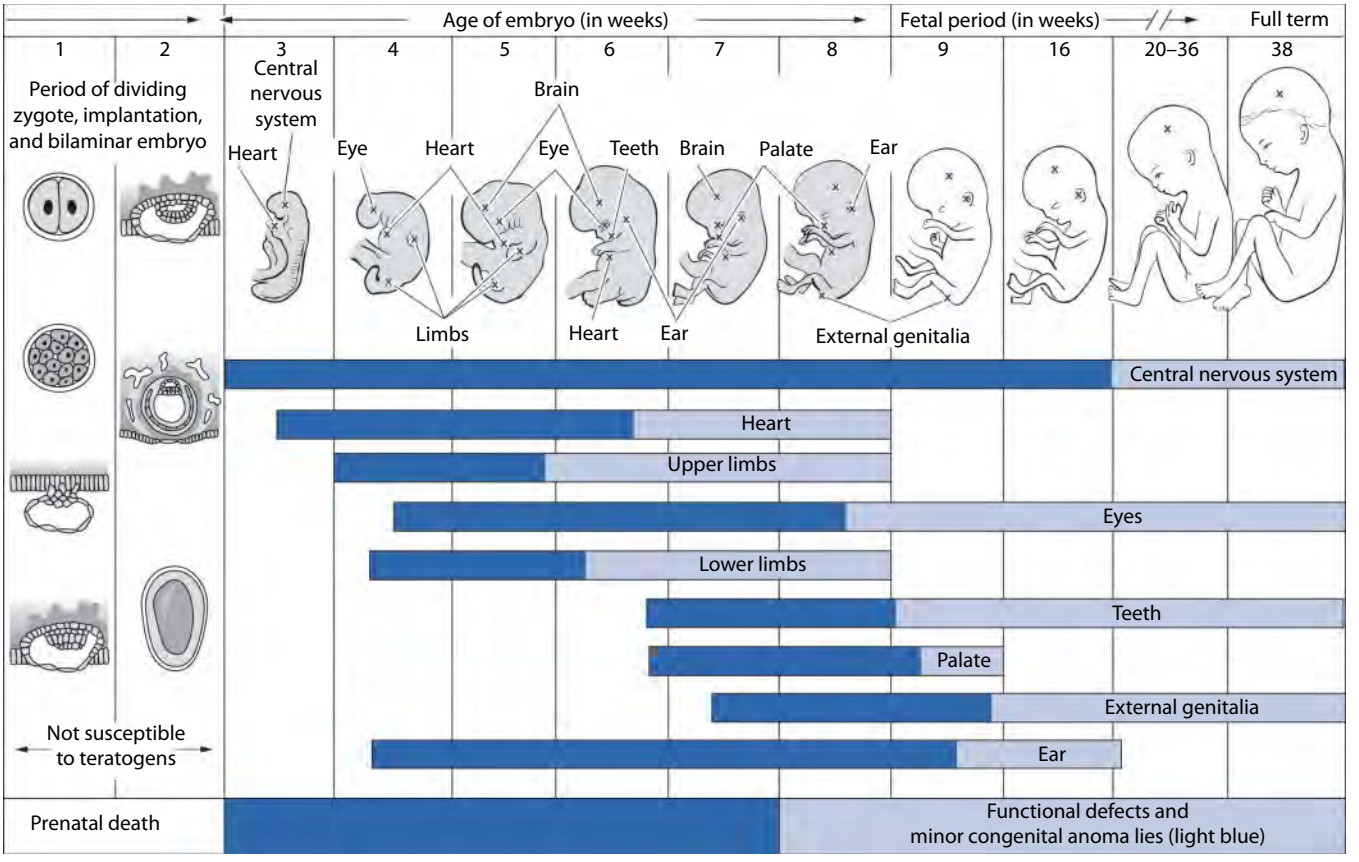


FIGURE 53-20. Teratogen susceptibility during periods of prenatal human development. During the first 2 weeks' gestation, damage from teratogen results in death of the conceptus, or damages only a few cells, allowing recovery by the embryo to develop without birth defects. *Dark blue* indicates highly sensitive periods when major defects may be produced (e.g., amelia, absence of limbs). *Light blue* indicates less sensitive time periods when minor defects may be produced (e.g., hypoplastic thumbs). Adapted from Moore KL, Persaud TVN. The developing human: clinically oriented embryology. 5th ed. Philadelphia (PA): W.B. Saunders Co.; 1973. p. 156.

smoke, aspirin, and retinoic acid (a vitamin A metabolite) are examples of an ever-increasing number of substances that are being identified as teratogens, causing cleft lip and palate as well as other craniofacial anomalies.[99] Fetal alchohol syndrome, characterized in part by maxillary and midface deficiency, has been diagnosed with disturbing frequency, now estimated at 1 in 750 births in the United States.[100] Teratogens have distinct mechanisms of action and are selective to certain target cells, but the severity of the resulting malformation is variable. It is speculated that the range of phenotypic effects caused by a teratogen is due to factors that include the concentration or method of delivery, the timing and duration of exposure, variations in susceptibility, and synergistic interactions among teratogenic compounds.[101] Even in the absence of any detectable malformations, serious long-term physical and mental development can result from drug intake during pregnancy.

When the fetal period begins during 9 weeks' gestation, environmental factors can still have a profound growth effect on the developing fetus. Maternal malnutrition adversely affects fetal growth.[102] Maternal diet composition is relevant, with a high-protein diet being associated with increased linear fetal growth and a high-fat diet linked to an increased birth weight. Maternal consumption of alcohol, recreational drugs, or tobacco all have an important negative influence on growth in utero as well as during the first year of life.[103–105] Even maternal exposure to passive tobacco smoke reduces fetal growth.[106] Frequent high maternal noise exposure has been shown to adversely affect prenatal growth, perhaps related to the stress imposed.[107] Maternal pathology such as rubella can cause a prenatal growth deficit with no long-term recovery.[108]

Intrauterine pressures can result in deformations or disruptions. Intrauterine restrictions can result in mild to severe deformations that can present as mild facial or cranial asymmetry. Some isolated forms of craniosynostosis, causing cranial deformations such as plagiocephaly, may be caused from intrauterine mechanical factors.[109] These deformations may resolve after birth with catch-up growth but usually require orthopedic or surgical intervention during infancy. Another deformation is the Robin sequence whereby retrognathia from posterior restraint of the mandible forces the developing tongue into a posterior position, often acting as a mechanical obstruction that prevents elevation of the palatal shelves, resulting in an isolated cleft palate. Severely affected newborns can have airway compromise that has been more recently treated by advancing the deficient mandible with distraction osteogenesis to avoid the need for a tracheostomy. A disruption is a typically more serious anomaly than a deformation, from the standpoint of both treatment and future growth, because it presents as a morphologic and functional defect that requires surgical repair. An example of a disruption is when a strand of torn amnion or amniotic band is swallowed by the fetus, resulting in a facial cleft that is not located at a site of embryonic fusion. There can be congenital absence of mandibular elevator muscles from unknown causes, resulting in growth deficiency affecting both the maxilla and the mandible on the affected side.

The hormonal regulation of fetal growth is not well understood. Fetal androgens appear to be growth promoters. At midgestation, the level of gonadotropin is similar to pubertal levels.[110] Although there is some evidence that estrogen promotes fetal bone development, there are also data that suggest that it inhibits fetal growth.[111,112] There is a marked and progressive increase in prolactin during late gestation. Before 12 weeks' gestation, maternal hypothyroidism can have long-term deleterious effects on hearing and intelligence, but neither maternal nor fetal hypothyroidism has an appreciable effect on fetal length or weight. However, when both conditions are present, linear growth is still unaffected but there is incomplete pulmonary, cardiovascular, and skeletal maturation.[113] Although poorly understood, insulin appears to have an important role in regulation and promotion of fetal growth. Maternal diabetes increases fetal length and weight, whereas fetal insulin deficiency results in decreased length and weight at birth.[114]

Although growth hormone (GH) is essential for postnatal growth, growth in utero and probably in the first 2 years of life is largely GH-independent.[115] Nevertheless IGF-I and -II play important roles in determining fetal growth, but the specific nature of these roles are not well understood. IGF-II is important in supporting fetal growth during early gestation, whereas IGF-I has a greater role during later gestation and especially in postnatal life.[116] The fetal roles of growth factors such as nerve, epidermal, and platelet-derived growth factors also remain unclear. Other fetal growth factors include hematopoietic growth factors, FGFs, vascular endothelial growth factor, and members of the TGF-β family.[117]

The placenta functions as an additional endocrine organ, providing a secondary source of hypothalamic, pituitary, adrenal, and gonadal hormones and growth factors.[118] Placental GH and lactogen can alter the production of maternal IGF-I.[116] Maternal IGF-I in turn affects placental nutrient transport, increasing fetal growth.[119] Lactogen regulates maternal glucose, amino acid, and lipid metabolism, facilitating nutrient transport to the fetus. Disruption of placental GH or lactogen production can occur from vascular disease, infection, or intrinsic placental abnormalities, impairing fetal growth.[120]

Postnatal Factors

The size of infants in the first months of life is more related to the prenatal environment than is parental height. If prenatal factors caused only mild growth attenuation and it occurred during the last trimester, postnatal catch-up growth is feasible.

Genetic

Heritability appears to have an effect on somatic growth, from a greater to lesser extent in the following order: skeletal length, skeletal breadth, weight, circumference, and skin folds.

By the same token, skeletal tissues respond less to changes in the nutritional environment than do soft tissues.[121] The timing and pace of postnatal maturation are also genetically controlled to a large degree. The extent to which heredity is the cause of postnatal growth that results in jaw discrepancies is controversial.[122] Whereas it appears that there is a substantial genetic component to skeletal characteristics, it apparently is low for dental characteristics.[123,124] There is evidence that malocclusions have shown modest increases in prevalence for the last millennium, with more significant increases during the last 150 years.[125,126] Contrary to the popular belief that greater ethnic and racial diversity are principally responsible for the prevalence of jaw discrepancies, population diversity results in only a modest additive effect with no significant increase in severe problems.[127] Some U.S. data support specific malocclusions being more prevalent in certain racial groups.[128] More extensive international epidemiologic studies are needed to properly characterize racial differences and confirm empirical convictions such as the greater prevalence of maxillary deficiency in East Asian populations. Perhaps two thirds of anteroposterior and vertical facial variation is genetically determined.[124,129,130] Genetic influence does play an important role in skeletal class III growth and excessive vertical facial growth.[131,132] There appears to be at least five autosomal dominant phenotypic subtypes of class III with genetic linkage to at least five loci that suggest a common upstream genetic element for both maxillary deficiency and mandibular prognathism.[133,134] GH receptor gene has been associated with mandibular height.[135] Overall, it is speculated that the majority of human facial skeletal variation is due to the genetic component, with the remaining due to environmental influence.[129,136,137] Functional forces have a crucial influence in altering craniofacial bone growth, and it is apparent that gene expression can be modified by muscular and soft tissue forces, representing epigenetic influences.[138,139] Although genetic influence is important, the membranous viscerocranium is determined to a great extent by functional influences, with these extrinsic factors having the greatest control over mandibular growth.

Environmental and Epigenetic

A multitude of postnatal environmental factors interact with genetic control mechanisms, including functional, traumatic, endocrine, nutritional, pathologic, psychological, cultural, and climatic or seasonal factors.

The functional environment is determined by neuromuscular behavior necessary for survival such as respiration, mastication, deglutition, speech, and posture. However, it is clear that functional influences at rest (i.e., postural activity or the presence of a pathologic mass) are the important determinants of craniofacial growth, not transient muscle contractions and mandibular movement.[140] Chronic pressure alters regional skeletal growth and may be used to improve or correct some craniofacial deformities.[141] Habitual behavior such as non-nutritive sucking and other oral or postural habits can exert such pressure if it is present with great enough frequency and duration, usually at least 4 to 6 hr/day.[142,143] Mastication limited to one side for sufficient duration can cause asymmetrical mandibular growth.[144] There is some evidence that diet consistency has an effect on mandibular morphology.[145,146] It has been popular to attribute changes in craniofacial dimensions from early to modern human civilizations to dietary changes from a coarse to a refined diet, but this relationship remains unclear. Certainly, it is apparent that more recent diet alterations in contemporary societies have reduced functional jaw demands, but it is not clear whether this change is accelerating the evolutionary trend toward reduced jaw size. The extent of masticatory muscular and dental development can modify the morphology of skeletal superstructures, including the temporal fossae and sagittal crests, the zygomatic arches, the lateral pterygoid plates, the angular and coronoid processes and rami of the mandible, and parts of the temporomandibular joints.[147] The size and function of masticatory muscles have been correlated with facial morphology, but the location of muscle attachments is probably more important in determining bone shape than the amount of activity.[148–150] Other studies have demonstrated an atrophic effect from muscle denervation.[151,152] However, it also is clear that external craniofacial bone growth nevertheless can occur in the absence of any muscle function.[153,154]

Growth deficiency due to neuromuscular deficits can occur in muscle weakness conditions such as muscular dystrophy. The difficulty in returning function to the area makes such conditions particularly resistant to treatment. However, if the muscle is normal, it appears that the normal force range of masticatory muscular function in the general population does not significantly affect facial growth.[155] Long-term impairment of nasal breathing historically has been viewed as a cause of long face deformity, but this assumption continues to be controversial.[156] Although there is some evidence that there is a change in posture with total nasal obstruction,[157] long-term presence of this condition is extremely rare in humans. There is less disagreement about a relationship between nasal obstruction and facial deformity than there is with the extent and duration of mouth breathing necessary to cause a deformity.[158]

Postnatal surgery during infancy for congenital malformations such as cleft lip and palate introduces scarring that is responsible for some midfacial growth attenuation. Typical surgery to close palatal clefts requires that mucoperiosteal flaps be raised and moved medially and posteriorly. This results in denuded bony areas that will heal with the formation of scar tissue bands of variable size and elasticity. This scar tissue usually connects across the maxilla and includes the palatal bones and possibly the pterygoid plates. It is thought that the presence of this scar tissue during postnatal growth compromises midfacial growth.[159,160] The longer postnatal surgery can be delayed, the less growth is affected. There is some evidence demonstrating brain morphology and neuropsychologic differences in nonsyndromic children affected by clefts.[161,162]

Natal or postnatal trauma to the craniofacial complex can modify growth if there is limitation of blood supply or

mechanical constriction due to scarring. Extensive midfacial trauma can cause midface growth deficiency as a result of the loss of intrinsic nasal septal growth or from a structural collapse that prevents normal morphologic expression of growth.[163] Trauma that results in scarring across circummaxillary sutures is rare and usually results in a unilateral growth deficit. Untreated burns of the head and neck can cause significant craniofacial dysmorphology.[164] Neurologic damage may lead to muscle paralysis that can alter craniofacial form due to decreased muscle function. There should be caution when considering early craniofacial reconstructive surgery, because the surgery itself may produce additional scarring that can exacerbate the growth attenuation. There is no evidence that the use of rigid plate fixation for trauma reconstruction causes restrictive growth effects in addition to the trauma alone.[165]

Mandibular condylar neck fracture is not uncommon in young children owing to the fragility of this area and the common occurrence of blows to the chin. These fractures almost invariably result in anterior displacement and resorption of the condylar head, yet regeneration of the condylar process and subsequent normal mandibular growth is common. (Lund, 1974) In 15% to 20%, however, there is some reduction in growth on the affected side, the severity usually related to the amount of soft tissue trauma and scarring, and likely is the most common cause of mandibular asymmetry in children.[166,167] With trauma involving the mandible, mandibular function must be maintained, in selective cases requiring physical therapy and the use of a functional appliance. As long as mandibular ankylosis is prevented, surgical open reduction should be avoided when treating condylar fractures in children.[168]

Endocrine disturbances originating from pathology or the environment are among the most potent regulators of postnatal growth. Mandibular growth is reduced by sex hormone deficiency.[169] The primary growth-promoting hormone, GH, is secreted by the pituitary and regulated by somatostatin and GH-releasing hormone release from the hypothalamus. Studies have demonstrated increased GH secretion throughout the day and night in the newborn. However, IGF-I levels are lower at birth and gradually increase during childhood and into adolescence, indicating an early immaturity in the feedback loop. Although the growth process in utero and in the first months after birth is more nutritionally dependent than GH-dependent, this changes during the first year of life, with full GH dependence attained during the second year. GH has been shown to have a direct stimulatory effect on cartilage growth, whereas IGF-I acts as a secondary stimulatory effector.[170]

GH has an important endocrine influence on modulating the size and relationships of the craniofacial structures.[171] GH-deficient children have excess subcutaneous fat and overall delayed facial and cranial base development, resulting in infantile, but proportional, facies. Dental development is delayed as well but to a much lesser degree than facial or somatic growth. In contrast, the craniofacial growth is disproportionate in an autosomal recessive condition known as Laron syndrome in which there is IGF-I deficiency in spite of increased serum GH levels.[172] There is a normal calvarium with small facial bones, resulting in the forehead appearing large and prominent relative to the small recessed face.[173] This suggests that some areas of the face are more directly affected by GH than by IGF-I. GH supplementation in GH-deficient children accelerates catch-up craniofacial growth.[174]

GH excess, usually a consequence of a pituitary adenoma, results in gigantism if it occurs before the end of adolescence and presents with overall larger craniofacial dimensions with coarser facial features. Acromegaly is the outcome if the GH excess is produced after adolescence, characterized by increased periosteal bone that includes cranial thickening, increased size of the frontal sinuses, prominent supraorbital ridges, and nasal enlargement as well as an enlarged tongue and renewed mandibular condylar cartilage growth, leading to mandibular prognathism and a broader mandibular arch (see Figure 53-18). Late mandibular growth also occurs in hemimandibular hypertrophy, a unilateral condition with an unknown cause that most commonly affects females after pubertal growth.

Hypothyroidism will decrease GH release and results in delayed bone and dental development.[175] The craniofacial outcome of this deficiency differs from GH deficiency primarily by the smaller cranium. Anabolic steroids increase craniofacial growth but may lead to excessive anterior maxillary growth in high doses.[176] Testosterone, GH, and IGF-I accelerate endochondral and intramembranous craniofacial skeletal growth as well as statural height. Estrogen appears to decrease endochondral growth.[177,178]

Although glucocorticoid production is necessary for normal growth, glucocorticoid therapy in the prepubertal child must be carefully managed to avoid its inhibitory effect on GH and IGF-I production, resulting in short stature.[179] Although there are no clinical studies indicating the effect on craniofacial growth, animal model studies have suggested a retarding effect on mandibular condylar cartilage growth and acceleration in dental eruption.[180,181]

Poor nutrition, hygiene, and health adversely affect growth. Insufficient caloric and protein intake is the most common cause of growth failure worldwide.[182] Growth deficiency from malnutrition is proportional to the severity of the nutritional deficit. Malnutrition is associated with increased GH but decreased production of IGF-I, reallocating calories from anabolic to survival requirements.[183] It is estimated that 55% of the morphologic variation of the cranium is due to nutritional factors.[184] Because of the early rapid growth of the brain, the cranium is affected more by infant malnutrition than the rest of the craniofacial complex. The size of the neurocranium decreases in rats subjected to malnutrition.[185] A diet deficient in calcium and vitamin D results in cranial dimensional changes in rats.[186] It is thought that maternal vitamin A deficiency alters endocrine function that causes a disturbance in chondrogenesis, reducing the cranial base.[187]

Nasomaxillary hypoplasia in humans can be related to maternal vitamin K deficiency, since this condition induced in rats causes limited nasal septal cartilage growth.[188] Protein malnutrition in rats decreases the length of the skull relative to the width.[189,190] Voluntary undernutrition has become more common during adolescence, especially with females trying to decrease weight for athletics and those with anxiety about being overweight. This may develop into extreme eating disorders such as anorexia or bulimia nervosa, which may result in impaired growth, delayed puberty, and osteopenia.[191] Obesity has been associated with larger maxillary and mandibular dimensions as well as bimaxillary prognathism.[192] Improved nutrition is considered the primary reason for a phenomenon termed the *secular trend* that has resulted in progressively increased growth rate and attainment of stature and earlier sexual maturation, particularly in industrialized cultures. The secular growth trend has had special relevance for the timing of orthopedic and surgical treatment for craniofacial skeletal discrepancies, resulting in earlier average intervention. There also is evidence that craniofacial skeletal proportions are becoming progressively taller and narrower during the last century.[193] Chronic disease such as congenital heart disease, malabsorption syndrome (e.g., chronic inflammatory disease, cystic fibrosis, celiac disease), chronic renal or liver disease, chronic anemia, inborn errors of metabolism, chronic infections (e.g., tuberculosis, acquired immunodeficiency syndrome), severe asthma, or other chronic pulmonary disease can adversely affect growth.[194] There are a variety of mechanisms causing the growth deficits from these conditions, including reduced nutritional intake, metabolic disbalance, hypoxia, chronic metabolic acidosis protein loss, and often the treatment for the pathology itself.[39] Medications that limit potential growth include chronic adrenal steroid therapy (used for asthma, nephritic syndrome, lupus, and other chronic diseases) and cytostatics (for cancer treatment). Irradiation of the head and face for childhood cancer can result in severe hypoplasia of soft and hard tissues.[195,196] If cranial irradiation is required in cases of leukemia and tumors of the CNS, hypothalamic function can be damaged, affecting the release of hypothalamic and pituitary hormones, notably GH.

Muscle dysfunction can adversely affect craniofacial growth through chronic excessive contraction or loss of function. Untreated torticollis results in facial asymmetry from growth restriction on the side affected by excessive contraction of neck muscles.[197] Loss of tonic muscle contraction experienced in muscular dystrophy and other muscle weakness conditions or due to congenital or post-traumatic CNS disturbance often results in an open mouth posture that leads to increased vertical eruption of posterior teeth, increased face height, and altered mandibular form and position. However, differences in biting forces in otherwise healthy individuals do not appear to make a difference in vertical facial form.[155]

Chronic psychological trauma, emotional deprivation, or psychosocial stress can have a profound effect on somatic growth, causing a functional and reversible GH deficiency, often mimicking growth disorders that are caused from endocrine or nutritional deficiencies.[198]

Cultural and seasonal factors can affect the rate of physical growth. Children in an urban environment tend to mature faster than those from rural settings, particularly in preindustrial societies. Physical growth tends to be more rapid in the spring and summer than in the fall and winter. Additional minor factors have been shown to have a significant influence on postnatal growth and development. These include climate, altitude, exposure to environmental pollutants, and noise.[199,200] Future research will increase our understanding of the role that these and other yet unidentified environmental factors play in altering human genetic growth potential.

In summary, most individuals with severe craniofacial skeletal discrepancies who do not have identifiable congenital malformations likely have a combination of genetic, epigenetic, and environmental determinants influencing their skeletal growth. Our knowledge of the genetic determinants will certainly make rapid progress with the identification of the human genome and advanced genetic diagnostic techniques. The progress of our knowledge of the environmental determinants and their interaction with the genetic control mechanisms will undoubtedly be more delayed and challenging to attain.

ORTHODONTIC, ORTHOPEDIC, AND ORTHOGNATHIC CLINICAL CONSIDERATIONS

Craniofacial Growth Assessment

An evaluation of physical development is needed when considering whether craniofacial orthopedic treatment or orthognathic surgery is indicated for an adolescent. There is a tendency to orthopedically treat females too late owing to a secular trend characterized by progressively earlier sexual maturation and adolescent growth, often before complete eruption of the permanent dentition. Correspondingly, there can be a tendency to orthopedically and surgically treat males too soon because they begin puberty on average 2 years later and continue adolescent craniofacial growth longer, often years after eruption of the permanent dentition. It is well recognized that orthognathic surgery will have a minimal effect on an individual's pattern of craniofacial growth, underscoring the importance of surgical correction of undesirable growth after it has occurred rather than expecting growth to be redirected while it is occurring. Instability of orthopedic as well as orthognathic surgical treatment usually has much more to do with post-treatment growth than surgical relapse.

One indicator for physical growth is skeletal maturation, which historically has been determined with a hand and wrist radiograph. This image provides a view of 30 bones with a predictable ossification sequence and still is considered the assessment standard for skeletal development. A somewhat less accurate, but acceptable, alternative, using standardized lateral cephalometric radiographs to assess

vertebral maturation, is being utilized more recently by orthodontists and oral and maxillofacial surgeons because it precludes the need for an additional radiograph.[201,202] It is important that the orthodontist and surgeon not rely solely on this variable to predict the likelihood of adequate remaining jaw growth for successful orthopedic treatment or minimal jaw growth for successful orthognathic surgical treatment. Although it may provide some idea of growth potential, it certainly cannot accurately predict the magnitude, timing, or direction of remaining growth.[203–205] There is some hope that saliva or blood samples may provide future reliable indicators for the timing, intensity, and end of pubertal growth.[206] Superimposition of serial lateral cephalometric radiographs remains the most reliable current method for demonstrating minimal remaining adolescent craniofacial growth. Even with assurance from cephalometric radiographs that adolescent growth is complete, orthodontists and surgeons need to appreciate that the craniofacial growth pattern exhibited before and during adolescence will continue to some extent after orthognathic surgery long into adult life, leading to some return of previous jaw discrepancies in certain individuals, particularly those who have a growth pattern characterized by vertical excess, mandibular excess, or asymmetry.

Orthopedic Treatment for Growth Modification

A harmonious aesthetic facial appearance and balanced dentoskeletal segments facilitating a functional occlusion are both goals that orthodontists and oral maxillofacial surgeons work to achieve by means of orthodontic treatment combined with orthognathic surgery. However, before a surgical correction is contemplated in a growing patient, a determination should be made as to whether the patient is a candidate for orthopedic treatment that may modify craniofacial growth to improve the skeletal imbalance to a favorable aesthetic and functional outcome without the need for orthognathic surgery. It is well known that craniofacial orthopedic devices can generate forces that cause stress in sutures capable of modifying suture growth.[207] As indicated earlier in the chapter, almost 50% of the total cumulative growth of the midface and mandible remains when a child is entering adolescence, making it possible to have an orthopedic treatment effect on the jaws before adolescent growth is completed.

In spite of over a century of clinical experience with orthopedic facial appliances, it remains controversial as to what extent craniofacial skeletal growth can be predictably and permanently modified by orthopedic treatment. Although consensus exists that there is an important genetic influence on the outcome of craniofacial growth, there is a wide range of views regarding the amount in which postnatal factors, particularly orthopedic treatment, influence this outcome. Views vary from the belief that orthopedic alteration of jaw relationships is predictable and stable to the contrasting opinion that skeletal growth is primarily determined genetically and cannot be significantly altered long-term by orthopedic treatment. The current consensus based on our best basic and clinical data is that clinically significant craniofacial orthopedic treatment is possible, depending on the type of orthopedic treatment, the patient compliance, and the extent of skeletal growth during and after active treatment. The best outcome of craniofacial orthopedic treatment occurs when there is only mild to moderate skeletal discrepancies in patients with short or average face height and mild to moderate dental crowding. The poorest outcome is in individuals with more severe jaw discrepancies, a long lower face height, and moderate to severe dental crowding. It is essential that orthodontists and surgeons bear in mind that dental compensation for the skeletal discrepancies is inevitable and some degree of facial aesthetic compromise may be a part of craniofacial orthopedic treatment. It has been proposed that the typical range of skeletal malocclusions include individuals with normal gene polymorphisms for signaling molecules and growth factors that attenuate the capacity of tissues and cells to reliably respond to orthopedic treatment.[208]

The efficacy of craniofacial growth modification has been a controversial subject for more than a century. At the onset of the 20th century, there was universal confidence by the orthodontic profession that forces applied through the dentition to the growing face could effectively treat craniofacial skeletal discrepancies. After the 1920s, there was a decline in this conviction by North American orthodontists. With the invention of the cephalostat, more precise skeletal assessment of treatment outcomes became possible during the 1950s. This resulted in renewed faith in facial growth modification, with the demonstration of skeletal changes from the use of extraoral force applied with a cervical headgear.

In Europe, there was less controversy regarding craniofacial growth modification efficacy throughout the first half of the 20th century. European orthodontists relied primarily on removable "functional" appliances, designed to provide forces from facial muscles and soft tissue function, for facial orthopedic treatment. The separate philosophic paths taken by European and American orthodontists united in the 1960s, resulting in a more global acceptance of either extraoral or intraoral appliances or a combination of both to facilitate craniofacial orthopedic treatment. This acceptance gained support and enthusiasm from results of basic research conducted during the 1970s using animal models. Although this enthusiasm reached its peak in the 1980s, it was considerably moderated in the 1990s from clinical experiences and the results of prospective clinical studies. There remains little argument that some craniofacial growth modification is feasible with specific skeletal discrepancies, but there continues to be controversy over the nature and extent of the skeletal change possible in individual patients as well as the optimal treatment timing and appliance type. In addition, a reliable and accurate method of predicting the direction, timing, and magnitude of craniofacial growth for an individual has not been devised.

The aim of craniofacial growth modification is to alter the growth pattern by changing the relationships of the jaws. If the skeletal unit is too large, the aim of the orthopedic

treatment is to attenuate or redirect its growth to improve its relationship relative to the opposing smaller jaw. If the jaw is too small, the growth modification treatment is aimed at enhancing or redirecting its growth relative to the larger skeletal unit. Virtually all of the craniofacial growth modification appliances until the 1990s have been "toothborne" to some extent, so that the orthopedic forces applied to the skeletal units also create stress to the teeth that results in some dental movement. Although the goal of craniofacial growth modification is to limit the changes to the skeletal units while minimizing movement of the teeth, the reality is that most treatment is predominantly dentoalveolar with a possible minor skeletal component.

There is growing evidence that the long-term success of many forms of growth modification requires that the treatment be continued until facial growth is nearly complete, making early treatment a less efficient way to treat many jaw discrepancies. The following discussion summarizes the facial orthopedic options we have for clinical application of our present understanding of craniofacial growth in the three planes of space.

Transverse Orthopedic Treatment

Because transverse growth reaches completion earlier than anteroposterior or vertical craniofacial growth, it follows that transverse skeletal problems should be addressed early. The most common transverse skeletal problem is maxillary constriction, which can be treated in the preadolescent child even as early as during the primary dentition. The most recent federal epidemiologic study, the Third National Health and Nutrition Examination Survey (NHANES-III) conducted from 1989 through 1994, indicates the prevalence of posterior crossbite is about 5% of the U.S. population.[209] Significant facial or mandibular asymmetry represents approximately 0.1% of the total population.[210] Although maxillary orthopedic expansion devices have been used since 1860, they fell out of favor for a few decades prior to the 1940s due to unsubstantiated concerns regarding their safety and effectiveness.[211] Orthopedic expansion of the maxilla can be achieved with a variety of toothborne appliances (Figure 53-21). These appliances apply moderate to high forces to the teeth that are transmitted as stresses to the maxilla, primarily distracting the midpalatal sutures but also producing less pronounced stresses to the sphenoid and zygomatic bones and other adjacent structures.[212] The midpalatal sutural expansion outcome is wider anteriorly due to the buttressing effect of more posterior maxillary structures. This anteroposterior differential in opening becomes more exaggerated when treating older children due to the greater posterior resistance with age. Within days after initial expansion, new bone forms, eventually depositing both perpendicular and parallel to the edges of the expanded sutures.[213] Although a large amount of the skeletal expansion relapses during retention, overall stability is good if the extent of sutural patency and magnitude of expansion are great enough. A potential additional benefit to improvement in interarch transverse compatibility is an increase in dental arch perimeter made possible by the maxillary orthopedic expansion.[214]

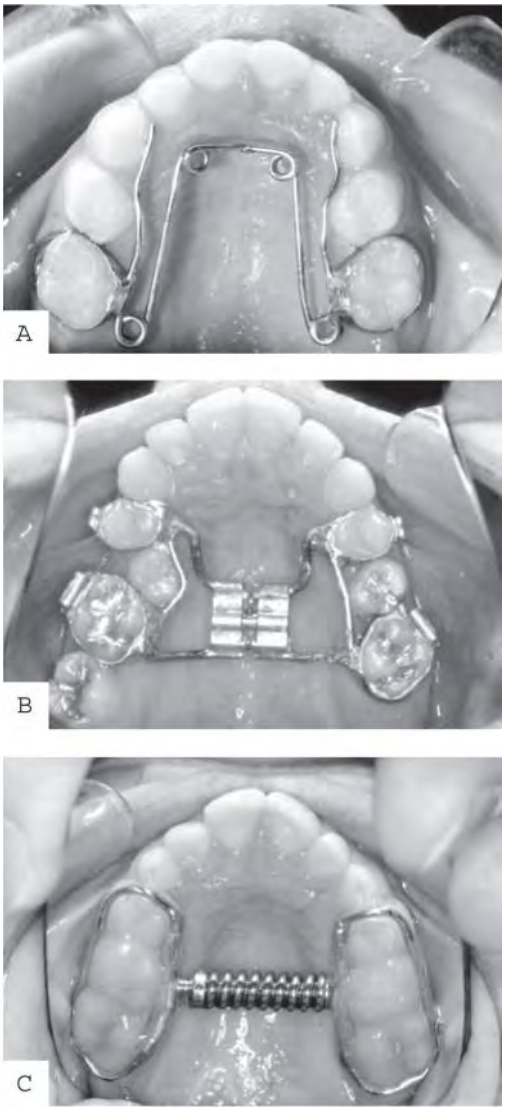


FIGURE 53-21. Types of maxillary orthopedic expansion appliances. **A,** Quad-helix: An effective skeletal expansion appliance in the primary dentition. **B,** Banded hyrax: This traditional jackscrew also can be used as an activation component for an appliance bonded to the maxillary posterior teeth. **C,** Bonded Minne expander: This spring-loaded component also can be used as an activation component for an appliance banded to the maxillary posterior teeth.

Although complete fusion of the midpalatal suture usually does not occur until the third postnatal decade, the process leading to fusion is a gradual one, characterized by progressive sutural interdigitation and ossification.[215] For this reason, more effective and parallel sutural separation, requiring less force and concomitant undesirable dental expansion, is possible in the younger child, especially before puberty, during a "phase 1" treatment in the mixed or early permanent dentition. Treatment before the peak pubertal growth velocity may result in greater long-term skeletal craniofacial transverse width.[216] Treatment may even be indicated as early as the

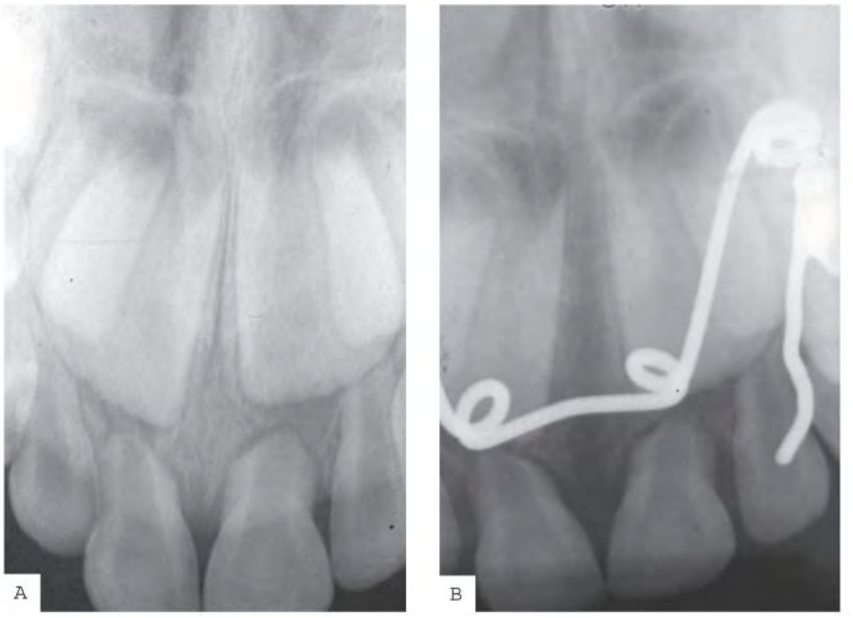


FIGURE 53-22. Occlusal radiographs demonstrate maxillary skeletal expansion with a Quad-helix in the primary dentition. Note distraction of the midpalatal suture. Before **(A)** and after **(B)** initial palatal expansion.

primary dentition in the presence of a large transverse functional shift. This compensatory functional problem can result in asymmetrical condylar positioning that may lead to asymmetrical mandibular growth and uneven remodeling of the glenoid fossae, possibly resulting in permanent facial asymmetry, even if the constricted maxillary arch is corrected at a later date.[217–219] Maxillary constriction without a transverse functional shift does not carry the same urgency and is conveniently treated closer to the onset of puberty during the early permanent dentition.[220]

Maxillary orthopedic expansion in late adolescent or postadolescent patients should be attempted with caution. Even if skeletal expansion is possible in these older patients, the extent of circum-maxillary and midpalatal sutural patency is limited enough to compromise stability of the treatment outcome. It is appropriate to confirm intermaxillary expansion with an occlusal radiograph in these patients, because the development of a midline diastema may only indicate bending of maxillary bones. If the expansion is limited to lateral tipping of maxillary posterior teeth, buccal alveolar bone height reduction and gingival recession may occur. It usually is more prudent to consider surgically assisted palatal expansion (SARPE) for late adolescent or postadolescent patients to avoid periodontal compromises and instability. One should keep in mind that SARPE expansion will create much greater skeletal expansion across the canines as opposed to the more posterior expansion achieved by a conventional osteotomy.

The expansion appliance can be banded or bonded to the maxillary posterior teeth with a spring-loaded or non–spring-loaded palatal jackscrew that usually is activated by the patient 0.5 mm/day, delivering from 2 to more than 10 pounds of force (this may increase to cumulative loads of 20 pounds or more with multiple activations in the absence of adequate sutural separation). The conventional description for the expansion induced with this appliance is "rapid palatal expansion." However, it is possible to affect slower expansion with less frequent activations (≤1 mm/wk), requiring more active treatment time but less retention time to ensure stability.[221] It is feasible to achieve skeletal expansion with simpler appliances such as a W-arch or Quad-helix that deliver only a few hundred grams of force, provided that the patient is in the primary or very early mixed dentition when the maxillary sutures are more patent or when a cleft of the hard palate is present (Figure 53-22).

Because all of these expansion appliances are toothborne, unwanted dentoalveolar expansion is an inevitable consequence.[222,223] An additional undesirable outcome is the long-term loss of about 30% of the skeletal expansion achieved during active treatment due to the rebound of stretched palatal tissues.[224] To compensate for these effects, maxillary expansion should be continued until adequate overexpansion is achieved, usually to the extent that the lingual cusps of the maxillary molars are opposing the buccal cusps of the mandibular molars (Figure 53-23). Once adequate expansion has



FIGURE 53-23. Maxillary overexpansion to compensate for dentoalveolar expansion and skeletal relapse. Note the lingual cusps of maxillary posterior teeth are occluding with buccal cusps of the mandibular posterior teeth.

been accomplished, at least 3 to 6 months of retention is necessary to permit new bone to fill in the spaces created by maxillary separation and to permit time for dissipation of reaction forces stored in the facial bones that promote relapse. It is important to recognize that transverse maxillary skeletal expansion is notoriously unstable and prone to relapse, whether it is achieved orthopedically or surgically.[225,226] Overexpansion accounts for some of the relapse and permits the orthodontist to transversely upright the posterior teeth in their alveolar housing without compromising the final transverse occlusal correction after retention. More recently developed osseointegrated attachments or temporary skeletal anchorage devices hold some promise for a means of expanding the maxilla without buccally tipping posterior teeth and may improve the stability of the skeletal change (Figure 53-24).[227] However, there is preliminary evidence that skeletal anchorage for maxillary orthopedic expansion does not offer significant benefits over conventional toothborne appliances.[228]

A less common transverse skeletal problem than maxillary constriction is asymmetrical mandibular deficiency, usually caused from a previous trauma associated with unilateral mandibular condylar fracture or hemifacial microsomia, a congenital facial asymmetry. In both of these conditions, the affected side exhibits growth deficiency relative to the unaffected or normal side, resulting in a mandibular deviation toward the affected side. If left untreated in a growing individual, the alveolar processes compensate with limited eruption of the maxillary posterior teeth on the affected side and excessive eruption of the maxillary posterior teeth on the unaffected side, resulting in an occlusal cant that is higher on the affected side. It is best to start orthopedic treatment with these individuals before pubertal growth, as early as patient compliance will permit. The goal is to maximize the growth expression on the deficient side and minimize dentoalveolar compensation. The orthopedic appliance of choice is an asymmetrical "hybrid" functional appliance that is constructed to posture the mandible forward on the affected side, bringing the chin to the midline.[229] Posterior dental eruption is attenuated on the unaffected side with a bite block, and eruption is facilitated on the affected side with a buccal shield and the absence of interocclusal acrylic (Figure 53-25). Because untreated mandibular asymmetries of this nature invariably worsen with growth, orthopedic treatment is considered successful if the asymmetry remains stable or improves. Treatment should not continue if progressive asymmetry is apparent in spite of reliable appliance use by the patient.

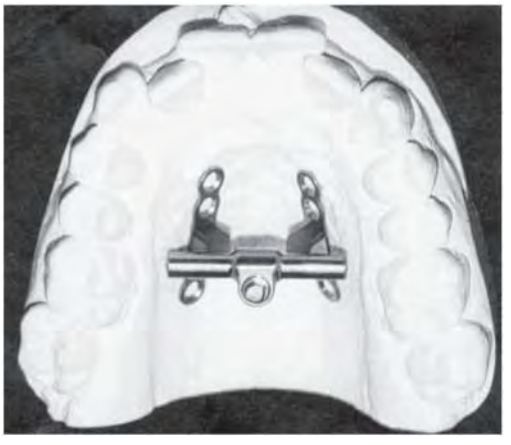


FIGURE 53-24. Bone-anchored palatal distraction appliance. Reproduced with permission from J Orofac Orthop 2003; 64:444.

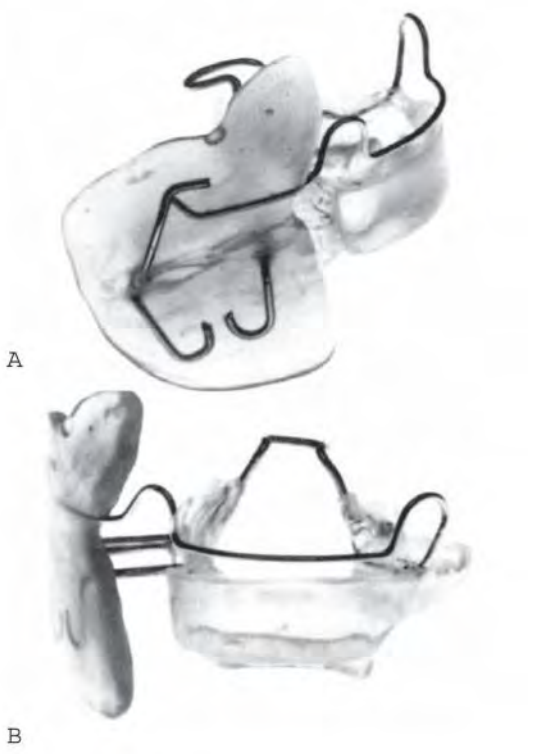


FIGURE 53-25. Asymmetrical "hybrid" functional appliance. **A,** Right lateral view. **B,** Frontal view. On the left, unaffected side, posterior dental eruption is restricted with an interocclusal acrylic block. On the right, affected side, the mandible is postured forward and posterior dental eruption is encouraged with a buccal shield and absence of an interocclusal acrylic block. (**A** and **B,** Reproduced with permission from Proffit WR, Fields HW. Contemporary orthodontics. 3rd ed. St. Louis (MO): Mosby; 2000. p. 370.)

Anteroposterior Orthopedic Treatment: Class II

A class II skeletal relationship can be the result of a retrusive and/or a deficient mandible, a protrusive or vertically excessive maxilla, or a combination of these skeletal problems. The prevalence of this type of malocclusion is approximately 15% to 20% of the U.S. population, with approximately 2% severe enough to be considered as handicapping.[209] Prospective clinical studies have supported that early growth modification during the mixed dentition may lead to an improvement in the skeletal class II malocclusion.[230–232] It should be kept in mind that regardless of whether orthopedic treatment is attempted during active facial growth, approximately 10% of patients ultimately require orthognathic surgery to fully correct the class II malocclusion.[232]

The headgear has been used as a means of class II orthopedic treatment in North America since the late 19th century

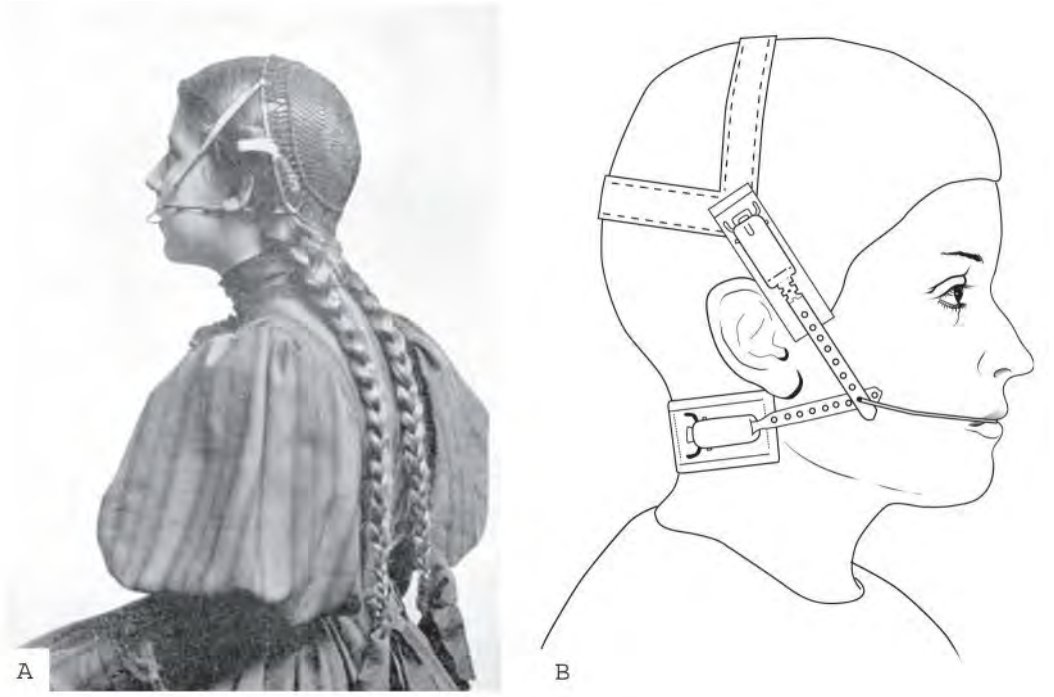


FIGURE 53-26. Headgear, past and present. An orthopedic force is directed posteriorly and superiorly to the maxilla, attenuating circum-maxillary sutural growth. **A,** Headgear from the late 19th century. **B,** Contemporary headgear appliance fabricated with more durable materials and with additional calibration and safety features, although the overall design has changed little in over a century. (**A,** Reproduced with permission from Angle EH. Treatment of malocclusion of the teeth. Philadelphia (PA): S.S. White Dental Manufacturing Co; 1907. p. 234; **B** adapted from McNamara JA, Brudon WL. Orthodontics and dentofacial orthopedics. Ann Arbor (MI): Needham Press, Inc.; 2001. p. 365.)

(Figure 53-26). An orthopedic force ranging from 16 to more than 32 ounces is delivered using elastic traction from the headgear to a cervical or cranial attachment for 12 to 14 hr/day, usually for 9 to 12 months. A safety release for the retractive mechanism and parent/patient education are essential to prevent potential traumatic injuries. Theoretically, the force is transmitted in a posterior and superior direction via the teeth through the maxilla to compress the circum-maxillary sutures, limiting or redirecting maxillary growth. Since the introduction of standardized cephalometric radiographs, many clinical studies have demonstrated that maxillary growth can be altered with the use of the headgear.[233–235] These clinical data have been supported by primate studies demonstrating that extraoral orthopedic force directed against the maxilla attenuates forward growth and alters bone apposition at the maxillary sutures.[236–239] Some retrospective studies suggest that mandibular growth may be enhanced as well. Because the headgear is a toothborne appliance, some maxillary dental retraction accompanies the skeletal change. Another dentoalveolar effect is the attenuation of maxillary molar eruption, resulting in anterior and superior mandibular rotation. There is some support for this being the only clinically relevant skeletal effect.[240] A significant treatment effect usually requires that a headgear be worn 12 to 14 hr/day. Human GH and other endocrine factors that promote growth and dental eruption are primarily released during the evening and night.[241–243] It is fortuitous that this is the only time of day that one can reliably expect an adolescent to wear a headgear. Because it is a removable appliance, few adolescents after the peak of pubertal growth will reliably wear the appliance.

The alternative orthopedic method for treating a class II skeletal relationship is the class II functional appliance, which has been used since the early 20th century in Europe and since the 1960s in North America. These appliances include the removable toothborne activator, bionator, and twin block, the removable and primarily tissueborne Fränkel (functional regulator) appliance, or the fixed toothborne Herbst appliance (Figure 53-27). All of these appliances position the mandibular condyles downward and forward away from the glenoid fossae. Theoretically, the distracted condylar positions reduce the normal compressive joint pressure on the growing condylar cartilage and the forward mandibular posturing alters muscle tension on the condyles, stimulating or accelerating the endochondral condylar growth more than would normally occur. There is some support from animal studies that a histologic increase in condylar growth can be achieved.[244–246] Retrospective clinical studies have both supported[247–251] and refuted[252–255] that clinically significant lengthening of the mandible can be achieved with functional appliances. However, prospective, randomized clinical studies have more definitively confirmed that there is no greater absolute growth reflected by the long-term treatment outcome.[256] Much of the skeletal change demonstrated with a functional appliance may result from forces against the maxilla, similar to those applied with a headgear, that are created from the stretched facial muscles and soft tissues

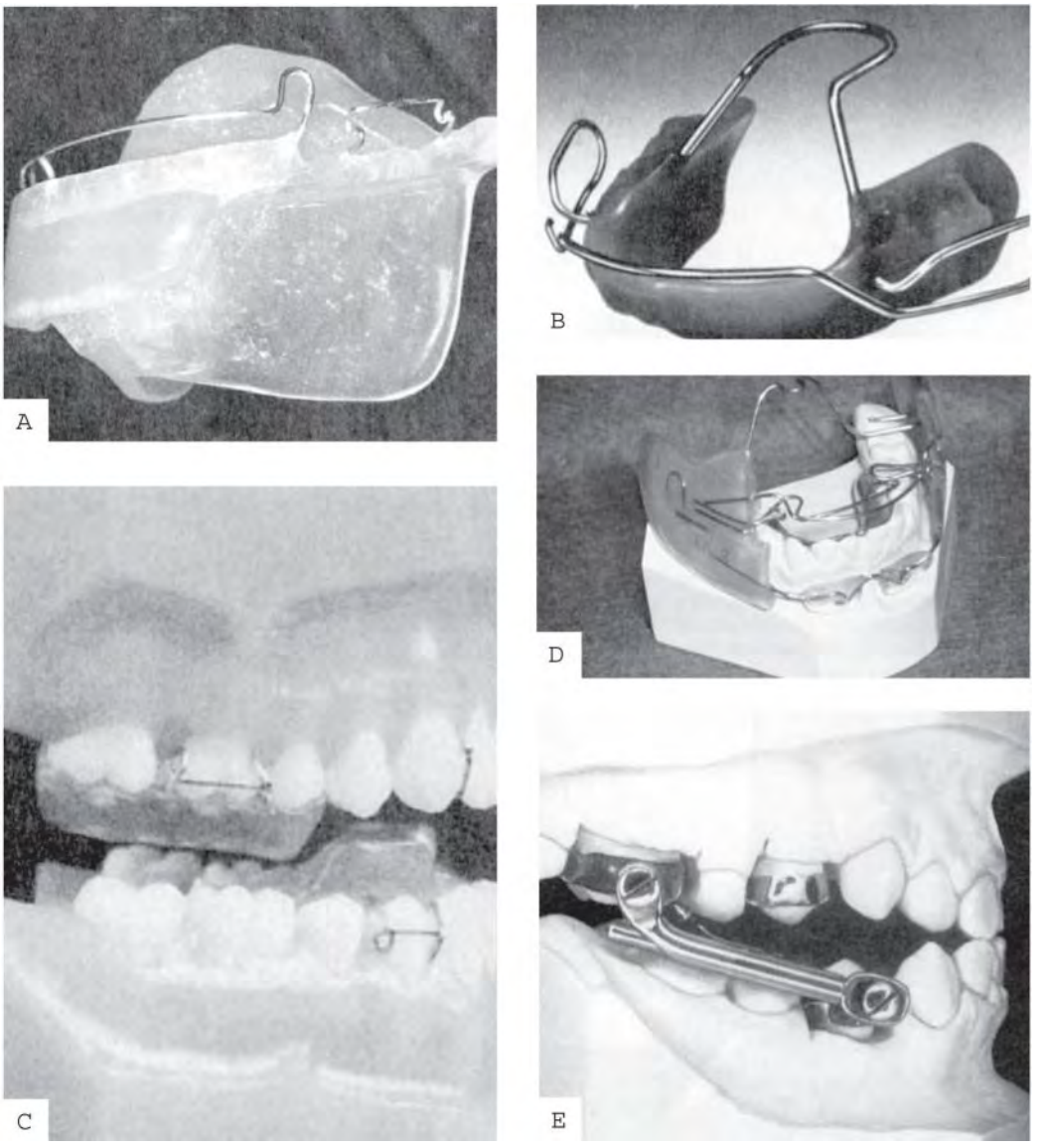


FIGURE 53-27. Class II functional appliances. All of these appliances position the mandible downward and forward, distracting the condyles from the glenoid fossae. This simultaneously creates posterior and superior forces on the maxilla from the facial muscles and soft tissues attempting to return the mandible to its normal position. **A,** Activator. **B,** Bionator. **C,** Twin block. **D,** Fränkel. **E,** Fixed Herbst. An alternative is to utilize occlusal acrylic and bond the maxillary portion of the appliance with a removable acrylic splint for the mandibular arch. (**A–E,** Reproduced with permission. A, C, and D from Bishara SE. Textbook of orthodontics. Philadelphia (PA): W.B. Saunders; 2001. p. 345. B from Graber TM, Vanarsdall RL. Orthodontics: current principles and techniques, 3rd ed. St. Louis (MO): Mosby; 2000. p. 488. E from McNamara JA, Brudon WL. Orthodontics and dentofacial orthopedics. Ann Arbor (MI): Needham Press, Inc.; 2001. p. 287.)

attempting to return the postured mandible back to its posterior and superior position.[257,258] Because of the toothborne nature of this appliance, dentoalveolar changes accompany the skeletal changes, including maxillary dental retraction and mandibular dental protrusion.

Retrospective clinical studies have demonstrated few differences in treatment outcome when comparing the skeletal response between headgear and functional appliance treatment.[251,259,260] In the last decade of the 20th century, three prospective, randomized class II clinical trials conducted in the United States. and Great Britain have all demonstrated that orthopedic treatment with either a headgear or a functional appliance in the mixed dentition results in an average improved short-term skeletal outcome. There is some evidence that there is more of a maxillary effect with headgears[232,260] and more of a mandibular effect with functional appliances.[231,232,260,261] Most functional appliances need to be worn for the same daily duration as the headgear (exceptions are the Fränkel and Herbst appliances, which are worn full time) for a significant treatment effect. Much like the headgear, dependable wear is more realistic during the evening and at night when the most active facial growth and dental eruption usually occur. However, like the headgear, its removable nature prevents it from being reliably worn by most adolescents after the peak of their pubertal growth.





FIGURE 53-28. Combination headgear and removable functional appliance. The interocclusal acrylic permits orthopedic forces to be transmitted further anteriorly and superiorly through the center of the maxilla. **A,** Intraoral part of the appliance with headgear tubes embedded in the interocclusal acrylic. **B,** Extraoral application of the appliance. (**A** and **B,** Reproduced with permission from Bishara SE. Textbook of orthodontics. Philadelphia (PA): W.B. Saunders; 2001. p. 341.)

In the late 1960s, when European and American facial orthopedic philosophies were becoming more fully integrated, a method was introduced in Europe using a headgear in combination with a functional appliance (Figure 53-28).[262] This approach was intended to provide greater cumulative skeletal growth effects than use of either appliance alone, but this has yet to be demonstrated by clinical studies.

Although there is greater general acceptance that treatment with a headgear or functional appliance may achieve an improved long-term treatment outcome, there continues to be controversy over the optimum treatment time in the growing child. It has already been demonstrated that orthopedic class II treatment in the very young child, in the primary or early mixed dentition, results in substantial relapse and recurrence of the original facial skeletal pattern by late adolescence.[263] However, there is great debate regarding the long-term efficacy of orthopedic treatment during later mixed dentition as the first phase of a two-phase treatment versus delaying orthopedic treatment until definitive orthodontic treatment during puberty, after the eruption of the permanent dentition. Many orthodontists historically have preferred the earlier first phase because there is substantial potential growth remaining, compliance in wearing the orthopedic appliance often is greater, and the arch space for the remaining erupting dentition may be improved. The advocates for a delayed one-phase treatment have contended that comparable skeletal treatment effects can be achieved during definitive orthodontic treatment without putting the patient through an unnecessary initial phase.[264,265] The three prospective, randomized class II clinical trials mentioned earlier have supported this position, demonstrating that early skeletal improvement achieved from these appliances seems to represent accelerated growth and can be used just as effectively later during pubertal growth.[266–268] They also showed that there was substantial individual growth variability with no reliable predictors for a favorable growth response identified and early treatment did not reduce the need for dental extraction or orthognathic surgery during the definitive phase. These studies indicate that there is no adequate additional benefit in treatment outcome to justify the greater burden to the patient, their parents, and the orthodontist, as represented by an early phase that precedes the definitive phase of orthodontic treatment.[256]

It can be concluded from past retrospective and more recent prospective clinical trials that headgear treatment tends to have more of a maxillary-restrictive effect whereas functional appliances have more of a mandibular-enhancing effect. Either approach can be satisfactory and should be selected on the basis of patient acceptance of the appliance and dentoalveolar side effects (there is more maxillary dental retrusion with headgear and more mandibular protrusion with a functional appliance). The American and British prospective, randomized trials have clearly demonstrated that class II orthopedic treatment during the mixed dentition is justified only in selected cases in which there is short face height with impinging anterior overbite, predisposed to root resorption or trauma risk coupled with excessive overjet, sufficient aesthetic/psychosocial concern by the patient, or a precocious adolescent growth that substantially precedes dental development.[269,270] Treatment at an earlier age has been further justified by the argument that there is lack of reliable means of predicting mandibular growth and that improved cooperation with a removable appliance usually is present with the younger patient.[271]

In summary, the most ideal patient for class II orthopedic treatment would be pubertal with facially inclined and spaced maxillary incisors, lingually inclined mandibular incisors, a mild skeletal discrepancy, and a short or normal anterior face height. Although improvement in skeletal discrepancy is expected, the class II correction usually is due to a combined response of both the dentoalveolar and the skeletal components. Both headgear and class II functional

appliance use can be effective in limiting downward and forward eruption of the maxillary molars. However, the functional appliance tends to promote upward and forward eruption of mandibular molars, which may complement the correction in deep overbite cases but is counterproductive in patients with a long face. Because class II orthopedic appliances are toothborne, there may be some unwanted dentoalveolar change, including retraction of maxillary anterior teeth and protraction of mandibular anterior teeth. This compensatory change may be undesirable if the skeletal discrepancy ultimately requires orthognathic surgery for correction. With the advent of osseointegrated attachments or temporary skeletal anchorage devices, there may be the future possibility of preventing unwanted dentoalveolar change by attaching a force system directly to these attachments rather than using toothborne attachments. Orthodontic retention after class II orthopedic treatment as a part of definitive orthodontic treatment may require the use of a class II functional appliance at night for up to 24 months, particularly if they have significant adolescent growth remaining.

Anteroposterior Orthopedic Treatment: Class III

A class III skeletal relationship can be the result of a retrusive and/or deficient maxilla, a large and/or prognathic mandible, or most often, a combination of these skeletal problems. The prevalence of this type of malocclusion is approximately 3% to 5% of the U.S. population, with approximately 0.3% severe enough to be considered as handicapping.[209] Since the late 19th century, when headgear was being used for class II skeletal problems, the chin cup was the appliance used for orthopedic treatment of skeletal class III problems (Figure 53-29). Theoretically, an orthopedic force is transmitted to the mandibular condyles, compressing the condylar cartilage and limiting endochondral growth in order to decrease the ultimate length of the mandible.[272] Primate studies suggest that mandibular growth can be impeded with heavy full-time forces directed against the mandibular condyles.(Janzen & Bluher, 1965) Full-time wear with such excessive forces is unrealistic with humans. Most clinical studies, using tolerable forces (12–16 oz/side) and realistic daily wear (12–14 hr), have demonstrated that mandibular growth is not restrained, but rather vertically redirected from chin cup wear, rotating the mandible down and back, resulting in decreased chin prominence while increasing face height.[273–276] Clinical studies also have suggested that the long-term stability of these changes is poor.[277,278] The stability is dependent on favorable facial growth after active treatment, which is unpredictable but usually results in a resumption of the original facial growth pattern and relapse of the skeletal discrepency during or after adolescent growth. An additional side effect from chin cup wear is more lingual inclination of lower incisors due to the force of the chin cup to the lower lip and base of the mandibular anterior alveolar process.[274] Although this effect may help in the dentoalveolar compensation for the skeletal discrepancy, it usually does so at the expense of lower incisor crowding and increased relative chin prominence. Treatment with a chin cup may be an acceptable option for an individual with mild mandibular excess associated with decreased facial height (only one third of the prognathic white population) but is contraindicated when there is a normal or excessive facial height, because the treatment outcome simply would be trading one deformity for another. East Asian populations may benefit more often from this treatment because they tend to have shorter face heights than whites. However, chin cup treatment for mandibular prognathism has very limited utility, with its benefits usually negated from subsequent growth and ultimately resulting in unnecessary additional treatment burden to the patient.

Class III functional appliances also have been developed, limiting eruption of mandibular posterior teeth and promoting eruption of maxillary posterior teeth and resulting in downward and backward mandibular rotation with increased facial



A



B

FIGURE 53-29. Class III chin cup appliance. A retractive orthopedic force is directed against the chin posteriorly and superiorly toward the condylar heads. **A,** Chin cup from the late 19th century. **B,** Contemporary chin cup, with minimal change in design, but fabricated with more comfortable and durable materials. (**A** and **B,** Reproduced with permission.) A from Angle EH. Treatment of malocclusion of the teeth. Philadelphia (PA): S.S. White Dental Manufacturing Co; 1907. p. 194. B from Proffit WR, Fields HW, Sarver DM. Contemporary orthodontics. 4th ed. St. Louis (MO): Mosby; 2007. p. 301.

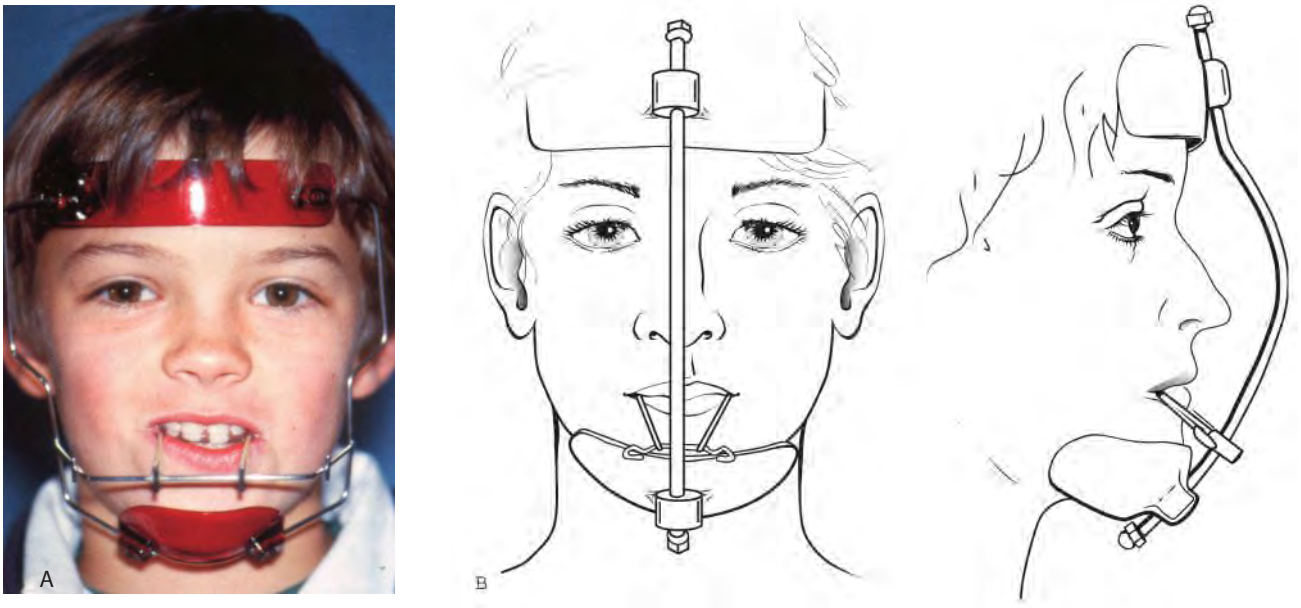


FIGURE 53-30. Class III maxillary protraction headgear, or facemask. A protractive orthopedic force is directed on the maxilla in a downward and forward direction, with the force being dissipated on the forehead and chin, distracting the circummaxillary sutures to augment AP maxillary growth. **A,** Delaire design. **B,** Petit design (frontal and lateral views). (**A,** Reproduced with permission from Proffit WR, White RP, Sarver DM. Contemporary treatment of dentofacial deformity. St Louis (MO): Mosby, 2003. p. 516. **B** adapted from McNamara JA, Brudon WL. Orthodontics and dentofacial orthopedics. Ann Arbor (MI): Needham Press, Inc.; 2001. p. 378.)

height. These functional appliances have few advocates owing to their effectiveness, similar to chin cup treatment, being limited to dentoalveolar changes and their inability to promote forward maxillary growth or attenuate mandibular growth.[279,280] Jean Delaire, a French dentist, was responsible for developing the protraction headgear or facemask, the most effective orthopedic appliance for skeletal class III problems since its introduction in the early 1970s (Figure 53-30).[281] Delaire recognized that the offending jaw in many of the skeletal class III problems was the maxilla, so he departed from the historic focus on the mandible and directed treatment at the retrusive or deficient maxilla. The appliance creates tension in the circum-maxillary sutures with elastics from the maxillary dental arch to a frame that uses the forehead and chin to dissipate the force anteriorly. Primate studies have demonstrated adaptive responses of the sutures to the stress from the distraction forces produced by this appliance.[213]

Depending on the developmental stage and size of the patient, a protractive force ranging from 2 to 4 pounds is applied to the facemask in an anterior and slightly inferior direction relative to the occlusal plane for 12 to 16 hr/day, usually for 6 to 9 months. Clinical studies have demonstrated clinically relevant maxillary skeletal protraction downward and forward on average with this appliance, with some concomitant protraction of the maxillary teeth due to the tooth-borne nature of the intraoral part of the appliance.[282–285] However, as with other forms of craniofacial orthopedic treatment, there is substantial variability and an unpredictable patient response to the appliance, ranging from no appreciable skeletal change to about 5 mm anterior maxillary movement.[286] It is important to achieve overcorrection of the anterior crossbite and anterior overbite because there is some relapse after discontinuation of treatment.[287] Additional effects of maxillary orthopedic protraction often include rotation of the maxilla downward in the posterior and upward in the anterior, downward and backward rotation of the mandible, and retraction of the mandibular incisors due to the reactive posterior force dissipated on the chin. These additional orthopedic and dentoalveolar changes that accompany maxillary skeletal protraction would be contraindicated for a class III pattern with excessive vertical development (long anterior face height and obtuse gonial angle) or where mandibular excess is the underlying cause of the problem.[288] In summary, the most ideal patient for maxillary orthopedic protraction would be younger than 8 years old and would have a mild skeletal discrepancy without mandibular prognathism, a short anterior face height, and retrusive maxillary incisors.

Although some clinicians have promoted the orthopedic distraction of the midpalatal suture to enhance subsequent anterior protraction with the facemask,[289] this has not demonstrated improved treatment efficacy.[290] The timing of facemask therapy often is recommended for patients in the primary to early mixed dentition (i.e., ages 4–8) owing to the increased patency of the maxillary sutures and compliance with appliance wear at this age.[291,292] As the patient ages, there is more interdigitation and ossification of the circummaxillary sutures, resulting in less skeletal and more dental response to the protraction forces. However, the earliest practical time for starting treatment is after eruption of first permanent molars owing to the poor anchorage provided by the primary dentition alone for the intraoral portion of the appliance. Although some clinical studies have found few differences between early and late treatment up until puberty,[292–295]

more controlled studies have shown that skeletal change diminishes after age 8 after which mostly tooth movement and backward mandibular rotation can be expected.[291] The limited skeletal change after this age is undoubtedly due to increased resistance from more mature sutures posterior and superior to the maxilla.

The stability of maxillary orthopedic protraction with a facemask is variable and dependent on whether mandibular growth ultimately overwhelms the skeletal improvement after active treatment. It should be expected that the original facial growth pattern will continue after treatment, often resulting in relapse of the skeletal discrepency. Approximately 30% of patients end up in anterior crossbite in late adolescence, most of whom will ultimately require orthognathic surgery to fully correct the class III malocclusion.[296,297] It should be kept in mind that long-term efficacy of facemask treatment has not been fully studied.

Toothborne class III orthopedic appliances, as with class II devices, result in dentoalveolar movements that accompany the skeletal changes. Treatment with the facemask causes protraction of maxillary anterior teeth and retraction of mandibular anterior teeth. The development of osseointegrated attachments or temporary skeletal anchorage devices may make it possible to transmit the orthopedic protraction force to intraoral skeletal attachments that prevent undesirable dentoalveolar changes.[298,299] Orthodontic retention for class III orthopedic treatment may require the use of a class III functional appliance at night for up to 24 months after definitive orthodontic treatment, particularly if they have post-treatment adolescent growth remaining. However, it is wise to wait before initiating the definitive treatment as close as possible to the completion of adolescent growth to minimize overcompensation for a skeletal discrepancy that ultimately requires orthognathic surgery. A particularly challenging aspect of treating skeletal class III problems (as well as vertical skeletal excess problems) is that short-term successful treatment, whether it occurs during or at the completion of adolescent growth, may relapse due to the continued class III growth pattern during adulthood.[300]

A recently developed novel method of class III orthopedic treatment, termed "bone-anchored maxillary protraction" (BAMP), has used maxillary posterior and mandibular anterior miniplates as skeletal anchorage in children 10 to 12 years of age (Figure 53-31).[301] Relatively light (150–250 g/side) full-time use of class III maxillomandibular intraoral elastic traction used with this method has produced remarkable maxillary skeletal protraction within about 12 months without the concomitant maxillary dentoalveolar protraction seen in more conventional facemask treatment (Figure 53-32). Preliminary data indicate that this treatment often results in anterior movement of the zygoma and maxilla and corresponding soft tissue with variable effects on the mandible in different individuals.[302, 303] There is evidence that treatment using this new method results in much larger maxillary advancement with better controlled vertical changes

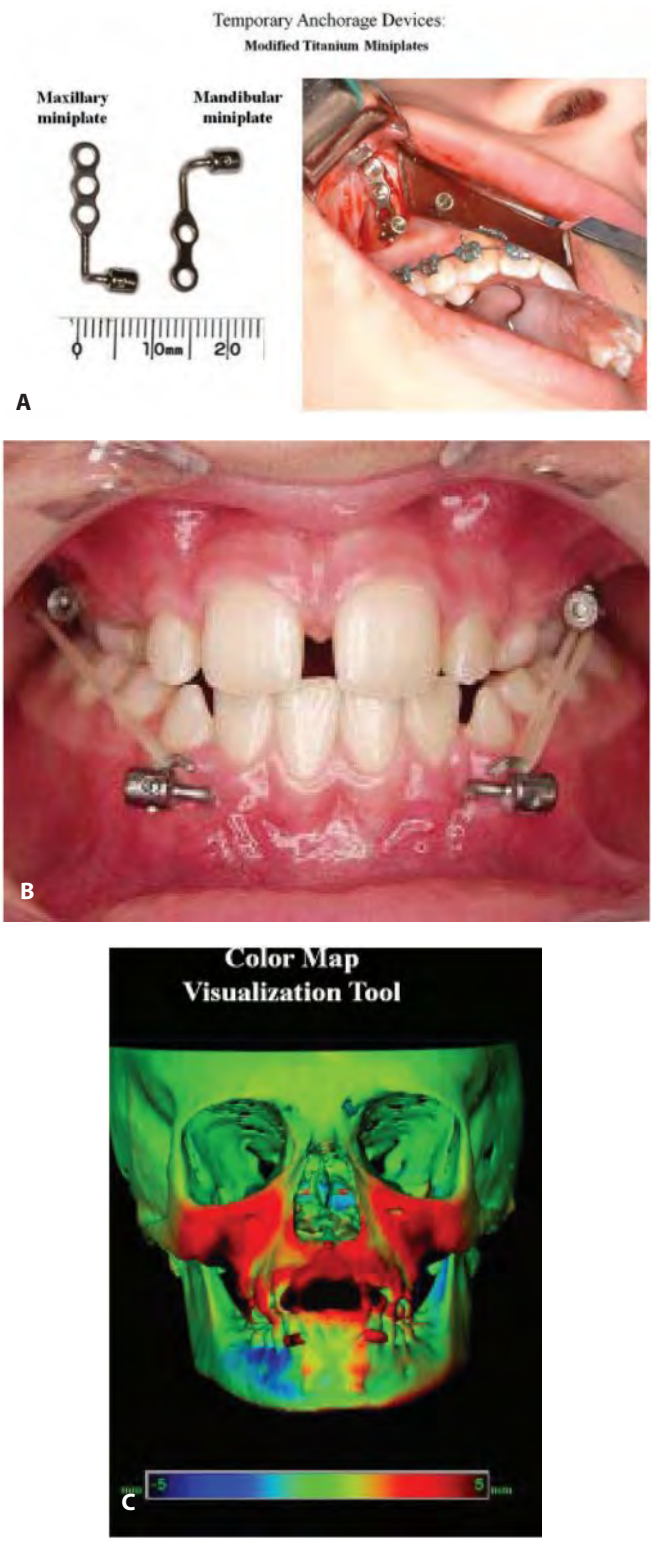


FIGURE 53-31. **A**, Modified titanium miniplates used as TADs for bone-anchored maxillary protraction (BAMP). **B**, Intermaxillary Class III elastics to customized attachment hooks on miniplates. **C**, Three-dimensional superimposition with color map tool used on individual patient. Areas on the red end of the spectrum represent outward movement during active treatment. Areas on the blue end of the spectrum represent inward movement during treatment. (**A-C**, Reproduced with permission from Am J Orthod Dentofacial Orthop 2010;137. pp. 276, 277).

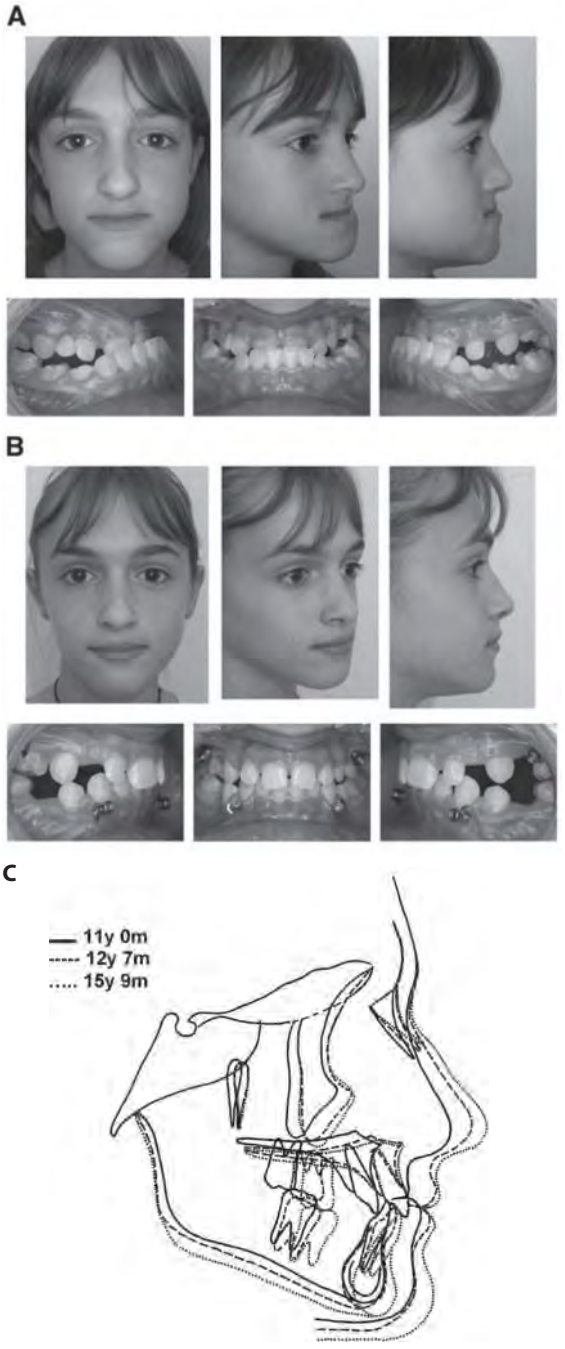


FIGURE 53-32. Class III orthopedic treatment with bone-anchored maxillary protraction (BAMP). A reciprocal force of 300 to 500 g is produced by maxillomandibular elastics, resulting in a maxillary force directed downward and forward and a mandibular force directed upward and backward. **A,** Before treatment. **B,** After treatment. **C,** Superimposed cephalometric tracings show the beginning of treatment, end of treatment, and follow-up. (**A–C,** Reproduced with permission from J Oral Maxillofac Surg 2009;67. pp. 2126, 2128.)

than the conventional orthopedic treatment with facemask. There may be a short window of treatment opportunity with this technique because mandibular miniplates cannot be placed before canine eruption and circum-maxillary sutural patency is more limited at older ages.

Vertical Orthopedic Treatment

Excessive vertical face height is characterized by a posterior maxilla that is inferiorly positioned, resulting in downward and backward rotation of the mandible, resulting in an increased mandibular plane angle and incompetent lips. Although most individuals with excess vertical face height exhibit anterior open bite, a minority will have excessive eruption of maxillary anterior teeth preventing open bite but worsening the excess vertical display of the maxillary gingiva and anterior teeth. The prevalence of excess vertical facial problems is less than 5% of the U.S. population with approximately 0.3% considered as handicapping.[209,210]

The orthodontist has not historically had very effective nonsurgical options to manage vertical skeletal problems. Orthopedic treatment strategy has historically been directed at restraining vertical maxillary growth and posterior dental eruption in order to promote anterior and superior mandibular rotation because it is usually associated with a class II skeletal discrepancy. A high-pull headgear is used to apply a superior intrusive force of 2 to 4 pounds to inhibit eruption of maxillary posterior teeth and compress circum-maxillary sutures to limit the downward development of the maxilla. An alternative to the use of high-pull headgear is a removable orthopedic appliance that incorporates interocclusal acrylic bite blocks in order to stretch the facial musculature and soft tissue beyond the normal resting vertical dimension, creating a reactive intrusive force against the mandibular as well as the maxillary teeth.[304,305] Neither of these methods has been very effective, perhaps owing to the extensive duration of wear required by the patient daily and throughout adolescent growth. Repelling magnets have been embedded in the opposing acrylic bite blocks to accentuate the intrusive force with no significant treatment benefit.[306–309]

High-pull headgear and interocclusal acrylic have been combined into one appliance with the hope that this approach may provide greater cumulative skeletal growth effects than use of either appliance alone.[262] Unfortunately, this combined treatment demonstrates no appreciable additional benefit in controlling excess vertical growth.[310]

In summary, neither the high-pull headgear or interocclusal acrylic alone or in combination offer appreciable effective treatment for vertical skeletal excess. A final challenge in the use of either or both of these orthopedic appliances to treat vertical maxillary excess is the inability to direct the orthopedic force in a straight vertical manner. Both methods include some posteriorly directed force to the maxilla that is not appropriate unless the maxilla is protrusive as well as inferiorly positioned. It also is counterproductive in a patient with maxillary vertical excess combined with a class III skeletal relationship. Just as the protraction headgear should not be used with this individual to address the anteroposterior skeletal problem, the high-pull headgear and/or interocclusal bite block are contraindicated in this same clinical situation to address the vertical excess.

The clinical introduction of temporary anchorage devices in the form of miniplates[311] or miniscrews[312,313] offers a new and potentially more successful way of orthopedically controlling mild to moderate vertical skeletal excess. Miniplates or miniscrews permit skeletal anchorage to intrude posterior teeth as a means of dentoalveolar compensation for the skeletal discrepancy. The posterior dental intrusion has shown short-term success in rotating the mandible upward and forward, closing mild to moderate anterior open bites. The extent to which posterior teeth can be orthodontically intruded by this means has not yet been determined. In addition, no substantial post-treatment data are yet available to confirm the long-term stability of this treatment approach. If this treatment takes place before the end of adolescent growth, it is likely that the continued vertical skeletal growth pattern will cause relapse. Even if this short-term successful treatment occurs after adolescent growth is complete, the continued vertical growth pattern during adulthood may lead to some return of the malocclusion. No studies to date have been able to demonstrate long-term stability with any nonsurgical orthopedic methods for correcting vertical maxillary excess, with or without an anterior open bite.[314] Achieving stability is particularly difficult owing to the extended duration of vertical facial growth continuing well beyond adolescence.[78] The long duration of active orthopedic treatment as well as duration of vertical growth makes the excessive vertical plane of space the most difficult to successfully manage by the orthodontist attempting facial growth modification.

Deficient vertical face height usually is a result of more posterior than anterior mandibular vertical growth with deficient vertical eruption of posterior teeth and anterior mandibular rotation, resulting in deep anterior overbite with lingual inclination of incisors. It is common for deficient face height to be associated with a mandibular or maxillary anteroposterior deficiency resulting in a class II or class III malocclusion. Orthodontic treatment directed at encouraging posterior eruption or extruding posterior teeth to improve facial height may include a functional appliance or a cervical headgear, in combination with an anterior bite plane, to disocclude posterior teeth. This may be successful with a class II malocclusion if upper molar eruption is limited, lower molar eruption is encouraged, and there is adequate mandibular growth to compensate for the resulting downward and backward mandibular rotation. It also may be successful with a class III malocclusion if upper molar eruption is encouraged, lower molar eruption is limited, and there is minimal mandibular growth after active orthodontic treatment. In contrast to the treatment stability of excess face height, long-term stability of treated deficient vertical face height is quite good.[315]

Conclusion

Facial orthopedic treatment may be effective in resolving mild to moderate skeletal discrepancies in some patients. The indications for craniofacial skeletal growth modification before adolescence is a maxillary deficiency in any plane of space or a progressive deformity, usually causing facial asymmetry. No growth modification can be justified for mandibular excess. Class II skeletal problems should not be addressed before adolescent growth except for a small number of selected cases involving trauma, root resorption risk, or psychosocial concerns. An orthopedic phase should be attempted with a specific treatment time frame established in order to assess treatment progress toward successful correction. This time frame must be honored in order to prevent protracted treatment with excessive dental compensations that need to be reversed for surgical correction. The orthodontist attempting growth modification must be mindful of the duration and extent of treatment to prevent excessive length and morbidity of the orthodontic treatment. If significant skeletal improvement is not being achieved within 6 to 8 months of starting orthopedic treatment, the case needs to be reevaluated and the growth modification treatment likely abandoned as a treatment choice.

It has become clear that there is great variability in individual treatment response, even when factoring out wear compliance and duration of treatment. It has not been possible to identify the variables to explain why some patients respond well and some do not demonstrate any significant skeletal improvement with treatment regardless of their facial morphology or the severity of their skeletal discrepancy. It is anticipated that future research will reveal the variables that will enable the clinician to better predict treatment response.

It also is expected that research will provide a greater understanding of the nature and extent of facial growth modification possible in individual patients as well as the type of appliance and timing of treatment to achieve the best outcome. The development of intraoral skeletal anchorage attachments holds promise for a future means of dissipating orthopedic forces to prevent unwanted dentoalveolar changes that at present occur with our toothborne appliances. Analogous attachments are at present undergoing clinical testing for surgically assisted orthopedic movements associated with distraction osteogenesis.[316,317] Recent and future advances in developmental biology and genomics hold great promise in increasing our understanding of the molecular and genetic mediators of craniofacial growth. This understanding will be crucial for us to make constructive modifications of our treatment methods to target these mediators in order to prevent or correct a craniofacial anomaly or developmental deformity.

Orthodontic Camouflage: Orthopedic Consequence versus Surgical Preparation

Orthodontic camouflage, rather than orthognathic surgery, may indeed be an appropriate treatment choice for some mild and moderate skeletal malocclusions in patients who are beyond pubertal growth. Successful camouflage is predicated on a favorable dental occlusion that will not be overly unfavorable to the patient's facial aesthetic appearance. Because aesthetic appearance is subjective in nature, it is essential to have the patient's and parent's perceptions dictate

whether camouflage is a reasonable option. Camouflage can be considered when there are normal vertical facial proportions and minimal crowding of teeth so that extractions can be used for retraction of anterior teeth rather than to simply alleviate crowding.

Most mild and some moderate skeletal class II malocclusions can be effectively camouflaged with extraction of two maxillary premolars and retraction of maxillary anterior teeth, leaving the posterior teeth in a class II occlusion. However, these cases should mainly be limited to those that present without significant dental crowding, some protrusion of the maxillary incisors, and where there is not significant maxillary gingival display on smiling. If the maxillary incisors are normally or palatally inclined before treatment, orthodontic retraction of these teeth may result in an even poorer aesthetic result than the original problem even if the occlusion is acceptable. The unaesthetic appearance includes not just the incisor inclination but often an unattractive retrusive upper lip and increased maxillary gingival exposure during smiling as well. This is why the recent introduction of palatal implants, miniplates, and miniscrews as skeletal orthodontic anchorage, which provide the opportunity for the orthodontist to retract maxillary incisors even further than was previously possible, is a mixed blessing.

It is more challenging to camouflage class III than class II skeletal malocclusions because considerable natural dentoalveolar camouflage (proclined maxillary incisors, lingually inclined mandibular incisors) is often already present before treatment. Unlike camouflage class II treatment, successful class III camouflage treatment is only limited to mild skeletal discrepancies. Additional maxillary incisor proclination may be unaesthetic, and further lingual inclination of mandibular incisors usually accentuates an already prominent chin. For this reason, extraction of mandibular premolars to permit more retraction of mandibular incisors to obtain positive overjet often compromises the aesthetic outcome. If interarch tooth size compatibility can be maintained, extraction of one mandibular incisor rather than two premolars may provide an acceptable compromise. It is crucial not to begin class III camouflage as a definitive treatment until completion of adolescent growth to minimize post-treatment relapse. Camouflage of an anterior open bite that is due to maxillary vertical excess has been notoriously unsuccessful in the past. It remains to be seen whether the newly introduced skeletal anchorage devices for posterior orthodontic intrusion offer stable long-term treatment of mild to moderate skeletal anterior open bite problems. An orthodontic technique for stably extruding anterior teeth has been introduced.[318] Unfortunately, this only exacerbates the excessive vertical display of maxillary gingiva and anterior teeth.

SUMMARY

The surgeon's understanding of craniofacial growth has an important impact on clinical treatment decisions to alter craniofacial morphology. This understanding is relevant to the appreciation of the role of orthopedic treatment in the prepubertal and pubertal patient to limit or preclude the need for corrective surgery at a later age. Clinically relevant modification of craniofacial growth is possible, but substantial advances will be necessary to elucidate how growth modification can be accomplished in a controlled and predictable manner to achieve an efficacious outcome. Optimal timing and stability of craniofacial surgery are dependent on a thorough appreciation of the sequence, timing, magnitude, and differential expression of craniofacial growth. The recent dramatic advances in developmental genetics and molecular biology, highlighted by complete mapping of the human genome, familial genetic studies, and gene targeting in mice and humans, promises an explosive increase to our understanding of the complex interactions of the genetic, epigenetic, and environmental influences that determine human craniofacial morphogenesis, prenatal development, and postnatal growth. A more thorough understanding of these influences and their interactions will be necessary to determine the best timing and method of clinical intervention to achieve the optimum treatment outcome.

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54

CHAPTER

Orthognathic Database Acquisition

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INTRODUCTION

Traditional methods of database acquisition remain the primary means of beginning the diagnostic and treatment planning phases of orthognathic surgery, although recent advances in imaging and improvements in accuracy have contributed to some improvements in this process. Prior to the new millennium, treatment planning in orthognathic surgery was based primarily on a system of clinical observations, a static set of records (dental models, two-dimensional radiographs), with the major goals of treatment directed toward satisfying lateral cephalometric goals (STOs [surgical treatment objectives]). These goals include the correction of specific measurements (sella-nasion–A point and A point–nasion–B point differences) or analytical norms (Steiner, Ricketts), or even comparison of lateral cephalometric film tracings of patients with craniofacial skeletal dysplasia[1–3] to templates having average skeletal proportions derived from longitudinal growth studies.[4] The most significant shortcoming of reliance on the lateral cephalometric film as the primary determinant of treatment goals is that it did not take into account the resting and dynamic hard-soft tissue relationships, which are the most critical aspects in treatment planning for both orthodontics and orthognathic surgery. Furthermore, cephalometric analysis quantifies dentoskeletal relationships in angular and linear measures, which are not entirely representative of the three-dimensioanl multidimensional interrelationship of craniofacial structures. In other words, the integumental soft tissue drape may, on occasion, be inconsistent with the underlying skeletal framework in an individual patient. Whereas the skeletal framework may be reasonably stable after adolescence, the soft tissues become more susceptible to maturational and age-related changes. The cephalometric approach to treatment planning is now considered "procrustean," after the story based on Greek mythology, in which Procrustes was an innkeeper with only one bed, and if a traveler was too tall for the bed, Procrustes would cut off the traveler's feet so that he or she would fit the bed. A traveler who was too short would be stretched on a rack to likewise fit the bed in length. This charming story relates to this chapter in that the inappropriate application of the same hard tissue lateral cephalometric analysis to all patients would result in all of our patients being crammed into the same bed, with the goal of creating patients with similar, or "normal," cephalometric measurements, but with poor final functional and aesthetic results.

The contemporary approach to the surgical-orthodontic treatment of dentofacial deformities illustrates the importance of the use of dentofacial proportionality instead of applying absolute linear or angular norms to individual patients. Individualized patient-specific treatment planning is the focus of this chapter.

CONTEMPORARY ORTHOGNATHIC TREATMENT PLANNING

The emphasis on orthognathic diagnosis and treatment planning will lead the clinican into a new era and methodology of patient analysis and establishment of treatment goals. In modern orthognathic surgery, treatment goals are determined through systematic clinical examination and quantification of the individual patient's dentofacial characteristics. Therefore, the purpose of this chapter is to introduce a method of systematic three-dimensional dentofacial analysis with emphasis on both static and dynamic relationships as well as both functional and aesthetic treatment objectives.

Problem-oriented treatment planning has served the orthodontist and oral and maxillofacial surgeon well in the past several decades by focusing on the problems in need of correction, including the identification of solutions for each problem. The natural progression of problem-oriented treatment planning should include both the identification of favorable attributes as well as the abnormal features and problems in facial proportions. The reason for this is the realization that focusing solely on the problems and their solutions may result

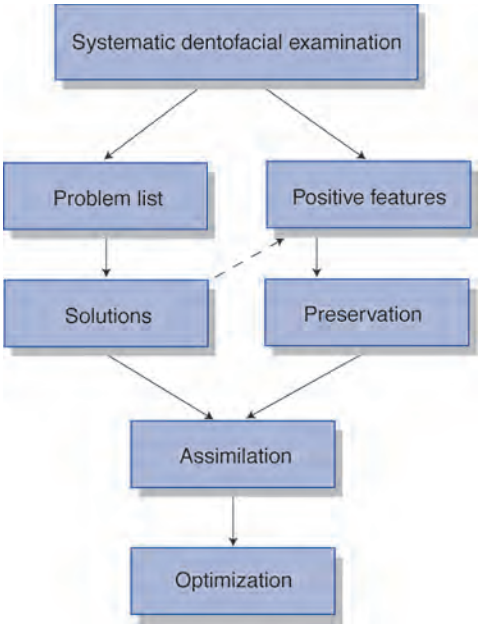


FIGURE 54-1. Contemporary treatment planning flow chart. After the clinical examination and databasing of the quantitative measurements, both problems and positive attributes are identified. Solutions for the problems are identified and the *dotted arrow* indicates that each potential solution can negatively affect a positive feature. Therefore, the advantage of measuring both problems and positive features permits the clinician to recognize the potential negative impact that any given solution has on the positive attributes. This "decision tree" leads to correction of the problems but also preservation of the positive attributes. The clinician then assimilates all of this information for optimization of treatment.

in a treatment plan that potentially has a negative effect on the positive attributes already present in a patient. A classic orthodontic example is one in which the extraction of maxillary premolars in the correction of a skeletal class II malocclusion, although satisfying functional and occlusal issues, may result in profile flattening and a negative effect on facial appearance. In orthognathic surgery, a good example is widening of the alar base secondary to maxillary advancement and/or impaction. The goal of orthognathic treatment is the correction or optimization of negative attributes, while at the same time preserving those attributes that are deemed favorable (Figure 54-1).

This systematic approach to clinical examination of the patient is essential for the development of an optimization-oriented database. All clinically detectable deviations from the optimal range fall into the two broad categories, function and aesthetics.

Oral Function and Dentofacial Deformity

Patients with severe discrepancies in the size and position of their jaws and teeth often have difficulty with oral function. Certain foods may be difficult to incise and chew, and speech may also be negatively affected by a jaw deformity. If the patient cannot bring the tongue and lips into the proper position, it may not be possible to produce a specific sound properly. Other than a careful clinical examination of the patient, it is doubtful that diagnostic tests of oral function could be performed adequately in the dental or surgical office. The relationship of temporomandibular joint (TMJ) problems to severe malocclusions and dentofacial deformities is complex, but important. Although there is some evidence that patients with specific types of malocclusion are more susceptible to TMJ problems (e.g., prevalence of internal derangement in class II deformities), the increased risk is relatively small.[5] In general terms, patients with dentofacial deformity are similar to patients with normal facial proportions with regards to the prevalence of TMJ problems.[6] In addition, speech and mastication are commonly affected negatively by dentoskeletal deformities that may affect tongue position and lip competence, and much research has focused on the effects of correction of the skeletal deformity on speech and bite forces, with mixed results.

Facial Aesthetics and Dentofacial Deformity

Appearance and dentofacial aesthetics can be divided into three subcategories: macroaesthetics, miniaesthetics, and microaesthetics (Figure 54-2). The specific aesthetic concerns of the patient can be elucidated through open-ended doctor-patient communication and then integrated into the diagnostic decision-making tree. The surgeon and orthodontist should be sensitive to the patient's aesthetic desires, balancing them against cultural and familial standards and norms. The physical burden of orthodotnic and surgical treatment is borne by the patient and must be considered when determining the extent of treatment intervention. For example, when deciding whether treatment should involve orthodontics alone, orthodontics and orthognathic surgery, or acceptable orthodontic camouflage, the patient should understand the risk-benefit ratio of any given treatment sequence.

DATABASE COLLECTION

Primary database collection begins with the clinical examination and is supplemented with static and dynamic recordings of the patient in three spatial dimensions. Record-taking should replicate the functional and aesthetic facial presentation of the patient. Findings from the clinical examination should be either confirmed or challenged by data obtained from the records. The analysis of the clinical database will generate a diagnostic summary and optimized problem list. An emerging soft tissue paradigm in surgical orthodontic treatment planning has refocused analysis on facial proportionality and balance versus reliance on normative data derived from cephalometrics.[7] The art of surgical orthodontics rests in the ability to envisage the patient's desired three-dimensional soft tissue outcome and then retroengineer the dental and skeletal hard tissues to produce such a change. The concept of retroengineering is explained later in this

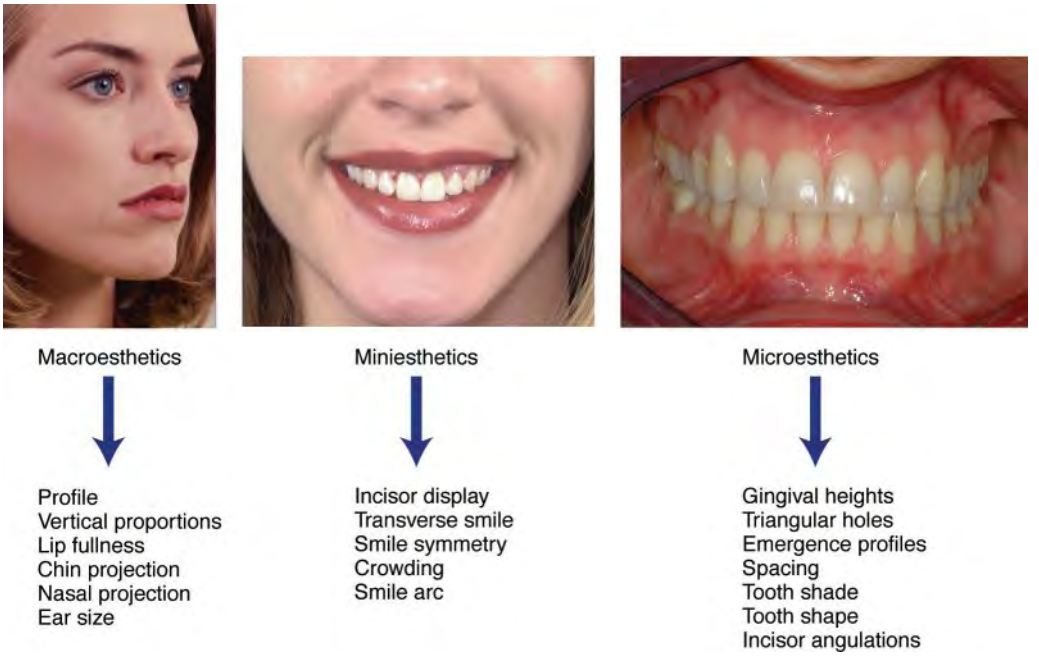


FIGURE 54-2. Recommended subcategories of appearance and aesthetics.

chapter under technologic applications to orthognathic treatment planning.

In today's clinical environment there are three methods of data collection. The first and most commonly used method includes still facial photography, dental study models, and lateral and posteroanterior cephalometric radiographs. The second is the use of databasing programs to document direct clinical measurement of the patient's resting and dynamic, or functional, relationships. The third involves the use of digital video to record the dynamics of facial movement. This methodology as it currently exists does not dynamically quantify movement, but emerging technologies should lead to research into the clinical application of quantification of dynamic facial movements.

Conventional Diagnostic Records

Standard orthodontic records have not changed significantly in many years, but contemporary records in surgical orthodontic treatment are changing rapidly. Surgical orthodontics demands three-dimensional treatment planning of all patients. In clinical practice, standard diagnostic records include digital photographs, two-dimensional radiographs, and dental study models (plaster, mounted or unmounted, or digitally scanned dental models [e.g., OrthoCAD]). The facial images, which are considered universally standard records, include a frontal view at rest, frontal smile, and profile at rest images. Whereas these orientations do provide an adequate amount of diagnostic information, they do not contain all of the information needed for three-dimensional visualization and quantification. Orthognathic surgery requires expansion of the database compared with conventional orthodontic treatment.

The suggested records can be divided into two groups: static and dynamic. The accepted facial photographic recordings should include frontal smile close-up, oblique (or three-quarter view) facial smile, oblique smile close-up, and profile smile, as well as bird's-eye and worm's-eye views, when indicated for facial syndromic or nonsyndromic asymmetries.[8]

Direct Measurement as a Biometric Tool

The goal of the clinical examination is to quantitatively assess soft and hard tissue attributes of the dentofacial complex and record what elements are satisfactory and which are in need of correction or optimization. Whereas clinical examination procedures vary greatly among practitioners, measurements should be thorough, systematic, and consistent, in an attempt to minimize the chance that something of importance will be overlooked. It is critical to avoid a situation in which the clinician performs a cursory examination, recording only brief notes regarding abnormalities observed, without recording any other descriptive data. This practice is often justified by the assumption that most diagnostic decisions can be made from the records after the patient leaves. This is a poor assumption for several reasons. First, static records cannot reflect the dynamic relationships that are important in the overall functional assessment of the patient. For example, the simple idea of the relationship of the upper incisor at rest and on smile is not reflected on radiographs or models and is poorly evaluated in photographs. Second, information that may have not looked important enough to document during a cursory examination may be important later and, therefore, would be unavailable because it was not recorded. Third, a thorough and comprehensive examination record that includes normal observations provides an

accurate medicolegal document. It is difficult for an unhappy patient to prove negligence when it is clear that comprehensive clinical information was obtained and utilized in the treatment planning phases. The more thorough and well documented the record is, the more valuable it is if problems arise after treatment.

Contemporary clinical examination uses a computer-databasing program to facilitate data entry, and these data are then merged into reports and treatment planning screens or forms.[9] Each clinical characteristic in the examination has a pop-up menu containing all of the possible descriptions for that particular trait (Figure 54-3). By using a computer interface, the surgeon or orthodontist saves valuable time in both the clinical examination and the diagnostic and treatment planning workup. The information is then stored for recall and analysis and can even have predefined parameters that identify problematic measurements automatically.



FIGURE 54-3. A computer databasing program facilitates data entry, and these data are then merged into reports and treatment planning screens or forms. Each clinical characteristic in the examination has a pop-up menu containing all of the possible descriptions for that particular trait.

As an example of how this interface facilitates the examination of dynamic hard-soft tissue relationships, the following frontal measurements may be performed systematically in evaluation of anterior dental display, both at rest and at smile:

- Philtrum height: The philtrum height is measured in millimeters from subnasale (the base of the nose at the midline) to the most inferior portion of the upper lip on the vermilion tip beneath the philtral columns. The absolute linear measurement is not particularly important, but what is significant is its relationship to the upper incisor and the commissures of the mouth. In the adolescent, it is common to find the philtrum height to be shorter than the commissure height, and the difference can be explained in the differential in lip growth with maturation.
- Commissure height: The commissure height is measured from a line constructed from the alar bases through subspinale, then from the commissures perpendicular to this line.
- Interlabial gap: The interlabial gap is the distance in millimeters between the upper and the lower lips, when lip incompetence is present.
- Amount of incisor show at rest: The amount of upper incisor display at rest is a critical aesthetic parameter because one of the inevitable characteristics of an aging tooth-lip relationship is diminished upper incisor show at rest and on smile. For example, an adult patient who displays 3 mm of gingival display on smile and 3 mm of upper incisor at rest should carefully consider only maxillary incisor intrusion or maxillary impaction to reduce gingival display, because reduction in gingival display also results in diminished incisor show at rest and during conversation (a characteristic of the aging face).
- Amount of incisor display on smile: On smile, patients will show either their entire upper incisor or only a percentage of the incisor. Measurement of the percentage of incisor display, when combined with the crown height measured next, leads the clinician to decide how much tooth movement is required to attain the appropriate smile for that patient.
- Crown height: The vertical height of the maxillary central incisors in the adult is measured in millimeters and is normally between 9 and 12 mm, with an average of 10.6 mm in males and 9.6 mm in females. The age of the patient is a factor in crown height because of the rate of apical migration in the adolescent.
- Gingival display: There is variability in what is aesthetically acceptable for the amount of gingival display on smile, but it is important to always remember the relationship between gingival display and the amount of incisor shown at rest. In broad terms, it is better for a patient to be treated less aggressively in reducing smile gumminess when considering that the aging process will result in a natural diminishment of this characteristic. A gummy smile is often more aesthetic than a smile with diminished tooth display.
- Smile arc: The smile arc should be defined as the relationship of the curvature of the incisal edges of the maxillary incisors and canines to the curvature of the lower lip in the posed social smile.[10,11] The ideal smile arc has the maxillary incisal edge curvature parallel to the curvature of the lower lip upon smile, and the term *consonant* is used to describe this parallel relationship. A *nonconsonant*, or flat, smile arc is characterized by the maxillary incisal curvature being flatter than the curvature of the lower lip on smile. The smile arc relationship is not as quantitatively measurable as the other attributes, so the qualitative observation of consonant, flat, or reverse smile arcs is generally cited.

It critically important to measure the previously described characteristics in orthognathic cases for a variety of reasons. The following case illustrates the significance of resting and dynamic soft tissue measurements, and how surgical or orthodontic treatment planning is determined by direct measurement as much as it is by indirect cephalometric radiographic analysis. The role of cephalometrics is discussed later in this chapter, and the emphasis is less on static comparisons to norms and more on its coordination with the soft tissue overlay of the individual face and the use of predictive algorithms to arrive at final macrotreatment decisions.

The patient in Figure 54-4 was a 16-year-old male who presented with a chief complaint of excessive gingival display on smile, or a "gummy smile" (see Figure 54–4A). He had finished orthodontic treatment 1 year ago, and the orthodontist believed that the only way to improve smile aesthetics further was to consider maxillary superior repositioning via a Le Fort I osteotomy. The oral and maxillofacial surgeon wanted to wait until growth completion, but the patient's mother pursued further investigation.

The examination revealed an excellent occlusion, and the macrorelations were also normal in terms of profile and facial proportions. The anterior tooth-lip relationships were as follows:

- Resting relationships (see Figure 54-4B)
  - Philtrum height: 25 mm
  - Commissure height: 25 mm
  - Maxillary incisor at rest: 2 mm
- Dynamic relationships (see Figure 54-4C)
  - Percentage of maxillary incisor display on smile: 100%
  - Maxillary incisor crown height: 8 mm
  - Gingival display on smile: 4 mm
  - Smile arc: consonant

It is useful to outline the various etiologies of excessive gingival display on smile and characteristics seen with each in order to demonstrate the decision-making process in problem-oriented treatment planning with optimization:

- Vertical maxillary excess: VME is characterized by a disproportionately long lower facial height, lip incompetence, excessive incisor display at rest, and excessive gingival display on smile.



FIGURE 54-4. **A–D,** Case illustration of direct measurement of lip-tooth-gingival relationships.

- Short philtrum: The philtrum height is shorter than the commissure, with excessive incisor display at rest, and a reverse resting upper lip line.
- Excessive smile curtain: Excessive animation of the upper lip on smile is seen, displaying more tooth and gingiva than desired.
- Short crown height: If the anterior incisor height is short, excessive gingival display may result.

In this case, VME was ruled out because facial proportionality was normal, no lip incompetence was present, and only 2 mm of upper incisor showed at rest. The second possibility, a short philtrum, was ruled out because the philtrum and commissure heights were the same and no reverse upper lip resting characteristics were noted. The third possibility, excessive curtain, was eliminated because the vermilion was adequate on smile and the margins of the commissure and philtrum even on posed smile. The fourth possibility, short crown height, was significant because the maxillary incisors measured to be only 8 mm in height.

Treatment options to decrease gingival display included maxillary impaction, orthodontic intrusion of maxillary incisors, or periodontal crown lengthening.

- Maxillary impaction: A 4-mm superior repositioning of the maxilla would decrease the gumminess of the smile, but would result in a –2-mm upper incisor show at rest, greatly hastening the aging characteristics of the face and smile.
- Orthodontic intrusion of the maxillary anterior teeth: This would likewise result in reduction of incisor display at rest but would also flatten the already consonant smile arc.
- Periodontal crown lengthening: The increase in anterior crown height decreases the gumminess of the smile (appropriate because the teeth are short) and optimizes treatment by *not* decreasing incisor display at rest and by maintaining the consonant smile arc.

After discussing all these options with the family, the family decided to proceed with the third and recommended option of crown lengthening, with an excellent aesthetic outcome (see Figure 54-4D).

In summary, this case demonstrates the new direction in dentofacial treatment planning, even though the final result was not an orthognathic treatment plan. This case was selected to make the point that through careful observation and measurement, the appropriate treatment plan was performed.

Digital Videography

Dynamic recording of patient's facial motion is accomplished with the use of digital videography.[12] This technology may be used to document and evaluate such characteristics as range of mandibular motion on opening and laterotrusive movements, deviations on opening, smile, and speech. Digital video and computer technology have primarily been used to record anterior tooth display during speech and smiling. Digital videos can be recorded in a standardized fashion with the camera at a fixed distance from the subject. It is recommended that these images be taken in a standard format with emphasis on natural head position, so that future analysis and research possibilities may be maximized. It is also recommended that video be taken in the frontal, oblique, and lateral dimensions. Clinically, an example of where this technology is most relevant is the patient with an asymmetrical smile. The question that arises is whether or not the patient has a dental asymmetry, skeletal asymmetry, or asymmetrical movement of the lip curtain during animation. The single smile photograph cannot corroborate the clinical impression gained during the data collection process. The video clip may be reviewed and evaluated during all planning phases of treatment as well as for comparison of the orthognathic treatment effects (Figure 54-5).

SYSTEMATIC CLINICAL EXAMINATION OF DENTOFACIAL DEFORMITIES

The importance of clinical observation and direct measurement of the interaction of hard and soft tissues in planning

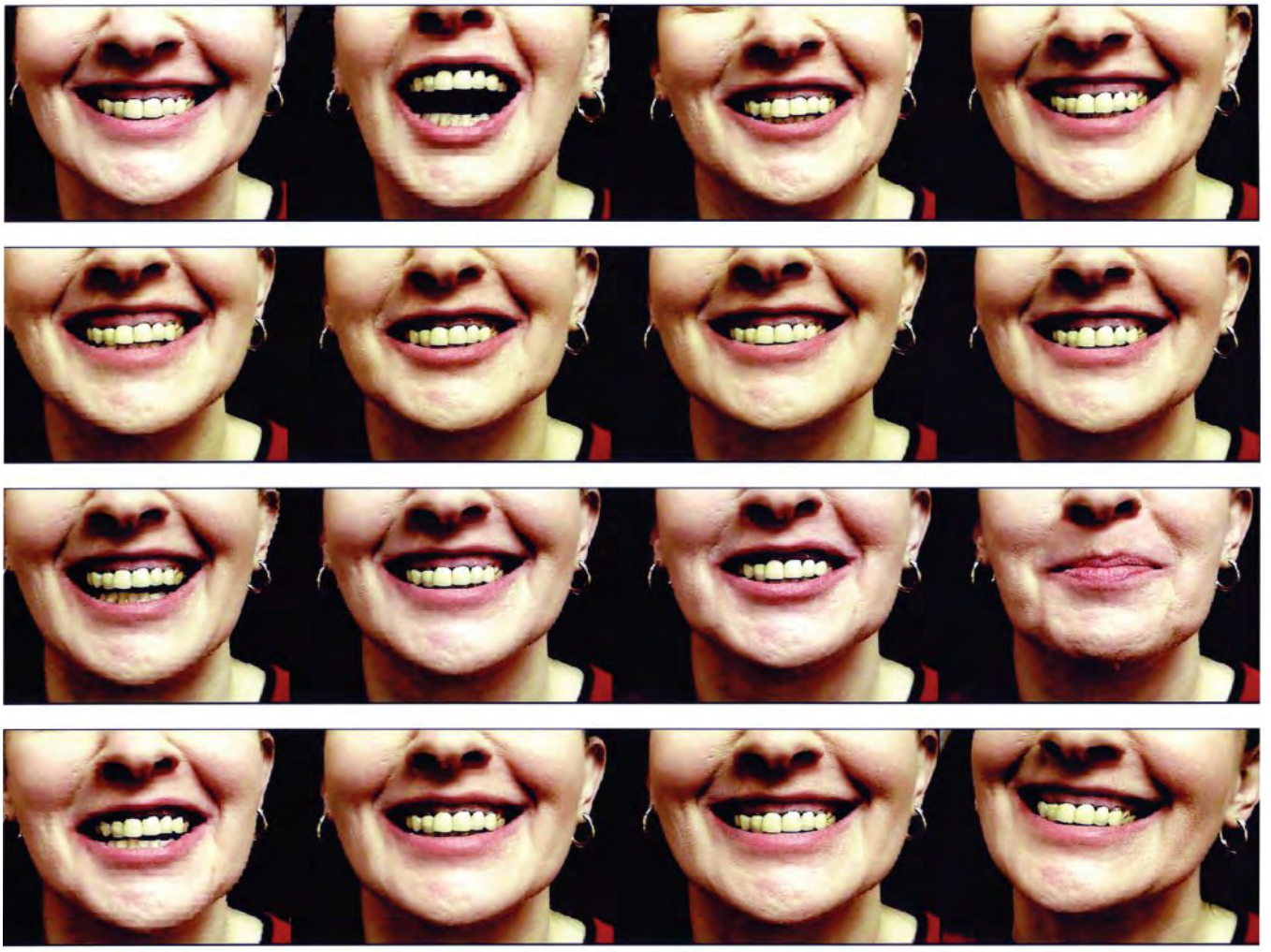


FIGURE 54-5. The video clip may be reviewed and evaluated during all planning phases of treatment as well as for comparison of the orthognathic treatment effects.

appropriate combined orthodontic and orthognathic treatments of dentofacial deformity has been discussed, and in this section, the components of the examination from the macro-, mini-, and microperspectives are described.

Macroaesthetic Examination: Frontal View

The facial areas for macroaesthetic examination, as investigated from the frontal view, can be summarized as follows:

- Vertical proportions
  - Facial heights:
  - Lower facial third
- Transverse proportions
  - Rule of fifths
  - Middle fifth
    - Inner canthi
    - Alar base
  - Medial two fifths
    - Outer canthi
    - Gonial angles of mandible
  - Outer two fifths
    - Ear deformity
    - Ear projection
  - Nasal anatomy
  - Alar base
  - Columella
  - Nasal tip
  - Dorsum
- Transverse symmetry
  - Nasal tip to midsagittal plane
  - Maxillary dental midline to midsagittal plane
  - Mandibular dental midline to symphysis
  - Mandibular asymmetry with or without functional shift
  - Maxillomandibular asymmetry
  - Chin asymmetry

The starting point for the macroaesthetic examination is the frontal perspective. Transverse and vertical relationships make up the major components of the frontal examination and analysis. As emphasized in the introduction, the *proportional* relationship of height and width is far more important



FIGURE 54-6. **A,** The mesocephalic facial type is characterized by equal vertical facial thirds. **B,** The brachycephalic facial type appears square with a diminished lower third. **C,** The dolichocephalic facial type appears ovoid with an increased lower third.

than absolute values in establishing overall facial type. Faces can be broadly categorized as either mesocephalic, brachycephalic, or dolichocephalic (Figure 54-6).[13] The differentiation between these facial types has to do with the general proportionality of facial breadth to facial height, with brachycephalic faces being broader and shorter in comparison with the longer and more narrow dolichocephalic faces. Generally, the most attractive faces tend to have common proportions and relationships that generally differ from normative values.[14]

Vertical Facial Proportions

The ideal face is vertically divided into equal thirds by horizontal lines adjacent to the hairline, the nasal base, and the menton (Figure 54-7A). Surgical orthodontic treatment is in a large part limited to the lower facial third. Measurement of the upper face is often hindered by the variability in identification of broad landmarks such as the location of the hairline and radix.

The clinical examination begins with the evaluation of lower facial height. In the ideal lower third of the face, the upper lip makes up the upper third, and the lower lip and chin compose the lower two thirds (see Figure 54-7B). Disproportion of the vertical facial thirds may be a result of many dental and skeletal factors, and these proportional relationships may help us define the contributing factors related to vertical dentofacial deformities.

In the following sections, case illustrations of orthognathic changes in vertical proportionality are presented.

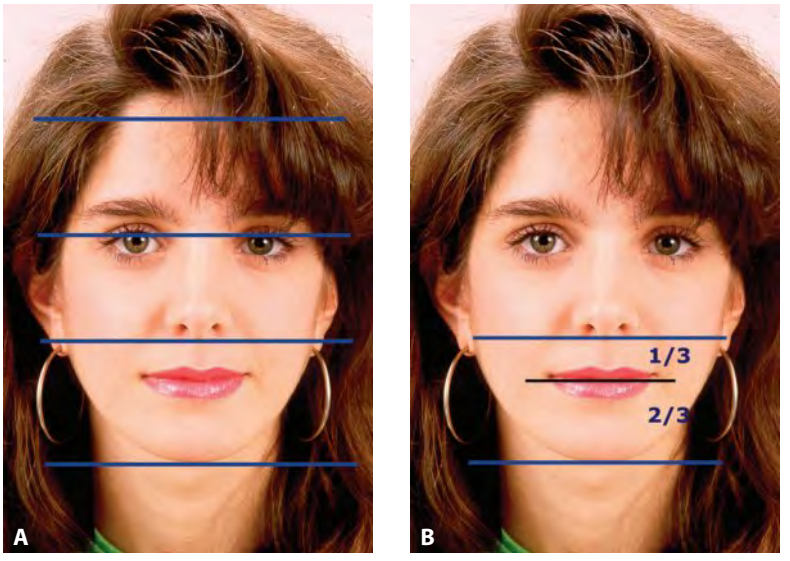


FIGURE 54-7. **A,** The ideal face is vertically divided into equal thirds by horizontal lines adjacent to the hairline, the nasal base, and menton. **B,** In the ideal lower third of the face, the upper lip makes up the upper third, and the lower lip and chin compose the lower two thirds.

Short Vertical Proportions

The patient in Figure 54-8 presented for correction of her class II deep bite secondary to her mandibular deficiency. Her anterior vertical relationships were characterized by a short lower facial third relative to her upper thirds (see Figure 54-8A and B). In addition, the lower third was comprised of a 45:55 vertical relation of the upper lip to lower lip and chin height. Recalling that the ideal proportions of the lower face are one-third upper lip and two-thirds lower lip and chin, the treatment plan was clearly a result of the direct clinical examination rather than any cephalometric standard. Other important clinical measurements entered into the decision process. Differential diagnosis for a short lower facial height included the following:

- Vertical maxillary deficiencies, which are then characterized by the following features:
  - Short lower facial third
  - Diminished maxillary incisor display at rest
  - Diminished incisor display on smile
- Diminished chin height, ascertained through the proportionality in the lower face rather than a linear cephalometric value
- Posterior dental collapse secondary to the loss of posterior dental support

The functional goal of mandibular advancement to correct the class II dentoskeletal relationship was obvious, but an aesthetic adjunctive consideration was a vertical genioplasty to optimize the macroaesthetics of her vertical facial thirds. The final diagnosis depended not only on the vertical facial proportionality but on the measurement of the resting tooth-lip relationships as well in order to more clearly define the etiology of the lower facial height. In this case, our patient displayed 3 mm of maxillary incisor at rest and all of her maxillary incisor on smile (see Figure 54-8C), which was inconsistent with vertical



FIGURE 54-8. **A,** This patient was referred for correction of her class II deep bite secondary to her mandibular deficiency. **B,** Evaluation of her anterior vertical relationships was characterized by a short lower facial third relative to her upper thirds. The lower third was comprised of a 45:55 vertical relationship of the upper lip to lower lip and chin height. The ideal proportions of the lower face are one-third upper lip and two-thirds lower lip and chin; thus the treatment plan was derived from the direct clinical examination of this patient rather than any cephalometric standard. **C,** Our patient displayed 3 mm of maxillary incisor at rest, and all of her maxillary incisors on smile, inconsistent with vertical maxillary deficiency. **D,** The treatment plan was orthodontic preparation for mandibular advancement and vertical genioplasty. **E,** The post-treatment frontal view demonstrates balance of the vertical facial thirds. **F,** The vertical incisor position at smile was maintained, while the lower facial third was vertically augmented. **G,** The profile view shows improved mandibular projection relative to the upper face, improved chin-neck angle, and improved chin-neck length.

maxillary deficiency. Because the chin height was short, the final diagnosis was mandibular deficiency with short chin height. Therefore, the recommended treatment plan was orthodontic preparation for mandibular advancement and vertical genioplasty (see Figure 54-8D) to increase the lower facial height (see Figure 54-8E-G).

Long Vertical Proportions

Long lower facial height is due to one of two possibilities: (1) VME or (2) excessive chin height. The clinical keys that may be associated with VME are gummy smile, open bite, lip incompetence, and steep mandibular plane as evidenced by gonial angle form. Excessive chin height is measured from the lower vermilion to the soft tissue menton. The clinical keys that may be associated with excessive chin height are lower facial third disproportionate from the one-third upper lip to two-thirds low lip and chin ratio and the absence of VME characteristics.

The patient in Figure 54-9A was referred for correction of an anterior open bite and a gummy smile. Our systematic examination revealed the following problem list and characteristics:

- Frontal proportions at rest
  1. Long lower facial third
  2. Disproportion of chin height with the upper lip occupying 25% of the lower facial third and the lower lip and chin occupying 75% of the lower third of the face
  3. Lip incompetence of 5 mm
  4. 8 mm of maxillary incisor display at rest
  5. Midsymphysis to right 3 mm with no functional shift
  6. Lip strain on closure (Figure 54-9B)

Clinical assessment of the frontal resting macroaesthetic evaluation: This patient had most of the macrocharacteristics of VME with a long lower facial height and excessive incisor display at rest. Excessive chin height was also a contributor to the lower facial height disproportion, as is evidenced by the upper lip and chin height clinical proportions.

- Frontal proportions on smile (see Figure 54-9C)
  1. 100% of maxillary incisor displayed on smile
  2. Excessive gingival display on smile with 3 mm gingival display at the right cuspid and 5 mm at the left with a transverse cant to the palatal plane
  3. Transverse cant to the maxilla with the left side down 2 mm more than the left

Clinical assessment of the frontal dynamic (smiling) macroaesthetic evaluation: A gummy smile was present but with normal incisor crown height. This would exclude cosmetic periodontal crown lengthening as the primary therapeutic choice for improvement of the gummy smile. The asymmetry of the maxilla is in compensation for the mandibular asymmetry and results in a canted frontal occlusal plane and smile line.

- Oblique at-rest facial observation (see Figure 54-9D)
  1. Excessive lower facial height
  2. Lip strain and excessive chin height
  3. Flat labiomental sulcus
  4. Nasal form judged to be quite adequate

Clinical assessment of the oblique macroaesthetic evaluation: The flattened labiomental sulcus was secondary to the excessive lower facial height, lip incompetence, and chin deficiency.

- Oblique smiling facial observation (see Figure 54-9E)
  1. No noticeable anteroposterior cant to the maxillary occlusal plane
  2. The smile arc was consonant
  3. The excessive gingival display was also evident on the oblique smile

Clinical assessment from the oblique smiling macroaesthetic evaluation: Because the smile arc was consonant, alteration of the palatal plane would not have been indicated through either surgery or orthodontic incisor repositioning.

- Profile evaluation (Figure 54-9F)
  1. Long lower facial third
  2. Long chin height
  3. Flat labiomental sulcus
  4. Lip strain on closure

Clinical assessment of the profile macroaesthetic evaluation: As expected from the frontal and oblique characteristics, the lateral profile reflected the overall skeletal and dental characteristics of VME, but the chin deficiency that became evident on the oblique view was clearly demonstrated on the profile view.

The functional problem of the anterior open bite in this nongrowing patient necessitated superior repositioning of the maxilla to correct the functional complaint (see Figure 54-9G-K). The exact surgical movements were directed by the clinical examination and measurements. Because the patient had the clinical diagnosis of VME, maxillary impaction was indicated, but some discretionary decisions were needed for appropriate position of the maxilla from the aesthetic standpoint. From the frontal dimension, the left side of the maxilla was impacted 2 mm more than the right in order to level the smile line. The differential degree of impaction reflected the degree of the maxillary compensation for the mandibular asymmetry.

Transverse Facial Proportions

The assessment of the transverse components of facial width is best described by the rule of fifths.[9] This method describes the ideal transverse relationships of the face. The face is divided sagittally into five equal parts from helix to helix of the outer ears (Figure 54-10). Each of the segments should be one eye distance in width. Each transverse fifth should be individually examined and then assessed as a complete group.

The middle fifth of the face is delineated by the inner canthus of the eyes. A vertical line from the inner canthus should be coincident with the alar base of the nose. Variation in this facial fifth could be due to transverse deficiencies or excesses in either the inner canthi or the alar base. For example,



FIGURE 54-9. **A,** This patient was referred for correction of an anterior open bite and a gummy smile. **B,** Lip strain on closure. **C,** Frontal proportions on smile with 100% of maxillary incisor displayed. There was excessive gingival display on smile with 3-mm gingival display at the right cuspid and 5 mm at the left with a transverse cant to the palatal plane. Transverse cant to the maxilla with the left side down 2 mm more than the left. **D,** Oblique at-rest facial observation with excessive lower facial height, lip strain and excessive chin height, and a flat labiomental sulcus. The nasal form was judged to be quite adequate. **E,** Oblique smiling facial observation with no noticeable anteroposterior cant to the maxillary occlusal plane. The smile arc was consonant. The excessive gingival display was also evident on the oblique smile. **F,** Profile evaluation with emphasis on a long lower facial third, long chin height, a flat labiomental sulcus, and lip strain on closure. **G–K,** The functional problem of the anterior open bite in this nongrowing patient necessitated superior repositioning of the maxilla to correct the functional complaint. The exact surgical movements were obtained from the clinical examination and measurements (see text).

hypertelorism in craniofacial syndromes can create disproportionate transverse facial aesthetics.

A vertical line from the outer canthus of the eyes frames the medial three fifths of the face, which should be coincident with the gonial angles of the mandible. Although disproportion may be very subtle, it is worth noting because our treatments can positively change the shape or relative proportion of the gonial angles.



FIGURE 54-10. The face is divided sagittally into five equal parts from helix to helix of the outer ears. The middle fifth of the face is delineated by the inner canthus of the eyes, the inner corner of the eye containing the lacrimal duct. A line from the inner canthus should be coincident with the ala of the base of the nose. A vertical line from the outer canthus of the eyes frames the medial two fifths of the face, which should be coincident with the gonial angles of the mandible. The outer two fifths of the face is measured from the lateral canthus to the lateral helix, which represents the width of the ears. Another significant frontal relationship is the midpupillary distance, which should be transversely aligned with the commissures of the mouth.

The outer two fifths of the face is measured from the lateral canthus to lateral helix of the ear, which represents the width of the ears. Unless this abnormality is part of the chief complaint, prominent ears are often a difficult feature to discuss with the patient because laypeople only recognize its effect on the face in severe cases. However, studies clearly indicate that large ears are judged by laypeople to be one of the most unaesthetic features, particularly in males. Otoplastic surgical procedures are relatively atraumatic and can dramatically improve facial appearance. In orthognathic cases in which this disproportion is noted by the clinician, we believel that failure to mention this feature violates informed consent. Therefore, otoplasty should be presented as a treatment option, whether received positively or not. These procedures can be performed on adolescents and adults as is illustrated in Figure 54-11.

Another significant frontal relationship is the midpupillary distance, which should be transversely aligned with the commissures of the mouth.[15] Although this is considered the ideal transverse facial proportionality, little can be done therapeutically to correct this disproportion, except in craniofacial synostosis such as Apert's syndrome.

Nasal anatomy in the transverse plane should also be assessed through proportionality. The width of the alar base should be approximately the same as the intercanthal distance, which should be the same as the width of an eye. If the intercanthal distance is smaller than an eye width, it is better to keep the nose slightly wider than the intercanthal distance. The width of the alar base is heavily influenced by inherited ethnic characteristics.

Asymmetry of the face is a somewhat natural occurrence. Systematic examination of the patient's facial symmetry should be directly measured in the frontal plane. The following measures compose this portion of the clinical examination.

Nasal Tip to Midsagittal Plane

Evaluation of the position of the nasal tip is performed by asking the patient to elevate the head slightly and then visualizing the nasal tip in relation to the midsagittal plane (Figure 54-12). Any deviation of the nasal tip should be noted in relation to the maxillary midline. The clinician should not make the mistake of treating the maxillary midline to a distorted nose. An attempt to obtain the etiology of nasal tip asymmetry is recommended. The patient should be



FIGURE 54-11. **A,** An otoplastic surgical procedure was recommended for this patient's prominent ears. **B,** The facial transverse fifths were improved, resulting in a dramatic facial improvement.

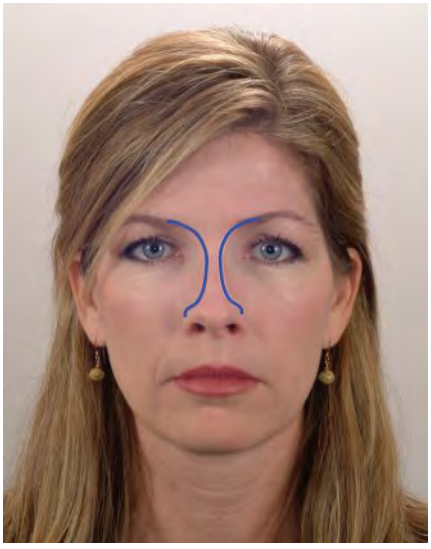


FIGURE 54-12. The "gull in flight" contour of the base of the nose.

questioned as to any previous history of nasal trauma or nasal surgery for a deviated septum. Patients may then be advised appropriately as to whether this deviation is severe enough to consider correction.

Maxillary Dental Midline to Midsagittal Plane

The maxillary dental midline should be recorded relative to the midsagittal plane. A discrepancy could be due to either dental factors or skeletal maxillary rotation. Maxillary rotation is a rarely occurring clinical finding and is usually accompanied by posterior dental crossbite. The dental features of maxillary midline discrepancies are discussed in both the miniaesthetic and microaesthetic perspectives.

Mandibular Dental Midline to Midsymphysis

The mandibular dental midline to midsymphysis relationship is best visualized by standing behind the patient and then viewing the lower arch from above (Figure 54-13). The patient should open her or his mouth in order for the clinician to view the lower arch and its relationship to the body of the mandible and symphysis. Lower dental midline discrepancies are usually due to tooth-related issues such as dental crowding with shifted incisors, premature exfoliation of primary teeth and subsequent space closure in preadolescents, congenitally missing teeth, or an extracted unilateral tooth. If the lower dental midline is not coincident with the midsymphysis, it usually indicates a dental shift. However, chin asymmetry should also be considered.

Mandibular Asymmetry with or without Functional Shift

Mandibular asymmetry is suspected when the midsymphysis is not coincident with the midsagittal plane. An important diagnostic factor is whether a lateral functional shift is present secondary to a functional shift of the mandible due to crossbite. When the patient is manipulated into centric relation, a bilateral, end-to-end crossbite usually is present, and as the patient moves the teeth into full occlusion, the patient must choose a side to move his or her mandible into maximum intercuspation. This lateral shift is indicative not of true mandibular asymmetry but of transverse maxillary deficiency and a resultant functional shift of the mandible.

True mandibular asymmetry is suspected when, in closure into centric relation, no lateral functional shift occurs. The truly asymmetrical mandible may be due to an inherited asymmetrical facial growth pattern or a result of localized or systemic factors. A thorough history of traumatic injuries and a review of systems of the patient will help ascertain potential etiologies of true mandibular asymmetry.

Chin Asymmetry

Facial asymmetry in some cases may be limited to the chin only. If the systematic evaluation of facial symmetry has dental and skeletal midlines and vertical relationships of the maxilla is normal and lower facial asymmetry is noted, the asymmetry may be isolated to the chin. Measurement of the midsymphysis to the midsagittal plane is a logical indicator of chin asymmetry, but the parasymphyseal heights should also be measured when chin asymmetry is suspected (Figure 54-14). The frontal view is recommended, but a view from the superior facial aspect (much like the evaluation of



FIGURE 54-13. If the lower dental midline is not coincident with the midsymphysis, it usually indicates a dental shift. However, chin asymmetry should also be considered.

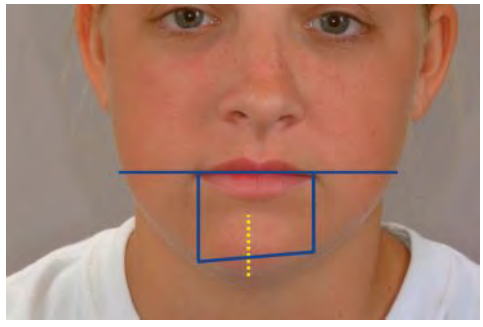


FIGURE 54-14. Measurement of the midsymphysis to the midsagittal plane is a logical indicator of chin asymmetry, but the parasymphyseal heights should also be measured when chin asymmetry is suspected.



FIGURE 54-15. Transverse tilting of the maxilla may be detectable cephalometrically but is most evident during the macroaesthetic examination.

the mandibular dental midline) with the mouth closed also affords the clinician excellent visualization of the chin to the body of the mandible and the midsymphysis.

Maxillomandibular Asymmetry

Mandibular asymmetry is often accompanied by maxillary compensation, which is reflected clinically by a transverse cant of the maxilla. This means that evaluation of mandibular deformity should now include the possibility of maxillomandibular deformity. Transverse tilting of the maxilla may be detectable cephalometrically but is most evident during the macroaesthetic examination (Figure 54-15). Clinically, one notes this, for example, as *right maxilla 4 mm more superior than left*. The transverse cant of the maxilla is often determined by the relative difference in gingival show present at the level of the canine moving posterior at smile. Differentiation between the macro- and miniaesthetic factors that are related to the transverse cant of the maxilla are discussed later.

Macroaesthetic Examination: Oblique View

The facial areas for macroaesthetic examination, as investigated from the oblique view, can be summarized as follows:

- Midfacial
  - Orbital position
  - Nasal form
  - Cheek/zygomatic form
- Lower facial
  - Lip form
    - Philtrum
    - Vermilion
  - Mandibular form
  - Chin projection

The oblique view (Figure 54-16A) in the macroaesthetic examination affords the surgeon and orthodontist another perspective for evaluating the facial thirds. With regard to the upper face, the clinician may view the relative projection of the orbital rim and malar eminence. Orbital and malar retrusion is often seen in craniofacial syndromes. Cheek projection is evaluated in the area of the zygomaticus and malar scaffold. Skin laxity and atrophy of the malar fat pad in this area may actually be a characteristic of aging and, therefore, seen in the older orthognathic population.[16] This area can be described as "deficient," "balanced," or "prominent." Nasal anatomy, which was described in the frontal examination, may also be characterized in this dimension.

Lip anatomy is also examined in the oblique and lateral views. The philtral area and vermilion of the maxillary lip should be clearly demarcated. The height of the philtrum should be noted as "short," "balanced," or "excessive." Vermilion display should be termed as "excessive," "balanced," or "thin."



FIGURE 54-16. **A,** Desirable definition of the chin-neck anatomy. **B,** A dolichofacial skeletal pattern with a steeper mandibular plane, not as aesthetic as in **A. C,** A brachyfacial pattern with an obtuse cervicomental angle secondary to submental fat deposition.

The relative projection of the maxilla and mandible can be assessed in the oblique view. Midface deficiency can result in increased nasolabial folding, relaxed upper lip support, and altered columella and nasal tip support.

One of the greatest values of the oblique view is visualization of the body and gonial angle of the mandible as well as the cervicomental area. The patient in Figure 54-16A illustrates a desirable definition of the chin-neck anatomy. The patient in Figure 54-16B has a dolichofacial skeletal pattern with a steeper mandibular plane, not as aesthetically pleasing as the previous illustration. The patient in Figure 54-16C demonstrates a brachyfacial pattern with an obtuse cervicomental angle secondary to submental fat deposition. Mandibular deficiency with associated dental compensation may produce lower lip eversion, excessive vermilion display, and a pronounced labiomental sulcus.

A characterization of mandibular form is also very important. The oblique view also demonstrates the effects of animation on the appearance of lip and chin projection. The patient in Figure 54-17 shows a moderate anterior divergence and facial concavity at rest, but during the smile, animation reveals an increased chin projection with excessive concavity.

Macroaesthetic Examination: Profile View

The facial areas for macroaesthetic examination, as investigated from the profile view, can be summarized as follows:

- Lower facial
  - Maxillomandibular projection or facial divergence
  - Lip form
    - Size
    - Projection
    - Labiomental sulcus
    - Chin projection

The last view in the macroaesthetic examination is the profile perspective. Natural head position is essential for accurate evaluation of profile characteristics. The patient should be instructed to look straight ahead and, if possible, into her or his own image in an appropriately placed mirror. The visual axis is what determines "natural head position." This axis very often, but not always, approximates the Frankfort horizontal plane. The classic vertical facial thirds should also be applied in profile view. An assessment of lower facial deficiency or excess should be noted.

The *nasolabial angle* describes the inclination of the columella in relation to the upper lip. The nasolabial angle should be in the range of 90 to 120 degrees (Figure 54-18A).[17] The nasolabial angle is determined by several factors: (1) the anteroposterior position of the maxilla to some degree; (2) the anteroposterior position of the maxillary incisors; (3) vertical position or rotation of the nasal tip, which can result in a more obtuse or acute nasolabial angle; and (4) soft tissue thickness of the maxillary lip that contributes the nasolabial angle, in which a thin upper lip favors a flatter angle and a thicker lip favors an acute angle.

The characterization of the lower face in profile (see Figure 54-18B) is measured by the relative degree of lip projection, the labiomental sulcus, the chin-neck length, and the chin-neck angle. Maxillary and mandibular sagittal position can be described by means of facial divergence. The lower third of the face is evaluated in reference to the anterior soft tissue point at the glabella. Based on the position of the maxilla and mandible relative to this point, a patient's profile will be described as "straight," "convex," or "concave" and either anteriorly or posteriorly divergent.

Lip projection is a function of maxillomandibular protrusion or retrusion, dental protrusion or retrusion, and/or lip thickness. The description of lip projection should include pertinent information from any of the previous sources. For example, a patient with lower lip protrusion may be maxillary (midface) deficient with dentoalveolar compensation including flared incisors and a thin maxillary vermilion display or simply may have a thick lower lip that appears protrusive.

The *labiomental sulcus* is defined as the fold of soft tissue between the lower lip and the chin and may vary greatly in



FIGURE 54-17. **A,** The amount of facial concavity and chin projection at rest is within acceptable limits. **B,** When this patient animates, an excessive amount of chin projection and facial concavity is revealed.

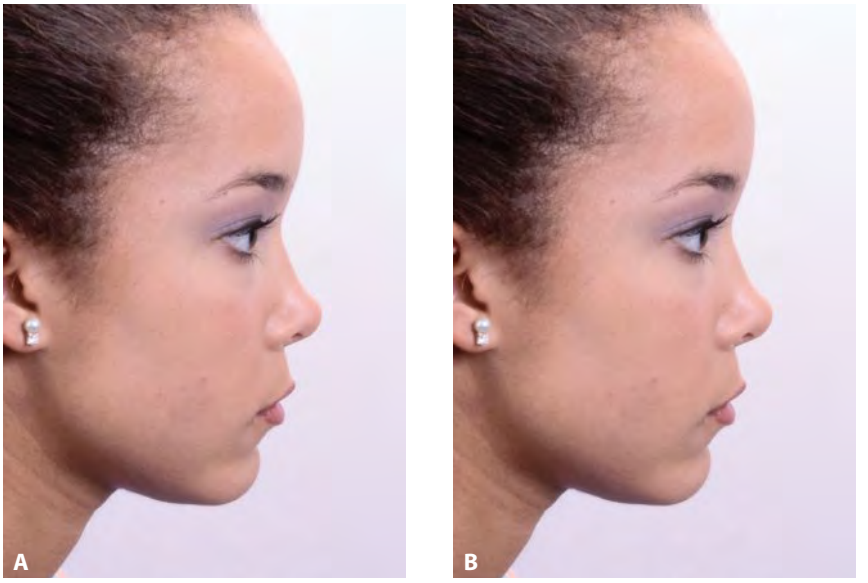


FIGURE 54-18. **A,** The facial profile view. Superiorly, the radix of the nose is characterized by an unbroken curve that begins in the superior orbital ridge and continues along the lateral nasal wall. The nasal dorsum is made up of both bony and cartilaginous tissues. The nasal tip is described as the most anterior point of the nose, and the supratip is just cephalic to the tip. The columella is the portion of the nose between the base of the nose (subspinale) and the nasal tip. **B,** The characterization of the lower face in profile is measured by the relative degree of lip projection, the labiomental sulcus, the chin-neck length, and the chin-neck angle.

form and depth. The clinical variables that can affect the labiomental sulcus include (1) lower incisor position, in which upright lower incisors tend to result in a shallow labiomental sulcus because of lack of lower lip projection, whereas excessive lower incisor proclination deepens the labiomental sulcus, and (2) vertical height of the lower facial third, which has a direct bearing on chin position and the labiomental sulcus. Diminished lower facial height will usually result in a deeper labiomental sulcus (just as in the overclosed full-denture patient), whereas a patient with a long lower facial third has a tendency toward a flat labiomental sulcus.

Chin projection is determined by the amount of anteroposterior bony projection of the anterior, inferior border of the mandible, and the amount of soft tissue that overlays that bony projection. The amount of profile chin projection is measured by the distance from pogonion (or Pg, the most anterior point on the bony chin) to soft tissue pogonion' (or Pg', the most anterior point on the soft tissue profile of the chin) and is not particularly alterable by surgical means. In the adolescent, the amount of chin is directly correlated to the amount of mandibular growth that occurs because the chin point itself is borne on the mandible as it grows anteriorly.

The angle between the lower lip, chin, and R point (the deepest point along the chin-neck contour) should be approximately 90 degrees. An obtuse angle often indicates (1) chin deficiency, (2) lower lip procumbency, (3) excessive submental fat, (4), retropositioned mandible, and (5) low hyoid bone position.

Another important measure in this area is the chin-neck length and chin-neck angle. The angle, also termed the *cervicomental angle,* has been studied extensively in plastic surgery and orthognathic literature.[18] Studies report that a wide range of normal neck morphology exists, and that the cervicomental angle may vary between 105 and 120 degrees, with gender being a major consideration. Age of the patient must be considered with regard to this area. Soft tissue "sag" due to the loss of skin elasticity during aging is a major cause of change in the cervicomental region. Weight gain is another important factor in the morphology of this area.

Miniaesthetic Examination: Frontal View

The facial areas for miniaesthetic examination, as investigated from the frontal view, can be summarized as follows:

- Vertical characteristics of the smile
  - Lip-tooth-gingival relationships
  - Gingival display on smile
  - Excessive gingival display on smile
  - VME
  - Short philtrum height
  - Excessive curtain
  - Short clinical crown height
  - Upright maxillary incisors
  - Inadequate gingival display on smile
  - Vertical maxillary deficiency
  - Diminished curtain
  - Short clinical crown height
  - Flared maxillary incisors
- Transverse characteristics of the smile
  - Arch form
  - Buccal corridor
  - Cant of the transverse occlusal plane

Vertical Characteristics

Lip-Tooth-Gingival Relationships

A key feature of vertical facial aesthetic characteristics is the relationship between the incisal edges of the maxillary incisors relative to the lower lip as well as the relationship between the gingival margins of the maxillary incisors relative to the upper lip. The gingival margins of the cuspids should be coincident with the upper lip, and the lateral incisors positioned slightly inferior to the adjacent teeth. It is generally accepted that the gingival margins should be coincident with the upper lip in the social smile. However, this is very much a function of the age of the patient, because children show more teeth at rest and gingival display on smile than do adults.[19]

Excessive Gingival Display on Smile

The vertical characteristics of facial miniaesthetics affect the relative amount of gingival display at rest and during animation. Gingival display is the amount of "gumminess" of the smile. Measuring the amount of gingival display on smile easily quantitates a "gummy" smile. The decision as to whether the amount of gingival display is an aesthetic problem in which treatment is desirable is a personal choice. Orthodontists and oral and maxillofacial surgeons tend to see the "gummy" smile as an unaesthetic characteristic, whereas laypersons attach importance only in the more extreme cases. The use of computerized graphic simulation of the frontal view of the smile is useful in counseling a patient and showing potential treatment changes. The individual is then able to guide the clinician and express opinions about what should and should not be corrected. Computer imaging not only provides the patient with a visual template for treatment but also provides the clinician with a testing ground for treatment options. The patient in Figure 54-19A exhibits excessive gingival display on smile, secondary to VME. The diagnosis of VME is confirmed by the facial characteristics of a long lower facial third, lip incompetence, excessive incisor display at rest, and excessive gingival display on smile. Superior repositioning of the maxilla was performed with excellent facial proportions and smile aesthetics (see Figure 54-19B).

The patient in Figure 54-20A also exhibited excessive gingival display but has normal vertical facial proportions. Her incisor crown height, however, is only 8 mm. The etiology of her "gummy" smile is not an orthognathic problem or an orthodontic problem but a cosmetic or periodontal problem. This diagnosis was confirmed and further visualized through computerized image modification (see Figure 54-20B and C), simulating the crown-lengthening procedure. Orthodontic intrusion of maxillary incisors would have reduced gingival display but would also have adversely affected the smile arc with concomitant flattening. This case example emphasizes differential diagnosis of gingival display issues and also emphasizes the optimization of unaesthetic facial traits while preserving those positive facial aesthetic attributes.

Transverse Characteristics

The three transverse characteristics of facial aesthetics in the frontal dimension are (1) arch form, (2) buccal corridor, and (3) transverse cant of the maxillary occlusal plane.

Arch form plays a pivotal role in the transverse dimension. Recently, much attention has been focused on using broad square arch forms in orthodontic treatment and orthognathic surgical treatment. In cases in which the arch forms are narrow or collapsed, the smile may also appear narrow and, therefore, present inadequate transverse smile characteristics. An important consideration in widening a narrow arch form, particularly in the adult, is the axial inclination of the buccal segments. Cases in which the posterior teeth are already flared laterally are not good candidates for dental expansion. Upright premolars and molars allow for a more bodily transverse expansion of the buccal segments in both adolescent and adult patients but are particularly important in the adult in whom sutural expansion is less likely. Orthodontic expansion



FIGURE 54-19. **A,** This patient exhibits excessive gingival display on smile, secondary to vertical maxillary excess. **B,** The actual post-treatment outcome.



FIGURE 54-20. **A,** This patient exhibits excessive gingival display but has normal vertical facial proportions. Her incisor crown height, however, is only 8 mm. The etiology of her "gummy" smile is not an orthognathic problem or an orthodontic problem but a cosmetic or periodontal problem. **B** and **C,** This diagnosis was confirmed and further visualized through computerized image modification, simulating the crown-lengthening procedure.

and widening of a collapsed arch form can dramatically improve the appearance of facial aesthetics and smile by decreasing the size of the buccal corridors and improving the *transverse smile dimension* (Figure 54-21). The transverse smile dimension (and the buccal corridor) is related to the lateral projection of the premolars and the molars into the buccal corridors. The wider the arch form is in the premolar area, the greater would be the portion of the buccal corridor filled.

As alluded to in the previous cases, arch expansion can have undesirable effects. Expansion of the arch form may fill out the transverse dimension of the smile, but two undesirable side effects may result and careful observation should be made to avoid these side effects wherever possible. First, the buccal corridor can be obliterated and create a "denture"-like smile. Second, when the anterior sweep of the maxillary arch is broadened, the smile arc may be flattened. Although it may not be possible to avoid these undesirable aspects of expansion, the clinician must make a judgment in concert with the patient as to what "trade-offs" are acceptable in the pursuit of the ideal facial aesthetic outcome.

The last transverse characteristic of facial aesthetics is the transverse cant of the maxillary occlusal plane. Transverse cant of the maxilla can be due to differential eruption and placement of the anterior teeth and skeletal asymmetry of the skull base and/or mandible resulting in a compensatory cant to the maxilla. Intraoral images or even mounted dental casts do not adequately reflect the relationship of the maxilla to the smile. Only frontal smile visualization permits the orthodontist to visualize any tooth-related asymmetry transversely.

Smile asymmetry may also be due to soft tissue considerations such as an asymmetrical smile curtain. In the asymmetrical smile curtain, there is a differential elevation of the upper lip during smile, which gives the illusion of transverse cant to the maxilla. This smile characteristic emphasizes the importance of direct clinical examination in treatment planning the smile, because this soft tissue animation is not visible in a frontal radiograph or reflected in study models. It is not well documented in static photographic images and is documented best in digital video clips.

Miniaesthetic Examination: Oblique View

Miniaesthetic examination from the oblique view involves two main areas:

- Orientation of the palatal and occlusal planes
- Smile arc

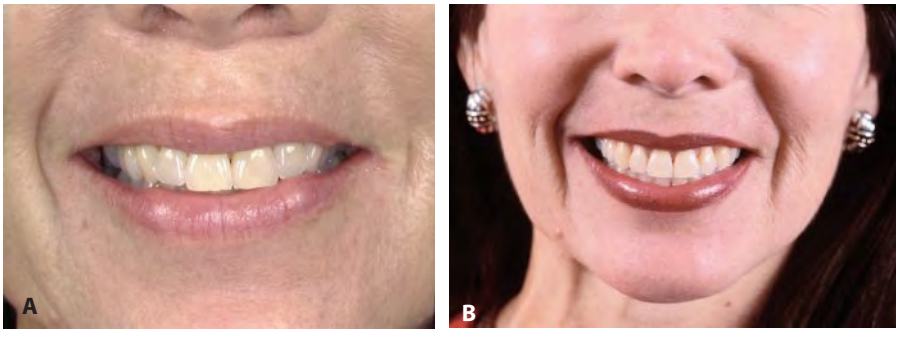


FIGURE 54-21. **A,** The transverse smile dimension in this patient was characterized by narrow arch form and excessive buccal corridor. In this adult, the axial inclinations of the molars and premolars were favorable for orthodontic expansion. **B,** The transverse smile dimension after orthodontic treatment.

The oblique view of the smile reveals characteristics of the smile that are not obtainable on the frontal view and certainly not obtainable through any cephalometric analysis. The palatal plane may be canted anteroposteriorly in a number of orientations. In the most desirable orientation, the occlusal plane is consonant with the curvature of the lower lip on smile (see discussion of *smile arc*, in the next paragraph). Deviations from this orientation include a downward cant of the posterior maxilla, upward cant of the anterior maxilla, or variations of both.[20] In the initial examination and diagnostic phase of treatment, it is important to visualize the occlusal plane in its relationship to the lower lip.

The *smile arc* should be defined as the relationship of the curvature of the incisal edges of the maxillary incisors, canines, premolars, and molars to the curvature of the lower lip in the posed social smile. The ideal smile arc has the maxillary incisal edge curvature parallel to the curvature of the lower lip upon smile, and the term *consonant* is used to describe this parallel relationship. A *nonconsonant*, or flat, smile arc is characterized by the maxillary incisal curvature being flatter than the curvature of the lower lip on smile. Early definitions of the smile arc were limited to the curvature of the canines and the incisors to the lower lip on smile because smile evaluation was made on direct frontal view. The visualization of the complete smile arc afforded by the oblique view expands the definition of the smile arc to include the molars and the premolars (Figure 54-22).

Miniaesthetic Examination: Profile View

The facial areas for miniaesthetic examination, as investigated from the profile view, can be summarized as follows:



FIGURE 54-22. The smile arc is best visualized in the oblique view and should be defined as the relationship of the curvature of the incisal edges of the maxillary incisors, canines, premolars, and molars to the curvature of the lower lip in the posed social smile. The 45-degree view permits visualization of vermilion display, lip fullness, and turgor not readily seen in another view.



FIGURE 54-23. The two miniaesthetic characteristics visualized in the sagittal dimension are overjet and incisor angulation.

- Overjet
- Incisor angulation
  - Upright maxillary incisors
  - Flared maxillary incisors
  - Retroclined mandibular incisors

The two miniaesthetic characteristics visualized in the sagittal dimension are overjet and incisor angulation (Figure 54-23). Excessively positive overjet is one of the most recognizable dental traits to the layperson. Adolescents tend to label unflattering names such as “Andy Gump” and “Bucky Beaver” onto children unfortunate enough to have inherited this dentoskeletal pattern. How overjet is orthodontically corrected involves macroelements such as jaw patterns and soft tissue elements such as nasal projection. Excessive positive overjet is not as readily perceived in the frontal dimension as it is in the sagittal dimension. Many class II patterns have very aesthetic smiles frontally but not when the patient’s smile is observed from the side. In class III patterns, the same phenomenon may be true, in that the smile looks aesthetic on frontal smile, but on the oblique or sagittal view, the overall appearance reflects the underlying skeletal pattern and dental compensation. The patient and parents have to decide with the clinician whether this is an acceptable outcome.

The amount of anterior maxillary projection also has great influence on the transverse smile dimension in the frontal view. When the maxilla is retrusive, the wider portion of the dental arch is positioned more posteriorly relative to the anterior oral commissure. This creates the illusion of greater buccal corridor in the frontal dimension. Overall, the sagittal cant of the maxillary occlusal plane in natural head position can influence the smile arc in the frontal dimension, affecting vertical characteristics. A negative cant of this plane will diminish the apposition of the incisal edges of the maxillary anterior teeth to the superior vermilion border of the lower lip at smile.

Dental Examination

The dental component of the clinical examination is the evaluation of any standing periodontal or cariogenic disease

process and the assessment of the patient's occlusion. The areas for dental examination can be summarized as follows:

- Alignment
  - Crowding
  - Spacing
  - Missing or supernumerary teeth
- Anteroposterior
  - Angle classification
  - Overjet
  - Compensation
- Bite depth
  - Anterior
  - Posterior
  - Compensation
- Transverse
  - Compensation
- Functional occlusal issues
  - Missing teeth and sequelae
  - Occlusal interferences and parafunction

Intra-arch and interarch relationships are described in the categories of dental alignment, anteroposterior occlusion, and bite depth. Clinically, the patient's occlusion should be examined in both a static and a dynamic sense.

The maxillary and mandibular dental arches are described as either "well aligned," "crowded," or "spaced." The extent of crowding or spacing is usually noted in millimeters. Individual teeth are described by virtue of their spatial position and degree of rotation. Therefore, an incisor could be described as "severely rotated" and "in linguoversion." Any congenitally missing, lost, or supernumerary teeth are noted. A description of teeth that have been severely worn or damaged due to trauma should be included.

In terms of the static occlusion, Angle's classification of the patient should be recorded. The Angle class I relationship is such that the mesiobuccal cusp of the maxillary first molar should rest in the buccal groove of the mandibular first molar. The Angle class II relationship exhibits a more anterior position of the mesiobuccal cusp of the maxillary first molar, The Angle class III relationship exhibits a more posterior position of the mesiobuccal cusp of the maxillary first molar. The degree of incisor overjet that accompanies an anteroposterior discrepancy should also be noted.

Concepts of Incisor Compensation

Incisor compensation in the sagittal view is very important in planning the presurgical orthodontics, yet not fully recognized by both orthodontists and surgeons alike. In most cases of skeletal dysplasia, whether in the range of surgical or nonsurgical treatment, dental compensation is a common feature. The forms and expression of this compensation are as complex as the myriad of dentoskeletal problems that exist, but common patterns are frequently encountered. In the diagnosis and proper treatment of these cases, the primary responsibility of the orthodontist is to recognize these compensations and eliminate or *decompensate* them. The range of which compensations are problematic is not concrete, so the surgeon and the orthodontist must decide how much compensation is acceptable and what is to be done for decompensation. Although we tend to think of these compensations as an anteroposterior consideration (incisor angulation problems), dental compensation can occur in all planes of space.

Class II and Class III Problems

The classic pattern of compensation in class II skeletal patterns is the proclination of the mandibular incisors and retroclination of the maxillary incisors. Conversely, class III skeletal dysplasias often feature retroclination of the mandibular incisors and proclination of the maxillary incisors. The orthodontist must recognize these compensations and decide what degree of compensation is acceptable and what requires substantive treatment. For example, if lower incisor flare in the class II patient is only moderate, what is the value of removing two mandibular premolars to upright the incisors? These decompensation decisions affect the treatment outcome in three basic ways: (1) inadequate incisor positioning can compromise buccal interdigitation; (2) incisor positioning can substantially affect the aesthetic outcome; and (3) in certain types of functional problems such as obstructive sleep apnea syndrome, aesthetic considerations have a lower priority compared with correction of the functional problem.

The effect of incisor angulation on buccal occlusal relationships was advanced and best expressed by Andrews.[21] In presurgical preparation for mandibular advancement, maxillary incisors that are not properly flared or mandibular incisors that are left overly flared may result in (1) insufficient overjet to provide for adequate advancement of the mandible from the aesthetic standpoint and (2) the inability to achieve desired class I buccal segments because the advanced nature of the lower incisor edge does not permit interdigitation of the buccal segments (Figure 54-24). The appropriate amount of incisor angulation can be determined either through cephalometric investigation or by simply holding study models in a simulated class I molar relationship.

Vertical Characteristics and Compensations

Bite Depth

The vertical component of the dental examination describes *bite depth*. A patient's anterior bite depth is the amount of maxillary incisor overbite relative to the mandibular incisors. Therefore, a patient can be described as having "an anterior open bite," "a satisfactory bite (25–50% overbite), or "an anterior deep bite." The posterior bite depth is usually characterized as being "open," "satisfactory," or "collapsed." The latter is seen when the patient is missing unilateral or bilateral posterior dental units.

Curve of Spee

Dental compensation in the vertical plane has to do with aberrations in the curve of Spee. The curve of Spee is measured

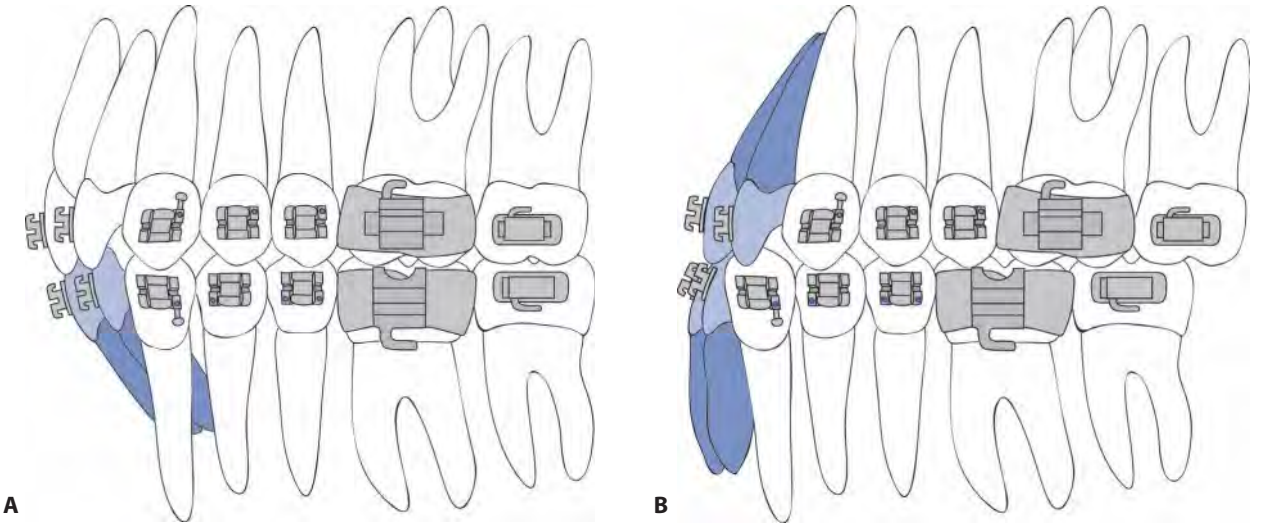


FIGURE 54-24. **A,** Inadequate decompensation in class II correction makes class I buccal segments not attainable because the flared lower incisors do not permit interdigitation of the posterior segments. **B,** Inadequate decompensation in class III correction makes class I buccal segments not attainable because the upright lower incisors or flared maxillary incisors do not permit interdigitation of the posterior segments.

by the arc extending from the cusp tips of the incisors posteriorly to the cusp tips of the molars in a sagittal view. Clinically, the study model can be placed on a flat surface and the cusp tips relative to that flat plane will give a rough estimate of the maxillary and mandibular curve of Spee. This is an important diagnostic feature of model analysis in recognizing potential pitfalls that may be encountered during orthodontic preparation for orthognathic surgery. For example, in a patient in whom the anterior segment is significantly superior (>2 mm) to the posterior segment, failure to recognize this occlusal plane differential may result in orthodontic flattening before surgery and postsurgical relapse, resulting in anterior open bite.[22]

Transverse Compensations

The class II patient often has narrowing of the maxilla in response to the narrower portion of the mandible being placed in the broader portion of the maxillary arch. In the class III patient, the maxillary posterior segments are often flared buccally in compensation for the wider portion of the mandible being placed into the narrower aspect of the maxilla. By holding the study models in a simulated class I relationship, these compensations can be easily recognized (Figure 54-25).

Functional Occlusal Issues

The last portion of the dental examination relates to dynamics of occlusal function. The clinician should ascertain whether the patient exhibits a discrepancy between maximum intercuspal position and retruded contact position in the anteroposterior dimension. In general, small differences exist in the vast majority of patients. Only large slides should be recorded. If the patient's dentition is mutilated, the clinician should note the resultant occlusal compensations. Any supererupted teeth will create lateral and anteroposterior interferences. A history of bruxism or other parafunctional habits will affect orthodontic appliances and will affect the type of retention used post-treatment.

Microaesthetic Examination

The microaesthetic portion of the clinical examination focuses on the morphology of tooth-to-tooth contacts and the surrounding intraoral tissues, summarized as follows:

- Dentogingival relationships
- Tooth form/tooth contact/gingival architecture



FIGURE 54-25. Transverse problems are first diagnosed by holding the study models together in a simulated class I relationship. The most commonly found transverse problem is that the maxilla is narrower than the mandible in cases similar to that of this patient who was being evaluated for class II correction by mandibular advancement.

As a structural unit, the dentogingival complex is defined by the relationship of the teeth to the alveolar bone and surrounding gingival and masticatory mucosa. The factors that influence the appearance of the dentogingival complex are the patient's periodontal status and past history of disease, the proximal and occlusal contacts of the teeth, the shape of the individual teeth, and the type of gingival architecture.

An assessment of the patient's current periodontal status is exceedingly important from an orthodontic and surgical point of view. The clinician should take an accurate dental history in order to ascertain whether the patient has had any periodontal disease and related treatment. Clinically, the teeth should be examined for plaque accumulation and any supragingival calculus. Patients who cannot maintain a satisfactory level of oral hygiene are at risk for gingival inflammation, attachment loss, and caries during presurgical orthodontic treatment. Periapical radiographs combined with a panoramic radiograph will reveal alveolar architecture and any evidence of horizontal or vertical bone loss. Suspected periodontal defects should be probed and the depths recorded. The extent of attachment loss and degree of tooth mobility will influence tooth movement.

Surgical treatment planning of the segmental Le Fort I osteotomy should consider gingival architecture in relation to maxillary segmentation. If the incisions are made mesial to the maxillary canines, the patient may lose the interdental papilla in between this tooth and the maxillary lateral incisor. By positioning the incisions distal to the maxillary canines, an obliterated papilla can be more easily camouflaged owing to the convexity of the canine.

COMPUTERIZED CEPHALOMETRIC PREDICTION

For computer image prediction, a digital model of the cephalometric tracing must be entered into computer memory. It is important that the radiograph be obtained in natural head position, with the teeth lightly together and in retruded contact position and the lips relaxed. The details of the digital model vary among the several currently available software programs but the similarities are more impressive than the differences. The more points in the digital model, the greater the anatomic fidelity of that model. Conversely, the more points that are digitized, the more time it takes to perform the digitization process.

A lateral image of the patient's profile, matching the cephalograms as closely as possible in head position and lip posture, must be captured and entered into the computer program (either directly via digital photography or by scanning a slide). Ideally, the radiograph and profile image would be taken simultaneously, although the hardware arrangement to do this does not yet exist.

The patient in Figure 54-26A presented for correction of a severe class II dentofacial deformity. After clinical examination and diagnostic records, digital image integration and algorithmic projections are used for consultation with the patient.

After the records are gathered, the next step in the treatment planning is to superimpose the profile image and radiograph, with the hard and soft tissues matched to each other as closely as possible. Most programs use the profile as the major method of image coordination. Once the images are coordinated, any cephalometric analysis can be displayed, although in contemporary surgical planning, the goal of treatment is not what the analysis indicates.

At that point, a "treatment screen" (see Figure 54-26B) provides the clinician with "handles" (the *blue squares*) by which selected sections of hard tissue can be moved (e.g., the mandible, the maxilla, or maxillary incisor segment); the procedures are similar to the use of templates and manual prediction. In this case illustration, surgical mandibular advancement is being contemplated. Dental compensation is present in the form of flared mandibular incisors, and decompensation is recommended to decompensate the dentition in order to increase the overjet, thus maximizing the magnitude of mandibular advancement. Simulation of lower first premolar extraction and lower incisor retraction is made on the treatment screen; the software applies its embedded algorithms for profile prediction and creates a new line drawing of the profile (see Figure 54-26C) reflecting the expected profile change after incisor decompensation. The algorithms may be ratios based on regressive equations and multiple correlations. They are not the same in all programs: the quality of the algorithms is the major determinant of how well or poorly the predicted profile matches the actual change produced by the treatment. The quantitative table on the right of Figure 54-26C provides to the clinician the measurements of the movements made on screen calibrated to actual movements required clinically to achieve the projected change.

After decompensation movements are simulated, the mandible is advanced on the treatment screen to ideal overjet, and the software then "warps" the original profile image to fit the prediction line drawing, producing an image that conveys much more visual information to the clinician and patient than the line drawing (see Figure 54-26D). As treatment is being planned, the amount of change is suggested until, within the limits of possible surgical change, it looks best. It is advantageous to include the patient in this process of adjusting the amount of change to provide an optimal outcome. In this case, a comparison image is generated so the patient may visualize the profile outcome expected with mandibular advancement (see Figure 54-26E). The profile was judged to be improved but was still clearly chin deficient. Simulation of chin advancement is then performed (see Figure 54-26F), not by using any cephalometric norm or predetermined value, but by simply using the facial outline as a guide. In other words, the chin is moved horizontally and vertically until it meets the approval of the patient. The projected final profile image is depicted in Figure 54-26G. The quantitative table reflects the exact movements in millimeters so that the surgeon and orthodontist have a precise plan for the amount of change needed to produce the desired result seen in Figure 54-26H. Presurgical planning using this



FIGURE 54-26. **A,** This patient presented for correction of a severe class II dentofacial deformity. **B,** A "treatment screen" provides the clinician with "handles" (the *blue squares*) by which selected sections of hard tissue can be moved (e.g., the mandible, the maxilla, or the maxillary incisor segment); the procedures are similar to the use of templates and manual prediction. **C,** Simulation of lower first premolar extraction and lower incisor retraction is made on the treatment screen. The software applies its embedded algorithms for profile prediction and creates a new line drawing of the profile reflecting the expected profile change after incisor decompensation. **D,** After decompensation movements are simulated, the mandible is advanced on the treatment screen to ideal overjet, and the software then "warps" the original profile image to fit the prediction line drawing, producing an image that conveys much more visual information to the clinician and patient than the line drawing. **E,** In this case, a comparison image is generated so that the patient may visualize the profile outcome expected with mandibular advancement. **F,** Simulation of chin advancement is then performed, not by using any cephalometric norms or predetermined value, but by simply using the facial outline as a guide. **G,** The projected final profile image. **H,** The actual treatment result.

methodology should eliminate "on-the-table" estimates of whether or not the patient needs "a bit more chin." It is ludicrous to make aesthetic treatment decisions with the patient under general anesthesia, horizontal, fully draped, paralyzed, and with a nasal tube in place.

An important consideration is the accuracy of the computer prediction process. Although it is far from perfect (some computer programs are more accurate than others), it is good enough to be clinically useful.[23–27] Chin predictions are usually quite accurate and those of the upper lip are reasonably good, whereas predictions of the lower lip can be problematic. As the data on which algorithms are based become more extensive, as different algorithms are applied when vertical and anterior changes occur, and as multiple regression equations replace simple ratios, accuracy can be expected to improve.[28]

It could be said that in this era of informed consent and bioethical decision making, the patient should be actively involved in the process of computer prediction and treatment planning. Cultural and familial traits may be important to the patient. Surgeons and orthodontists tend to want to "optimize" all patients to the prevailing aesthetic norm, which diminishes any ethnic variation in dentofacial appearance.

SYNTHESIS OF AN OPTIMIZED PROBLEM LIST

The data derived from the systematic clinical examination and analysis of patient records are synthesized into a diagnostic

optimized problem list. Essentially, there are two branches in the problem-solving tree: aesthetics and function. Thus, the diagnostic problem list should be subdivided into the categories of macroaesthetic problems, miniaesthetic problems, microaesthetic problems, and functional problems. All recognizable problems that are relevant to the patient's chief complaint should be rank-ordered. Lastly, each problem should be evaluated in terms of its therapeutic modifiability

Conceptually and operatively, the orthodontist and surgeon have to visualize the desired solution to the specific problem and then assess whether the given solution will negatively affect some other dentofacial feature at the same time. The concept of facial optimization involves the preservation of as many positive elements as possible, while harmonizing those elements that fall short of the aesthetic and functional needs of the patient. The problems that might exceed the limitations of treatment or perhaps have a poor therapeutic prognosis should be described. Informed consent and bioethical treatment of the surgical patient requires that the clinician explain the risk-benefit considerations of the proposed treatment strategy. The goal of the systematized clinical examination and optimized problem-oriented diagnosis is to record and analyze the data in such a way that the required treatment becomes implicit in the description of the problem.

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CHAPTER

Orthodontics for Orthognathic Surgery

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An understanding of the role of the orthodontist, as well as specifics of orthodontic therapy as it relates to orthognathic surgery, is essential for the oral and maxillofacial (OMF) surgeon. Moderate to severe occlusal discrepancies and dentofacial deformities in adolescents and adults usually require combined orthodontic treatment and orthognathic surgery to obtain optimal, stable, functional, and aesthetic results. The basic goals of orthodontics and orthognathic surgery are to (1) satisfy the patients' concerns, (2) establish optimal functional outcomes, and (3) provide good aesthetic results. To accomplish this, the orthodontist and the OMF surgeon must be able to correctly diagnose existing dental and skeletal deformities, establish an appropriate treatment plan, and properly execute the recommended treatment. The orthodontist is limited, to a great extent, by growth, and although the orthodontist can move teeth and, to some degree, the alveolar bone, she or he does not have any appreciable effect on the basal bone of the jaws. The orthodontist's role is to align the teeth relative to the maxillary and mandibular jaws, whereas the OMF surgeon is responsible for surgically repositioning the jaw(s) and associated structures.

It is very important to listen to, and understand, the patient's concerns. Empathetic listening from the first appointment and throughout the treatment will build trust, improve communication, and help provide a quality end result for all parties involved. Comprehensive analysis of the patient and the complete orthodontic records (cephalograms, panoramic films, computed tomography, photographs, dental models, occlusal registration, and face-bow transfer) is important for diagnosis and development of the presurgical orthodontic goals. Although detailed analysis of the patient's facial and jaw structures from a clinical and radiographic perspective is vitally important, the focus of this chapter is the teeth and orthodontic considerations in preparation for orthognathic surgery and postsurgical orthodontic management. Other important factors in diagnosis, treatment planning, and outcomes, such as patient concerns, psychosocial factors, masticatory dysfunction, airway problems, speech difficulties, temporomandibular joint (TMJ) pathologies, and comprehensive orthognathic surgery workup are not discussed in this chapter. Note that the normal values provided in this chapter are not absolutes for every patient because of individual size, morphologic variances, and racial and ethnic differences. They are provided as a guide to help the clinician evaluate each individual patient. Establishing an all-inclusive diagnosis is paramount to developing a comprehensive prioritized treatment plan. The orthodontist must determine the orthodontic goals based on the pretreatment findings and on the projected treatment outcome. This chapter first presents orthodontic diagnostic information, then orthodontic treatment considerations, followed by postsurgical orthodontic management.

CLINICAL EXAMINATION AND DENTAL MODEL ANALYSIS

From an orthodontic standpoint, in evaluating the occlusion and other dental factors, the clinical and dental model analyses, when correlated with the cephalometric analysis, provide the most information for proper diagnosis and treatment planning. There are 13 basic clinical and dental model evaluations that are helpful for making these determinations:

1. **Arch length:** This measurement correlates the mesiodistal widths of the teeth relative to the amount of alveolar bone available and aids in identifying the presence of crowding



FIGURE 55-1. Arch length analysis measures mesiodistal widths of the teeth relative to the amount of alveolar bone available to identify the presence of crowding or spacing. The curved wire shows ideal cuspid and incisor tip position relative to the basal bone.

or spacing. This helps determine whether teeth need to be extracted or whether spaces need to be either created or closed (Figure 55-1). Clinical and dental model assessment, when correlated to the cephalometric analysis, will aid in determining arch length requirements. Generally, class II patients will tend to have more crowding in the mandibular arch and less in the maxillary arch, whereas class III patients may have spacing in the mandibular arch but a tendency for crowding in the maxillary arch.

2. **Tooth size analysis:** This assessment relates the mesiodistal width of the maxillary teeth compared with the mandibular teeth. A tooth size discrepancy (TSD) causes an incompatibility of dental alignment and may occur in the anterior teeth, premolars, and molar regions. Approximately 40% of patients with dentofacial deformities will have an anterior TSD affecting the anterior six teeth of the maxillary and mandibular arches (the mandibular arch is commonly too large compared with the maxillary arch), usually owing to small maxillary lateral incisors. In such cases, proper tooth alignment, with all spaces closed, often precludes the establishment of a good class I cuspid-molar relationship with treatment, and instead, a class II end-on cuspid-molar occlusal relationship may result. Occasionally, the maxillary anterior six teeth may be too large for the mandibular anterior teeth, creating an excessive anterior overjet when in a class I cuspid relationship. Determination of a TSD pretreatment will provide the best opportunity to correct the TSD during the presurgical orthodontic phase of treatment. Explaining to the patient before treatment that small maxillary lateral incisors may need restorative crown enlargement to maximize the quality aesthetic and functional outcome is important, so that the patient is aware from the onset of the time and financial commitment necessary for treatment. The normal mesiodistal widths of each of the permanent teeth are recorded in Tables 55-1 and 55-2. Variations from the norm may create difficulties in tooth alignment and occlusal interdigitation.

Bolton's analysis is a method used to correlate the widths of the maxillary and mandibular anterior six teeth. Needle-point calipers can be used to measure each individual tooth at its widest dimension and successive holes punched into a tablet for each of the anterior six teeth for each arch. Then, a measurement from the first to the last holes will give the summation of mesiodistal widths of the anterior six teeth for each arch (Figures 55-2 and 55-3). The summation of the mesiodistal widths of the maxillary anterior six teeth, divided into the combined width of the mandibular anterior six teeth, yields a value called the *intermaxillary (Bolton's) index*. The average index (percentage) is 77.5 ± 3.5.[1] A simple conversion of this factor would be to measure the width of the mandibular anterior six teeth and then multiply that sum by 1.3. This results in a calculated ideal maxillary arch width. The difference between the calculated and the actual maxillary arch

TABLE 55-1. **Maxillary Mesiodistal Teeth Diameters***

	Central Incisor	Lateral Incisor	Cuspid	First Bicuspid	Second Bicuspid	First Molar	Second Molar
Males	8.9 (0.59)	6.9 (0.64)	8.0 (0.42)	6.8 (0.47)	6.7 (0.37)	10.6 (0.56)	9.5 (0.71)
Females	8.7 (0.57)	6.8 (0.64)	7.5 (0.36)	6.6 (0.46)	6.5 (0.46)	10.2 (0.58)	8.8 (0.73)

*Measurements in millimeters (standard deviation [SD]).

Adapted from Moyers RE, van der Linden FPGM, Riolo ML, McNamara JA, editors. Standards of Human Occlusal Development. Ann Arbor: The University of Michigan. The Center for Human Growth and Development; 1976; pp. 53–94.

TABLE 55-2. **Mandibular Mesiodistal Teeth Diameters***

	Central Incisor	Lateral Incisor	Cuspid	First Bicuspid	Second Bicuspid	First Molar	Second Molar
Males	5.5 (0.32)	6.0 (0.37)	7.0 (0.40)	6.9 (0.63)	7.2 (0.47)	10.7 (0.60)	10.0 (0.67)
Females	5.5 (0.34)	5.9 (0.34)	6.6 (0.34)	6.8 (0.70)	7.1 (0.46)	10.3 (0.74)	9.5 (0.59)

*Measurements in millimeters (standard deviation [SD]).

Adapted from Moyers RE, van der Linden FPGM, Riolo ML, McNamara JA, editors. Standards of Human Occlusal Development. Ann Arbor, MI: The University of Michigan. The Center for Human Growth and Development; 1976; pp. 53–94.



FIGURE 55-2. Bolton's analysis. Needle-point calipers are used to measure each tooth at the contact-point level to aid in tooth-size analysis.

width values determines the TSD (see Figure 55-3). This evaluation is very helpful in determining presurgical orthodontic and surgical goals. TSDs can also occur in the premolar and molar areas (normally the same maxillary and mandibular teeth are similar in size) in which the mandibular teeth may be significantly larger than the maxillary teeth.

Bolton's analysis is not perfect and functions only as a guide in assessing the tooth size compatibility of the anterior teeth because it does not take into consideration the labiolingual thickness of the incisors, the axial inclination of the teeth, or the thickness and prominence of the marginal ridges. A thin labiolingual dimension of the maxillary incisors may compensate for small TSDs, but thicker than normal dimensions or prominent marginal ridges may preclude a class I cuspid relationship even though Bolton's index is normal. An accurate dental model orthodontic wax setup or computer rendering may achieve a more accurate assessment.

3. **Incisor inclination:** This refers to the inclination of the maxillary and mandibular incisors relative to their respective basal bones. The dental models are correlated to the cephalometric analysis and the ideal axial inclination of the incisors determined (Figure 55-4). The incisor inclination analysis contributes to the determination of whether extractions are necessary, spaces need to be created or eliminated, and what mechanics are required to align and level the arches or segments of the arches. The key is to remove any anterior dental compensations by repositioning the incisors in their proper position and inclination over basal bone.
4. **Arch width analysis:** This refers to the evaluation of the intra-arch transverse widths between the maxillary and

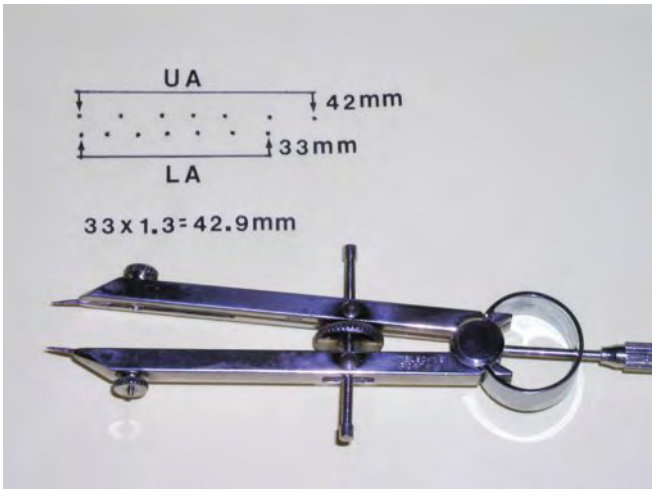


FIGURE 55-3. Bolton's analysis. Seven successive holes are punched into a tablet for the contact points of each of the anterior six teeth for each arch. The distance from the first hole to the last hole is the summation of mesiodistal widths of the anterior six teeth in each arch. Multiplying the summation of the mandibular anterior six teeth (LA) by 1.3 yields the calculated arch width for the maxillary anterior six teeth (UA). Subtracting the actual maxillary anterior arch width from the calculated width yields the tooth-size discrepancy (0.9 mm, in this example).

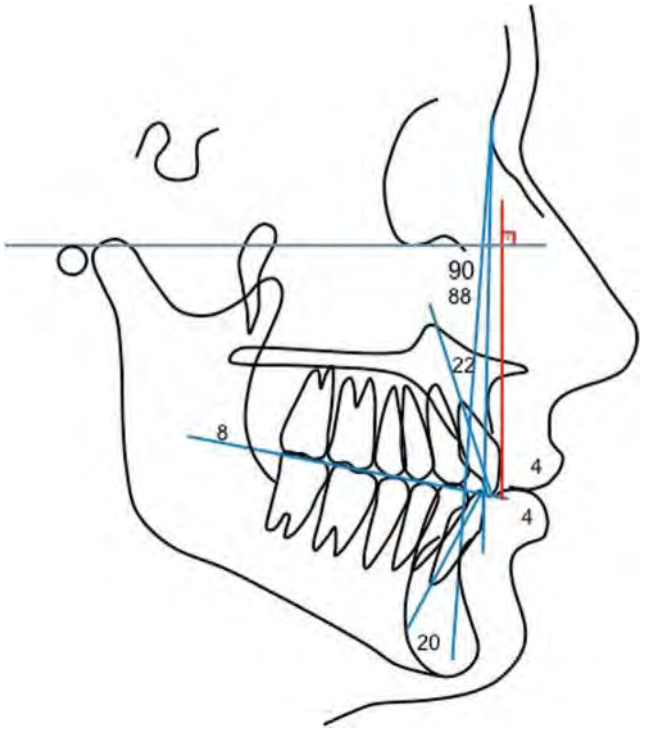


FIGURE 55-4. Cephalometric analysis. Normal maxillary depth angle is 90 degrees ± 3 degrees and mandibular depth is 88 degrees ± 3 degrees. Normal occlusal plane angulation is 8 degrees ± 4 degrees. Normal maxillary incisor inclination to the nasion point A (NA) line is 22 degrees ± 2 degrees with the labial surface of the incisor being 4 mm ± 2 mm anterior to the NA line. The angle between Frankfort horizontal plane and a line drawn tangent to the facial aspect of the maxillary central incisor crown should be at 90 degrees ± 2 degrees. Normal mandibular incisor inclination to the nasion point B (NB) line is 20 degrees ± 2 degrees with the labial surface of the incisor being 4 mm ± 2 mm anterior to the NB line.

TABLE 55-3. **Maxillary Arch Width* Measurements in millimeters (standard deviation [SD])**

	Cuspid	First Bicuspid	Second Bicuspid	First Molar	Second Molar
Males	32.3 (1.7)	36.7 (2.0)	41.5 (2.5)	47.1 (2.8)	52.3 (3.4)
Females	31.2 (2.45)	34.6 (3.2)	39.3 (2.2)	44.3 (2.3)	49.3 (2.8)

*All measurements at centroid.

Adapted from Moyers RE, van der Linden FPGM, Riolo ML, McNamara JA, editors. Standards of Human Occlusal Development. Ann Arbor, MI: The University of Michigan. The Center for Human Growth and Development; 1976; pp. 53–94.

TABLE 55-4. **Mandibular Arch Width* Measurements in millimeters (standard deviation [SD])**

	Cuspid	First Bicuspid	Second Bicuspid	First Molar	Second Molar
Males	24.8 (1.3)	32.8 (1.5)	37.6 (2.3)	43.0 (2.7)	49.0 (2.3)
Females	23.1(2.0)	31.8 (1.4)	36.8 (1.3)	41.7 (2.3)	47.2 (2.1)

*All measurements at centroid.

Adapted from Moyers RE, van der Linden FPGM, Riolo ML, McNamara JA, editors. Standards of Human Occlusal Development. Ann Arbor, MI: The University of Michigan. The Center for Human Growth and Development; 1976; pp. 53–94.

the mandibular arches. The average maxillary and mandibular arch widths for adults are listed in Tables 55-3 and 55-4.[2] These averages are only guidelines and do not account for patient size or racial or ethnic differences. However, from a practical standpoint, a good way to analyze the arch width is to relate the models to the occlusal position that is to be achieved with the surgical correction and then assess the transverse relationship. For example, if a patient has a class II occlusion, position the models in a class I cuspid-molar relation and evaluate the transverse width relationship. Similarly, a patient with a class III occlusion is evaluated by positioning the models into a class I cuspid-molar relationship. When a class II relationship is shifted to a class I relationship, the maxilla may be narrow and require expansion. In some cases, it may be indicated to evaluate the transverse relationship by placing the models into a class II molar position to determine whether a class I cuspid and class II molar relationship (this would require maxillary bicuspid extractions) would be best for that particular patient; this may be beneficial when there is significant crowding in the maxillary arch and no crowding in the mandibular arch. Transverse discrepancies will influence the presurgical orthodontics and dictate the surgical procedures required.

5. **Curve of Spee:** This evaluates the vertical position of the anterior teeth compared with the posterior teeth. This assessment can be determined by placing the occlusion of the maxillary dental model on a flat plane; the incisors should be about 1 mm above the flat plane (Figure 55-5A). Placing the occlusion of the mandibular dental model on a flat plane should see the mandibular incisors elevated 1 mm above the midbuccal teeth. A significant accentuated curve of Spee in the maxilla is usually associated with an anterior open bite and a reverse curve associated with an anterior deep bite. An accentuated curve of Spee in the mandible (see Figure 55-5B) is commonly associated with an anterior deep bite and a reverse curve associated with an open bite. Accentuated or reverse curves of Spee will influence whether the curve in each arch requires correction, and if so, whether the correction will be achieved by orthodontics, with or without extractions, opening spaces, or surgical intervention.

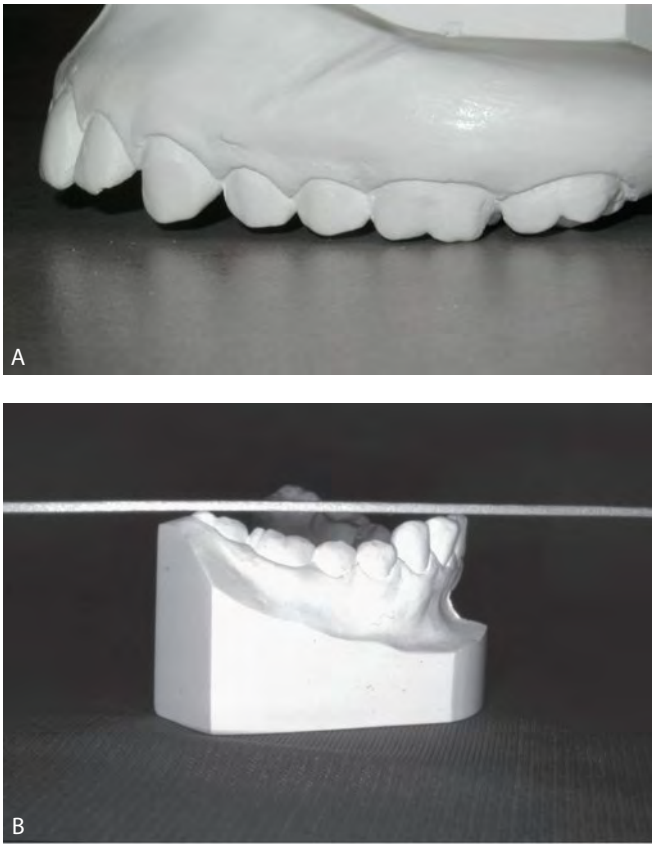


FIGURE 55-5. Increased curve of Spee in the maxillary arch **(A)** and in the mandibular arch **(B).**

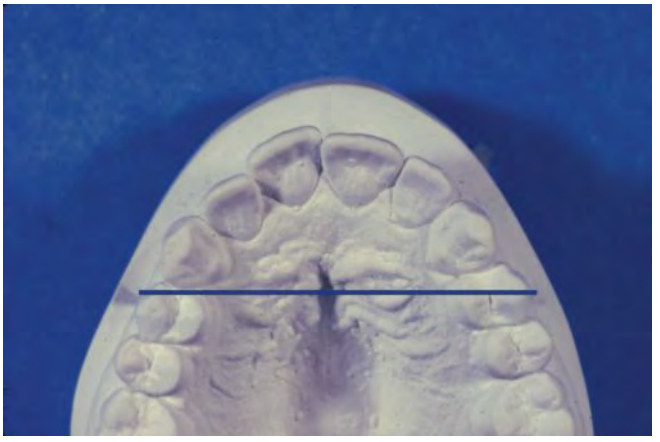


FIGURE 55-6. Tooth arch symmetry. The left cuspid is significantly more anteriorly positioned in the arch compared with the right cuspid.

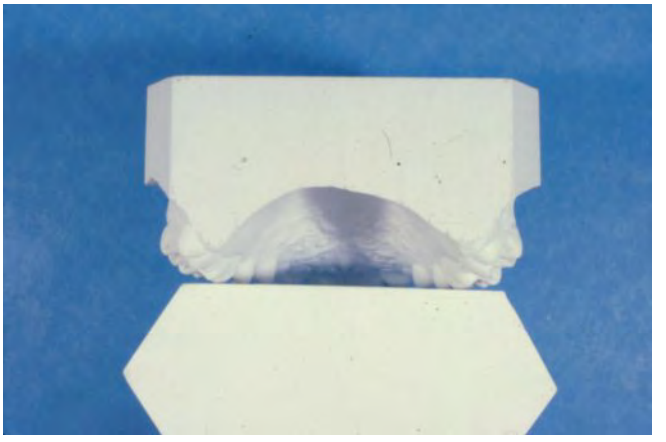


FIGURE 55-7. Curve of Wilson. This assesses the mediolateral position of the occlusal surfaces of the maxillary and mandibular posterior teeth.

6. **Cuspid-molar position:** This identifies the Angle classification and dental interrelationships. It is usually preferable to have a class I cuspid-molar relationship as an optimal outcome result; however, a class II molar relationship is acceptable. A class III molar relationship is less desirable because the mandibular first molar functions against the maxillary second bicuspid, although this may be indicated in select cases.
7. **Tooth arch symmetry:** This compares the left with the right side symmetry within each arch. There may be a significant asymmetry within the arch, such as a cuspid on one side being more anteriorly positioned in the arch than the cuspid on the opposite side (Figure 55-6). This problem often occurs with a unilateral missing tooth. Also, vertical asymmetries can occur with individual teeth, sections of the dentoalveolus, or the entire dental arches, creating a cant in the transverse occlusal plane. Correcting these types of asymmetrical conditions may require special orthodontic mechanics, unilateral or asymmetrical extractions or creation of arch space, and/or segmental surgical procedures.
8. **Curve of Wilson** (degree of buccal tooth tipping): This evaluates the mediolateral position of the occlusal surfaces of the maxillary (Figure 55-7) and mandibular posterior teeth. If the occlusal surfaces of the maxillary or mandibular posterior teeth are tipped too far buccally, it may be difficult to achieve a proper occlusal interdigitation relationship. In the presence of a transverse maxillary deficiency with preexisting increased curve of Wilson and posterior cross-bites, it is very difficult, if not impossible, to correct the problem orthodontically, orthopedically, or even with surgically assisted rapid palatal expansion (SARPE). The curve of Wilson will usually get much worse with these mechanics. In these types of cases, surgical expansion by multiple maxillary osteotomies may be indicated to decrease the curve of Wilson.

   When the mandibular posterior teeth are tipped buccally, it is often related to macroglossia or habitual tongue posturing. Orthodontic lingual tipping of the posterior teeth is very difficult when macroglossia is present and will likely be unstable. A reduction glossectomy may be indicated before orthodontics in order to permit a more stable final orthodontic result.
9. **Dental problems**, including missing teeth, severe caries, extensive dental restorations, or root resorption: These must be identified because they may influence treatment design. If a tooth is nonrestorable and requires extraction, it must be determined whether the extraction space requires orthodontic closure or the space maintained for later dental prosthetic reconstruction. In some cases, it may be helpful to maintain the condemned tooth to improve stability during surgical alignment of the jaws or segments thereof, with extraction after surgery. Crowns on previously restored teeth may need to be refabricated after orthodontics and orthognathic surgery, because the crown anatomy may need to be changed for proper occlusion with the new dental relationships. Evaluation for the presence of developmentally short roots or root resorption is necessary because this will have an effect on orthodontics and specific design of the surgical procedures. Determination of salvageable teeth and restorative requirements is an integral component in the planning and treatment of patients.
10. **Tooth ankylosis:** If undiagnosed, ankylosed teeth can have devastating effects on presurgical orthodontics. Tooth ankylosis, the fusion of alveolar bone and cementum, results from damage to the periodontal ligament (PDL). An ankylosed tooth may be identified by failure to move with orthodontic forces (Figure 55-8), failure of a tooth to erupt, submerged or incomplete tooth eruption (Figure 55-9), or lack of eruption of a tooth compared with adjacent teeth and alveolar bone growth. The most sensitive diagnostic test is percussion, performed by taping the crown with the end of a mouth mirror, in which the ankylosed tooth has a high, clear, solid metallic sound. A normal tooth has a dull sound, being protected

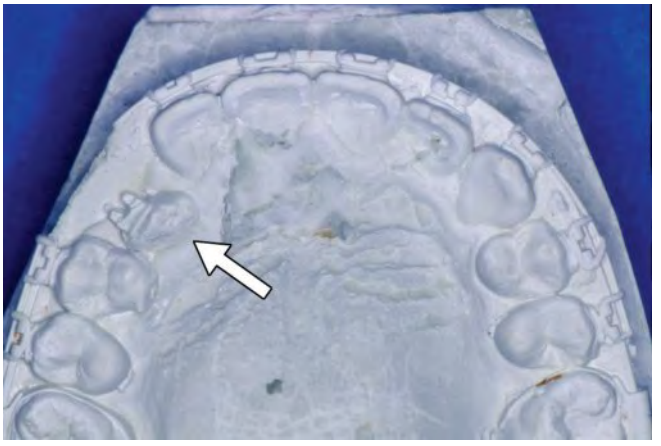


FIGURE 55-8. Dental cast shows a palatally displaced cuspid, unresponsive to orthodontic mechanics, indicating probable ankylosis.



FIGURE 55-9. Panorex shows incomplete eruption of primary tooth without a permanent successor, indicating ankylosis.

by the PDL. However, an erupted tooth with an impacted tooth directly against it may also have a solid sound to percussion. Normal multirooted teeth present a more solid sound than single-rooted teeth. Therefore, percussion testing should be compared with similar teeth (i.e., test bicuspids vs. bicuspids, molars vs. molars, using both sides of the arch). An ankylosed tooth lacks any mobility. Over 90% of ankylosed teeth are deciduous, most often, the second molar followed by the first molar.[3] Ankylosed primary teeth are not susceptible to resorption by the follicle of the underlying permanent tooth and may result in impaction of the permanent tooth.[3]

Ankylosed teeth can cause significant problems with jaw growth and development. Early ankylosis results in noneruption or partial eruption, resulting in incomplete development of the alveolar process.[4] Permanent teeth may be displaced from normal eruption pathways with resulting loss of alveolar bone height. The failure of an ankylosed tooth to erupt may allow adjacent teeth to drift and permit supereruption of the tooth in the opposing arch. Ankylosed teeth do not respond to orthodontic forces and can create significant orthodontic problems when malaligned and tied into the orthodontic arch wire (Figure 55-10).[5] The ankylosed tooth functions as an anchor and, in active uncontrolled orthodontics, will move adjacent teeth to align with its position, with subsequent development of an occlusal, alveolar, and possibly, a visible facial deformity.

11. **Periodontal status:** This is very important, because preexisting periodontal pathologies could be exacerbated during orthodontic and orthognathic surgical treatments.[6] Factors that can adversely affect the health and outcome of the periodontal tissues as well as the orthodontics and orthognathic surgery include smoking, excessive consumption of alcohol or caffeine, habitual patterns such as bruxism and clenching, preexisting con-

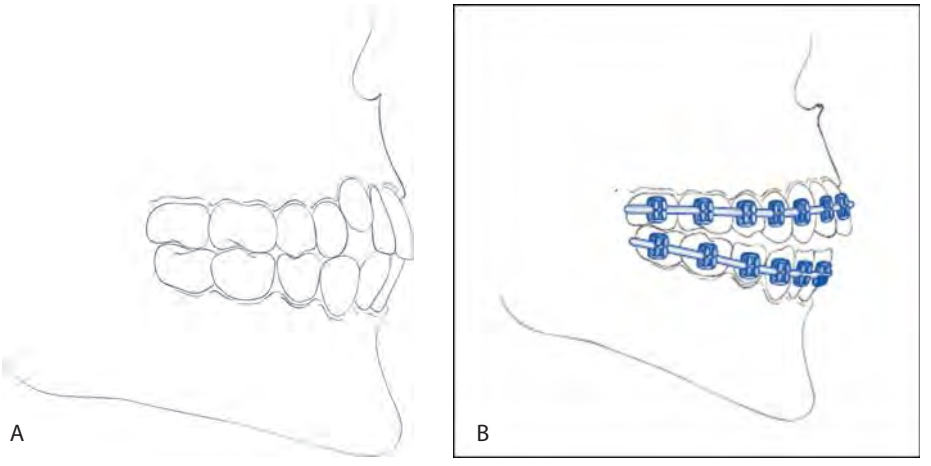


FIGURE 55-10. **A,** This illustration depicts a partially submerged ankylosed maxillary cuspid. **B,** If tied into an active straight arch wire, the adjacent teeth will be orthodontically moved toward the ankylosed tooth, resulting in the development of a significant malocclusion (in this case, an open bite due to incisor intrusion).



FIGURE 55-11. Periodontal concerns. A lack of attached gingiva in the lower incisor region before initiation of orthodontics if left untreated will cause severe gingival retraction and loss of supporting bone. Gingival grafting should be considered before initiation of orthodontics.



FIGURE 55-12. Macroglossia may cause anterior open bite, diastema between the teeth, accentuated maxillary curve of Spee, and reverse mandibular curve of Spee.

nective tissue/autoimmune diseases, diabetes, malnutrition, and other diseases that could affect the local tissue blood supply perfusion and wound healing. Any pretreatment of acute or chronic periodontal disease should be addressed before the orthodontics and surgery. The lack of attached gingiva around the teeth (most commonly seen in the mandibular anterior arch) can cause gingival retraction, loss of bone, and loosening of teeth if orthodontics is initiated and the mandibular incisors are tipped forward (Figure 55-11). Gingival grafting may be indicated before orthodontics to provide attached gingiva so as to prevent these problems. Good communication between the periodontist, the orthodontist, and the OMF surgeon is of utmost importance.

Orthodontics can help prepare interdental osteotomy sites by tipping the roots of the adjacent teeth away from each other to increase the interosseous space between the roots. A number of studies have demonstrated that interdental osteotomies have a minimal effect on the periodontium when they are properly performed.[7–11] Having healthy stable dental tissues to work with during the orthodontics and surgery will maximize the periodontal outcome as well as the overall outcome. The failure to recognize preexisting periodontal pathology, identify risk factors, poor performance of surgery, and/or lack of attention to detail could result in significant periodontal problems as well as other problems that could compromise the final result.

12. **Tongue assessment:** An enlarged tongue (macroglossia) can cause dentoskeletal deformities and instability of orthodontic and orthognathic surgical treatments and create masticatory, speech, and airway management problems. There are a number of congenital and acquired causes of true macroglossia, including muscular hypertrophy, glandular hyperplasia, hemangioma, lymphangioma, Down syndrome, and Beckwith-Wiedemann syndrome. Acquired factors include acromegaly, myxedema, amyloidosis, tertiary syphilis, cysts or tumors, and neurologic injury.[12] Specific clinical and cephalometric features may help the clinician identify the presence or absence of macroglossia, although not all of these features are always present. Specific clinical features of macroglossia include the following (Figure 55-12):

- Grossly enlarged, wide, broad, flat tongue
- Open bite (anterior or posterior)
- Mandibular prognathism
- Class III malocclusion with or without anterior or posterior cross-bite
- Chronic posturing of the tongue between the teeth at rest (rule out habitual posturing of normal-sized tongue)
- Increased curve of Wilson of the maxillary posterior teeth
- Reverse curve of Wilson of the mandibular posterior teeth
- Accentuated curve of Spee in the maxillary arch
- Reverse curve of Spee in the mandibular arch
- Increased transverse width of the maxillary and mandibular arches
- Diastema, with increased incisor inclination, in the mandible and/or maxilla
- Crenations (scalloping) of the tongue
- Glossitis (due to excessive mouth breathing)
- Speech articulation disorders
- Asymmetry in the maxilla or mandible associated with an asymmetrical tongue
- Difficulty with mastication and swallowing (severe cases)
- Instability in "stable" orthodontic mechanics or orthognathic surgeries
- Airway difficulties (obstructive sleep apnea) due to oropharyngeal obstruction
- Chronic drooling

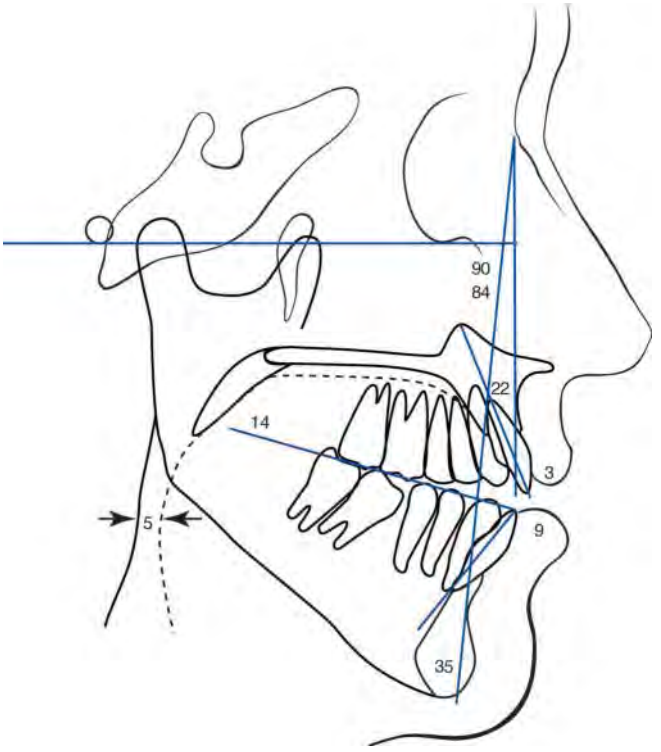


FIGURE 55-13. Macroglossia. Cephalometric analysis shows mandibular dentoalveolar protrusion and proclination of the mandibular anterior teeth. The tongue fills the oral cavity *(dotted line)* and the oropharyngeal airway is decreased (normal distance from posterior aspect of tongue to posterior pharyngeal wall is 11 mm; in this case, 5 mm).

Cephalometric radiographic features commonly seen with macroglossia (Figure 55-13) include the following:

- Tongue filling the oral cavity and extrudes through anterior open bite
- Mandibular or bimaxillary dentoalveolar protrusion
- Increased inclination of the maxillary and mandibular anterior teeth
- Disproportionately excessive mandibular growth
- Decreased oropharyngeal airway
- Increased gonial angle
- Increased mandibular plane angle
- Increased mandibular occlusal plane angle

Most open bite cases are not related to macroglossia. In fact, it has been established that closing open bites with orthognathic surgery will allow a normal tongue (which is a very adaptable organ) to readjust to the altered volume of the oral cavity, with little tendency toward relapse.[13,14] Most anterior open bite deformities that develop in the teenage years or later are commonly related to TMJ pathology.[15] However, if true macroglossia is present with the open bite, then instability of the orthodontics and orthognathic surgery will likely occur, with a tendency for the open bite to return. Pseudomacroglossia is a condition in which the tongue may be normal in size, but it appears large relative to its anatomic interrelationships. This can be created by (1) habitual posturing of the tongue; (2) hypertrophied tonsils and adenoid tissue displacing the tongue forward; (3) low palatal vault, decreasing the oral cavity volume; (4) transverse, vertical, or anteroposterior deficiency of the maxillary and/or mandibular arches decreasing oral cavity volume; and (5) tumors that displace the tongue. Pseudomacroglossia must be distinguished from true macroglossia because the methods of management are different.

13. **TMJ assessment.** Jaw deformities requiring orthognathic surgery often coexist with TMJ pathology. Unrecognized or untreated TMJ pathologies are one of the primary factors leading to postsurgical complications, resulting in poor quality and unpredictable outcomes. TMJ surgery may be required in these coexisting situations to obtain the highest quality results for patients. However, the TMJ surgery should be done before the orthognathic surgery in separate operations or perform the TMJ surgery followed by the orthognathic surgery at the same operation.[1,2] It is important that TMJ problems be identified and properly managed in the orthognathic surgery patient.

DIAGNOSTIC PROBLEM LIST AND PRIORITIZED TREATMENT PLAN

Before an orthodontic and surgical treatment plan can be properly developed, a diagnostic list of the existing problems is established based on patient concerns; a clinical, radiographic, dental model analyses; and other indicated evaluations. This will include all findings relative to musculoskeletal and dental imbalances, occlusal problems, aesthetic concerns, TMJ pathology, myofascial pain problems, missing teeth, crowns, bridges, endodontically treated teeth (occasionally ankylosed), periodontal problems, other functional disorders as well as any other medical factors that may affect treatment outcomes. The prioritized treatment plan is formulated from the diagnostic problem list and may include one or more ideal treatment plans and several other treatment options that may have one or more compromises, but may be appropriate based upon each individual patient situation. All potential treatment options should be presented to the patient; the final determination is made by the patient and the orthodontist and OMF surgeon.

PRESURGICAL ORTHODONTIC GOALS

The basic presurgical orthodontic goals are

- Align and position the teeth over the basal bone.
- Avoid excessive intrusion or extrusion of the teeth.
- Decompensate teeth.
- Avoid unstable expansion of the arches.
- Avoid class II and class III mechanics (unless required for dental decompensation).
- Perform stable and predictable orthodontics.
- Avoid orthodontic movements that can be performed more predictably with surgery.

Relative to the position of the maxillary and mandibular incisors, the ideal presurgical orthodontic goals are

1. Position the long axis of the maxillary central incisors approximately 22 degrees ± 2 degrees to the nasion-point A (NA) line, with the labial surface of the incisors 4 mm anterior to the NA line relative to a normally positioned maxilla and normal occlusal plane angle (see Figure 55-4).
2. The aesthetic crown angle is a line drawn tangent to the facial aspect of the maxillary central incisor crown extending through Frankfort horizontal plane. The angle created should be at 90 degrees ± 2 degrees. This places the crown into the best aesthetic position (see Figure 55-4).
3. Position the long axis of the mandibular central incisors 20 degrees ± 2 degrees to the nasion-point B (NB) line with the labial surface of the incisors 4 mm anterior to that line relative to a normally positioned mandible and normal occlusal plane angle (see Figure 55-4).
4. Satisfy arch length requirements (with regards to crowding or spacing).

Assessment of the ideal position of the maxillary and mandibular incisors to the NA and NB lines, respectively (see Figure 55-4), is the most convenient and practical method to establish the presurgical orthodontic goals for the incisors. However, these presurgical orthodontic goals may be different if the occlusal plane angle is to be altered surgically. Removal of dental compensations is helpful before surgery so that maximum skeletal correction can be achieved. An exact orthodontic treatment plan, including the specific mechanics and anchorage requirements necessary to position the teeth to satisfy the presurgical orthodontic goals, must be developed and executed.

INITIAL SURGICAL TREATMENT OBJECTIVE

The *surgical treatment objective (STO)*, also known as a *cephalometric prediction tracing*, is a two-dimensional visual projection of the changes in osseous, dental, and soft tissues as a result of orthodontics and orthognathic surgical correction of the dentofacial and occlusal deformity. The purpose of the STO is threefold: (1) establish presurgical orthodontic goals, (2) develop an accurate surgical objective that will achieve the best functional and aesthetic result, and (3) create a facial profile objective that can be used as a visual aid in consultation with the patient and family members. A prediction tracing of the anticipated presurgical orthodontic dental movements is created by placing an acetate sheet over the original cephalometric tracing and retracing the teeth into the position they will be placed with the presurgical orthodontics, based on the goals and available mechanics (Figure 55-14A). The initial STO is then constructed with the teeth in their presurgical orthodontic final position.

The STO has significant importance in two phases of treatment planning: (1) the initial STO is prepared before treatment to determine the orthodontic and surgical goals and (2) the final STO is prepared after the presurgical orthodontics are completed but before surgery to determine the exact vertical and anteroposterior skeletal and soft tissue

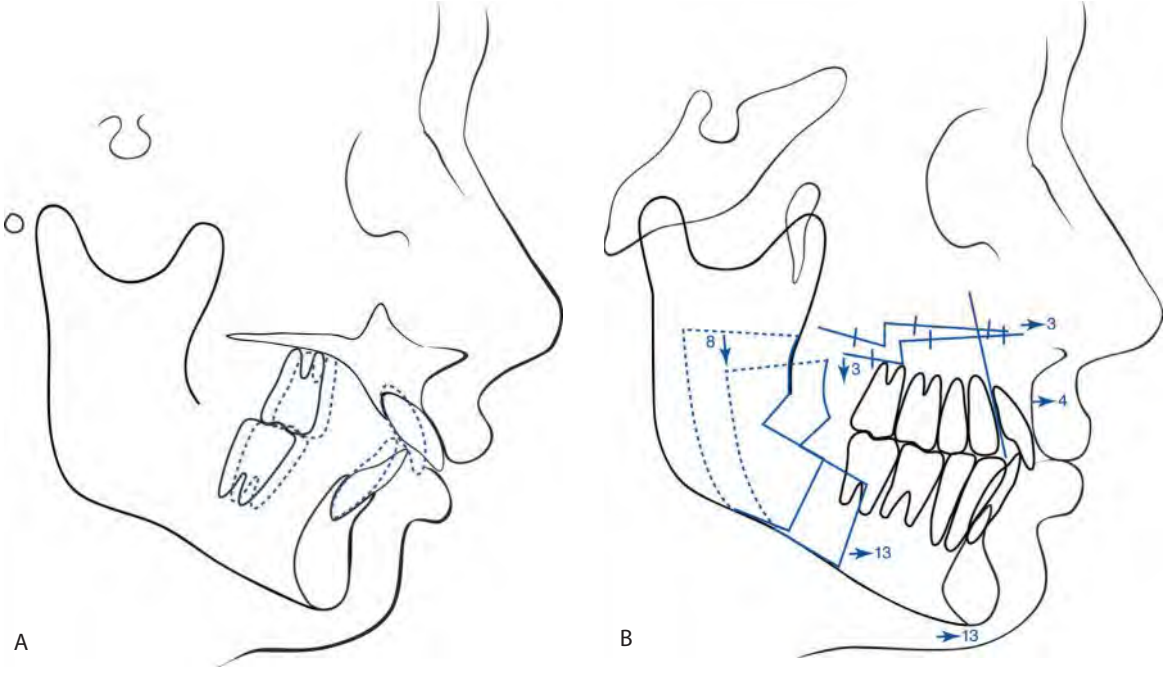


FIGURE 55-14. **A,** Presurgical orthodontics. The predicted orthodontic tooth movements are traced on acetate paper overlying the original lateral cephalometric tracing. The *solid lines* show the original position of the teeth. The *dashed lines* shoe the new position of the teeth after simulated extraction of four first bicuspids and orthodontic closure of the spaces. **B,** Surgical treatment objective (STO). This is an example of a completed final STO showing the predicted outcome of the presurgical orthodontics and the anticipated surgical treatment. The *arrows* and *numbers* indicate the direction and millimeters of movement.

movements to be achieved (see Figure 55-14B). These determinations can also be done through computer software programs, but these techniques may be less accurate than a manual STO because these programs have only the central incisor and first molar present to represent the entire dentition. However, the software programs are adequate for single-jaw and double-jaw planning when the maxilla is repositioned as a single unit. The STO is invaluable to the orthodontist and surgeon in establishing treatment objectives and projected results, acting as the blueprint for the entire treatment plan.

DEFINITIVE INTERDISCIPLINARY TREATMENT PLAN

The definitive treatment plan is formulated after consideration of all possible treatment options, and it is finalized based upon the patient's concerns, clinical evaluation, radiographic analysis, dental model evaluation, TMJ assessment, initial STO, and other relevant evaluations. Although the specific treatment plan may vary throughout the course of treatment, the general sequencing of the treatment that may be involved in a typical patient is described in the following sections.

Dental and Periodontal Treatment

Any indicated periodontal or general dental care related to maintaining teeth or improving dental health should be performed before orthodontics and surgical intervention. The objective is to maintain as many teeth as possible and maximize the health of the periodontium. Temporary crowns and bridges should be placed where necessary for the orthodontic and surgical phases of the treatment. Permanent crowns, inlays, and bridges should be constructed and inserted after the surgery and orthodontics have been completed. This gives the restorative dentist the opportunity to provide escapement grooves, cuspid protection, and incisal guidance for optimum function and aesthetics. Initial periodontal management may include scaling, root planing, and curettage, eliminating pockets, as well as grafting procedures to provide adequate alveolar bone and attached gingiva, especially in the anterior mandible. Occasionally, in patients with several missing teeth, osseointegrated implant placement before orthodontics and orthognathic surgery may provide anchorage for orthodontics and additional dental units to help in repositioning the jaw structures at surgery.

Presurgical Orthodontics

The orthodontist is responsible for positioning the teeth to the most desirable position over basal bone in preparation for surgery. The development of prescription brackets and straight wire orthodontic techniques has helped simplify orthodontics. Most prescription bracket systems are designed to tip the cuspid roots distally, creating space between the roots of the lateral incisors and the cuspids. In cases requiring segmentalization of the maxilla, this interdental space may be adequate through which to perform interdental osteotomies; but if inadequate, additional room can be created by tipping the lateral incisor roots mesially and the cuspids more distally. Bonded brackets are clean and eliminate interdental spacing problems created by circumferential bands. Bonded brackets with the currently available resins are quite adequate for orthognathic surgery procedures. However, inaccurate placement of the brackets on the teeth can result in undesired rotations, vertical discrepancies between teeth, malalignment of marginal ridges and labial surfaces of adjacent teeth, and unfavorable root positions. Careful placement of brackets is paramount in helping to achieve high-quality results.

Nickel-titanium or similarly shaped memory arch wires can be advantageous for many orthognathic cases to aid in presurgical orthodontic dental alignment goals. However, there are cases in which shape memory wires could be detrimental, such as in an anterior open bite with an accentuated maxillary curve of Spee. The use of nickel-titanium wires or any type of straight wire in these cases can create unstable orthodontic movements such as extrusion of teeth and buccal tipping of the molars as a result of reciprocal forces. Stainless steel wires (SSWs) with compensating bends (Figure 55-15A), or sectional wires (see Figure 55-15B), may provide a better controlled mechanical force in these types of cases.

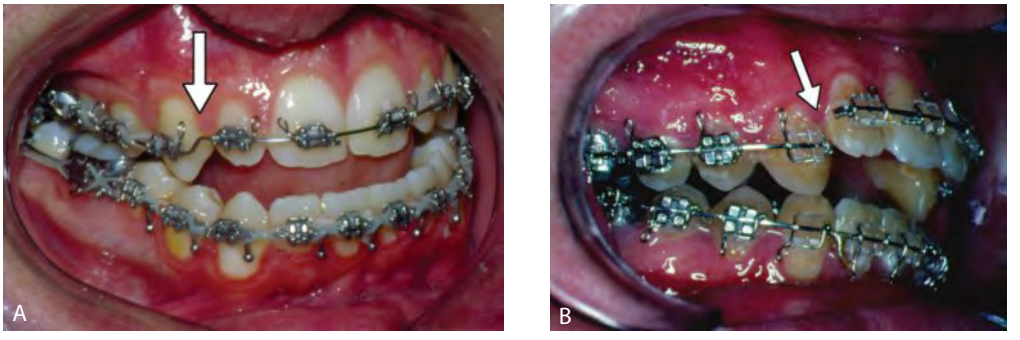


FIGURE 55-15. **A,** Compensating steps *(arrow)* in the orthodontic arch wire to align the anterior teeth at an elevated level compared with the posterior teeth to eliminate extrusion or intrusion of teeth that may result in unstable orthodontic movements. **B**, Sectioning the arch wire *(arrow)* is another approach to align teeth at independent levels to avoid extrusion or intrusion of teeth. However, sectional wires may decrease positional control of teeth adjacent to the ends of the cut wire.

The type of arch wire, and how long each is left in place, is critical and must be carefully monitored by the orthodontist.

Lingual orthodontic appliance are sometimes requested by patients for aesthetic reasons. Lingual appliances can work satisfactorily for single-piece maxillary osteotomies and mandibular osteotomies but will require the placement of brackets or buttons on the lateral aspect of the teeth at surgery to facilitate intraoperative intermaxillary fixation for application of rigid skeletal fixation to stabilize the osteotomies and to use postsurgical elastics, if necessary. When segmentation of the maxilla is required, lingual appliances present a greater challenge. Construction of palatal or occlusal covering surgical splints is much more difficult because of distortion of the lingual appliance on the surgical models that interfere with the fit of the splint at surgery. In addition, postsurgical changing of the arch wire is more difficult, uncomfortable, and painful for the patient. It may be several months after surgery before the lingual arch wire can be changed. It is much more difficult to finalize the occlusion and may require labial appliances to finalize the occlusal results.

To follow are basic presurgical orthodontic factors that commonly must be addressed in preparing patients for orthognathic surgery. It is important to avoid interarch class II mechanics (i.e., class II elastics, growth appliances, TMJ "disk recapturing" splints, Herbst appliances) unless they are specifically required during the presurgical orthodontics (e.g., to correct arch asymmetry to decompensate mandibular arch with lingually inclined mandibular incisors). Long-term class II mechanics positions the mandibular condyle downward and forward in the fossa and may allow hypertrophy (thickening) of the TMJ bilaminar tissues (Figure 55-16). This same condition can occur in patients with a "Sunday" bite. In these situations, after surgical mandibular advancement, the bilaminar tissue will slowly thin out over time, causing a slow relapse of the mandible toward a class II relationship. In addition, posturing the mandible forward for an extended time could result in foreshortening of the anterior articular disk attachments, increasing the risk of TMJ articular disk displacement postsurgery.

If a patient has been treated with long-term class II mechanics or has a "Sunday" bite, it may be an advantage to use light class III mechanics for a few months presurgery to eliminate the hypertrophied bilaminar tissue and to decompensate for any unstable orthodontics that may have been created. If the TMJ articular disk does become displaced, it would be better to have that occur before surgery because the articular disk can be surgically repositioned and stabilized with high predictability at the same time as the orthognathic surgery.[16–19] Attempts to recapture a TMJ displaced disk with splint therapy before surgery could be detrimental to the patient relative to outcome stability and pain. In most cases, nonsurgical disk "recapturing" procedures have proved clinically unsuccessful.

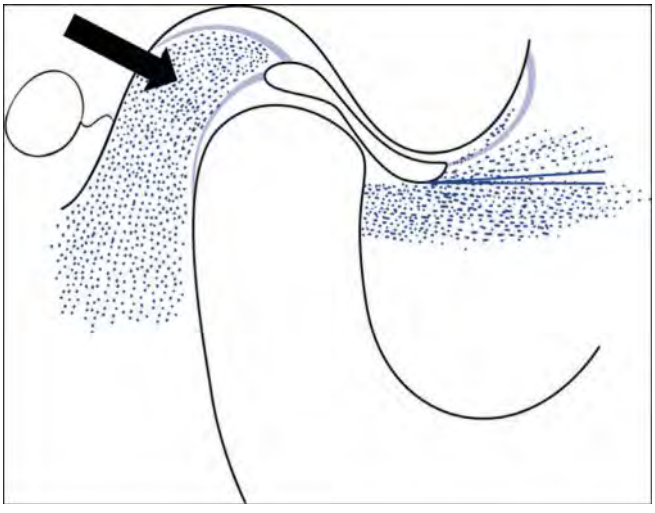


FIGURE 55-16. Temporomandibular joint (TMJ) effects of long-term class II mechanics, anterior repositioning splints, growth devices, or "Sunday" bite relationships can cause hypertrophy of the posterior bilaminar tissue *(arrow),* positioning the condyle downward and forward in the fossa. After mandibular advancement, this tissue will slowly thin out, and the condyles will move posteriorly in the fossa, causing a shift of the mandible and occlusion toward a class II relapse position.

TREATMENT OPTIONS FOR SPECIFIC ORTHODONTIC PROBLEMS

This section presents specific dental malrelationships and the orthodontic and surgical treatment options for consideration. Comprehensive assessment of the patient and developing treatment objectives will aid in selecting the appropriate treatment.

Adjustment for TSD

Usually, TSDs occur because of small maxillary lateral incisors, making the combined mesiodistal width of the maxillary anterior six teeth too small to fit properly around the mandibular anterior six teeth, so that when the teeth are properly aligned, an end-on class II cuspid relationship will result (Figure 55-17). If Bolton's analysis indicates a significant



FIGURE 55-17. Tooth size discrepancy (TSD). These casts show well-aligned and leveled teeth in each arch, but because the maxillary laterals are small creating a TSD, even with the best possible occludal fit, there is an end-on class II occlusion due to the TSD.

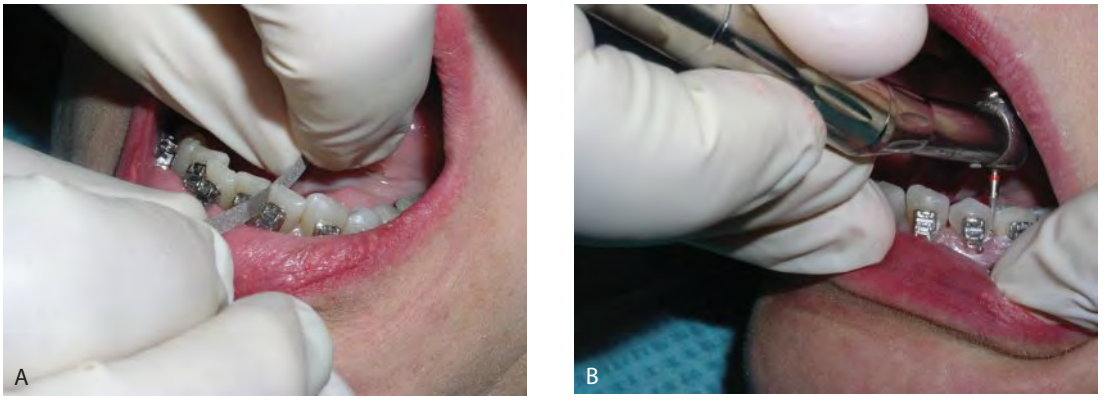


FIGURE 55-18. Interproximal reduction. Mesiodistal tooth width can be reduced with a diamond strip **(A)** or a thin cylindrical diamond bur **(B)**, and the space created can be closed orthodontically.

TSD, presurgical orthodontic adjustments can usually correct the discrepancy and aid in providing a solid class I cuspid relationship at surgery and in the final outcome. TSDs can also occur in the bicuspids and molars, with the maxillary teeth usually being too small compared with the mandibular teeth. The following are treatment options that can be used to correct TSDs.

Interproximal Tooth Size Reduction

Interproximal tooth size reduction (IPR) reduces the mesiodistal dimension of the involved teeth. Because most TSDs involve larger mandibular anterior teeth compared with the maxillary anterior teeth, slenderizing the mandibular anterior teeth can address the issue (Figure 55-18). Approximately 10% to 12% of the mesiodistal width can be safely removed from each tooth with 50% of the interproximal enamel remaining. Up to 3 mm of IPR can usually be safely achieved in the mandibular anterior six teeth. Slenderizing the mandibular anterior teeth is an advantageous procedure, in which the maximum width of the incisors is toward the incisor edge, particularly in the presence of crowding and/or increased inclination of the mandibular incisors. It may not be advantageous if the mandibular anterior teeth are in a normal or lingual inclination, because closing the resultant spaces will further decrease the incisor inclination and may adversely affect orthodontic stability and aesthetics. This technique is not indicated when the contact points are positioned toward the gingiva, because this could result in tissue strangulation with loss of papilla and interdental bone, creating significant periodontal issues. In the rare case in which the maxillary teeth are too large for the mandibular teeth, the maxillary teeth can be slenderized, but this is best used when the maxillary teeth are crowded and/or overangulated and the individual crowns are wider than normal (see Table 55-1).

When TSDs occurs in the bicuspid and/or molar area, IPR of the mandibular teeth will usually correct the problem, unless the IPR will cause excessive retraction of the mandibular anterior teeth. If this appears to be a potential outcome, careful closure of the spacing by loosing (slipping) posterior anchorage (using mechanics that will move the posterior teeth forward instead of the anterior teeth backward) may solve the problem. This approach may include class II mechanics to provide forward forces on the posterior teeth or moving one tooth at a time on each side. Dental implants or temporary anchoring devices (TADs) placed in the posterior or anterior dentoalveolus could provide stable anchorage to aid in applying mechanics necessary to push the posterior teeth forward.

Creation of Arch Space

Additional arch space can enlarge the circumference of each involved arch. Because TSDs are often related to small maxillary lateral incisors, creating space around the maxillary lateral incisors may be a logical approach. A simple technique involves placement of coil springs between the cuspids and the lateral incisors and, if needed, between the lateral incisors and the central incisors to open spaces (Figure 55-19). At the end of treatment, the lateral incisors can be built up by bonding, veneers, or crowns. This technique can also be used in the mandibular arch when the mandibular anterior teeth are too small compared with the maxillary anterior teeth. In either arch, this technique is most applicable when the teeth are decreased in inclination, because opening space will increase the axial inclination of



FIGURE 55-19. Orthodontic coil springs between teeth can create spaces around the lateral incisors to correct tooth size discrepancies. Post-treatment, the lateral incisors can be built up by bonding, veneers, or crowns.

the incisors. It may not be indicated when the maxillary or mandibular incisors are increased in inclination or crowded, because the resultant increased inclination may be unstable and cause untoward periodontal changes. However, if there is significant crowding or increased inclination of the incisors requiring extraction of bicuspids, during closure of the bicuspid spacing by retraction of the anterior teeth, space could be created around the lateral incisors.

When maxillary incisors are already increased in inclination, it is not feasible to open spaces during the presurgical orthodontics. In this situation, performing interdental osteotomies between the maxillary cuspids and the lateral incisors will permit opening space at surgery and the incisors can also be uprighted to decrease their axial inclination. A maximum 3 mm of spacing (1.5 mm on each side) can usually be acquired with this approach.

When the TSD occurs in the bicuspid or molar area, space can be opened around the maxillary bicuspids and/or molars to compensate for the tooth mass deficiency. Bonding, veneers, or crowns can then be placed to eliminate the created space.

Altering Axial Inclination of Incisors

This technique can affect the labial circumference of the anterior teeth. Increased axial inclination slightly increases the arch length, and decreased axial inclination slightly decreases it. Application of this technique would result in increasing the maxillary incisors' inclination above normal and decreasing the mandibular incisors' inclination below normal. This technique can accommodate small TSD differences but may place the teeth in a compromised position relative to stability and aesthetics.

Surgery can alter the axial inclination of the anterior teeth. In the maxillary arch, interdental osteotomies between the lateral incisors and the cuspids, and in the mandibular arch anterior subapical osteotomies, will provide a means to alter and control axial inclination of the incisors.

Altering Mesiodistal Angulation of Maxillary Incisors

Tipping the roots of the maxillary central incisors distally away from each other alters the position of the contact points, making the intercontact distance on each tooth slightly wider. This can be used only for small differences. However, it then usually requires recontouring of the distal aspect of the incisor edges and could cause a soft tissue void between the mesial contact points and the gingival tissues ("the black triangle"), creating an aesthetic concern for the patient. This technique is rarely recommended.

Extraction of Mandibular Incisors

This technique should be used only for large TSDs (5 mm) and only if there is significant crowding and/or significant increased inclination of the mandibular incisors. Removing a mandibular incisor usually creates a significant space (the width of the tooth), and closure of that space may significantly decrease the axial inclination of the mandibular incisors. In addition, it may cause a decreased transverse width between the cuspids, resulting in relative narrowing of both maxillary and mandibular arches, affecting the dental aesthetics. Extraction of a mandibular incisor may produce an increased overjet. If the patient has a good maxillary arch but mandibular crowding and increased incisor inclination, large TSD, and an end-on or slight class III anterior occlusion, the single mandibular incisor extraction may be the treatment of choice. An alternative in cases with large TSDs would be to slenderize the mandibular anterior teeth and create spacing around the maxillary lateral incisors.

A surgical alternative for a large TSD, when the teeth are not crowded and have good axial inclination, would be to extract the mandibular incisor and perform a vertical ostectomy through the mandible at the extraction site and rotate the segments together to eliminate the extraction space (Figure 55-20). This would prevent further decreased inclination of the incisors with subsequent orthodontics but will narrow the transverse dimension of the anterior mandibular dental arch.

Correction of Maxillary Anterior Crowding or Increased Inclination

Increased inclination and/or crowding of maxillary anterior teeth are most commonly seen in patients with maxillary deficiency (hypoplasia). The following treatment methods can be used to correct this type of situation:

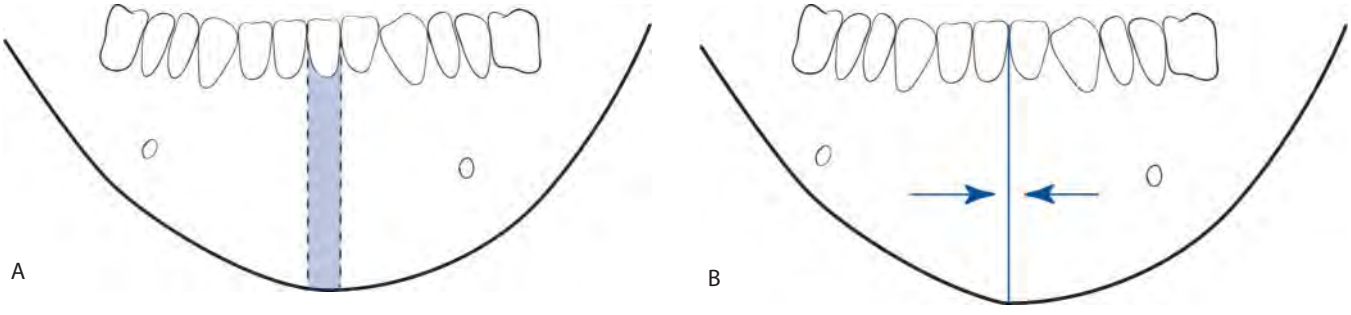


FIGURE 55-20. With a TSD of 5 mm or greater, in the presence of well-aligned teeth in proper inclination, the TSD can be managed by removing a mandibular central incisor and performing a vertical midline ostectomy **(A)** with space closure **(B)** and stabilization with a bone plate and screws.

IPR and Incisor Retraction

This technique involves removal of tooth structure at the contact points and is applicable when there is a rare reverse TSD with the maxillary anterior teeth too large for the mandibular anterior teeth. Usually, up to 3 mm of tooth structure can be safely removed from the contact area of the maxillary anterior six teeth with a margin of 50% of enamel remaining at the contact areas. However, this could make the maxillary incisors slightly smaller in size unless they are significantly oversized in the first place.

Extraction and Incisor Retraction

First or second bicuspids can be extracted depending on the amount of crowding, the anchorage requirements, and the amount of retraction of the incisors necessary. Every 1 mm of incisor retraction will require 1 mm of space on each side of the arch. Therefore, if the orthodontic goal is to retract the maxillary incisors by 3 mm, 6 mm of maxillary arch space will be required to accomplish this. Extracting first bicuspids will result in greater incisor retraction, because six multirooted posterior dental units (compared with six single-rooted anterior dental units) provide greater posterior anchorage. Extracting second bicuspids will result in less incisor retraction, because four posterior dental units (compared with eight anterior units) provide less posterior anchorage so that the posterior teeth will move forward a greater amount compared with first bicuspid extractions. The occlusal plane angle will also affect the posterior anchorage. Low–occlusal plane angle cases will have greater posterior anchorage stability, even with second bicuspid extraction, than will high–occlusal plane angle cases. High–occlusal plane angle cases will have less posterior anchorage stability, even with first bicuspid extraction, than low–occlusal plane angle cases. These factors are probably related to bite force influences. The amount of crowding may also influence which teeth to extract.

Distalization of Posterior Teeth

This objective can be accomplished using pendulum-type appliances, headgear, class II mechanics, osseointegrated dental implants, or TADs (i.e., implants posterior to molars, zygomatic implants, palatal implants, or buccal or lingual cortex implants). Distalizing maxillary posterior teeth can be augmented with class II mechanics but should be used only short term and discontinued several months before surgery to minimize postsurgical skeletal relapse potential that can occur with the use of long-term class II mechanics and the subsequent adverse effects on the TMJs. Another option is to distalize one tooth at a time on each side of the arch, beginning with the second molars (2 teeth moved against 12 anchor teeth). Another feasible approach is to use osseointegrated dental implants or TADs to distalize the maxillary arch, with implants either placed in the zygoma buttress, posterior to the second molars, or attached to the buccal cortex.

Anterior Maxillary Segmental Osteotomies

This technique permits uprighting of the anterior teeth but will cause the apical base of the segment to shift forward relative to the incisor edges unless teeth are extracted to reposition the incisal edges of the anterior teeth posteriorly. Careful assessment of the profile aesthetics is necessary to determine whether the patient can aesthetically benefit from this change. The interdental osteotomies should be done between the lateral incisors and the cuspids because this offers the best control in uprighting the segments (Figure 55-21), vertically leveling the segments, and correcting arch asymmetry and also allows opening of space between the lateral incisors and the cuspids (≤3 mm with 1.5 mm/side) that can be used for correction of crowding or TSD.

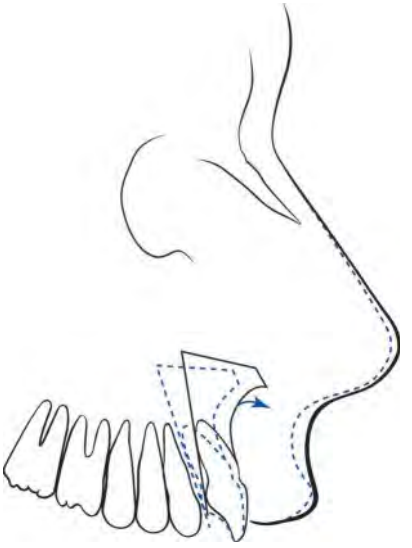


FIGURE 55-21. An anterior maxillary segmental osteotomy can be used to upright maxillary incisors, but the dentoalveolar base will rotate anteriorly if no teeth are extracted. Because this will affect the position of the nose and upper lip, careful evaluation of facial aesthetics is necessary to determine whether this approach is appropriate. *Dashed line* represents the original position of the anterior maxilla, and the *solid line* represents the uprighted segment and associated soft tissue changes.

Maxillary Expansion by Orthodontics, Orthopedic Palatal Expansion, and SARPE

These techniques will increase arch length and may allow retraction of the anterior teeth. However, they will also increase the curve of Wilson because the transverse width of the maxillary arch increases because the crowns of the posterior teeth will expand three times as much as the palate expands (Figure 55-22). In addition, with SARPE, the palate moves inferiorly The expanded arches may not be as orthodontically stable, requiring long-term or permanent retention.

Correction of Mandibular Anterior Crowding or Increased Inclination

Increased inclination and/or crowding of mandibular teeth occurs most often with mandibular deficiency (hypoplasia). The following treatment options can be used to correct these types of conditions.

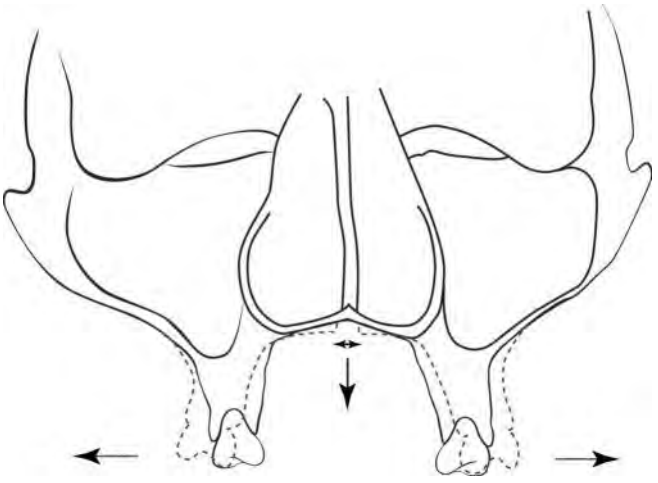


FIGURE 55-22. Maxillary expansion by orthodontics, orthopedics, or surgically assisted rapid palatal expansion (SARPE) will cause an increase of the curve of Wilson. Even with SARPE, the occlusal surface will expand three times as much as the palate expands, thus increasing the curve of Wilson. The palate also moves inferiorly.

IPR and Incisor Retraction

This technique involves removal of tooth structure at the contact points and is most applicable when there is a TSD with the mandibular anterior teeth being too large for the maxillary anterior teeth. Up to 3 mm of tooth structure can be safely removed from the contact areas of the mandibular anterior six teeth with a margin of 50% of enamel remaining at the contact areas. Subsequent retraction will decrease the axial inclination of the incisors providing that no major crowding is present.

Extraction and Retraction

First or second bicuspids can be extracted depending on the degree of incisor inclination, the amount of crowding, the anchorage requirements, and the amount of retraction of the incisors necessary. Every 1 mm of incisor retraction will require 1 mm of space on each side of the arch. Therefore, if the orthodontic goal is to retract the mandibular incisors by 3 mm, 6 mm of mandibular arch space will be required to accomplish this. Extracting first bicuspids will result in greater incisor retraction, because six multirooted posterior dental units (compared with six single-rooted anterior dental units) provide greater posterior anchorage. Extracting the second bicuspids will result in less incisor retraction, because four posterior dental units (compared with eight anterior units) provide less posterior anchorage, so that the posterior teeth will move forward a greater amount compared with first bicuspid extractions. The occlusal plane angle will also affect the posterior anchorage. Low–occlusal plane angle cases will have greater posterior anchorage stability, even with second bicuspid extraction, than will high–occlusal plane angle cases. High–occlusal plane angle cases will have less posterior anchorage stability even with first bicuspid extraction than low-angle cases. These factors are probably related to bite force influences. The amount of crowding may also influence which teeth to extract. If there is a large TSD (>5 mm), extraction of a mandibular incisor could be considered.

Distalization of Posterior Teeth

The mechanics to accomplish this include intra-arch, inter-arch, extraoral, or implant mechanics. Class III mechanics (i.e., elastics, headgear) can be used to distalize the mandibular teeth but may increase loading on the TMJs and could initiate TMJ problems. Another option is to distalize one tooth at a time on each side of the arch, beginning with the second molars (2 teeth moved against 12 anchor teeth). However, this technique takes a lot of time. The placement of dental implants or TADs posterior to the molar teeth or in the posterior buccal cortex could facilitate retraction without appreciably increasing the load to the TMJs.

Anterior Mandibular Subapical Osteotomies

This technique permits uprighting of the anterior teeth but will cause the apical base of the segment to shift forward relative to the chin (Figure 55-23), unless teeth are extracted at the time of surgery to reposition the incisal edges of the anterior teeth posteriorly.

Bilateral Mandibular Body Osteotomies

This technique will permit uprighting of the anterior teeth and forward rotation of the chin (Figure 55-24), unless teeth are extracted. Without extraction, bilateral body bone grafting will be required to provide bony continuity between the segments and facilitate healing. This technique would be indicated only if the chin is anteroposteriorly deficient before surgery.

Mandibular Symphyseal Distraction Osteogenesis

This technique, usually performed with a midline vertical osteotomy, will allow expansion of the dentoalveolus and

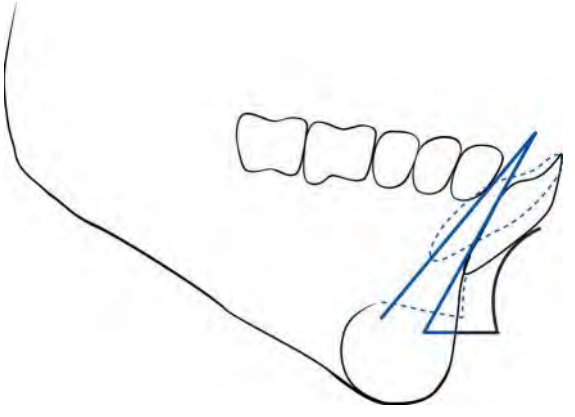


FIGURE 55-23. The anterior mandibular subapical osteotomy can be used to upright the mandibular anterior teeth, causing the apical base of the segment to shift forward relative to the chin, if teeth (bicuspids) are not extracted at the time of surgery. This may or may not be desirable. A chin augmentation may be required to achieve optimal aesthetics.

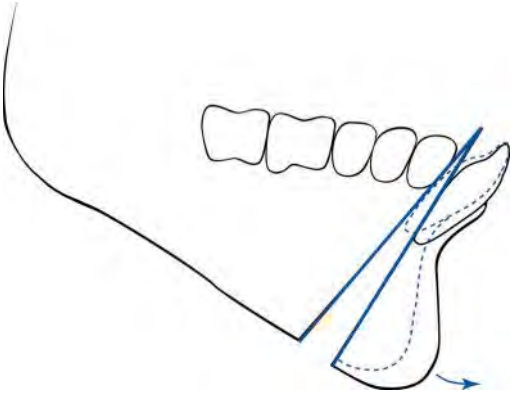


FIGURE 55-24. Bilateral body osteotomies can be used to upright mandibular anterior teeth, but the chin will rotate forward unless teeth are extracted. A gap created in the mandibular body area will require bone grafting unless teeth are extracted (first bicuspids) to allow the mandibular anterior teeth to move posteriorly, thus decreasing the forward movement of the chin.

widening of the mandibular arch, providing room to retract and/or align the teeth. This is an excellent treatment method to gain space for major arch length discrepancies. However, it is done as a prerequisite surgery to achieve the orthodontic goals before the major orthognathic surgery. Orthodontic preparation may be necessary before performing the midline vertical osteotomy. The roots of the central incisors (or the adjacent teeth, wherever the osteotomy is to be performed) must be tipped away from each other to make room for the interdental osteotomy. This can be accomplished by placing the mesial aspect of the bracket higher than the distal aspect on each of the central incisors. Placing a short segment straight arch wire will then tip the roots distally, creating space to safely perform the vertical interdental osteotomy (Figure 55-25). If a toothborne distraction device is used, orthodontic treatment on any other teeth should *not* be initiated until adequate healing of the distraction area has occurred (~4 mo from initiation of the distraction). Otherwise, it may result in developing dental mobility and orthodontic instability, with the teeth expanding more than the basal bone. This can result in transverse dental relapse postdistraction with less expansion of the dental arch than desired. Bone-borne devices are not affected by predistraction orthodontics.

Correct Decreased Inclination of Maxillary Incisors

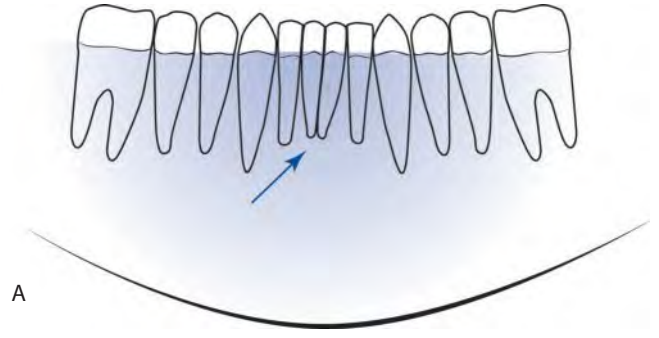
Decreased inclination of maxillary incisors is most commonly seen in class II division 2 malocclusions or with missing teeth in the arch. The following approaches can be used to correct this type of condition.

Correct Crowding

Crowding of the maxillary anterior teeth can accompany vertically inclined teeth. Therefore, correcting the crowding will increase the incisor inclination.

Open Space

In class I and class II patients, decreased inclination of the incisors may be present because of previous extractions (i.e., bicuspids), congenitally missing teeth, previous trauma resulting in loss of teeth, or small maxillary anterior teeth (i.e., small maxillary lateral incisors). Opening space in the bicuspid areas, if the problem exists because of previous extractions, can correct this problem and provide additional



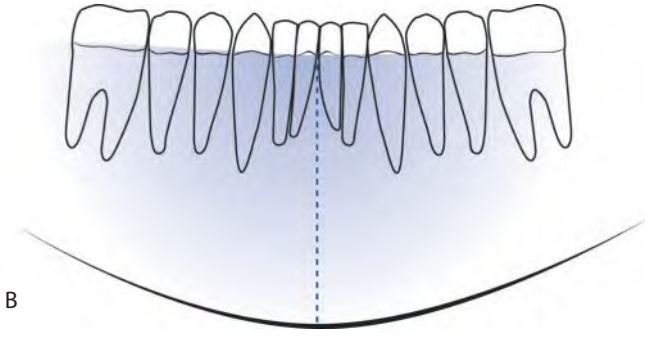




FIGURE 55-25. Mandibular symphysis distraction osteogenesis. **A,** Often, the incisor roots are very close together. **B,** Space must be created between the roots of teeth adjacent to the intended midline vertical osteotomy. **C,** Placing brackets on only the central incisors with the mesial aspect of the brackets higher than the distal aspect on each tooth and placing a short straight wire segment will tip the roots distally away from each other, creating space to perform the vertical interdental osteotomy.



FIGURE 55-26. Coil springs to create arch space. Retroclined incisors may result from a division 2 malocclusion, crowding, missing dental units, or small teeth. Space can be created using coil springs that tip the incisors forward. **A,** Small maxillary laterals and missing mandibular bicuspids. **B,** Coiled springs create interdental spaces around the maxillary laterals (to correct an anterior TSD) and in the mandibular first bicuspid area (to replace a missing tooth and increase inclination of anterior teeth). **C,** The spaces around the maxillary lateral can be eliminated by bonding, veneer, or crown. In the mandibular arch, the space can be eliminated by surgical ostectomy, or replacement of the missing tooth with a bridge or dental implant.

dental units for a more complete occlusal result. The use of coil springs usually works well for this situation. If the problem is in the lateral incisor area, opening space can help correct the TSD as well as increase the incisor inclination (Figure 55-26).

Interarch Mechanics

The use of class III mechanics (i.e., elastics) can increase maxillary incisor inclination. However, the class III mechanics can be detrimental by overloading the TMJs.

Interdental Osteotomies

An anterior maxillary subapical osteotomy or segmentalized Le Fort I osteotomy will permit rotation of the anterior teeth to increase their inclination. However, significant room must be created between the roots of the adjacent teeth (lateral incisors and cuspids) at the osteotomy areas. Because bone removal between the teeth may be required, there is an increased risk of damage to the adjacent teeth. If the maxilla requires surgical expansion, segmentalization between the lateral incisors and the cuspids will allow the anterior segment to rotate posteriorly between the expanded posterior segments with fewer requirements for bone removal, if required at all.

Correct Decreased Inclination of Mandibular Incisors

Decreased inclination of mandibular incisors is more commonly seen in patients with a prognathic mandible or with missing teeth. The following treatment methods can be used to correct this condition.

Correction of Crowding

Crowding of the mandibular anterior teeth often accompanies vertically inclined teeth. Therefore, correcting the crowding will increase the incisor inclination.

Creation of Arch Space

In class I and class II patients, decreased inclination of mandibular incisors may be present because of previous extractions, congenitally missing teeth, previous trauma resulting in loss of teeth, or small mandibular teeth. In class III patients, decreased inclination of incisors may be present owing to an excessive amount of alveolar bone compared with the size of the teeth. If bicuspids are missing, opening space in the bicuspid areas can correct this problem and provide additional dental units for a more complete occlusal result. The use of coil springs usually works well for this situation (see Figure 55-26).

Occasionally, a mandibular incisor may be missing for various reasons. Viable options include opening appropriate space around the remaining three incisors and building up the crowns by bonding, veneers, or crowns. This technique works best if there is a TSD that is less than the width of the missing tooth. However, the maxillary dental midline will be in the center of a mandibular incisor. Another option would be to open space in the area of the missing tooth and replace it with a dental implant or bridge. This technique may work best when there is no TSD with a full-size dental replacement.

Interarch Mechanics

The use of class II mechanics (i.e., elastics, Herbst appliance) can increase mandibular incisor inclination. However, long-term class II mechanics can be detrimental to outcome stability and results, because of the potential untoward effects on the TMJs.

Interdental Osteotomies

An anterior subapical osteotomy or bilateral anterior body osteotomies will permit rotation of the anterior teeth to increase their inclination. However, significant room must be created between the roots of the teeth adjacent to the osteotomy areas. Because bone removal between the teeth may be required, there is an increased risk of damage to the adjacent teeth.

Correction of Excessive Curve of Spee: Maxillary Arch

This condition is most often seen with anterior open bite situations and high–occlusal plane facial types. Careful assessment

of the curve of Spee is important because using only orthodontic mechanics to correct this condition may not be very stable. An increased curve of Spee usually makes it difficult to get the occlusion to fit together. The condition can be addressed by the following treatment options.

Anterior Tooth Extrusion

Conventional orthodontics with straight wire techniques will tend to extrude the anterior teeth and, as a byproduct, will tip the molars buccally, increasing the curve of Wilson. These dental changes may be unstable and fraught with relapse potential.

Midbuccal Tooth Intrusion

This is a very difficult technique, unless high-pull headgear or osseointegrated implants or TADs are used to provide intrusive forces. Placing TADs palatally and bucally would provide the best force vector for the intrusion of these teeth.

Extraction and Retraction

Extraction of maxillary first or second bicuspids with retraction will usually decrease the curve of Spee, providing the incisors are increased in inclination in the first place.

Orthodontic, Orthopedic Palatal Expansion, or SARPE with Retraction

Expansion of the maxillary arch by any of these techniques will increase the arch length and allow some retraction of the anterior teeth. In late adolescence or adulthood, SARPE may provide better stability than the other two techniques. However, note that the curve of Wilson will increase because the expansion at the occlusal level compared with the palate will be a 3:1 ratio.[20]

Orthognathic Surgical Correction

The maxilla can be orthodontically aligned in segments by aligning the four incisors at a different level, compared with the posterior teeth, to avoid extrusion, intrusion, and buccal tipping of teeth. Placing compensating vertical steps between the lateral incisors and the cuspids (see Figure 55-15A) will accomplish alignment at different levels. For some cases, the vertical positional difference may occur between the cuspids and the bicuspids or could occur asymmetrically on one side of the arch compared with the other side. The step in the arch would then be made between the appropriate teeth. Another technique involves cutting the arch wire into two or more segments and aligning groups of teeth in individual units (see Figure 55-15B). However, it may be more difficult to control rotations and root position, particularly of the teeth adjacent to the ends of the segmented wires, compared with using a continuous wire with compensating vertical steps. The arch can then be leveled surgically with a three-piece maxilla performing osteotomies between the lateral incisors and the cuspids. If a segmental arch wire has been used, the cuspid crowns tend to tip palatally presurgery and may require surgical expansion of the cuspids to a greater extent than using a continuous arch wire with a vertical step. The three-piece Le Fort I osteotomy, with interdental osteotomies performed between the lateral incisors and the cuspids, will permit repositioning of the anterior segment independent of the posterior segments (Figure 55-27). The anterior segment can be reoriented vertically and anteroposteriorly, and the axial inclination of the incisors can be changed to correct the curve of Spee and achieve the best interdigitation of the segments.

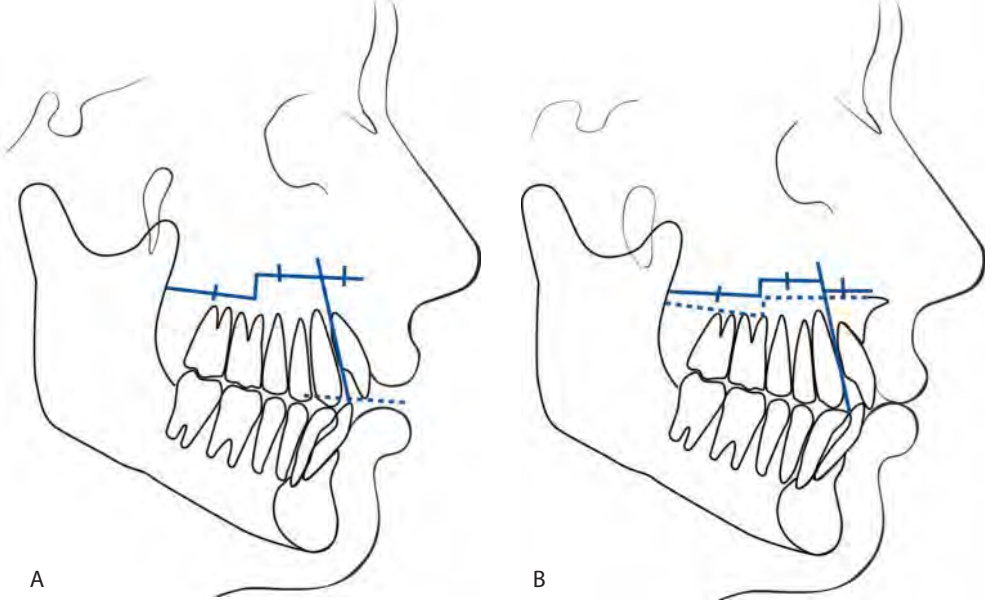


FIGURE 55-27. Surgery for correction of an excessive curve of Spee. **A,** Aligning the maxilla in segments with the incisors at an elevated level compared with the posterior teeth will permit interdental osteotomies to be performed. **B,** Surgical leveling of the occlusal plane *(dotted line)* from a predictability and stability standpoint is superior to orthodontic means alone, particularly when no extractions are performed.

Correction of Excessive Curve of Spee: Mandibular Arch

An accentuated curve of Spee in the mandibular arch most often occurs in anterior deep-bite relationships. The condition can be addressed by the following treatment options.

Mandibular Anterior Tooth Intrusion

Intrusion mechanics can predictably inferiorly position mandibular anterior teeth approximately 2 mm. Beyond 2 mm, the vertical relapse approaches 60%. With accentuated curves of Spee, the contact area of the teeth will be at a different level in which the teeth are more narrow, below the normal contact level. Therefore, for every 1 mm of leveling of the mandibular arch, the mandibular incisor edges will move forward 0.6 mm to 1 mm as the contact points align. Any crowding of the arch will further contribute to flaring of the incisors. Intruding teeth will decrease the anterior mandibular vertical height and must also be taken into consideration so that the anterior mandibular height is not excessively shortened.

Midbuccal Tooth Extrusion

Extrusion of midbuccal teeth may be more stable than intrusion of anterior teeth. However, this technique is difficult to perform without special considerations. If the patient's malocclusion has the bicuspids and first molars in occlusion, extrusion will be virtually impossible. However, constructing a splint or using turbos that will open the bite and engage only the mandibular anterior teeth and second molars, with the bicuspids and first molars out of contact, will permit extrusion of the midbuccal teeth. Another alternative would be to correct the accentuated curve of Spee after the mandible and occlusion are surgically repositioned, placing the incisors and molars into proper contact, and then extrude the midbuccal teeth postsurgery. With this approach, the molars may tip distally and the arch may widen.

Interdental Osteotomies

An anterior subapical osteotomy (Figure 55-28) or bilateral anterior body osteotomies (Figure 55-29) will permit downward repositioning of the anterior teeth, with very stable

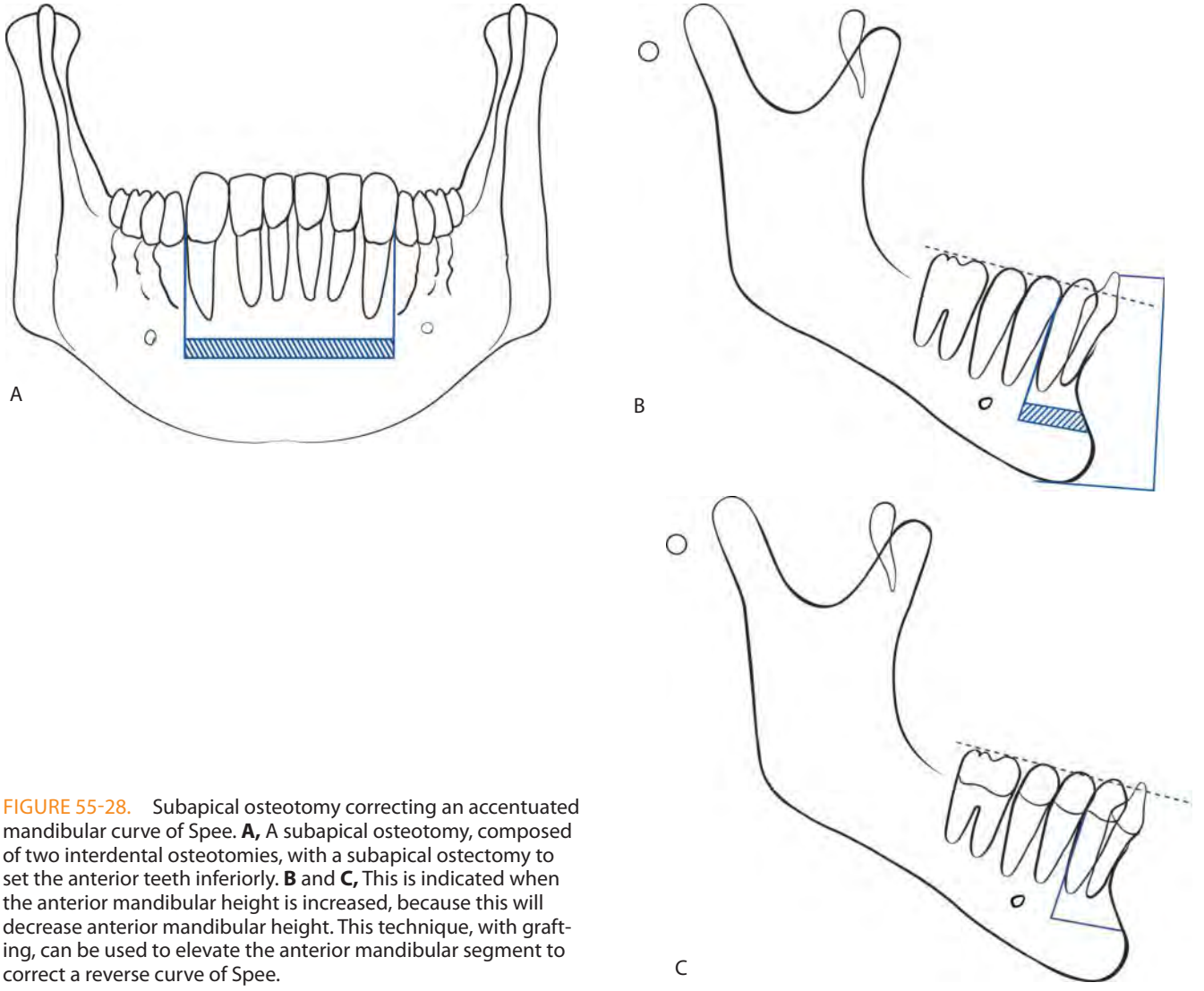


FIGURE 55-28. Subapical osteotomy correcting an accentuated mandibular curve of Spee. **A,** A subapical osteotomy, composed of two interdental osteotomies, with a subapical ostectomy to set the anterior teeth inferiorly. **B** and **C,** This is indicated when the anterior mandibular height is increased, because this will decrease anterior mandibular height. This technique, with grafting, can be used to elevate the anterior mandibular segment to correct a reverse curve of Spee.

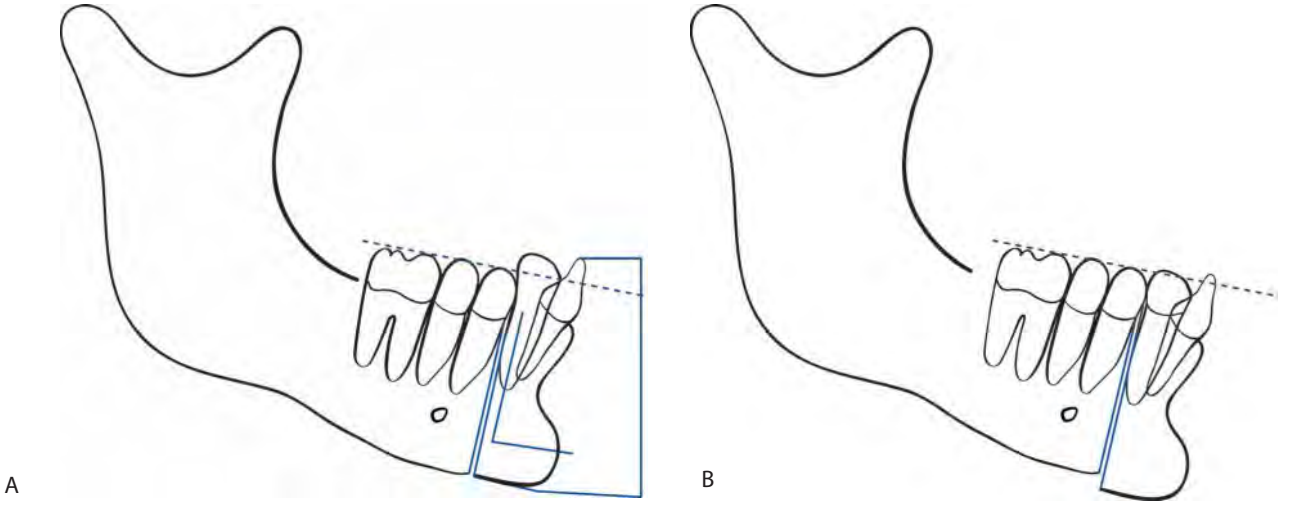


FIGURE 55-29. **A** and **B,** Bilateral mandibular body osteotomies will permit leveling of an excessive curve of Spee without shortening the vertical height of the mandible and are indicated when the mandibular anterior dental height is normal or slightly decreased vertically. This can also be used to correct a reverse curve of Spee by elevating the anterior segment.

results when the surgery is properly performed. If the anterior vertical height of the mandible is excessive, the subapical osteotomy would be indicated because it will shorten the anterior mandibular height by the amount that the incisors are lowered. Bilateral anterior body osteotomies would be indicated when the vertical height of the anterior mandible is normal or less, so that the anterior height remains unaltered while the curve of Spee is corrected (Figure 55-29).

Correction of Reverse Curve of Spee: Maxillary Arch

Reverse curves of Spee are more commonly seen in division 2 malocclusions and in vertical maxillary deficiencies with an anterior deep bite. The maxillary incisors are commonly in a decreased axial inclination. Crowding may or may not be present. The condition can be addressed by the following treatment options.

Correction of Crowding or Division 2 Relations

Eliminating crowding and division 2 dental positions will tip the incisors forward, increasing the incisor axial inclination and decreasing the reverse curve of Spee. These movements will usually fill out the upper lip but may decrease the maxillary tooth–to–lip relationship. Maxillary incisors may become intruded with a straight wire technique.

Midbuccal Tooth Extrusion

This technique is difficult if the midbuccal teeth are in occlusion with mandibular teeth. However, the bite can be opened with a splint or turbos that affords contact on only the maxillary second molars and anterior teeth, with the maxillary midbuccal teeth out of contact. The midbuccal teeth (bicuspids and first molars) can then be extruded into position to improve the curve of Spee.

Creation of Arch Space

If the reverse curve of Spee is related to missing teeth or TSDs, spaces can be opened to aid in increasing the axial inclination of the incisors and decreasing the reverse curve of Spee. These spaces can then be eliminated by bonding, crown and bridge, or dental implants and crowns (Figure 55-26).

Interdental Osteotomies

Multiple maxillary osteotomies can be performed so that the maxilla can be repositioned in segments, enabling leveling of the arch. Presurgical orthodontics should be designed to align the teeth at different vertical levels to facilitate the surgery and minimize orthodontic relapse potential. It is usually easiest and most applicable to make the osteotomies between the lateral incisors and the cuspids. This may particularly be indicated when the maxilla must be repositioned anyway and maxillary expansion is also required. Performing a three-piece segmented maxillary osteotomy will then allow vertical alteration between the anterior and the posterior segments to level the curve of Spee.

Correction of Reverse Curve of Spee: Mandibular Arch

This condition is most commonly seen in patients with macroglossia, habitual tongue posturing, or tongue thrust, with an associated anterior open bite. The following techniques can be used to correct this type of condition.

Anterior Tooth Extrusion

Extrusion of anterior teeth may not be very stable long term and, without permanent retention, could result in re-intrusion and redevelopment of an anterior open bite.

Midbuccal Tooth Intrusion

This technique may be accomplished with osseointegrated implants or TADs placed as anchors in the buccal and lingual

cortical bone of the dentoalveolus. However, no studies support the stability long term.

Extraction and Retraction

If the mandibular incisors are significantly increased in inclination, with or without crowding, bicuspid extractions can be performed and the incisors retracted, which will decrease the reverse curve of Spee.

Bonding the Mandibular Anterior Teeth

This technique can be used to level the arch by building up the incisors, increasing the crown height. However, care must be taken not to exceed a safe crown-root ratio and/or create an aesthetic compromise.

Interdental Osteotomies

Anterior subapical (see Figure 55-28) or anterior bilateral mandibular body osteotomies (see Figure 55-29) can be used to elevate the anterior teeth. If the anterior mandibular height is short, the subapical osteotomy can also be used to increase the anterior height of the mandible. If the anterior mandibular height is normal, the bilateral anterior body osteotomies will permit elevation of the anterior teeth while maintaining the anterior height of the mandible.

Anteroposterior Arch Asymmetry (Maxilla or Mandible)

Anteroposterior arch asymmetry, when the cuspid on one side of the arch is anterior to the cuspid on the opposite side of the arch, is fairly common in patients with dentofacial deformities. Arch asymmetries can be related to developmental abnormalities, missing teeth, or ankylosed teeth. Dental midlines may not align with the facial midline. The condition can be addressed by the following treatment options.

Unilateral Extractions

In some cases, unilateral extraction and retraction will correct the problem. The decision must be made as to which tooth to extract. Extraction of a first bicuspid will allow greater anterior retraction compared with extracting a second bicuspid. This extraction would be indicated only if there were significant increased inclination of the incisors, crowding, and/or significant midline dental shift.

Unilateral Creation of Arch Space

This technique would be indicated if a tooth is missing, there is significant decreased inclination of the incisors, and/or the midline is significantly deviated to one side.

Interarch Mechanics

This technique can be effectively used by incorporating class II mechanics on one side and class III mechanics on the opposite side. Anterior cross-arch elastics can also be helpful. If only one arch is involved, maximizing anchorage in the other arch is very important so that an asymmetry does not develop in the normal arch. Osseointegrated implants or TADs can be used as anchors to correct asymmetry in an arch without having to use interarch mechanics.

Segmental Osteotomies

Osteotomies can be used in the maxillary arch by segmentalization of the maxilla and advancing one side more than the other side. Osteotomies in the mandibular arch to correct arch asymmetry can become somewhat complex. Anterior subapical osteotomies with removal of a unilateral tooth can correct some large discrepancies (6–9 mm). However, the subapical osteotomy may need to be combined with ramus sagittal split osteotomies and a unilateral or bilateral body osteotomy, with or without extraction, to shift the occlusion into a symmetrical position. These types of movements require a high degree of surgical skill, but can provide high-quality outcomes.

Divergence of Roots Adjacent to Interdental Osteotomy Sites

When interdental osteotomies are planned, it may be necessary for the orthodontist to tip the adjacent tooth roots away from the area of the planned osteotomy to prevent damage to the teeth (Figure 55-30). If the roots are too close together, postsurgical periodontal problems may develop with possible loss of interdental bone and teeth. Creating interdental space between the roots significantly improves the margin of safety. This can be easily achieved by selective bracket placement. For the tooth mesial to the osteotomy, the bracket is slightly rotated so that the mesial aspect of the bracket is positioned slightly more gingivally than the distal aspect of the bracket (Figure 55-31). Conversely, the distal tooth bracket is positioned so that the distal aspect of the bracket is placed slightly more gingivally than the mesial aspect of the bracket. With a straight wire technique, the roots will diverge.

Postsurgically, periapical radiography may be necessary for the orthodontist to check for rebonding the adjacent teeth brackets to ensure proper root angulation at completion of treatment.

Extraction versus Nonextraction Orthodontic and Surgical Treatment

The decision to extract or not to extract can sometimes be difficult. A number of factors may contribute to this determination:

Increased Inclination Anterior Teeth

Increased inclination anterior teeth may require extraction to set the teeth over basal bone. However, if the arch is to be expanded or teeth slenderized with IPR for treatment of a TSD, for example, extraction may not be necessary.

Crowding

This is a common indicator, particularly with major crowding or increased inclination of the incisior teeth. However, if

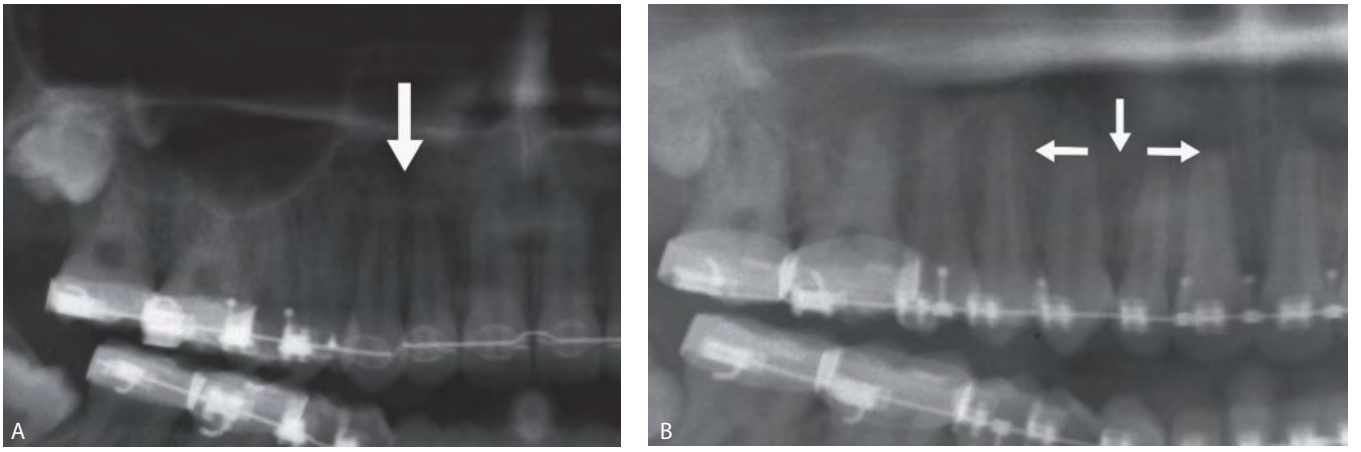


FIGURE 55-30. Interdental osteotomies. **A,** Panorex demonstrates inadequate room between the roots of the lateral incisors and the cuspids, and performing osteotomies with roots in this position may cause periodontal problems and loss of teeth. **B,** Adequate spaces for interdental osteotomies can be created by selective bracket placement on adjacent teeth.

crowding is mild to moderate, widening of the arch or teeth slenderizing for TSD may eliminate the need for extraction.

Tooth-Size Discrepancy

TSDs of significant magnitude may indicate the need for extraction, particularly if the TSD of the anterior mandibular teeth is 5 mm or greater and the mandibular incisors are increased in inclination and/or crowded, in which case a mandibular incisor extraction could be considered.

Curve of Spee

Accentuated curves of Spee in the maxillary arch usually have overangulated maxillary incisors, and reverse curves of Spee in the mandibular arch usually have increased inclination of mandibular incisors. Extraction of bilateral first or second bicuspids and retraction will result in leveling of the arches. However, arch expansion, when indicated, may create enough room so that extractions are not necessary.

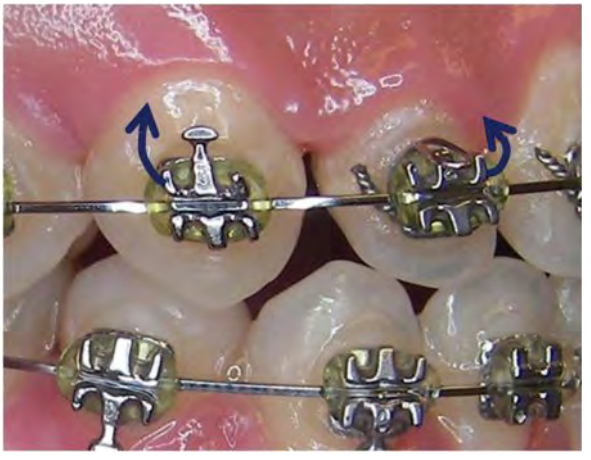


FIGURE 55-31. Selective bracket placement can create adequate interdental space for osteotomies. On the tooth mesial to the osteotomy, the mesial aspect of the bracket is rotated slightly gingival, and on the distal tooth, the distal aspect of the bracket is rotated slightly gingival, and a straight wire will then cause root divergence.

Arch Asymmetries

With significant anteroposterior arch asymmetries, unilateral or bilateral asymmetrical extractions (i.e., first bicuspid on one side and a second bicuspid on the opposite side) may be indicated when there is coexisting crowding, increased inclination of incisors, or midline shift.

Arch Width Coordination of Maxilla and Mandible

In some cases. transverse arch width discrepancies can be corrected with stable and predictable orthodontic movements, but in other cases. orthodontic correction may be very unstable and fraught with relapse. It must be determined whether to correct width problems by orthodontics, orthopedics, SARPE, or surgical expansion. Even with SARPE using a fixed device, the palate expands only approximately one-third the amount of the expansion that occurs at the occlusal level, thus increasing the curve of Wilson.[20] For example, if the maxilla is expanded with SARPE and the expansion at the occlusal level is 6 mm, the expansion at the palatal level will only be 2 mm (see Figure 55-22). Patients with reverse curves of Wilson in the maxillary arch may benefit more from these techniques, but those with a pretreatment accentuated curve of Wilson may have unfavorable results, with subsequent difficulty getting the buccal cusps to interdigitate. The following predictable changes will occur with maxillary arch expansion by orthodontic, orthopedic, or SARPE procedures.

1. The bite may open anteriorly, particularly if the maxillary incisors have significant initial vertical inclination. If the maxillary incisors are overangulated, the bite may deepen anteriorly as the spacing is closed.

2. Buccal tipping of the maxillary posterior teeth will increase the curve of Wilson, because the lingual cusps will move downward relative to the buccal cusps. This may make it very difficult to properly interdigitate the buccal cusps orthodontically. Therefore, these techniques are not recommended, especially when there is a preexisting accentuated curve of Wilson.
3. Long-term or perhaps permanent retention may be necessary to counterbalance the orthodontic relapse potential seen in a high percentage of these patients.
4. In late adolescent and adult patients, SARPE will likely be necessary to expand the maxilla orthopedically because the midpalatal suture is usually closed.

Surgical expansion of the maxilla at the time of the Le Fort I procedure using multiple segmentation of the maxilla, stabilization with bone plates and palatal or occlusal splints, and autogenous bone or hydroxyapatite synthetic bone grafting along the lateral maxillary walls can provide a good outcome. This technique when properly performed is very stable and eliminates the orthodontic relapse potential inherent with the other techniques.

Missing Teeth

Teeth can be missing from the arches for a number of reasons such as congenital absence, uneruption, previous orthodontic extractions, extractions for periodontal or dental pathology, and trauma. In some cases (i.e., congenital absence of maxillary lateral incisors, previous inappropriate bicuspid extraction), opening space to accommodate replacement teeth may be indicated. This is most applicable when the incisors are decreased in inclination without appreciable crowding. If the incisors are already increased in inclination and/or crowding is present, opening space orthodontically may be detrimental to stability and periodontal health. In this situation with missing maxillary lateral incisors, the cuspids can be used as lateral incisors but may require considerable recontouring to aesthetically and functionally conform to lateral incisor morphology. Although this cuspid substitution can work well for missing lateral incisors, it is done less frequently now that dental implants are so predictable and successful, thereby allowing the canine to be placed in its normal and more functional position.

When conditions permit, opening space for replacement teeth can be accomplished by appropriate mechanics to achieve the required space. Surgery can also be used to create spacing in some areas. In the mandibular arch, distraction osteogenesis can be used to create space. The missing teeth can then be replaced with dental implants, bridges, or partial dentures, for example.

Correction of Tooth Rotations

Bracket placement and arch wire adaptation are the primary keys to correcting rotated teeth, and it is usually best to achieve these corrections presurgery. However, if the malrotations do not interfere with the establishment of the desired dentoskeletal relationship, the rotations can be corrected postsurgery. Severe rotations may require supracrestal fiberotomy to prevent relapse and improve permanent retention. This can often be done at the time of orthognathic surgery.

Management of Short Roots

The two most common causes of short roots are developmental or root resorption. There are a numbers of reasons for root resorption including (1) orthodontic tooth movement with high forces; (2) presence of multinucleated odontoclasts possibly stimulated by transforming growth factor-Beta, cytokins, and interleukins; (3) systemic factors such as nutritional abnormalities, metabolic bone disease, age, and use of drugs; (4) systemic or innate predisposition including genetics; (5) impairment of alveolar bone resorption; (6) endocrine disturbances such as hyperphosphotemia, Papillon-Lefreve syndrome, Paget's disease, hyperparathyroidism, hypophosphatasia, Gaucher's disease, Turner's syndrome, or anachoresis; (7) inflammation of collagen in the periodontal ligaments; and (8) idiopathic root resorption. When these conditions are present pretreatment, orthodontic mechanics must be designed to place minimal forces on the teeth and minimize tooth movement with no extrusion, intrusion, or bodily repositioning because these forces may increase root resorption. Surgical management may require segmentation of the maxilla and/or mandible so that sections of the maxillary and mandibular alveolar bone and teeth can be moved independently to achieve a good dental interrelationship, minimizing pre- and postsurgical orthodontic forces. The interdental osteotomies, when performed properly, should have no adverse affect on root resorption.

Management of Ankylosed Teeth

Treatment of ankylosed teeth depends on (1) whether the tooth is primary or permanent, (2) the surrounding dentition, (3) the eruption status, (4) tooth position and orientation, (5) the time of onset and diagnosis, (6) the age of the patient, and (7) the treatment goals. Some of the considerations are discussed in the following sections.

Ankylosed Primary Tooth

This can impede the development and eruption of the permanent successor. If a primary tooth has a permanent successor, treatment is immediate extraction followed by space maintenance until the permanent tooth erupts. If no permanent successor is present and the primary tooth ankylosis occurs at an early stage in jaw growth and development with submergence of the tooth eminent, treatment includes extraction and space maintenance.[21] If the ankylosis occurs late with no permanent successor, the occlusal and proximal contacts can be reestablished with restorative dentistry to provide aesthetics and function with perhaps many years of service.[22]

It is important to diagnose and treat the ankylosed tooth before the adolescent growth phase. Retaining an ankylosed tooth during jaw growth leads to arrested development of the alveolar ridge. The severity of alveolar growth loss depends on the amount of facial growth left at the time that the ankylosis occurs. Timing the removal of an ankylosed tooth just at the start of the pubertal phase of adolescent growth may achieve the treatment objective of maintaining alveolar ridge height while allowing the tooth to remain long enough to act as a space maintainer and aesthetic temporary.[23]

Ankylosed Permanent Tooth

An unrecognized ankylosed permanent tooth tied into the arch wire can result in a significant malocclusion (Figure 55-32). There are several ways of treating the permanent ankylosed tooth. If ankylosis of the permanent tooth has an early onset during eruption, the tooth should be luxated, allowing for further eruption.[2] If repeated luxation proves ineffective, the tooth should be extracted to prevent submergence. If the onset of ankylosis occurs late in the normal eruption pattern, the tooth should be luxated. If the attempt is unsuccessful and the tooth does not submerge, it may be vertically restored on growth maturity. A composite buildup or crown can be added to a partially erupted ankylosed tooth to level and align the arch.[22] A deeply unerupted ankylosed tooth, primary or permanent, may be left undisturbed unless it is infected, alters the alveolar bone growth potential, constitutes an immediate threat to the occlusion or adjacent teeth, or would impede the placement of an osseointegrated implant.[3]

Other treatment options include extraction followed by reimplantation, osseointegrated implant, or prosthetic replacement.[24] The patient's developmental age is very important in considering replacing an ankylosed tooth with an osseointegrated implant. The implant will have the same effect on growth of the alveolar ridge as the ankylosed tooth and, therefore, should be considered for placement after alveolar growth is essentially complete.[25]



FIGURE 55-32. An ankylosed first molar tied into the arch wire has prevented alveolar development and created a posterior open bite.

Proffit[21] suggests surgical luxation of the tooth with extraction forceps disrupting the cementum-bone fusion followed by immediate orthodontic traction to move the tooth into position. Luxation involves breaking the bony bridge of ankylosis without damaging the apical nutrient vessels. This procedure forms fibrous inflammation tissue in the reparative process. This tissue forms a false periodontal membrane, and tooth eruption may resume. Orthodontic movement should begin immediately. Complications include possible crown, root, and alveolar fractures, loss of viability and vitality, as well as reankylosis. When an ankylosed tooth is impacted, a similar technique can bring an impacted tooth (usually canines) into the arch. Exposure involves surgical uncovering, application of orthodontic bonding, and tension forces applied to direct the tooth into occlusion. However, if the tooth becomes reankylosed, the orthodontic forces will intrude adjacent teeth.

Orthodontics for Surgical Management of Ankylosed Teeth

Presurgical orthodontics may be indicated to create adequate space (minimum of 2–3 mm) between the roots of the adjacent teeth to safely accommodate interdental osteotomies around the ankylosed tooth. Spacing is best assessed with cone beam or periapical radiographs. The ankylosed tooth is left out of the arch wire, and all other teeth are properly aligned. If orthognathic surgery is required to correct a dentofacial deformity, the orthodontics are performed in the traditional manner, but the ankylosed tooth must remain out of the arch wire, unless it aligns well with one of the dental segments. After surgery, orthodontic mechanics can be initiated immediately to help get the mobilized dental segment with the ankylosed tooth into the best possible position.

Segmental Osteotomies

Performing single-tooth osteotomies or sectional arch osteotomies with mobilization of the segment will permit immediate repositioning of the ankylosed tooth (Figure 55-33), or facilitate repositioning by distraction osteogenesis (DO).

In select cases in which an ankylosed primary molar is present, without a successor, a treatment option is to remove the ankylosed tooth and eliminate the extraction space by performing a vertical body ostectomy in conjunction with a mandibular ramus osteotomy and advance the posterior teeth and mandibular body forward (Figure 55-34). This eliminates the need for osseointegrated implants and extensive dental reconstruction.

Final Presurgical Orthodontic Preparation

As presurgical orthodontic treatment progresses, new diagnostic records (lateral cephalograms, pantomograms, dental models) are taken to determine the feasibility and timing of surgical procedures. This will also aid the orthodontist in identifying specific areas that may need to be addressed in completing the presurgical orthodontic goals (i.e., sectional leveling of the arch segments, marginal ridge alignment,

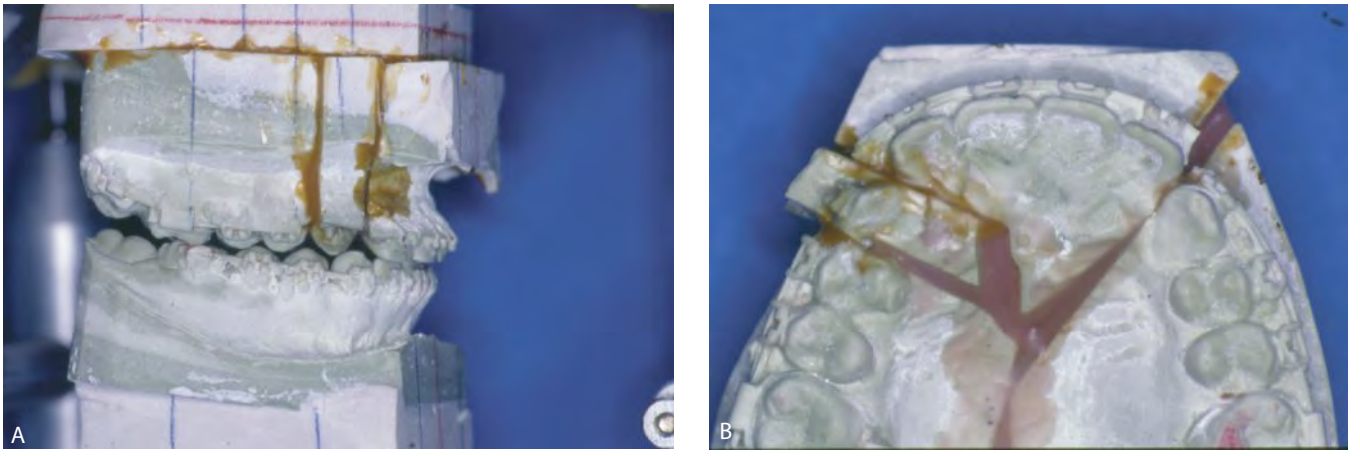


FIGURE 55-33. Single-tooth osteotomies can be performed as isolated cases or in combination with multiple maxillary osteotomies to allow individual movement of the dental osseous segments or application of immediate distraction osteogenesis (DO). **A** and **B,** This case had an ankylosed maxillary right cuspid (Figure 55-8) treated with segmental maxillary osteotomies including a single tooth segment containing the right cuspid.

vertical dental alignment, buccal surface alignment, additional TSD correction).

During surgery, the jaws are usually wired together once or twice, as each jaw is independently mobilized and stabilized with rigid fixation. To facilitate wiring the jaws together as well as provide a means of using postsurgical elastics if required, fixtures attached to the brackets or arch wires are usually necessary. Fixtures attached to the brackets are dependent on the manufacturer but may include ball hooks built onto the brackets, T pins, and K (Kobayashi) hooks (Figure 55-35). Fixtures attached to the arch wire include crimped-on hooks and soldered pins (Figure 55-36). Hooks built onto the brackets are preferred, followed by the other hooks placed on the brackets (T pins, K hooks). The least preferred are the hooks on the arch wire. The reason is that if postsurgery elastics are required for an extended time, the elastics and hooks on the arch wire will activate the arch wire, possibly creating unwanted orthodontic forces and movements (i.e., tipping the crowns lingually and the roots buccally). This undesirable torquing occurs to a much lesser degree when the hooks are directly on the brackets.

When the maxilla or mandible are to be segmentalized, it may be better for the orthodontist to section the arch wire (see Figure 55-15B) and bend the ends inward at the predetermined osteotomy areas immediately before surgery, or the surgeon can cut the wire at surgery.

The best type of arch wire to place before surgery is a rectangular SSW that fills the bracket slot. For example, with

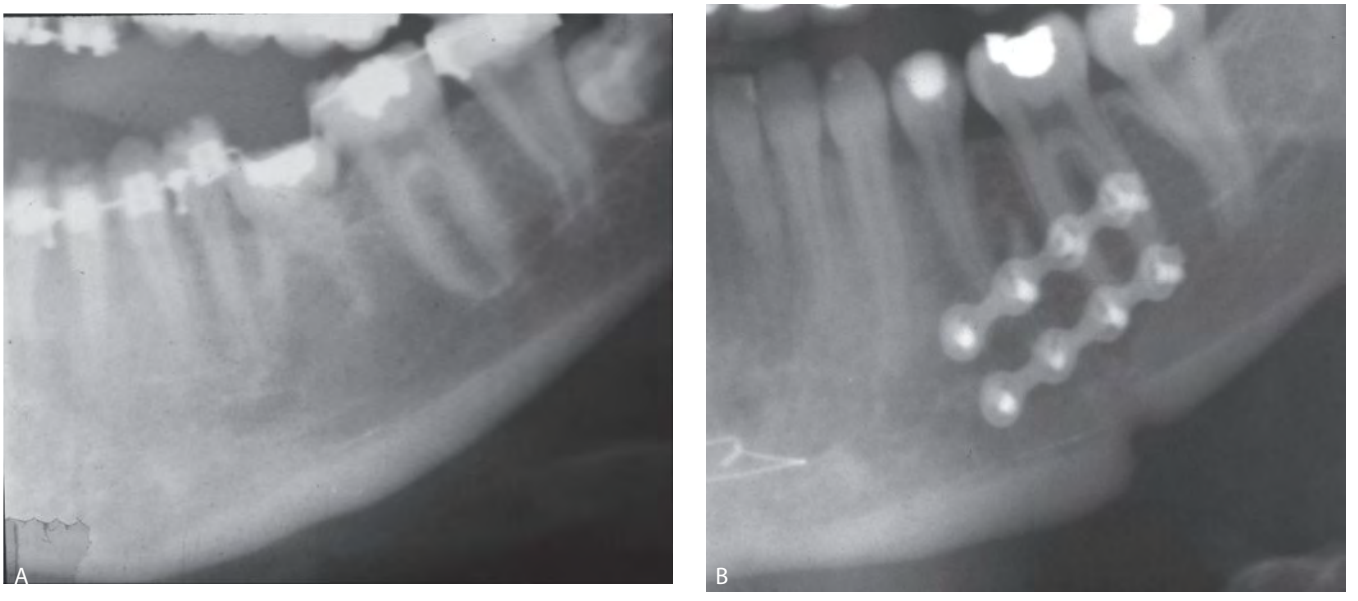


FIGURE 55-34. **A** and **B,** An ankylosed submerged primary tooth without a permanent successor can be treated with extraction of the primary tooth as well as a vertical body ostectomy with a mandibular ramus sagittal split osteotomy to advance the posterior teeth forward to eliminate the ankylosed tooth and space. This eliminates the need for an osseointegrated implant or a fixed prosthesis.

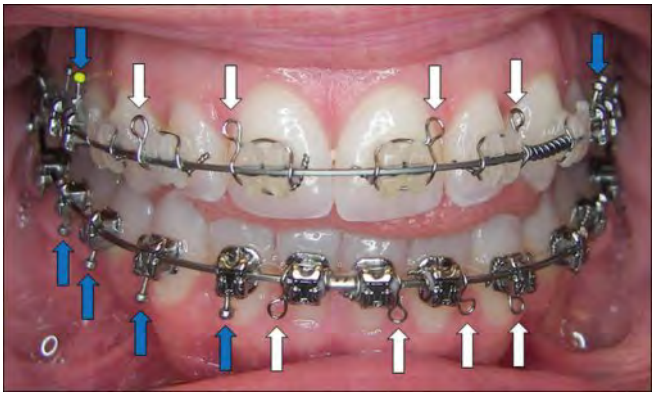


FIGURE 55-35. Orthodontic hooks. Ball hooks built onto the brackets *(blue arrows)* provide the best stability. Other options include T pins and K (Kobayashi) hooks *(white arrows)* or other methods to provide attachments directly on the brackets.

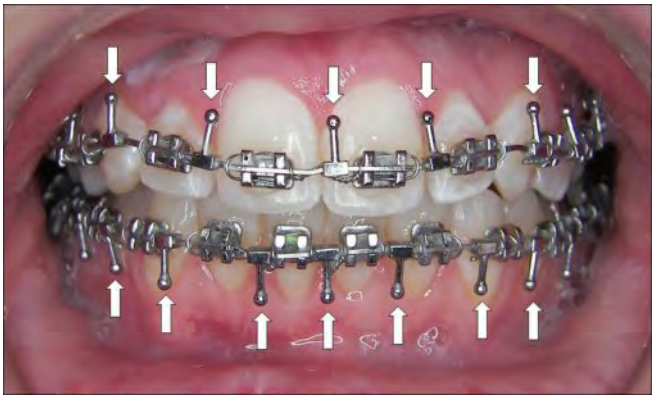


FIGURE 55-36. Soldered pins or crimped hooks *(white arrows)* onto the arch wire can also be used but are not preferred because the use of postsurgical elastics will activate the arch wire, possibly creating unwanted orthodontic movements.

an 18 slot, a 17 × 25-gauge wire is recommended, and for a 22 slot, a 21 × 25-gauge wire is indicated. This will help stabilize the individual dental units together as a whole arch or in segments when segmental surgery is required. The final wire should be placed 2 to 3 months before surgery.

POSTSURGICAL ORTHODONTIC TREATMENT

At the completion of surgery, a surgical stabilizing splint may be present and wired to the upper teeth and light elastics ($3^1/_2$ oz) are usually placed between the upper and the lower arches in the cuspid and molar areas and/or elsewhere, if indicated. If the occlusion is good, elastics may not be necessary if appropriate rigid fixation was used at surgery. Surgical stabilizing splints are indicated to provide the following: (1) stability in multiple segmental surgery of the maxilla or mandible, (2) transverse stability when the maxilla and/or mandible have been expanded or narrowed, (3) occlusal support when key teeth are missing (i.e., first, second, and third molars are missing in a quadrant), and (4) a means to interdigitate the occlusion if teeth are severely worn or missing. There are two basic types of maxillary splints for orthognathic surgery; the *palatal splint* (Figure 55-37) and the *occlusal coverage splint* (Figure 55-38). If the maxilla and mandible are single pieces and the teeth can be appropriately interdigitated, a final splint is usually not necessary in single- or double-jaw surgery.

Palatal Splint

This horseshoe-shaped splint (see Figure 55-37) is preferred because (1) it provides excellent stability, (2) the occlusal interrelationship can be maximized and observed (the teeth and occlusal relationships are not covered and hidden by the splint), (3) it is easier to keep the teeth and splint clean, (4) orthodontic treatment can be performed with the splint in place, including arch wire changes (see Figure 55-37B), (5) it can be left in place for extended time (2–3 mo or longer





FIGURE 55-37. The horseshoe-shaped palatal splint is constructed to help stabilize the maxillary segments to enhance healing. **A,** The splint design does not cover the occlusal surfaces but fits snugly around the palatal aspect of the tooth crowns. The splint is stabilized to the maxilla with 28-gauge stainless steel wire (SSW) placed through the splint and circumferentially around the first molars and first bicuspids, or as in this case, the second bicuspids, because the first bicuspids are missing. **B,** The wires are twisted on the labial side of the teeth *(white arrows)*. The splint design allows for maximal interocclusal fit at surgery as well as transverse and intersegmental stabilization. Orthodontic mechanics can be performed with the splint in position.

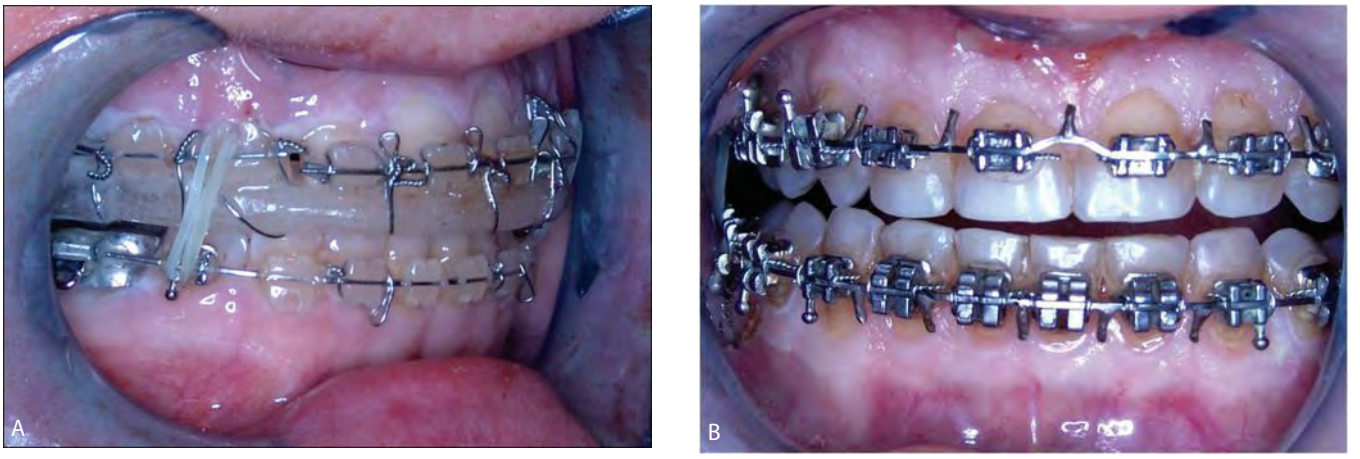


FIGURE 55-38. **A,** The occlusal coverage splint fits between the occlusal surfaces of the maxillary and the mandibular teeth. It is usually stabilized to the maxillary arch with wires placed through the buccal flange of the splint and around the adjacent orthodontic brackets on the teeth. **B,** Upon removal of the splint, a malocclusion can occur (as in this case) as a result of a defect in the occlusal surfaces of the models, teeth not set fully into the splint, occlusal interferences, or a warped splint. This may require considerable orthodontic efforts to optimize the occlusion.

if necessary), and (6) after removal, the splint can be modified to function as a retention splint by adding clasps. When making the splint, the palatal tissues beneath the splint must have wax relief of approximately 1 to 2 mm on the dental model so the splint does not impinge on the soft tissues. If the splint is constructed to sit against the soft tissues, a major risk of vascular compromise to the maxilla could occur resulting in a potential disastrous outcome. The splint is stabilized to the maxilla with light, 28-gauge SSW placed through the splint and circumferentially around the first molars and first bicuspids (second bicuspids if the first bicuspids are missing) and wires twisted on the labial side of the teeth (see Figure 55-37). The anterior teeth are not tied into the splint because the anterior segment could become displaced because of the cingulum morphology that will displace the segment superiorly and posteriorly. At surgery, the anterior segment is stabilized with bone plates.[26]

Occlusal Coverage Splint

The occlusal coverage splint is the most common type of maxillary splint used and has the following advantages: (1) it is easy to construct, (2) it provides transverse stability, and (3) it can "lock-in" the occlusion perhaps better than the palatal splint (see Figure 55-38A). However, the occlusal coverage splint presents the following possible complicating factors: (1) the clinician cannot see the occlusal interrelationship of the teeth with the splint in place, (2) an occlusal interference caused by the splint could result in a malocclusion (see Figure 55-38B), (3) postsurgical orthodontics can be much more difficult, (4) the arch wire cannot be changed with the splint in place, (5) orthodontics cannot be performed with the splint in place, 6) oral hygiene is more difficult to maintain, and (7) it can cause discoloration, decalcification, and caries of the teeth where covered by the splint. The splint is usually stabilized to the maxillary arch with wires placed through the buccal flange of the splint and around the adjacent orthodontic brackets on the teeth.

Mandibular Splint

Mandibular splints may be indicated for the following reasons: (1) they assist in stabilizing the mandible and segments after body osteotomies, subapical osteotomies, symphysis and sagittal split osteotomies, and other procedures, (2) they provide transverse stability if the mandible is expanded or narrowed by osteotomies or distraction osteogenesis, and (3) they temporarily replace missing teeth (Figure 55-39). Splints for providing transverse stability can be constructed to fit around the lingual aspect of the mandibular crowns, similar to the maxillary palatal splint. Therefore, these splints can be maintained in position for an extended time (2–3 mo or longer) if required. The splints can be secured to the mandibular arch by placing retention wires through the splint and circumferentially around the first molars and first bicuspids (second bicuspids if the first bicuspids are absent) (see Figure 55-39). Using this splint design, orthodontic arch wire changes and mechanics can progress with the splint remaining in place. Clasps can be placed on the splint after removal of the surgical retention wires so that the splint can be used as a removable retainer.

Splint Removal

This process requires cutting and removing the retention wires and then removing the splint. For most cases, the palatal or occlusal coverage splints can be removed at 1 month postsurgery. An exception is in maxillary expansions (>3–4 mm), in which the palatal splint can remain in position for 2 to 3 months or longer if required. After removal of the splint,

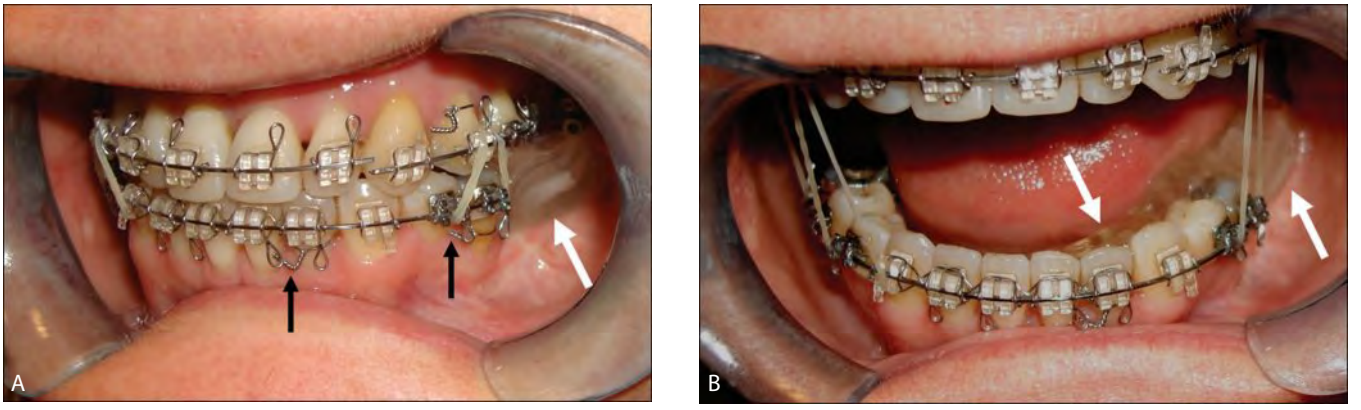


FIGURE 55-39. **A,** A mandibular splint *(white arrows)* can be constructed similar to the palatal splint design. It may be indicated to stabilize a segmented mandible, provide transverse stability, and temporarily replace missing teeth (as in this case). **B,** The splint can be stabilized to the mandibular teeth with wires placed through the splint and circumferentially around the adjacent teeth and twisted on the labial side *(black arrows).*

a transpalatal arch bar, removable palatal splint, or heavy labial arch wire followed by postorthodontic retainers can be used to maintain the maxillary expansions for an extended time period. The palatal splint can be modified to function as a removable splint by adding retention clasps. Maxillary or mandibular arches that have been expanded orthodontically, orthopedically, surgically assisted, or by osteotomies will require long-term transverse stabilization post-treatment to ensure a predictable outcome.[26]

Postsurgical Follow-up

The surgeon and/or orthodontist should see the patient on a weekly basis, or more often if indicated, for the first 1 to 2 months postsurgery to check the occlusion and patient progress and make any indicated changes in the elastic or orthodontic mechanics to maximize the occlusal interrelationship. If the occlusion fits properly, elastics may not be necessary. Then, as long as the occlusion remains stable, the time span between appointments can be increased. The surgeon and orthodontist must coordinate this postoperative patient management.

Patient Positioning at Follow-up

Patient position is important when evaluating postorthognathic surgery occlusal relationships. Patients should be in an upright or slightly reclined position; not in a horizontal position as used for routine orthodontic treatment. The mandibular condyles may set more inferior and posterior than usual, particularly shortly after surgery. Conversely, if the surgeon was somewhat rough with the surgery or TMJ surgery was done simultaneously, there may be intercapsular edema that could cause the mandible to be postured slightly forward. If there was preexisting TMJ pathology that was not corrected, patients may "splint" by holding the mandible forward to decrease pressure on the TMJs, altering the centric relation occlusion. Of particular concern is when TMJ surgery has also been performed at the same operation as the orthognathic surgery. The capsular ligaments have been cut and with the patient in a reclined position, the condyles can slide posterior and inferior to the centric relation position, yielding a false class II end-on occlusal relationship. Setting the patient in an upright or only slightly reclined position and pushing gently upward at the angles of the mandible should seat the condyles into a centric relationship to more accurately evaluate the occlusal interrelationship.[26]

Aggressive Postsurgical Orthodontics

If the occlusion is not "perfect" initially postsurgery, the surgeon and/or orthodontist must aggressively apply the appropriate elastic mechanics. Relatively light strength elastics ($3^1/_2$ oz) will usually be adequate to correct the situation. However, occasionally, heavier elastics may be necessary. This can be accomplished by doubling-up the $3^1/_2$-oz elastics or using a stronger elastic. Delays in addressing postsurgical occlusal imbalances will result in much greater difficulties in correcting the problem at a later time. If the occlusion was good at the completion of surgery, but a shift has occurred or open bite developed, this is often due to minor posterior occlusal changes that have caused interference. If light elastics do not reapproximate the occlusion, placing heavy elastics in the appropriate direction for 30 minutes to an hour may settle the occlusion into position so that light elastics can then be used again. Although heavy-strength elastics are occasionally necessary, it is unusual to require them when the surgery has been properly and accurately performed. Occasionally, equilibration of the teeth may be necessary to settle the occlusion into position. If there is a major postsurgical malocclusion, assessment of postsurgical lateral cephalograms and TMJ tomograms may help determine whether it is a correctable problem orthodontically or whether additional surgical intervention is required. If required, the sooner the surgical intervention is performed, usually the better situation for the patient and surgeon.

Postsurgical Elastic Traction

Postsurgical interarch elastics are indicated for the following reasons: (1) to maximize the occlusal fit; (2) to provide orthodontic forces to correct postsurgical occlusal discrepancies; (3) to take stress off the muscles of mastication to improve patient comfort immediately postsurgery; (4) to finalize the occlusion; and (5) to minimize edema in the bilaminar tissues if simultaneous TMJ surgery was performed. Controlling the occlusion postsurgery can generally be accomplished using elastics with one or more of the following vectors depending of the situation; that is, class II, class III, vertical, box, triangle, trapezoidal, rhomboidal, cross-arch, anterior tangential, or others. Usually, only light-force elastics of $3^1/_2$ ounces with sizes of $^1/_8$", $^3/_{16}$", $^1/_4$", and $^5/_{16}$" are needed because teeth and bone segments (maxilla particularly) can move faster postsurgery. If the occlusion fits together very well, elastics may not be necessary, unless the patient requires elastics for improved comfort by providing vertical support for relieving tension on the muscles of mastication. As soon as possible postsurgery, patients should be instructed on the placement and removal of their elastics to facilitate their dietary intake, oral hygiene, and jaw exercises. When TMJ surgery is simultaneously done with the orthognathic surgery, using light class III directional elastics for a few days postsurgery will minimize TMJ edema. If postsurgical TMJ edema develops, the occlusion can shift toward a class III end-on relationship. Class III elastics will then be necessary to reapproximate the occlusion into a class I relationship. It is usually easier to minimize the TMJ edema immediately after surgery with class III elastics than to recapture the occlusion later.[26]

Vertical Elastics

The use of anterior vertical elastics must be closely monitored. Although often necessary initially postsurgery to provide vertical support to the jaws to decrease masticatory muscle tension and to maximize the occlusal fit, these elastics can also create unwanted dentoalveolar changes (Figure 55-40). Patients that may be predisposed to these unwanted changes include (1) patients with nasal airway obstruction that was not corrected at surgery and who continue to be mouth breathers; (2) patients who habitually hold their jaws apart or in their work or play activities talk a lot requiring the jaws to be held open; and (3) patients with short roots. Holding the jaws apart for extended periods of time with anterior vertical elastics will extrude the anterior teeth and, over time, increase the alveolar bone height. Also, a degree of DO may occur in the maxilla. These changes may result in the development of posterior open bites, premature contact of the incisors, increased upper tooth-to-lip relationship, and increased mandibular and maxillary anterior vertical height. Avoiding these factors may include one or more of the following: (1) perform stable presurgical orthodontics and accurate surgery to decrease requirements for vertical elastics; (2) discontinue vertical elastics as soon as possible; (3) decrease daily time requirements for wearing elastics, particularly during activities in which the jaws will function in an open position; (4) use very light vertical therapeutic clenching to minimize adverse vertical forces; (5) correct nasal airway obstruction and sleep apnea issues by performing the appropriate surgical procedures so the patients can breathe through their noses instead of requiring mouth breathing.

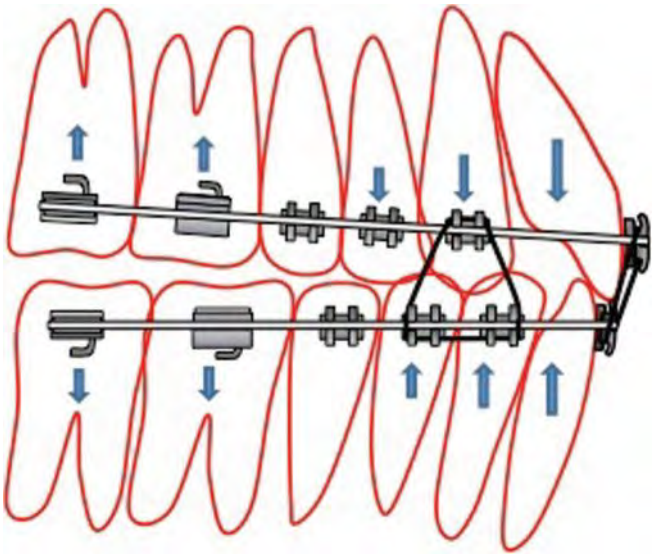


FIGURE 55-40. The use of anterior vertical elastics must be closely monitored. These elastics can create undesirable dentoalveolar effects including development of posterior open bites, premature contact of the incisors, increased tooth-to-lip relationship, and increased mandibular and maxillary anterior vertical height.

Power Chains

Avoid the use of power chains (Figure 55-41A) in the upper arch postsurgery. The use of power chains is a relatively common technique in the orthodontic finishing phase of treatment. However, power chain forces can create increased torque on the incisors, tipping the incisor edges posteriorly and tipping the root tips anteriorly (see Figure 55-41 B and C). This can cause premature contact of the incisors with subsequent end-on incisor relationship and development of posterior open bites. This can be a major problem, particularly in patients in whom spacing was created in the anterior arch around the lateral incisors to compensate for the TSD. When this problem develops, the power chain should be removed immediately and mechanics reversed to tip the incisor edges forward and the root tips posteriorly. This may involve reopening space between the lateral incisors and the cuspids that may have been inadvertently closed. This spacing can be eliminated postorthodontically with bonding, veneers, or crowns on the lateral incisors.

Changing Arch Wires

Segmental osteotomies in the maxilla or mandible commonly require the arch wire to be cut into sections. Crimping the wire ends, placing a bead of acrylic over the ends, or using

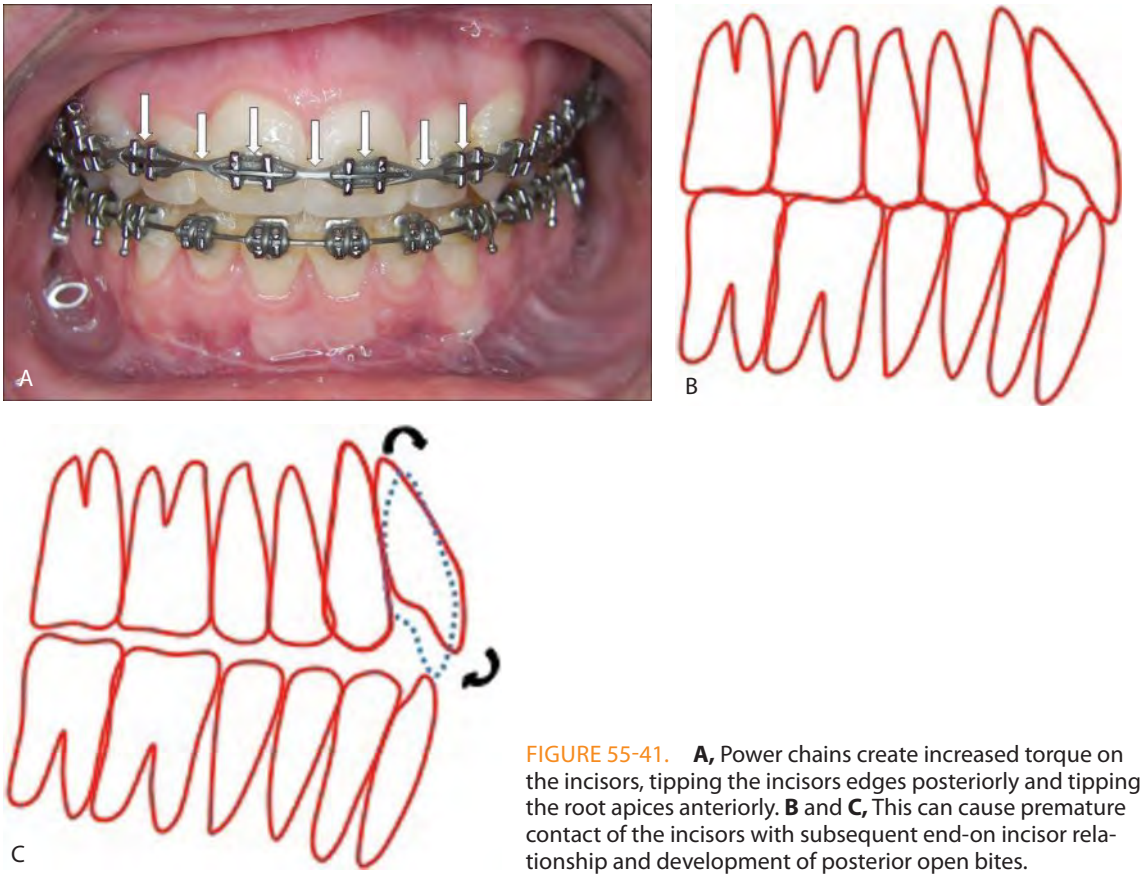


FIGURE 55-41. **A,** Power chains create increased torque on the incisors, tipping the incisors edges posteriorly and tipping the root apices anteriorly. **B** and **C,** This can cause premature contact of the incisors with subsequent end-on incisor relationship and development of posterior open bites.

wax postsurgery to cover the cut ends of the wire are techniques to protect the lips and cheeks from trauma caused by the cut wire ends. Generally, the arch wires can be changed at about 4 to 6 weeks postsurgery, at about the same time that the splint is ordinarily removed. It is usually too uncomfortable for patients to tolerate the arch wire change before that time. If a palatal splint is in place, it can remain in place because it should not interfere with the arch wire change and the splint can continue to help with maxillary transverse stability. Occlusal coverage splints usually require removal before the arch wire can be changed.

Dental Arch Spacing

The correction of significant anterior TSDs may require the creation of space in the upper arch between the lateral incisors and the cuspids and/or the lateral incisors and the central incisors to ensure a good class I occlusal fit with proper overjet/over bite relationship (see Figure 55-26B). It is very important in the postsurgical patient management phase that the space is maintained for subsequent bonding, veneers, or crowns (see Figure 55-26C). DO NOT CLOSE THE SPACE! because this can result in malocclusion. A common postsurgery orthodontic practice is to close interdental spaces. This can create major occlusal problems by downward and backward retraction of the maxillary incisors resulting in an end-on incisor relationship, posterior open bites, downward and backward rotation of the mandible, increased stress on the TMJs, and pain. If this situation occurs where the spaces have been closed, the spaces need to be opened again with forward rotation of the incisors to improve the occlusal fit with subsequent restorative dentistry to eliminate the spaces with bonding, veneers, or crowns.

Tooth Movement

Teeth move more rapidly for approximately the first 6 months postsurgery as compared with presurgery. Relative to tooth movement, the orthodontist can usually accomplish in 1 to 2 weeks postsurgery what took 4 to 6 weeks presurgery. This is because the bone metabolism and bone "turnover" increase substantially after surgery, allowing more rapid bone resorption and apposition in response to orthodontic forces, so the teeth move more quickly. In addition, with segmental maxillary surgery, the individual bone segments can also move some, providing increased flexibility of the teeth and bone segments.

Finishing Orthodontics

It is important for the patient to keep the orthodontic appliances on for a minimum of 4 to 6 months postorthognathic surgery, when appropriate rigid fixation techniques are used, to allow for initial bone healing as well as finish aligning,

leveling, and stabilizing the occlusion. It usually takes 4 months to complete the initial postsurgical bone healing phase to where the maxilla and mandible should be skeletally stable.[27] If inadequate rigid fixation was used or the maxillary bone was exceptionally thin, or uncontrolled clenching/bruxism is present, orthodontic appliances may be required for a significantly extended time. The greater and more complex required surgical movements and presurgical orthodontic treatment mechanics, generally the longer the time requirements for the postsurgical orthodontic management. The orthodontist will determine when the occlusion is maximized and stabilized and the patient is ready for debanding and retainers.

Orthodontic Retainers

For patients who have undergone orthognathic surgery, rigid retainers (Hawley or wraparound types) without occlusal coverage (Figure 55-42A) are recommended to provide transverse support to the width of the maxillary and mandibular dental arches, maintain the dental alignment, and allow for maximal interdigitation of the occlusion (see Figure 55-42B). Occlusal coverage retainers (see Figure 55-42C) should not be used because of vertical separation of the occlusion as this can create a malocclusion, usually with open bite situations anteriorly or posteriorly, unilaterally or bilaterally. In addition, occlusal coverage retainers are often flexible (see Figure 55-42D) and do not provide any transverse stability, resulting in transverse relapse in cases of surgical or orthodontic expansion or narrowing of the dental arches that can result in anterior open bite and posterior cross-bite relationships.[26]

Relapse

Orthodontic or surgical instability and relapse can also cause changes and is particularly related to transverse or vertical relapse of the orthodontically expanded maxilla or unstable

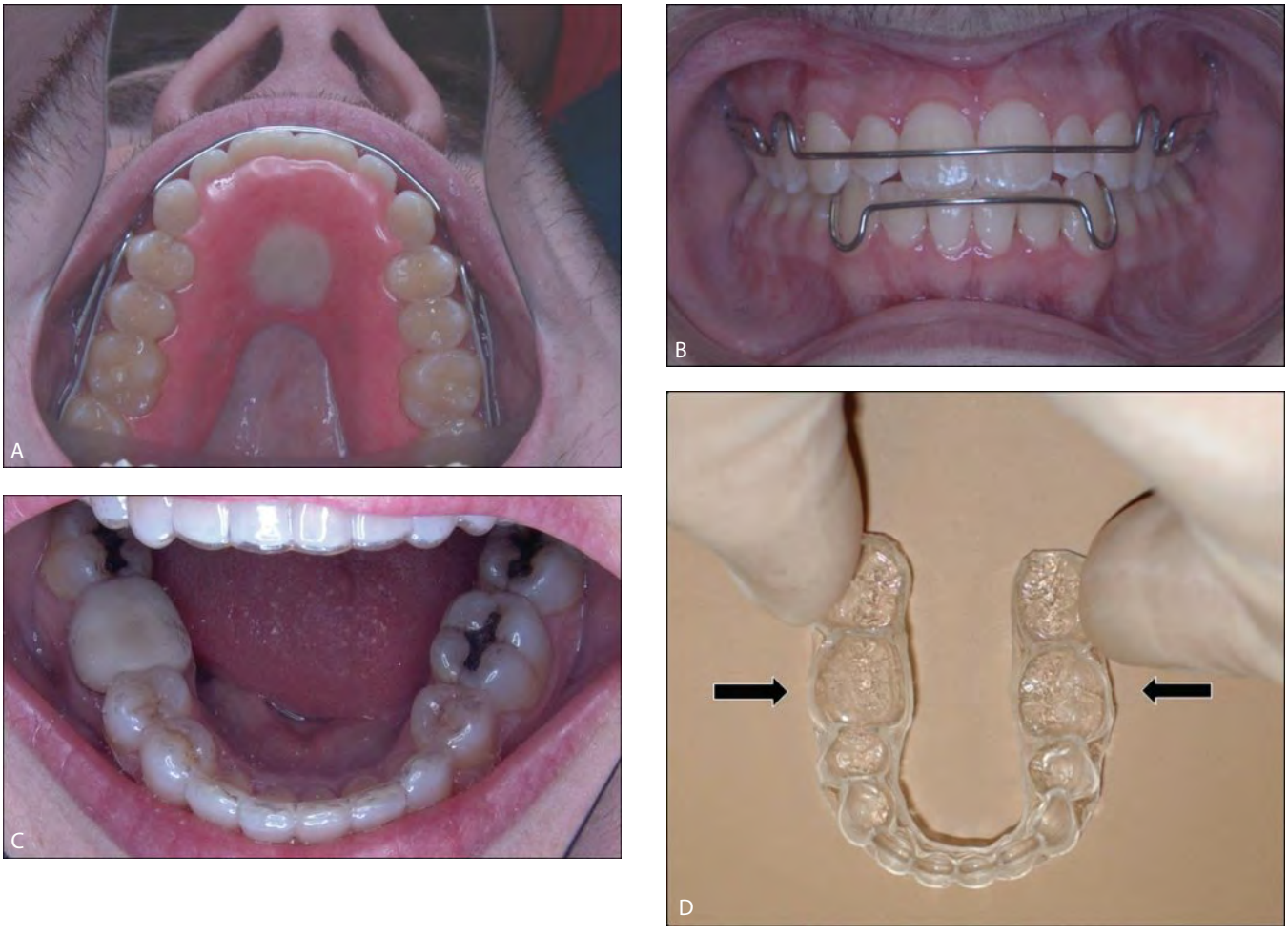


FIGURE 55-42. **A,** For patients who have undergone orthognathic surgery, rigid retainers (Hawley-type) without occlusal coverage, are recommended to provide transverse support to the width of the maxillary dental arch as well as maintain the dental alignment. **B,** These retainers should allow maximal interdigitation of the occlusion. **C,** Occlusal coverage retainers should not be used because of vertical separation of the occlusion as this can create a malocclusion. **D,** Occlusal coverage retainers are usually flexible and do not provide any transverse stability resulting in transverse relapse in cases of surgical or orthodontic expansion or narrowing of the dental arches that can result in anterior open bite and posterior cross-bite relationships.

dental extrusions or intrusions. Transverse collapse of the maxillary dentition will usually cause posterior cross-bites and dental interferences as well as an anterior open bite. Transverse stability is paramount to provide predictable treatment outcomes. Also, inappropriate orthodontic closure of spaces created for correcting TSDs is another orthodontic factor contributing to unwanted postsurgical occlusal changes. Performing careful stable orthodontics, providing good surgical results, no indiscriminate closing of spaces, and appropriate retention, should prevent these types of problems, providing that the TMJs are healthy and stable.

Postsurgical orthodontic patient management is a very important aspect of the treatment for providing optimal patient outcomes. Immediate postsurgical orthodontics should be approached aggressively to maximize high-quality occlusal results in the shortest time frame. However, orthodontic appliances should remain for a minimum of 4 to 6 months postsurgery to get through the primary bone healing phase. This will help minimize orthodontic and surgical relapse potential. Splints play an important role in providing stability for the segmentalized maxilla at surgery and postsurgery. Splints can usually be maintained in position for 1 month or longer for large arch expansions.

The TMJs are a vital component of orthognathic surgery but are often ignored. Failure to diagnose and properly treat TMJ conditions can result in orthodontic and orthognathic surgery relapse, unsatisfactory results relative to functional and aesthetic outcomes, as well as pain and headaches. Proper management of TMJ pathology can provide highly predictable treatment outcomes with usual significant reduction in pain and improved jaw function.

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CHAPTER

Model Surgery and Virtual Planning for Orthognathics

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INTRODUCTION

Traditional model surgery is one of the first examples of preoperative surgical simulation and template fabrication widely used to guide orthognathic surgical movements. The complex three-dimensional movements of the maxilla, mandible, and chin achieved with orthognathic surgery necessitate the significant precision that can be obtained through this process, if care is taken when performing each sequential step. Models are used throughout the course of the patient's treatment, beginning with the pretreatment planning stage, proceeding to an immediate preoperative analytical model surgery, and ultimately resulting in splint construction that is transferred intraoperatively to the orthognathic procedure. The diagnostic information gained from the pretreatment clinical facial and dental measurements, radiographic assessment, and model analysis is integrated to establish a treatment plan. The articulated anatomically mounted models can be utilized in this pretreatment planning stage. These help in the determination of the type of surgery needed and can direct the presurgical orthodontic movements and decompensations.

Presurgical records that provide the ability to surgically simulate surgery include dental impressions, a bite registration with the patient in centric relation (CR), a facebow transfer, and facial measurements made from a standardized clinical examination. Standardized clinical photos and a standardized cephalometric film are also obtained, but their analysis and use are addressed elsewhere in the text. The dental models are placed on a semiadjustable articulator using the CR bite registration and facebow transfer. The treatment plan is expressed in the model surgery that simulates the proposed surgical changes. These models are used to fabricate the occlusal wafers (splints) that facilitate jaw positioning during the actual surgery.

Advances in technology have begun to revolutionize the preparation and performance of orthognathic surgery. Imaging and software innovations have brought fully computerized three-dimensional treatment planning, virtual dental models, virtual simulated surgery, and computer-assisted manufacturing of surgical splints or custom onlay implants. A virtual three-dimensional model of the patient can be created and interactive software can be used to provide preoperative evaluation and treatment planning as well as simulated surgery and splint fabrication Virtual surgical planning and splint manufacturing is not always indicated for straightforward routine orthognathic cases, but these advances currently lend themselves well to the correction of complex facial asymmetry cases.[1] Outcome studies on the accuracy of virtual surgical simulation in orthognathic surgery and its cost-benefit analyses continue to be explored.

TRADITIONAL IMMEDIATE PREOPERATIVE ANALYTICAL MODEL SURGERY

Presurgical Clinical Database

The clinical examination of the patient is the first, and most important, step in the orthognathic surgical workup. A comprehensive variety of measurements are obtained that can characterize the patient's skeletal deformity. These measurements reflect not only the position of the maxilla and mandible but also help identify the symmetry of other facial structures. The clinical measurements used to identify the three planes of space—transverse, vertical, and anteroposterior (AP)—can be categorized into those measured at facial frontal view, facial lateral view, and oral examination (Figure 56-1). A small millimeter ruler is used to make most linear measurements and an angle ruler can be utilized for angle measurements.

The transverse measurements include the evaluation of the midlines—relating the facial midline to the maxillary dental midline, the maxillary midline to the mandibular

Orthognathic Physical Examination Database			
Transverse	**Date:**	**Date:**	**Date:**
Maxilla to face			
Mandible to maxilla			
Chin to maxilla			
Occlusal plane			
Mandibular angles			
Arch width			
Vertical			
Crown length			
Upper lip length			
Upper lip—resting			
Upper lip—speech			
Upper lip—smiling			
Open bite/overbite			
Anteroposterior			
Overjet			
Nasolabial angle			
Nasal contour			
Labiomental fold			
Chin			

FIGURE 56-1. Clinical orthognathic physical examination database.

midline, as well as the chin point to the maxilla. In patients with a notable deviation of their nasal structure or in those with hemifacial asymmetries, there will be added complexity in evaluating midlines. In these instances, utilizing a glabellar mark with a skin marker and holding a perpendicular plumb line from this point will help to measure facial and dental midlines. Occlusal cant is measured at both the maxillary canines and the first maxillary molars. It is quantified by measuring from each orbital rim or medial canthus to the tip of the maxillary canine on the same side. The difference between the two sides defines the cant (e.g., 1.5 mm down at the left maxillary canine). Whereas occlusal cant is often found in the maxilla with mandibular adaptation, there may be an isolated mandibular cant in rare instances. Another measurement of the asymmetry in the transverse plane is assessment of the symmetry of the left and right mandibular angles as measured from the most lateral aspect of the infraorbital rims.

The measurement of maxillary and mandibular arch widths is accomplished through the oral examination and on study models. In areas in which a tooth cross-bite exists within the mouth, hand articulating the study models will reveal whether this represents a true arch width discrepancy or is merely a reflection of the relative skeletal discrepancy manifested by the position of the mandible or maxilla. When a unilateral cross-bite is observed clinically, more often than not the cross-bite is actually bilateral, but the mandible slides to one side upon closure to achieve better interdigitation of the teeth. The examiner should carefully manipulate the mandible to a seated condylar position and then close the teeth together to determine where the first point of contact occurs. This can be crucial in determining whether the mandible is truly asymmetrical or has deviated from a bilateral end-to-end occlusion to a unilateral full cross-bite in "centric occlusion." This assessment can also be made on carefully mounted models.

The maxillary central incisors are key to treatment planning in orthognathic surgery. Their preoperative position must be assessed when the patient is smiling, speaking, and most important, in repose. In addition, any additional gingival show must be noted and quantified. Open bite in the area of the central incisors must be measured preoperatively as well as the length of the upper lip. Overbite, positive or negative, should be noted pretreatment and after orthodontic decompensations. The importance of this evaluation is to detect any orthodontic closure of a pretreatment open bite that may relapse after completion of all treatment. In addition, the nasolabial angle and the labiomental fold often help assess the soft tissue contour that accompanies the jaw relation discrepancies. One must also be mindful of the nasal contour while treatment planning upper jaw procedures. Typically, maxillary advancements or impactions widen the alar base and elevate the nasal tip. Concomitant procedures can be performed to correct significant nasal functional and aesthetic concerns such as osseous recontouring, alar cinch, turbinate reduction, and septoplasty.

The position and structure of the chin play a major role in the final aesthetic perspective of most patients. Although model surgery may or may not reveal the final position of the chin, it will predict the final position of "B" point, which can then be used with the cephalometric analysis and prediction tracing to predict final chin position. Thus, a preoperative assessment of the chin position at baseline is important to assess the need for change.

Presurgical Records

Dental Impressions

Dental impressions are obtained with alginate at the final presurgical records appointment. They must be obtained after all preoperative orthodontic tooth movement is complete and the surgical stabilizing arch wires have been in place for several weeks and are passive. Recently placed surgical arch wires cause tooth movement, which may continue to take place after the impressions are obtained. Any tooth movement that occurs after the impressions are made will lead to inaccuracies in how well the splint fits intraoperatively. The dental impressions must include the occlusal surfaces of each of the teeth and be without voids or alginate tears. The impressions are poured up using dental die stone and a dental vibrating platform for a hard and precise model. Indentations may be placed within the base of the cast in order to allow for future separation and re-indexing of the cast from its plaster mounting.

Facebow Transfer

The transfer of the maxillary cast to the articulator by using a facebow (Figure 56-2) gives a reliable estimation of the distance between the dentition and the intercondylar hinge

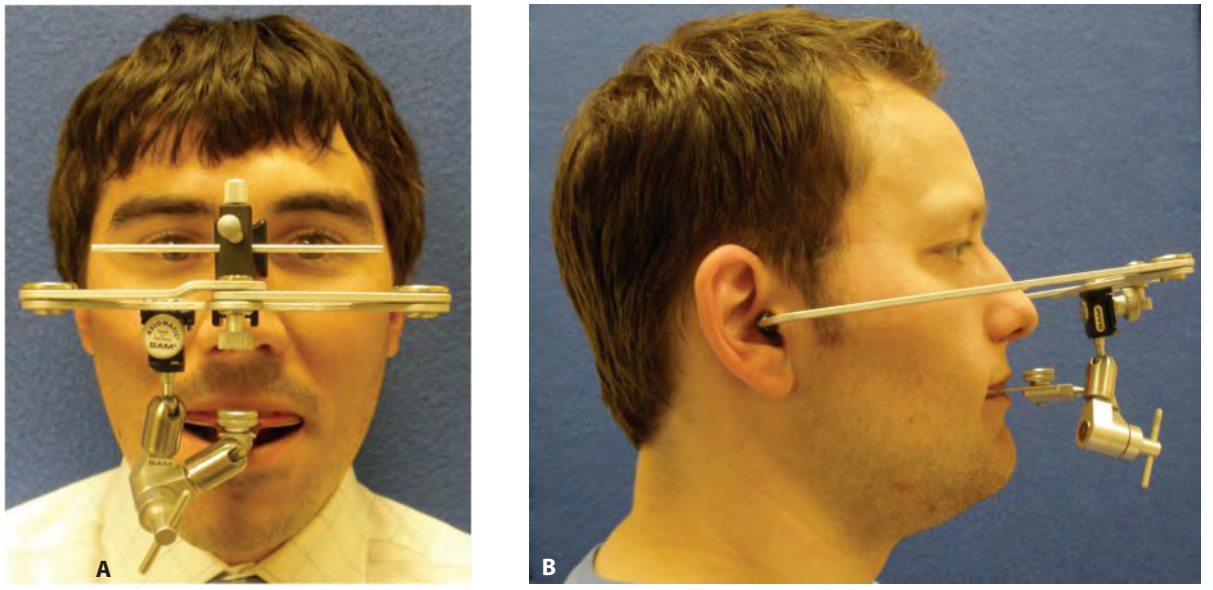


FIGURE 56-2. **A,** The Student Articulator of Munich (SAM) facebow is used to relate the maxillary occlusal plane to the axis-orbital plane (AOP). **B,** The AOP is formed by the intercondylar axis of the mandible and the lowest point of the inferior orbital rim known as the *orbitale*. This plane is approximately 7 degrees different from true Frankfurt horizontal (FH). This often results in cases in which the maxillary occlusal plane is too steep when mounted.

axis. This is important when vertical movements of the maxilla and/or mandible are planned, because autorotation changes the position of the jaws in both vertical and horizontal dimensions. The more accurately the maxillary model is mounted with respect to the true hinge axis, the more accurate will be the information provided about the horizontal and vertical movements of the jaws during model surgery. Traditionally, a facebow device is used to register the three-dimensional relationship of the maxillary dental arch to the Frankfort horizontal plane (FHP) using either the patient's external auditory meatus or the condylar heads (depending on the requirements of the facebow) as the posterior reference.

It is universally accepted that the upper arm of the articulator represents Frankfort horizontal (FH). However, this assumption can introduce error. The semiadjustable articulator used for model surgery was originally created for use in prosthetic dentistry. Its facebow was designed to transfer the relationship of the maxilla to the terminal hinge axis of the mandible. To accomplish this, the posterior end of the facebow was aligned to the terminal hinge axis (middle of the condyle), and the anterior end was aligned to orbitale. These points defined a plane called the *axis-orbital plane (AOP)*, which is 7 degrees off from FH. Modifications have been made for surgical cases to minimize this error. Facebows equipped with an adjustable nasal rest and an infraorbital pointer provide an additional point of reference. The pointer arbitrarily lowers the anterior part of the facebow below orbitale by 6.8 mm, thereby reducing the inclination of the occlusal plane. This facebow registration is transferred to the articulator to position and mount the maxillary dental model.

There are other reasons for the inaccuracy in the traditional facebow mounting of dental models[2]: (1) the position of the patient's external auditory meatus or mandibular condyles (depending on the reference for the facebow) may be asymmetrical from side to side compared with the fixed symmetrical position of the facebow mounting rods on the articulator; (2) owing to anatomic variances, the patient's FHP as determined by the facebow may be significantly different than the fixed FHP of the articulator; (3) the facebow may be improperly positioned on the patient, or facebow components could shift when tightening the bolts, nuts, and/or screws during the registration procedure; (4) cranial base and jaw aberrations may be present that are not reproducible on the articulator; (5) anatomic structures may be absent (i.e., hemifacial microsomia), rendering the facebow mounting totally arbitrary; and (6) shifting of the facebow components can occur during the mounting of the maxillary model. Despite these limitations, a carefully obtained and clinically correlated arbitrary facebow is essential to mount the models correctly without compromising the patient's surgical results.

CR Bite Registration

The mandibular dental model is then mounted with a CR bite registration. This registration is usually obtained with either wax or polyvinylsiloxane (PVS; Figure 56-3). An accurate registration of the relationship between the maxilla and the mandible *independent of the occlusion* must be obtained for proper mounting and model surgery to be carried out. This has historically been termed *centric relation (CR)* and a record of CR can be obtained in a number of manners. The definition of CR, however, is controversial and frequently misstated. The definition of CR underwent a change in terminology in 1987 from a mandibular position in which the condyles are seated in the most "posterior



FIGURE 56-3. Centric relation (CR) bite registration in wax and in polyvinylsiloxane (PVS).

superior" position to one in which they are placed in the most "superior anterior" position in the glenoid fossa. The most recent Academy of Denture Prosthetics' "Glossary of Prosthodontic Terms" (GPT) from 2005 defines CR as a maxillomandibular relation in which the condyles articulate with the thinnest avascular portion of their respective disks with the complex in the anterosuperior position against the slopes of the articular eminences.[3] It is essential to locate a mandibular position that is reproducible (without the assistance of the patient) and that can be reliably accomplished both preoperatively and intraoperatively. This position must also be transferable to the articulator in order for model surgery planning to be accomplished accurately. If an inaccurate bite registration is obtained from the patient preoperatively, this will be transferred to the articulated models, leading to inaccurate model surgery and resulting in intraoperative error. Obtaining an accurate and reproducible bite registration in CR is essential if the surgeon is to avoid this error.

The clinical methods to obtain a correct CR registration vary.[4] The "chin point guidance" procedure involves the patient opening wide while the interocclusal record material is applied over the occlusal surfaces of the mandibular teeth. The surgeon then gently manipulates the chin and guides the mandible closure until the mandibular incisors have encountered the material and the patient is held in this position until the material has set.

The effects of the material used to record CR (i.e., wax) have also been considered as a potential source of introducing error. The error introduced is directly proportional to its thickness and it is suggested that the wax between the distal molars should be as thin as possible. The wax should not be punctured and should retain its rigidity to avoid distorting contacts.

Mounting Dental Models for Simulated Surgery

Dental articulators allow the surgeon to measure surgical moves and carry out multidimensional surgical simulation. Semiadjustable hinge axis articulators commonly used by oral and maxillofacial surgeons and orthodontists permit visualization of the dentoalveolar units, their relationship to one another, and to a limited degree, the position of these structures relative to other parts of the craniofacial skeleton. Typically, the upper member of a semiadjustable or fully adjustable articulator is said to represent one of the cephalometric planes (i.e., FH), and the intercondylar distance is set to approximate that measurement in the average adult. The diagnostic utility of the articulator is enhanced when predictive mock surgery is carried out on mounted dental casts.

In order to ensure that the articulator is calibrated correctly, it is appropriate to periodically calibrate the instrument with a split cast check. To perform the split cast check:

1. Set the horizontal condylar inclination at 30 degrees and the Bennett side shift at 5 degrees (average setting) on each side.
2. The incisal pin should be at the zero mark or less.
3. Attach the metal portion of the split cast to the lower member and attach the plaster portion of the split cast to the upper member (Figure 56-4).
4. The two portions of the split cast should fit together without any discrepancy (Figure 56-5).
5. If these two parts do not fit together, make sure the incisal pin is out of contact with the incisal table and check the articulator for wax or plaster debris under the split cast or in the condylar housing area.

Mounting the maxillary cast using facebow transfer (Figure 56-6) requires that the angle between the occlusal plane of the maxillary model and the upper member of the articulator are the same as the occlusal plane angle. If the maxillary model is mounted at a different angle, the intermediate splint utilized in combined maxillary and mandibular cases will convey an aberrant position during surgery. Comparison of the maxillary plane angle (once the facebow is attached to the articulator) to patient's cephalogram may be required in the laboratory when mounting the maxillary cast. Accurate mounting of the mandibular cast using a CR interocclusal registration is mandatory.

The base of the mounting and the cast are best prepared for separation by application of a layer of separating media



FIGURE 56-4. Calibration of a SAM articulator with a split cast. The metal portion of the split cast has been attached to the lower member and the plaster portion has been attached to the upper member.

at the time of mounting. They also may be made in two separate colored stone products. For instance, the model may be poured in green stone and the mounting in white dental plaster. The base must be smooth and parallel to the base of the cast so that measuring marks can be made easily and smoothly. Trimming of maxillary cast should be parallel to the AOP. The incisal guide pin setting is recorded after mounting.

Marking and Measuring Final Models and Surgical Simulation

Mandibular Surgery

Isolated Mandibular Surgery (Sagittal Split Osteotomy or Vertical Ramus Osteotomy only)

Once the models have been correctly mounted, they are marked with points for measuring. When mandibular



FIGURE 56-5. The two portions of the split cast should fit together without any discrepancy.

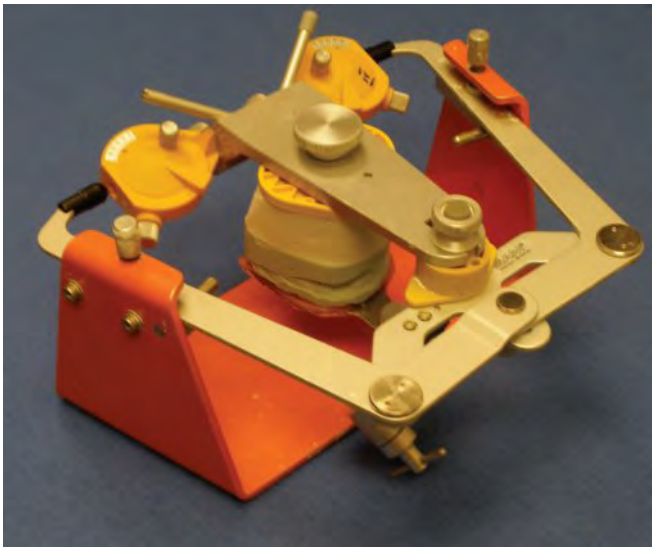


FIGURE 56-6. Mounting the maxillary model on a SAM articulator using a facebow transfer.

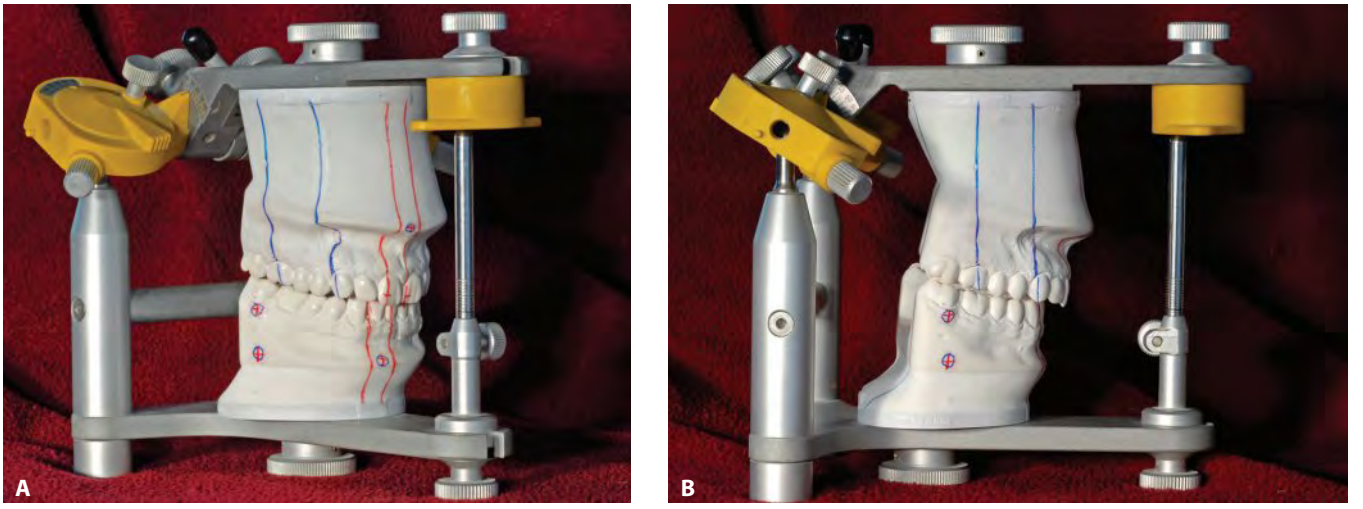


FIGURE 56-7. **A** and **B,** Mandibular models marked for surgery with representative points made on the mandibular cast at the region of the vertical corticotomy or Dal Pont and at the mandibular incisors and genial region.

surgery alone is planned, the mandibular anterior, posterior, or rotational movements are dictated by the maxillary dentition. Because of this, many surgeons use a simple hinge (nonadjustable) articulator to mount the final models. Although the maxillary position will remain constant throughout surgery, there are several benefits to using a semiadjustable articulator and facebow mounting even in straightforward cases. This provides the surgeon with information regarding the distance of the surgical move at the osteotomy site and possible proximal segment positioning challenges, especially when correcting large mandibular asymmetries.

In cases of isolated mandibular surgery, representative points are made on the mandibular cast at the region of the vertical corticotomy or Dal Pont and at the mandibular incisors and genial region (Figure 56-7). These points in the Dal Pont should be spaced as widely as possible in order to glean the most information about the move. Vertical lines are made at the posterior of the cast descending from the retromolar pad inferiorly.

There are two options to obtain the preoperative measurements now that the models are marked. One way utilizes an Erickson model platform (Great Lakes Orthodontic Products, Ltd., Tonawanda, NY), which is an orientation block with an attached caliper that allows for marking and measuring models to an accuracy of 0.01 mm. With the model secured on a model block, the platform and caliper allow for preosteotomy and postosteotomy measurements in an AP dimension and a vertical dimension (Figure 56-8). This technique eliminates the parallax error that may be introduced by use of the second measuring option, the freehand ruler technique measuring AP to the articulator's pin (Figure 56-9).

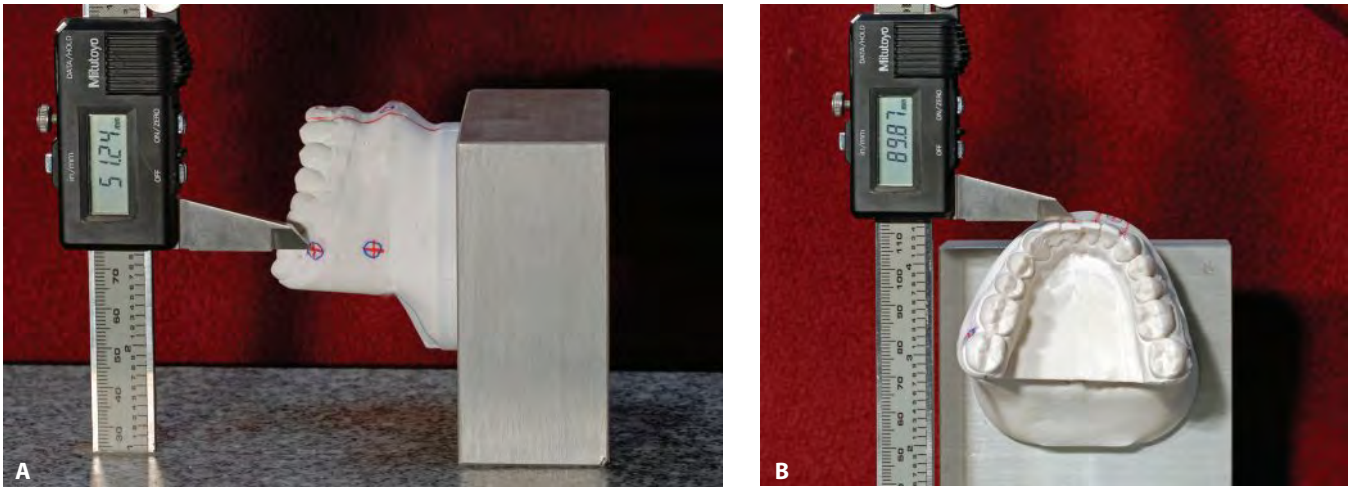


FIGURE 56-8. **A** and **B,** The use of a model platform with the model secured on a model block allows for preosteotomy and postosteotomy measurements in an anteroposterior dimension. Precise thin cross-hatch marks are made on the mandibular cast at the region of the site of the mandibular osteotomy, central incisors, and genial region.



FIGURE 56-9. Utilizing the freehand technique to measure anteroposterior distance at the inferior border of the anticipated right Dal Pont osteotomy. Parallax error is introduced when viewing the same object (in this case the markings on a ruler) from different places.

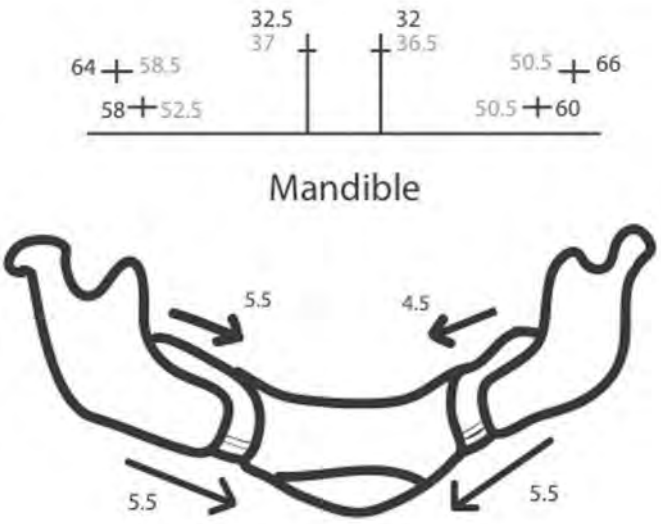


FIGURE 56-10. Mandibular roadmap for an isolated mandibular bilateral sagittal split osteotomy (BSSO) planned for 5.5-mm advancement on each side. The *numbers denoted in black* are made before the surgical move and the *numbers in gray* are made after remounting the mandible in its prescribed final occlusion. The differences between the two numbers reflect the surgical move at that point.

Parallax error results when viewing the same object (in this case, the markings on a ruler) from different places. Attempts to minimize this error include placing a straight black line on the pin platform (Figure 56-9) and measuring consistently to the anterior portion of the pin while viewing from a position at which the black line becomes a point (perpendicular to the ruler). The measurements obtained are recorded onto a laboratory data sheet (orthognathic roadmap) that can be taken to the operating room for reference (Figure 56-10).

The mandibular model can then be separated from its original cast and repositioned to the planned occlusion. Setting of bite should take into consideration the surgery that will be done (bilateral sagittal split osteotomy [BSSO] or intraoral vertical subcondylar ramus osteotomy [IVRO]) and should involve close communication with the patient's orthodontist. The mandibular model is secured to the maxillary with hot glue or sticky wax. The model is then remounted with plaster and a final acrylic surgical splint is fabricated. Postoperative measurements can then be obtained that will inform the surgeon of the distance the mandible is being advanced or set back on each side (the surgical move). In patients in whom an asymmetry is being corrected, a view of the models from a posterior vantage point will help identify regions in which proximal and distal segment interferences can be anticipated (Figure 56-11).

Maxillary Surgery

Isolated Maxillary Surgery

Pretreatment model surgery is essential when contemplating maxillary surgery alone and very useful when planning two-jaw surgery. Pretreatment model surgery permits the evaluation of the maxilla and the mandible whether the mandible is autorotated without surgery or also osteotomized. If the goals of the surgery can be accomplished with a single-jaw surgery, the orthodontic preparation and patient's expectations can be influenced appropriately.

In this scenario, the mandible will serve as a template for final position of the maxilla. A clinical example would be a single-piece Le Fort I maxillary osteotomy for correction of posterior maxillary excess resulting in a skeletal class I open bite malocclusion. The models are mounted as previously discussed. A semiadjustable articulator is strongly recommended for these cases. The advantage of using such an articulator lies in its simulation of the appropriate arc of autorotation of the mandible when properly mounted. When performing isolated maxillary impaction, the incisal pin will be shortened.

Marking the midline is the first step in marking models. This can be done by making lines parallel to the articulating

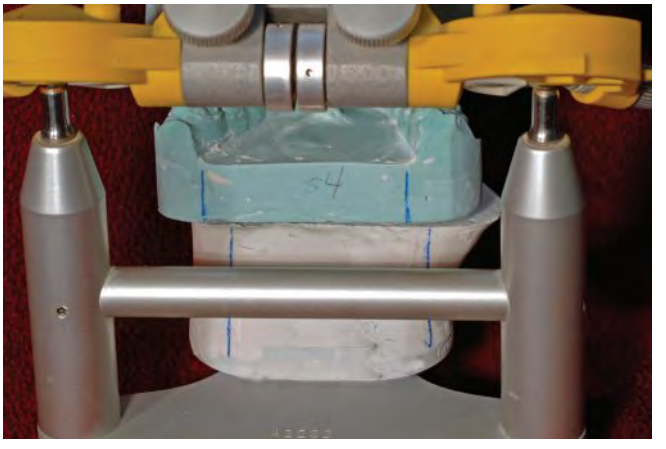


FIGURE 56-11. A view of the mandibular model from a posterior vantage point after surgical move will help identify regions in which proximal and distal segment interferences can be anticipated.

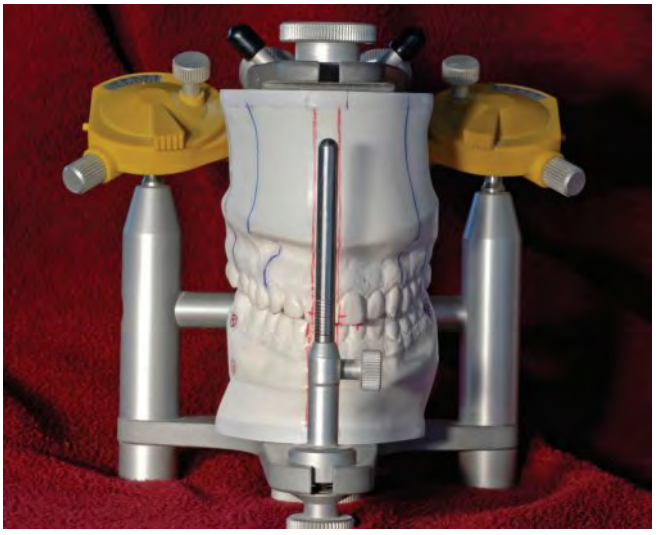


FIGURE 56-12. Red vertical lines are drawn on each side of the midline, using the articulating pin as a guide. The position of these lines is entirely dependent on a correct facebow transfer and the maxillary midline should correlate to the clinical examination.

pin on either side of it from a frontal view, avoiding paralleling errors (Figure 56-12). This step is done on both the maxilla and the mandible mounted together in the presurgical occlusion. A mounted maxillary model requires measurements in the AP, vertical, and transverse planes. This can be accomplished with the maxilla removed from the articulator or while on it. Where the models are measured will depend upon which of the two options is used to measure these distances: the model block or a freehand technique (Figure 56-13). When measuring vertical distances on the model block, it is necessary to "zero" the electronic caliper at the level of the mounting base before obtaining a measurement. The freehand technique uses a caliper for vertical measurements and a ruler for AP measurements. Vertical measures of the maxilla are made at both the central incisors at premarked points, both of the canine cusp tips, and the mesial-buccal cusp tips of the first molars. In addition, the cast should be vertically measured at both tuberosities. AP measurements are then recorded at the central incisors and a point representing the anterior nasal spine on the base of the cast (Figure 56-14). Measurements obtained with the "freehand" technique are made to a point on the pin of the articulator (to which all measurements should be consistently made).

The maxillary cast is now separated from its base and positioned in the proper occlusion with the mandibular cast. In cases of superior repositioning of the maxilla, it will be necessary to remove additional plaster from the mounting to provide clearance for the superior repositioning of the maxilla. A layer of wax or hot glue is added to the gap between the base and the model. Any AP changes are usually limited by the mandibular position. The incisal guide pin is adjusted as needed to provide the proper vertical measurement at the maxillary incisor. Shortening of the incisal guide pin height in maxillary impaction cases will allow the proper arc of autorotation of the mandible to this newly positioned maxilla. The final measurements are recorded (Figure 56-15) and the roadmap is prepared (Figure 56-16). A final splint is made to this occlusion.

Vertical Measurements in Maxillary Surgery

Despite meticulous presurgical planning and model surgery, vertical positioning in maxillary surgery will depend entirely upon intraoperative measurements and will not be ensured from only model surgery or surgical splints. How best to control the positional change of the maxilla has been debated. The unfortunate use of the terms "internal" and "external" reference marks has led to confusion because "internal" has been used to describe an antiquated technique of scribing

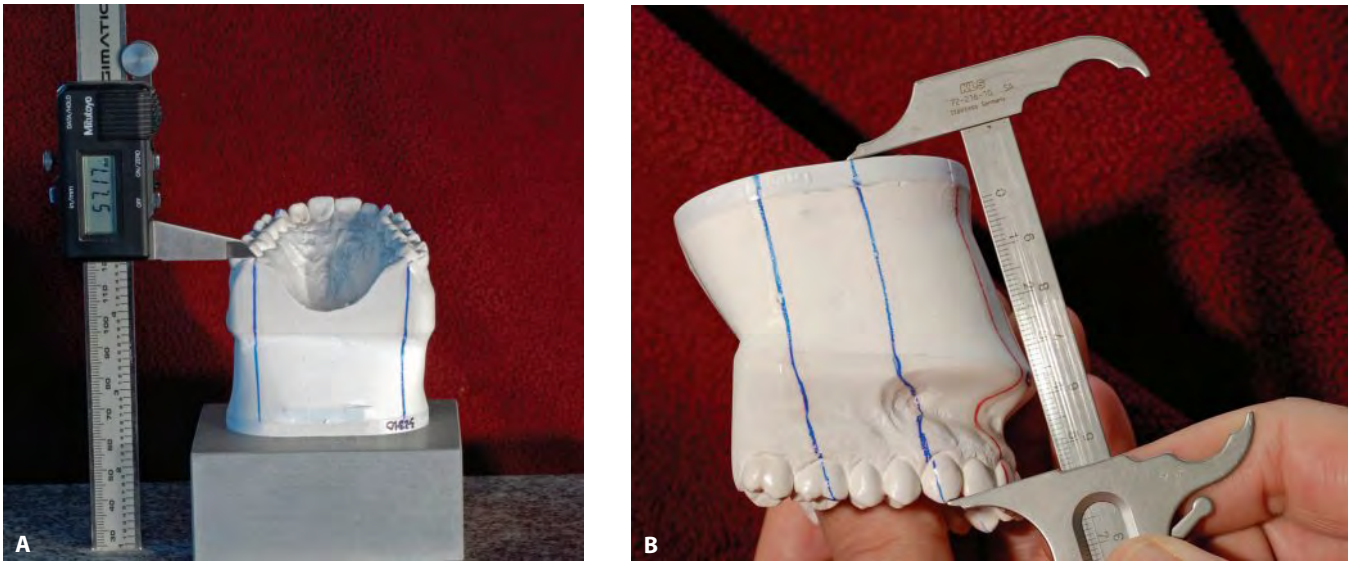


FIGURE 56-13. **A** and **B,** Vertical measurements may be obtained with either a model block **(A)** or a handheld caliper **(B).**



FIGURE 56-14. **A** and **B,** The freehand technique measures anteroposterior distance from a point marked on the maxillary right central incisor to the anterior of the mounting pin.

lines above and below the osteotomy in an attempt to measure the vertical position of the maxilla intraoperatively. The term "external" has come to mean placing a referencing device somewhere outside of the wound above the osteotomy and measuring to the mobilized maxilla, usually the central incisor brackets. But the issue is not whether the reference marks are inside or outside of the mouth or the wound, but rather that they are superior to the osteotomy and are in stable bone. The use of superior bony reference marks (SBRMs), inside or outside of the wound, has proved to be more accurate than scribing lines along the osteotomy. This makes outside of the wound reference marks unnecessary if SBRMs are placed as described later. The technique presented here provides four SBRMs in two bilateral locations. Measurement is made from the SBRM to the gingival crest rather than orthodontic brackets, which can be loosened resulting in the loss of that measuring point.

Method 1: Utilizing four SBRMs for point-to-point intraoperative measurements This method simultaneously employs two different manners of measuring. Preoperatively, the Erickson model block is used for pure horizontal or vertical measurements whereas bilateral "point-to-point" measurements are made with a Boley gauge or calipers that correlate to intraoperative measurements. Premovement measurements at the canine and molar areas bilaterally are repeated postmovement, in a point-to-point fashion, using a Boley gauge to measure vertical change.

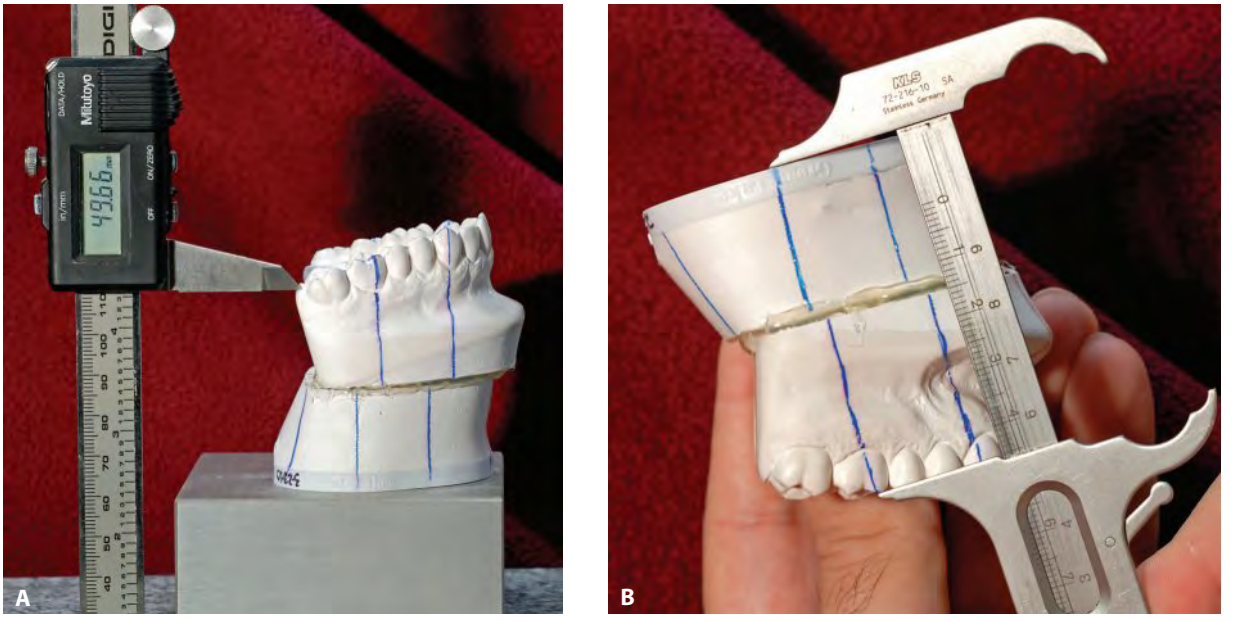


FIGURE 56-15. **A** and **B,** Final measurements are made at the left and right maxillary incisor, canine, and first molar teeth, as well as at the tuberosity and anterior nasal spine region. These new measurements reflect the change in vertical positioning.

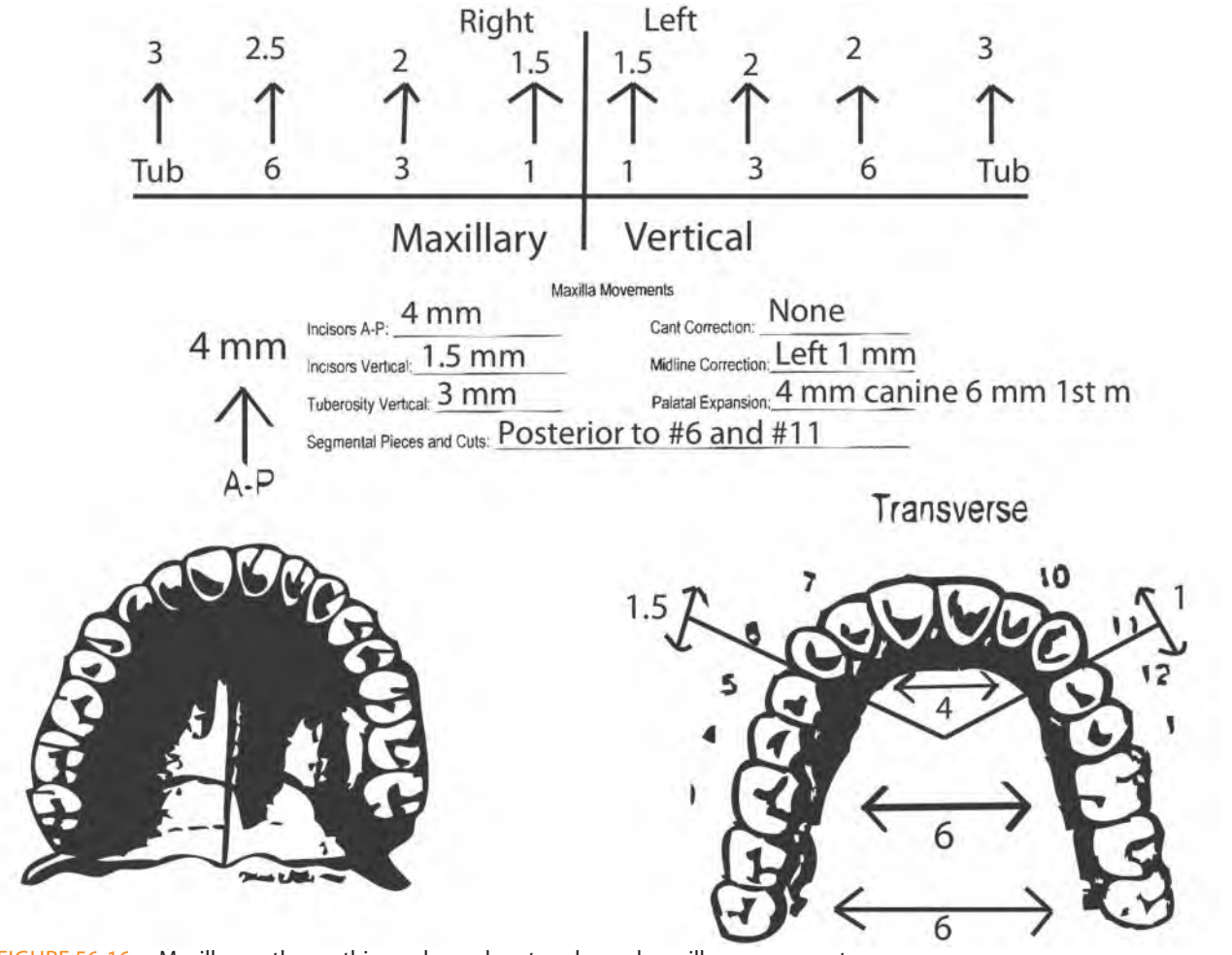


FIGURE 56-16. Maxillary orthognathic roadmap denotes planned maxillary movements.

Intraoperatively, the model surgery measurements are replicated. SBRMs are made bilaterally on the maxilla. These are made well above the planned osteotomy line in the piriform and buttress regions above the canine and molar. The difference in the two measurements sets from the models is utilized intraoperatively, in a point-to-point fashion, with a caliper, obtaining the same difference in the preoperative and postoperative measurements on the maxilla. This point-to-point measurement transferred from the models to the surgical measurements eliminates error encountered as a result of AP movement.

This method does not require a reference marker outside of the surgical wound and gives bilateral information regarding the three-dimensional maxillary moves. The steps necessary for this technique at the time of model surgery are as follows:

1. Mark a stable point on the models above the canine and first molar teeth bilaterally—a total of four stable points (see Figure 56-17, points *A* and *P*).
2. Mark a corresponding point on the canine and molar teeth bilaterally. The incisal edge is usually used. It is not important that this point be the same as that used at surgery so long as it is stable from before model surgical positioning until after (see Figure 56-17, points *B* and *C*).
3. Measure and record the distances from stable point to the tooth point for each of the four pairs (see Figure 56-17).
4. Measure the anterior maxilla from FH to the upper incisor using either the model block or the freehand technique (Figure 56-18).
5. Perform model surgery to the desired three-dimensional position (Figure 56-19).
6. Remeasure all five distances and record the difference between pre- and postmodel surgeries (Figure 56-20).

Note: The *differences* between the pre- and postmodel surgery measurements at the piriform rims and the first molars are the important values to have at the time of surgery, not the absolute values (Figure 56-21). The FH to upper incisor measurement is to place the model at the desired vertical and is not needed at surgery.

The steps completed for this technique at the time of surgery are as follows:

A. Place SBRMs by drilling small holes in the piriform rims and zygomatic buttresses bilaterally well above the planned osteotomy (Figure 56-22A).
B. Measure and record the distance from these holes (SBRMs) to the gingival crest of the canines and first

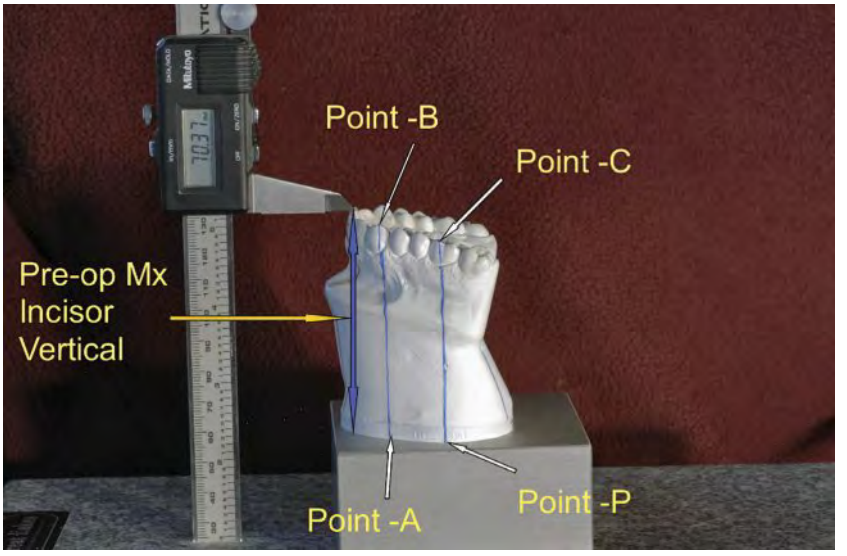


FIGURE 56-17. Measurement of preoperative maxillary vertical at right canine and mesial buccal cusp tip of right first molar with a model block.



FIGURE 56-18. Measurement of preoperative maxillary vertical at the right central incisor with a model block.

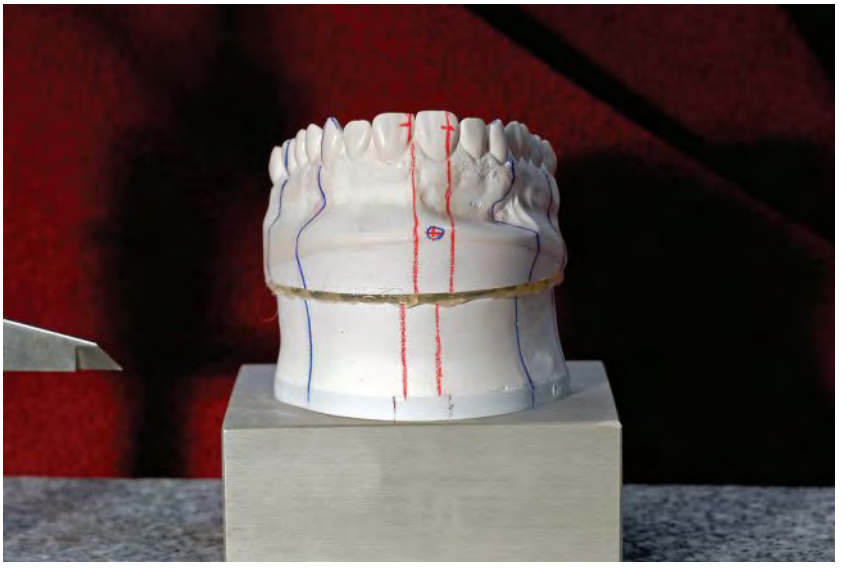


FIGURE 56-19. Cut and repositioned maxillary model placed onto the model block for postoperative measurement.

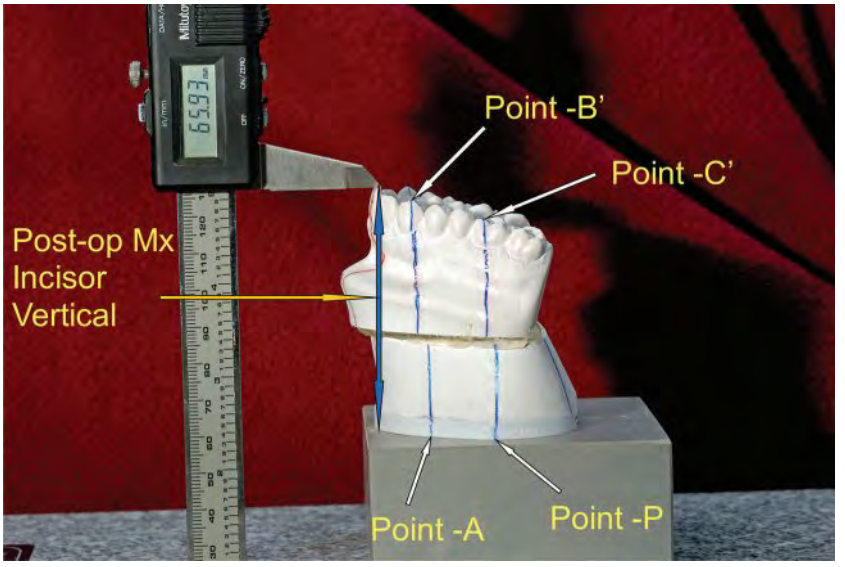


FIGURE 56-20. Postoperative vertical measurements are obtained in the same fashion as was done before cutting and repositioning the maxilla. Here, the new vertical measurements are obtained at the right canine and mesial buccal cusp tip of right first molar with a model block. The differences between the pre- and postoperative numbers should reflect the treatment plan.

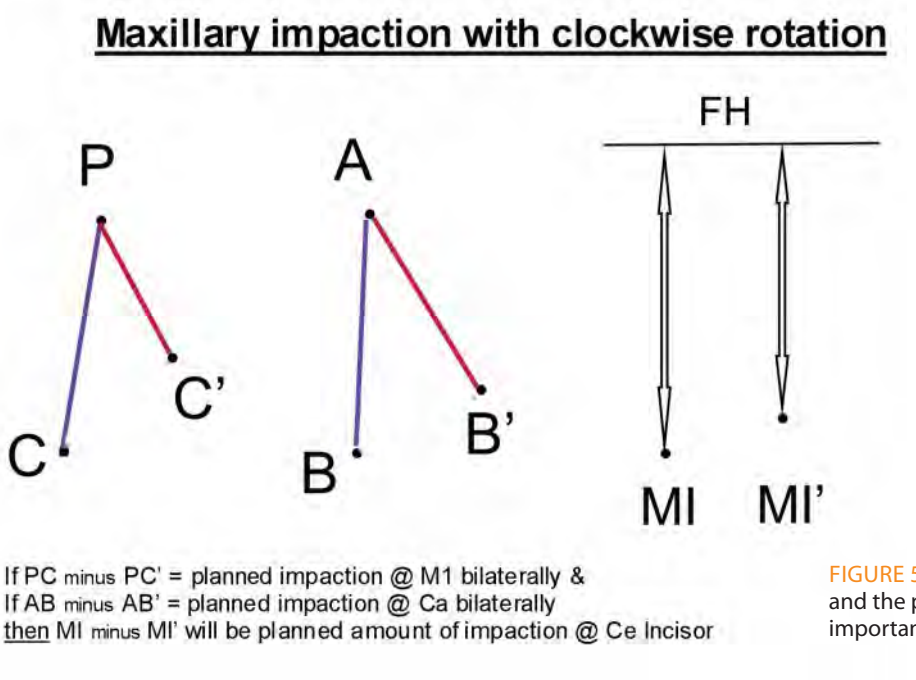


FIGURE 56-21. The differences between the pre- and the postmodel surgery measurements are the important values to have at the time of surgery.

molars bilaterally. Again, it is *not* important that this point on the teeth is the same as the model surgery so long as it is a stable, reproducible point (see Figure 56-22B).

C. After mobilization, position the maxilla so that the four *differences* measured at model surgery match those differences measured intraoperatively (Figure 56-23).

If the maxilla is positioned according to this method, the anterior maxillary vertical will be as planned. No SBRM outside of the wound is needed and the canine and molar vertical positions will also be as planned (see Figure 56-21).

Method 2: Utilizing one midline superior bony external reference mark This external reference point method provides a single measurement to assess proper vertical positioning of the maxilla and the anterior maxillary teeth. All other movements (AP and transverse) are provided from the model surgery and prefabricated surgical splints. After anesthetic induction and patient positioning and preparation are completed, the face is widely draped to allow for visualization of the nose. A 0.062 Kirschner pin (K wire) is placed in the nasal bones at the nasion of approximately 8 to 12 mm depth, and a measurement is obtained from the fixed wire to the maxillary central incisor orthodontic bracket before maxillary mobilization with a Boley gauge

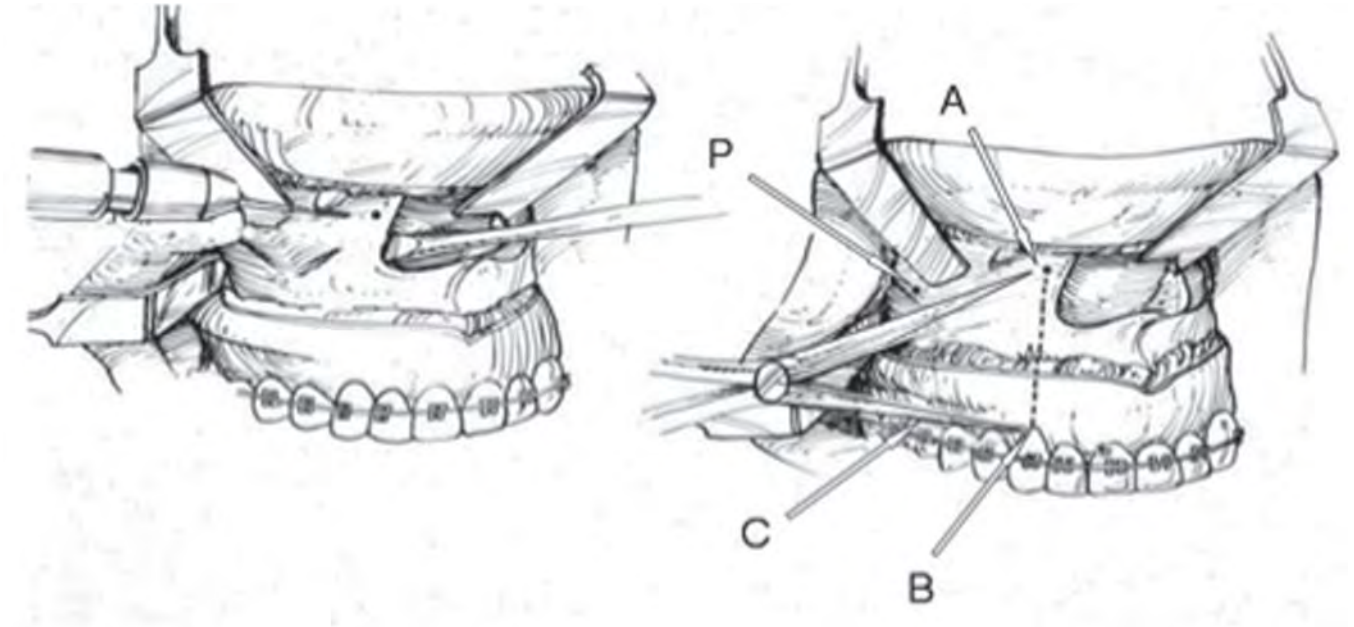


FIGURE 56-22. Intraoperative placement of superior bony reference marks (SBRMs) by drilling small holes in the piriform rims and zygomatic buttresses bilaterally well above the planned osteotomy with a 703 straight fissure bur.

or Perkins device. Measurements are then repeated after the osteotomy and positioning of the maxilla until the planned change in vertical is achieved. The use of external reference points is also an accurate and predictable technique to measure and confirm vertical maxillary position.

Segmental Maxillary Surgery

When performing model surgery for a segmental maxillary procedure, differences in the workup begin with the obtaining dental casts. It may be desirable to have more than one maxillary cast to test the planned segmentalization to

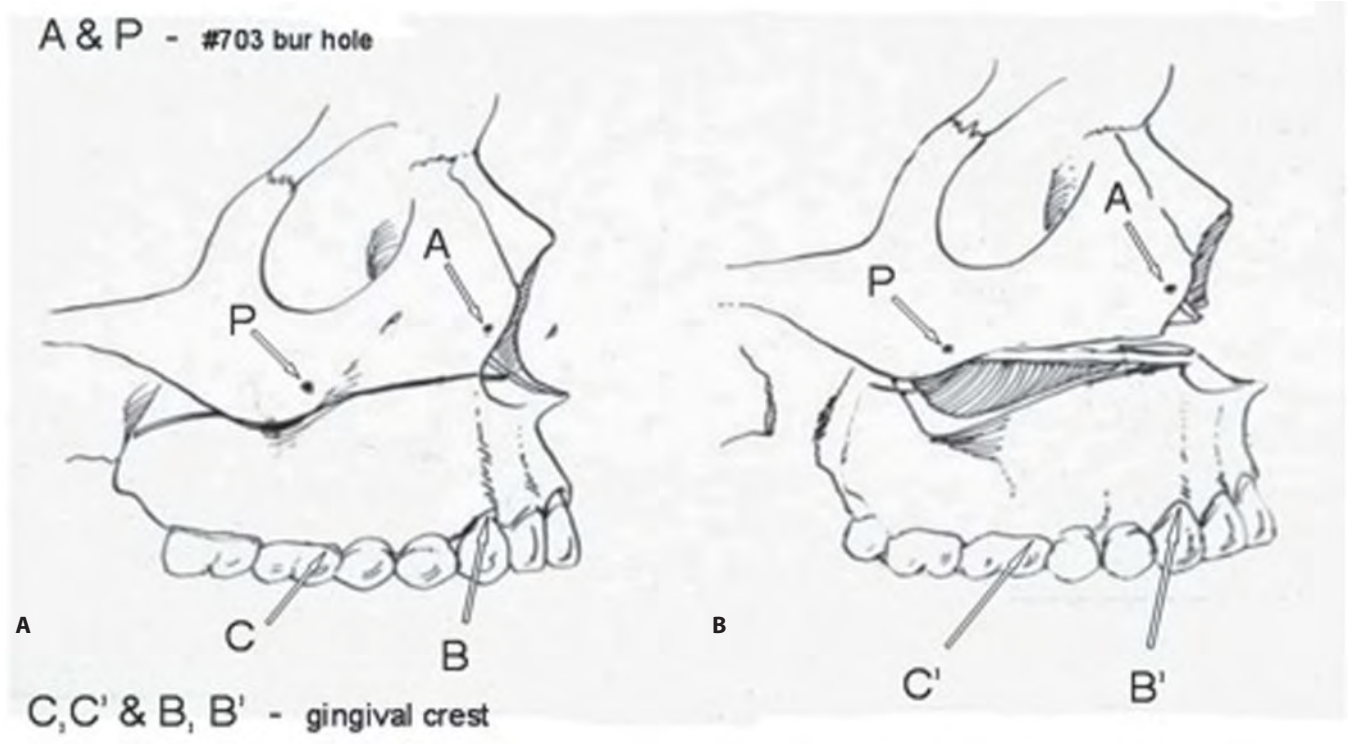


FIGURE 56-23. **A** and **B,** After mobilization, the maxilla is positioned so that the four *differences* measured at model surgery match those differences measured intraoperatively.

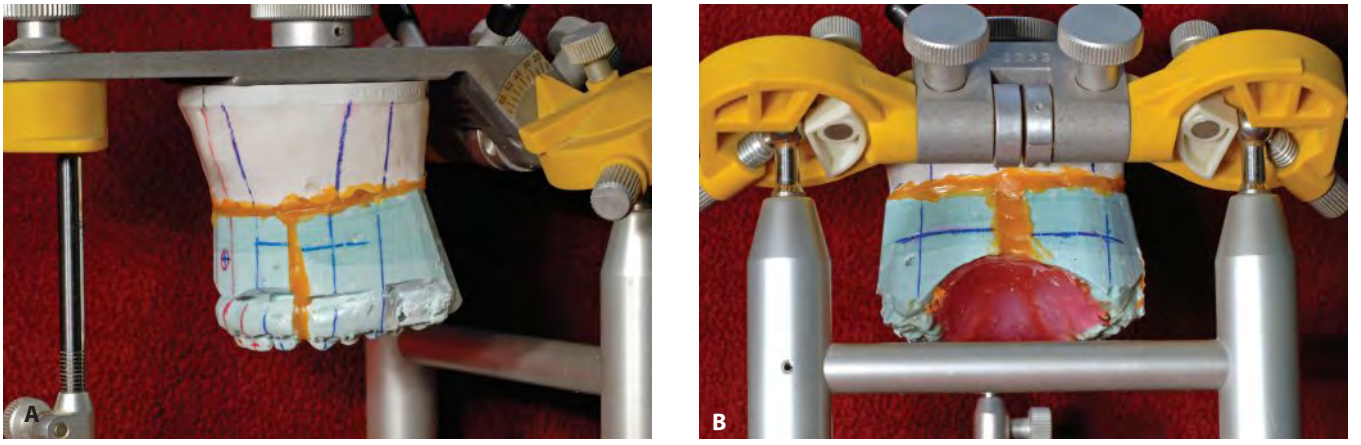


FIGURE 56-24. **A** and **B.** Additional markings placed on the maxillary cast for segmental surgery. Vertical lines should be drawn on both sides of the planned interdental osteotomy. Because segmentalization is often done on either side of the canine, some of these lines will already be present, but a line at the tooth on the opposite side of the interdental osteotomy must also be placed. In addition, horizontal lines should be placed across the posterior of the cast between the vertical tuberosity lines and also between the vertical lines at the interdental cuts. These lines are used for points of measurement and also to aid in appreciation of any tipping of the model of vertical changes in these areas.

determine whether the surgical cast should, in fact, be sectioned in a particular manner. Periapical radiographs should be made of the interdental osteotomy sites.

Marking and measuring of the surgical cast also has some variation (Figure 56-24). The casts are marked and measured as any other maxillary cast plus some additional markings and measurements. Vertical lines should be drawn on both sides of the planned interdental osteotomy. Because segmentalization is often done on either side of the canine, some of these lines will already be present, but a line at the tooth on the opposite side of the interdental osteotomy must also be placed. In addition, horizontal lines should be placed across the posterior of the cast between the vertical tuberosity lines and also between the vertical lines at the interdental cuts. These lines are used for points of measurement and also to aid in appreciation of any tipping of the models or vertical changes in these areas.

Transverse measurements are made with a Boley gauge at the intersection of the vertical and horizontal lines at the backside of the model, at the mesiobuccal cusps of the first molar, and at the canine. If there is an interdental cut behind the canine, this measurement should be made at the premolar. Additional measurements are made at the cusps of the teeth on either side of the interdental osteotomy and at the roots (the intersection of the vertical and horizontal lines at the interdental cut). Knowing the measurement at the cusp and root of the teeth is important to ensure that the planned surgery will not impinge upon the tooth roots. Even with separation at the cusps, the roots could converge with certain tipping movements.

After all marking and measuring is complete, the maxillary cast is sectioned in the areas of the interdental osteotomy. This is done with the thinnest diamond disk available and, in the area closest to the teeth, is best done by just scoring the cast with a #15 blade and breaking this last bit of the cast along the score (after sectioning the remainder of the cast). This will prevent any removal of stone in areas of the teeth and creating an unrealistic simulation of the surgery. Once the cast is sectioned, the next step is establishment of the maxillary arch form

The proper maxillary arch form is the set on the mandible. Feasibility of the planned movements is determined by comparing the postmovement measurements to the pre sectioning measurements described previously. Once the maxillary arch form is set, the remainder of maxillary moves can be done is the same fashion as for a nonsegmental surgery.

In a single-jaw case, the final interocclusal splint with a transpalatal strap (which is sufficiently relieved so as not to impinge on the palatal mucosa and blood supply) is fabricated (Figure 56-25). Alternatively, a palatal style splint can be fabricated, without occlusal coverage, and a separate interocclusal splint can be fabricated to position the remainder of the maxillary movements. In a two-jaw orthognathic case, which involves segmental maxillary surgery, the final splint must be made first in this manner. It must be trimmed and polished to completion. Because this splint will be wired into place on the maxilla and left, postsurgically, for a period of time, the intermediate splint is made as a "piggy-back" splint or a "splint within a splint." That is, the upper surface will articulate with the final splint in the final maxillary position, and the lower surface will articulate to the uncut maxilla. The highly polished final maxillary splint must be adequately lubricated to prevent curing to the intermediate splint.

Combined Maxillary and Mandibular Surgery

Examination and orthognathic workup of a patient will often reveal a skeletal deformity in both the maxilla and the mandible. If the maxilla is the first jaw to be mobilized in combined surgery, the following steps are carried out in the model surgery.

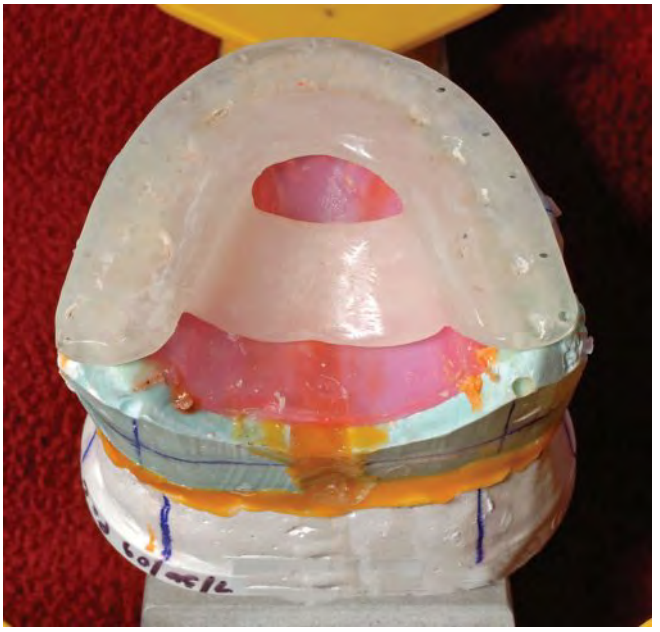


FIGURE 56-25. Final interocclusal splint with a transpalatal strap, fabricated over a layer of wax to provide relief and not impinge on the palatal mucosa and blood supply once inserted and secured. Holes are made in the flange to allow for the splint to be secured to the orthodontic brackets.

The models are articulated as per the previously described standard mounting technique utilizing a facebow transfer and CR registration. The articulator's pin is not changed at all during the case. The previously described vertical and horizontal measurements are made on the casts and recorded on the models and plaster as well as on the laboratory data sheet (orthognathic roadmap).

The maxillary cast is then separated from the base. The base of the cast is trimmed sufficiently to accommodate impaction, advancement, cant correction, or rotation. The new position of the maxilla must be considered in all three planes of space. The movement is correlated to the clinical database and photos to place the maxillary midline in concordance with the facial midline. Any cant correction is achieved by correcting vertical measurements that may be unequal at first. For instance, if on clinical examination, the maxillary left canine is 2 mm lower than the right canine, this is leveled in the final position of the cast. Relating the clinical database and cephalometric prediction tracing, the maxilla is then moved in an AP dimension to the prescribed position. The vertical position of the maxilla is also prescribed by the clinical orthognathic database.

A number of methods can be used to hold the maxillary cast in this new position while making the movements and verifying through measurements. Three small balls of white dental wax can be used during the "move and measure" phase followed by placing yellow sticky wax in between the model and the base to secure the maxilla to the base more securely in that position once the correct position is obtained. Some clinicians use dental plaster whereas others utilize a glue gun for the same purpose.

Caution must be exercised while making multidimensional movements to constantly reevaluate the measurements in all planes and also ensure that the heels of the cast are not kicked off in one direction that would be difficult to replicate during surgery. This is especially possible while attempting to correct the midline. As the anterior part of the cast moves to one side or another, it is easy for the heels to move in the opposite direction (sometimes in a manner that will be difficult to accommodate at the time of surgery). This can be avoided by placing the fulcrum of rotation as anterior as possible.

With the maxilla secured in its desired position, it will provide the surgeon its orientation to the unoperated mandible. Following the placement of maxilla in its new prescribed position; the mandibular cast is separated from its base, placed into the planned occlusion, and a new base is created for it, preferably in a stone of a different color as opposed to the original mounting. This will be the final position of the mandible. Ideally, occlusal adjustments that are needed are performed before impressions are obtained; however, if this was unanticipated, these adjustments are now accomplished on the casts and marked with red pencil in order to facilitate replication at the beginning of surgery.

A final splint is fabricated first in this position. Once that splint is complete, the mandibular model can be repositioned onto the initial base and the intermediate splint is constructed. Postsurgical measurements are then obtained and recorded on the roadmap for surgery.

Sequence of bimaxillary orthognathic surgery The model surgery for bimaxillary surgery will depend upon which sequence the surgeon will perform the osteotomies in the operation. The model surgery will be performed in the same sequence to allow for correct intermediate splints to be fabricated. The majority of surgeons prefer to operate on the maxilla first, fixate it against the unoperated mandible using an intermediate splint, and then move the mandible to occlude with this new maxillary position for the final maxilla-mandible relation. Specific situations in which this sequence may be altered and the mandible operated on first include cases in which there will be counterclockwise rotation of the maxillomandibular complex and a large (temporary intraoperative) anterior open bite created if the maxilla is moved first.[5] Other times, the mandible may be operated first purely as a matter of surgeon preference.

Splint Fabrication

Most surgical splints are made with cold cure acrylic material. An ideal splint must be thin, yet strong, and devoid of interferences, air bubbles, and imperfections (Figure 56-26). Most clinicians prefer coverage of the splint to include at least the first molar tooth. However, this may allow extrusion of the second molars before splint removal; therefore, coverage of all teeth in the arch is recommended.

The steps involved in splint fabrication begin with application of separating media on the casts. A self-cure acrylic

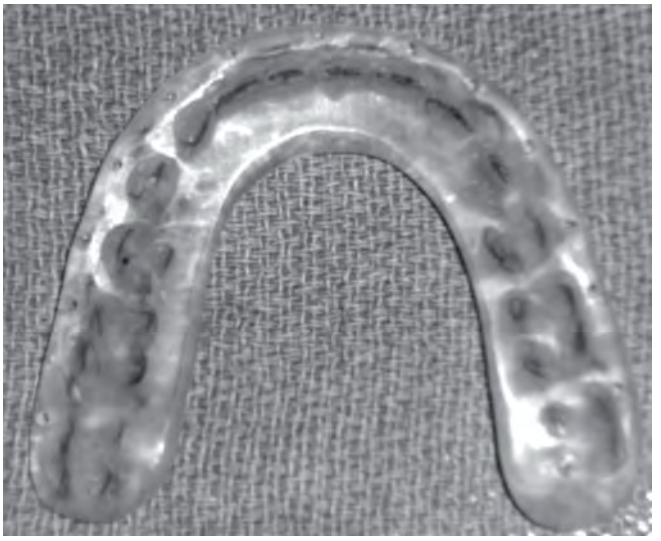


FIGURE 56-26. A final occlusal splint for an orthognathic surgical procedure that does not require a segmentalized maxilla. The regions that will accommodate the incisal tips of the teeth are marked with pencil to facilitate trimming. Holes are placed between the teeth within the flange of the splint for wires to be passed.

form is created and pressed between articulated casts. It is then cured in a pressure cooker and trimmed. Marks may be made with a pencil at the regions of the cusp tip indentations to prevent excessive trimming. Once the splint has been trimmed to be sufficiently thin, it is polished and small holes are made in the flange to allow for passing wires for assisting in intraoperative maxilla-mandible fixation.

While performing bimaxillary procedures that involve a segmental maxilla, the intermediate splint is a composite splint. This splint is constructed to fit over the final splint that remains attached to the maxilla (Figure 56-27). In segmental maxillary surgery, the final splint is often created with an additional palatal strap to prevent collapse of segments in the immediate postoperative phase. This splint is often left in place for several weeks to allow support for the

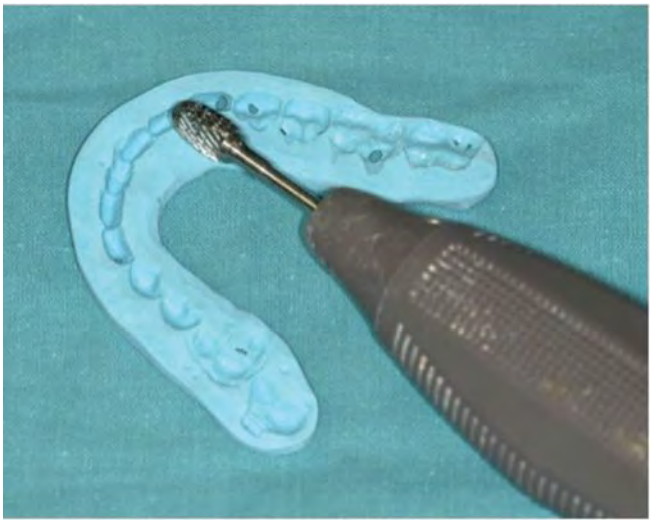


FIGURE 56-27. PVS intermediate splint trimmed, final occlusal splint surface.

segments. In cases that do not involve segmentalization, a splint fabricated from PVS may be used for both the intermediate and he final splint (Figure 56-28). When segmental maxillary surgery is performed, the final splint must be a rigid acrylic or composite splint, but the intermediate "piggy-back" splint may still be PVS. A PVS splint requires much less time and material to create and offers similar results (Figure 56-29).

Three-Dimensional Virtual Model Surgical Simulation

Introduction

Traditional model surgical planning has provided oral and maxillofacial surgeons with the ability to meticulously plan and perform orthognathic surgical procedures. Limitations and opportunities for the introduction of error through traditional model planning remain, however. The stone models are three-dimensional representations of the dentoalveolar





FIGURE 56-28. **A,** Intermediate and final splint. **B,** Intermediate splint *(red)* and final splint *(blue)* interdigitation on mounted casts.



FIGURE 56-29. Intermediate splint fabricated with PVS and interdigitation to the acrylic final splint for this segmental bimaxillary case.

structure but cannot reflect the maxillofacial bony anatomy. Error can be introduced through an inaccurate facebow transfer, bite registration, mounting, or in obtaining model measurements. The entire process, even in experienced surgeons, is time- and labor-intensive. It also involves assimilating multiple unrelated data sets (such as the cephalometric film and clinical photos), which are two-dimensional representations. The use of a cephalometric radiograph analysis and cephalometric prediction tracings provides some level of simulation and prediction but is extremely limited because they are two-dimensional representations.

Advances in three-dimensional medical imaging and virtual surgical simulation software for orthognathic surgery have enabled a major breakthrough and allowed unprecedented virtual diagnosis, treatment planning, and evaluation of treatment outcomes of maxillofacial deformities. Virtual simulation allows for inclusion of the bony anatomy into the treatment plan, not relying solely on the position of the teeth and allows the surgeon to be able to

- Analyze three-dimensional anatomic discrepancies in pitch, roll, and yaw.
- Analyze how occlusal movements affect surrounding bony anatomy.
- Utilize simulated postoperative soft tissue and bony predictions for patient education and surgical resident teaching purposes.
- Investigate multiple surgical approaches for the same patient.
- Digitally create intermediate and final orthognathic splints using computer-aided design and computer-aided modeling (CAD/CAM) technology.
- Reduce surgeon planning time, especially for complex cases.

History

Xia and coworkers are the first group who used computer-aided surgical simulation (CASS) for treatment planning of complex craniomaxillofacial deformities.[6] The first step of the CASS process is to create a composite skull model. This is accomplished with a bite jig that is used to relate the upper and lower dental casts to each other and to support a set of fiducial markers. Fiducial markers are selected points on an image that are used as a frame of reference in locating objects or in positioning (registering) images containing the same markers. After the bite jig is created, a computed tomography (CT) scan of the patient's craniofacial skeleton is obtained with the patient biting on the same bite jig. Scanning the stone dental models with a laser surface scanner then creates digital dental models. The markers are then used to register (superimpose) the digital dental models on to the three-dimensional CT skull model. The result is a computerized composite skull model with an accurate rendition of the bone and the teeth.

The second step of the CASS is to quantify the deformity with cephalometric analyses and virtual anthropometric measurements. The third step in the process is to simulate the surgery on the computer by moving the bony segments to the desired position. Using this software, the maxilla and mandible can be repositioned in all three planes of space. Hence, deformities of yaw, pitch, and roll can be accurately corrected in a virtual environment. The final step is to transfer the virtual plan to the operating room through surgical splints and templates that are created using a specialized CAD/CAM technique.

Whereas a number of CAD/CAM programs are currently commercially available for applications in craniofacial surgery and orthognathic surgery, CASS with the use of Simplant OMS (Materialise Dental, Ann Arbor, MI) software and the Medical Modeling Corporation (Golden, CO) are discussed and used as an example to illustrate a case sequence here.

Sequence of Virtual Simulated Surgery for an Orthognathic Surgical Procedure

1. The patient is clinically physically examined in the same fashion as one would for carrying out a traditional analytic model surgery and the same orthognathic database measurements are obtained and analyzed.
2. Digital photos of the patient in repose from the frontal, left, right, and while smiling, are obtained with the patient in a natural head position (NHP).
3. High-quality alginate impressions are obtained of the maxillary and mandibular dentition and stone casts are poured.
4. A final desired postoperative occlusion registration splint is fabricated from the models. This has the maxillary and mandibular teeth placed in their postoperative relationship. A cut and repositioned maxillary stone model is required if a segmental maxilla is planned.
5. A patient-specific bite jig is then created with the patient in CR. The jig will be used for the purpose of aligning a laser scan of the stone models and allows for detailed reorientation of the CT scan to the patient's NHP.
6. The jig is attached to a facebow and the patient's NHP is identified. Three Euler angle (pitch, roll, and yaw) readings (in degrees) are recorded with a gyroscope. The NHP may be established by asking the patient to look at a wall

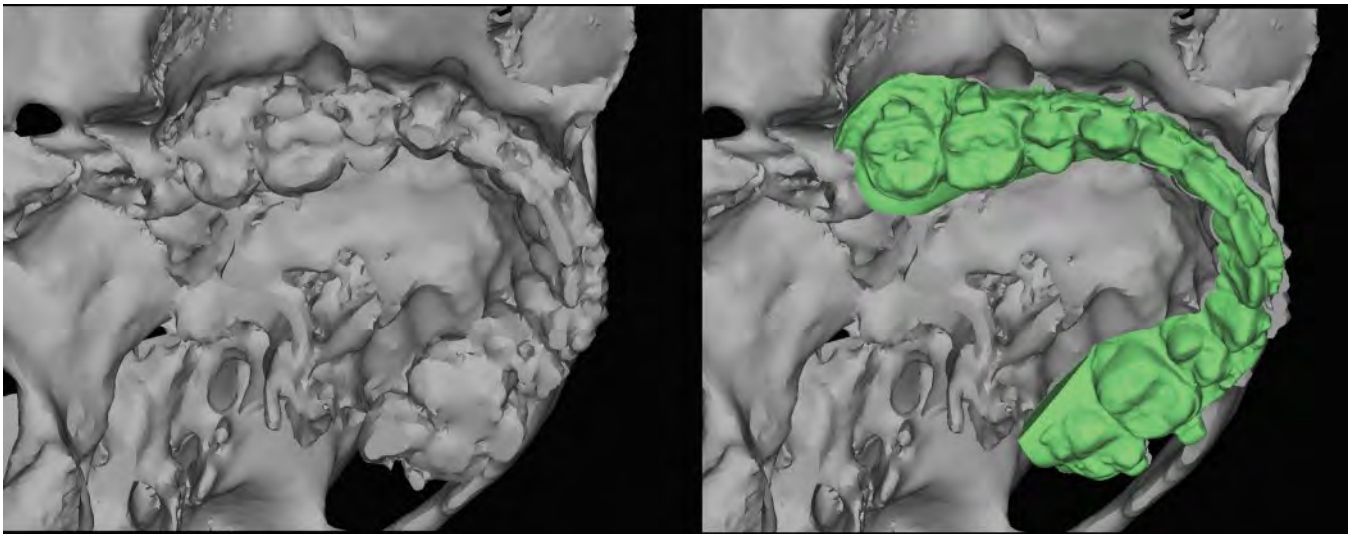


FIGURE 56-30. For an accurate occlusal surface, high-resolution laser scans of stone models are taken and integrated into the computed tomography/cone beam computed tomography (CT/CBCT) scan using fiducial marker registration.

without looking at a specific point or by the patient looking into his or her own eyes in a mirror with a plumb line hanging down the center of the mirror. If the patient has a tendency to orient his or her head forcefully (seen predominantly in asymmetry cases), the surgeon should intervene as necessary.

7. Following the appropriate CT scan protocol, a standard CT or cone beam computed tomography (CBCT) scan is obtained. This scan is obtained with the patient occluding on the bite jig, in CR, attached to the facebow.

Image Acquisition for Three-Dimensional Virtual Orthognathic Surgery

The surgeon must consider three important concepts in preparing data for successful digital orthognathic planning:

1. CT technology currently lacks the ability to capture the teeth and their occlusal surfaces with high accuracy. To produce accurate CAD/CAM splints, it is necessary to replace inaccurate occlusal surfaces from CT or CBCT scans with either a high-resolution laser scan or CT scan of the stone models (Figure 56-30).
2. Reorientation of the patient's CT/CBCT scan is needed to reflect their NHP for more accurate planning. This is accomplished utilizing the preoperative gyroscope Euler angle readings (Figure 56-31).
3. The quality of the CT scan is the most important aspect of creating a patient-specific anatomic model. The patient must wear the bite jig and attached fiducial facebow throughout the entire scan. For a standard CT, scans should be acquired at a high spatial resolution with thin (0.75–1.25 mm) image slices, a field of view of 20.0 to 25.0 cm, 0-degree gantry tilt with the patient's occlusal plane parallel to the gantry, and no motion artifact. The scans should be archived in DICOM (digital imaging and communications in medicine) format on a CD-R.

The digital clinical photos, upper and lower stone casts, clinical orthognathic database measurements, the acrylic bite jig, final occlusion registration, CT datasets, and the gyroscopic natural head position readings are then shipped to a software engineer for computer rendering.

Virtual Treatment Planning of Orthognathic Surgery

The software engineer then creates digital dental models by scanning the stone casts with a laser surface scanner. The digital dental casts are integrated into the digital CT skull using a best-fit model (Figure 56-32). This software offers the possibility of planning surgical procedures in multiplanar and three-dimensional views. Therefore, segmentation, measurement, repositioning, and importing tools are incorporated. All planning steps are based on virtual segmentation procedures, which are necessary for performing repositioning. By using predefined Le Fort I and sagittal (or vertical ramus) osteotomy lines, the upper (Figure 56-33) and lower jaws (Figure 56-34) can be segmented and virtually moved in all three planes of space.

A surgical plan is outlined and executed during a web conference with a software engineer. The maxillary and mandibular osteotomies are performed and movements are made with the patient's composite CT scan in the previously defined NHP. Deformities of yaw, pitch, and roll can be virtually corrected and accurately assessed using precise angular and linear digital measurements. Any inaccuracies in the virtual plan can then be corrected based on the virtual image analysis.

Transferring Virtual Treatment Planning to the Operating Room

After the surgical plan has been finalized, it is necessary to transfer the plan to the patient at surgery. Similar to traditional model surgery, dental splints are created for this purpose, but in this case, the intermediate (see Figure 56-33) and final (see Figure 56-34) digital surgical splints are fabricated by CAD/

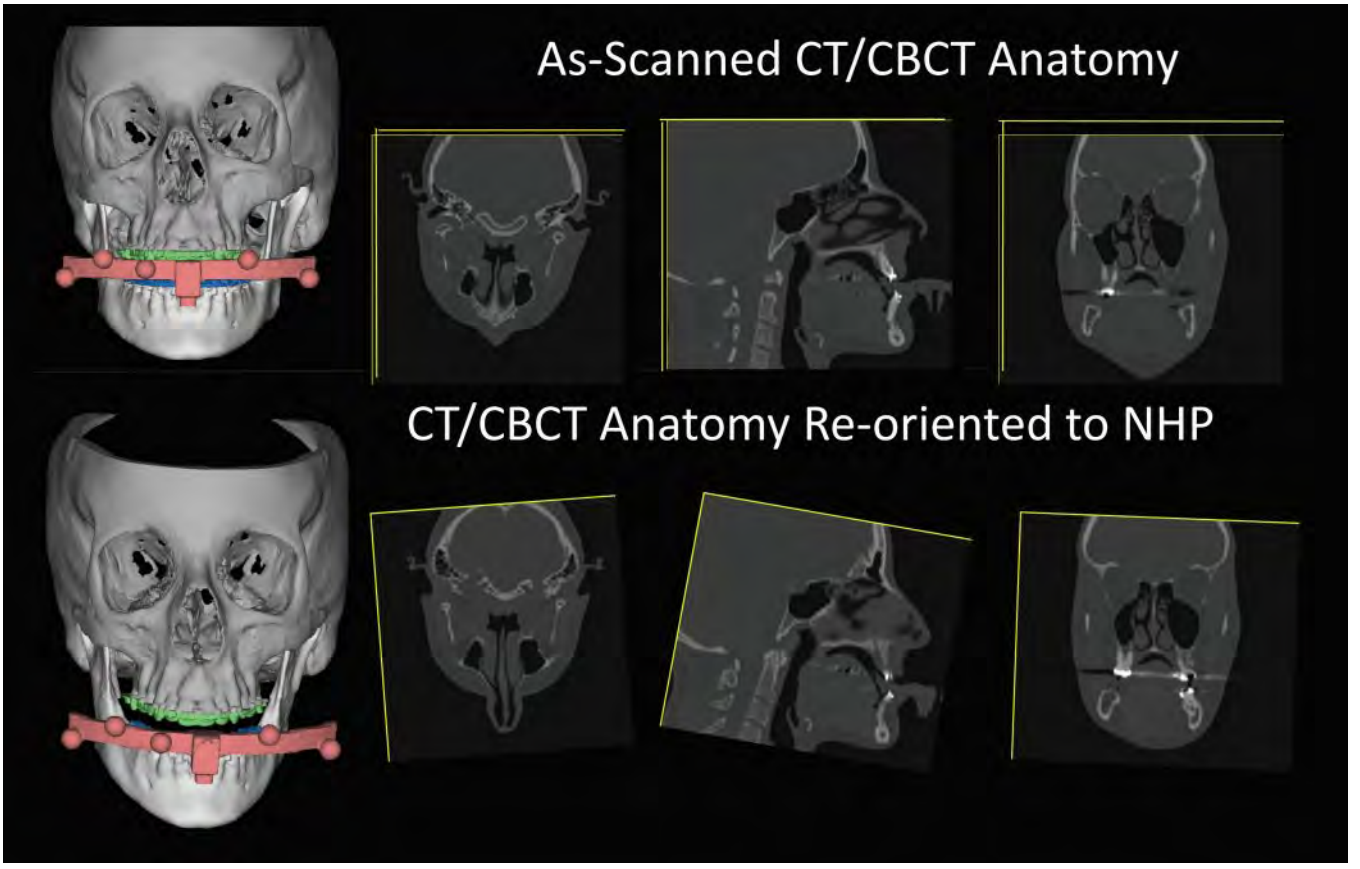


FIGURE 56-31. The patient is CT/CBCT scanned in centric relation wearing a facebow device and neutral head posture (NHP) is clinically recorded by the surgeon using a gyroscope device. These orientation measurements are applied to the CT/CBCT scan to reorient the patient's digital data into the proper NHP.

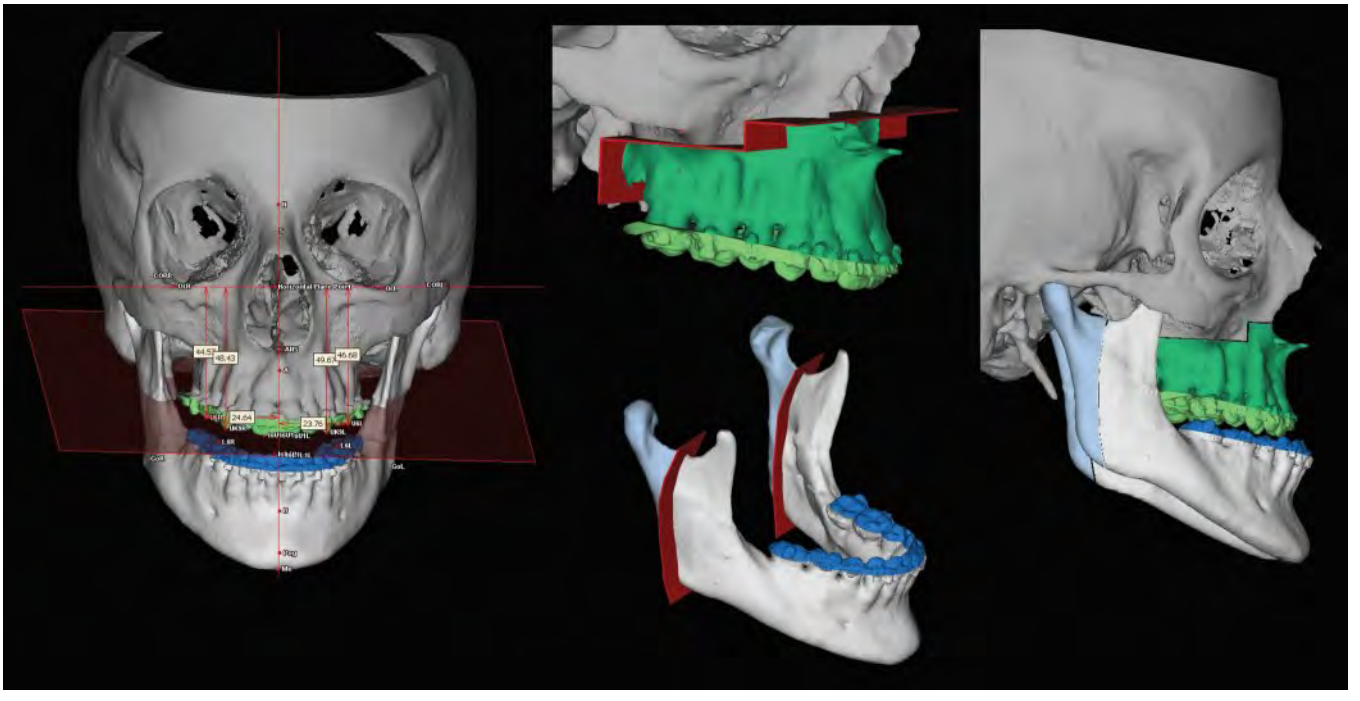


FIGURE 56-32. The patient's asymmetry is quantified using digital bony landmarks and measurements to orthogonal reference planes. Digital osteotomies are performed on both the upper and the lower jaws.

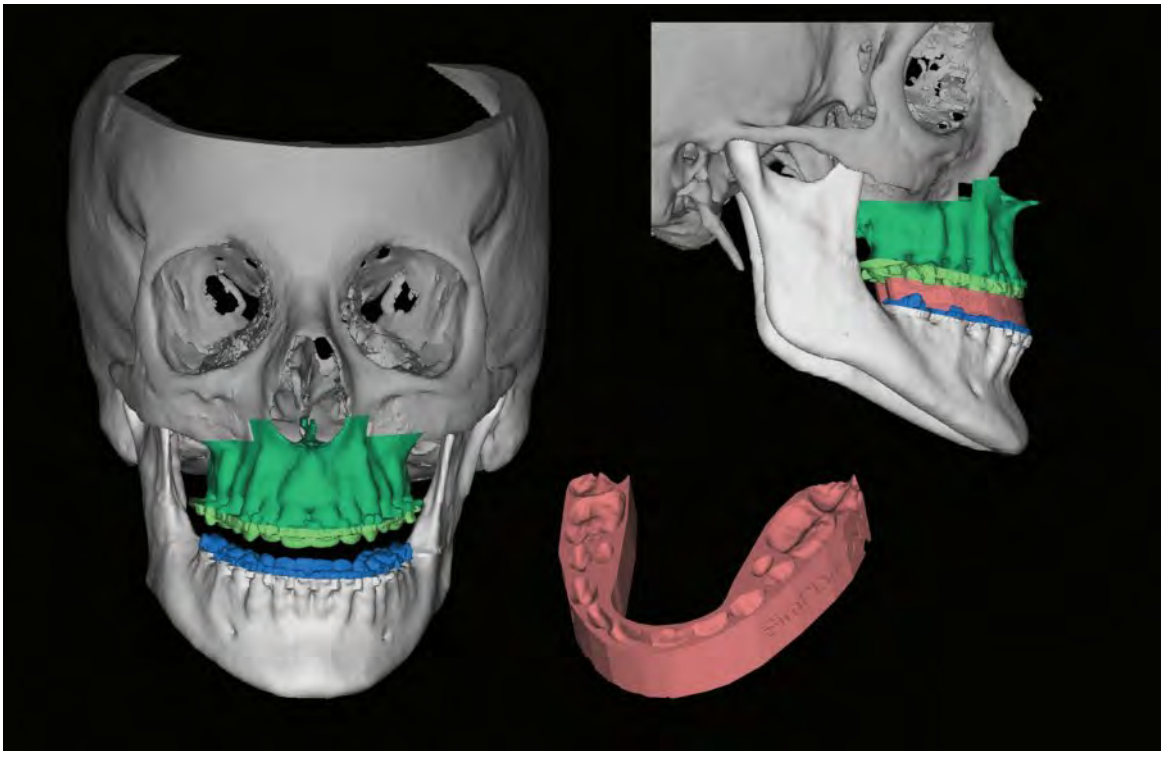


FIGURE 56-33. The maxilla is moved to its final position, and a computer-aided design and computer-aided modeling (CAD/CAM) intermediate splint is designed from the digital plan.

CAM technique. In procedures that require bone grafts to achieve symmetry, the computerized mirror-imaging technique can be used to provide a mirror image of the geometry from the healthy side to the affected side. The difference between the two sides can then be computed, resulting in a digital template that is used at surgery to harvest the graft and sculpt it or the implant. Vertical measurement is accomplished in either of the two methods mentioned earlier.

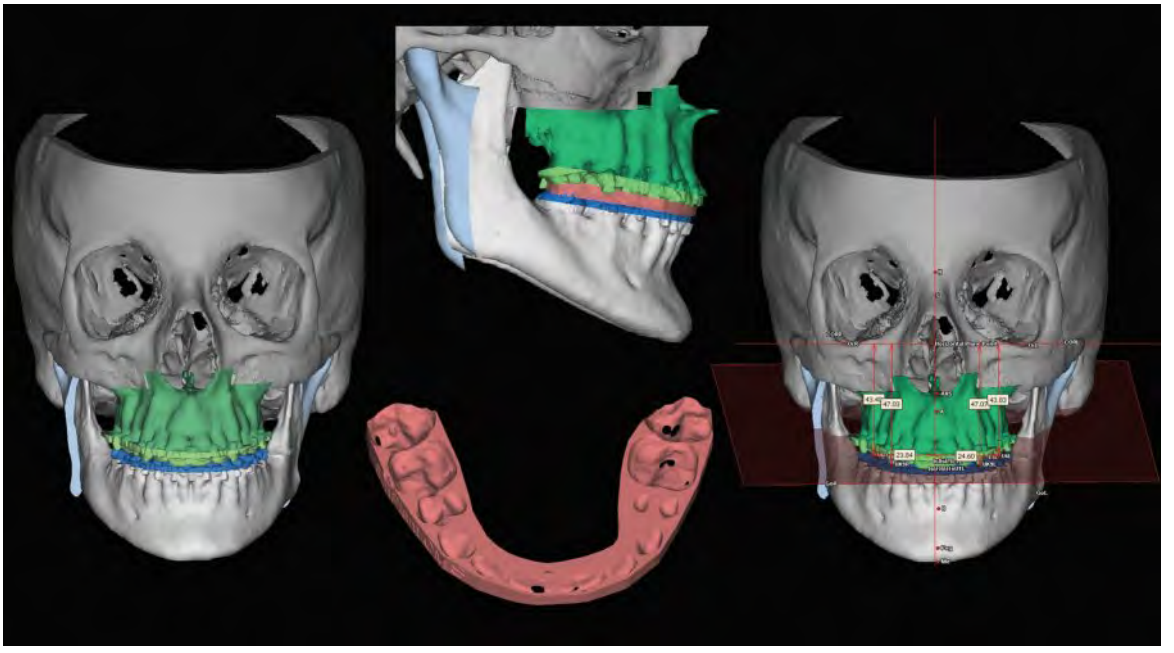


FIGURE 56-34. The mandible is moved into final occlusion and a final position CAD/CAM splint is designed from the digital plan. The simulated final position of the bony anatomy can be assessed using digital cephalometric analyses.

Much can be gained planning these cases in a virtual manner, similar to virtual planning of implant cases. The stumbling blocks at this time are need for the CT scan and the overall cost of the process.

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CHAPTER 57

Mandibular Orthognathic Surgery

Dale S. Bloomquist, DDS, MS, and Jessica J. Lee, DDS

The development of mandibular osteotomies for correction of dentofacial deformities closely parallels the advancement of oral and maxillofacial surgery as a specialty more than any other group of surgical techniques. From Hullihen in 1849, who was the first to describe a mandibular osteotomy, to Obwegeser in 1955, who developed the sagittal osteotomy of the vertical ramus, there has been dramatic progress in the techniques of mandibular osteotomies. After Obwegeser's original paper in German, and especially since his description of techniques in the English literature, orthognathic surgery has seen dramatic changes in use as well as refinement of the osteotomies. Although the development of osteotomy techniques is ongoing, the purpose of this chapter is to describe the most commonly used surgical procedures for the mandible and also to emphasize the refinements in technique that have been the result of the most recent clinical as well as basic science research.

HISTORY

Hullihen[1] corrected a burn patient with an anterior open bite and mandibular dentoalveolar protrusion with an intraoral osteotomy, similar to an anterior subapical osteotomy (Figure 57-1). His efforts did not stimulate much interest, because it was almost 50 years later when Angle[2] described a body osteotomy done by V. P. Blair[3] (Figure 57-2A) for a patient with mandibular horizontal excess. This technique, with minor modifications, was advocated until the 1970s. Since then, the only major modifications in the body osteotomy that have occurred are a greater emphasis on preserving the inferior alveolar nerve (IAN) and a change to an intraoral approach.

The horizontal osteotomy of the vertical ramus popularized by Blair[4] (see Figure 57-2B) was performed through an extraoral approach. As with many of the early mandibular procedures, a horizontal bone cut was made above the lingula and was described for correction of both mandibular horizontal deficiency and horizontal excess. An intraoral technique was not suggested until Ernst[5] discussed his procedure approximately 25 years later. This method of correcting mandibular deformities was used for almost 60 years, but because of its lack of postoperative stability, it has fallen into disuse.

The subcondylar osteotomy (see Figure 57-2C), a description of which was first reported by Limberg[6] as an extraoral technique, has undergone relatively minor refinements to the intraoral vertical subcondylar osteotomy (VSO) that is used today. There has, however, been a substantial number of osteotomy designs involving the vertical ramus that begin in the sigmoid notch, which has led to some confusion in the nomenclature of what is a fairly closely related group of ramus osteotomies. The names that have been developed have generally been based upon the length and direction of the cuts made in the posterior portion of the vertical ramus. The subcondylar osteotomy was used to describe the condylar neck osteotomies of Kostecka[7] and Moose.[8] Generally, longer cuts that extended to the posterior border above the angle, such as described by Limberg, Thoma, and Robinson, were referred to as "oblique osteotomies."[5–10] Shira, however, coined the term "oblique sliding osteotomy" for this particular surgical procedure.[11] Finally, Caldwell and Letterman[12] described a vertical osteotomy of the mandibular ramus that included a cut from the sigmoid notch to the inferior border, in front of the angle of the mandible. The cut was posterior to the mandibular foramen, and a portion of the lateral cortex

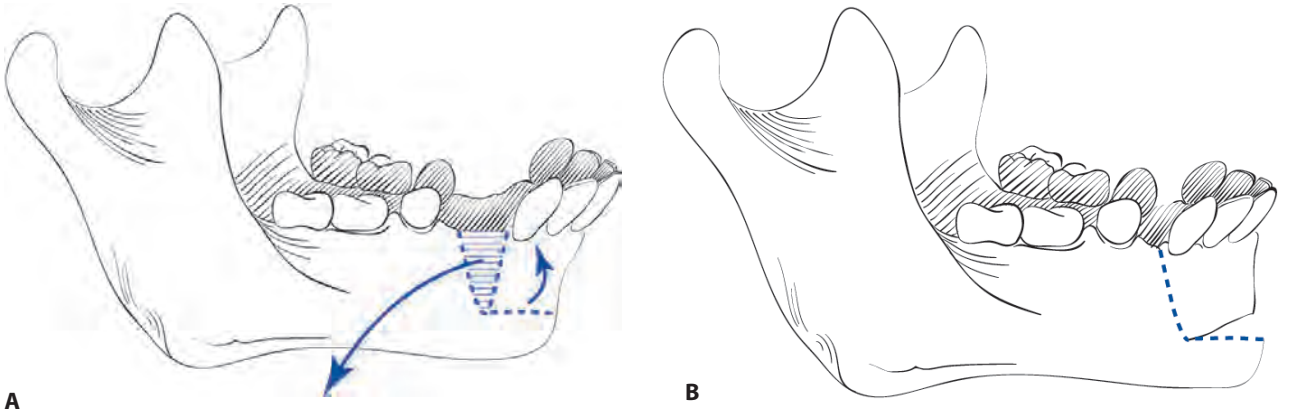


FIGURE 57-1. Hullihen's mandibular subapical osteotomy. (Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1416.)

of the distal fragment was decorticated to allow a larger area of bony contact. In general, these latter two groups of osteotomies are now called "vertical osteotomies," but some semantic differences still persist. Specifically, the terms *vertical subcondylar osteotomy (VSO)* and *vertical ramus osteotomy (VRO)* are still used interchangeably in the literature. Primarily, this type of osteotomy was designed for correction of mandibular horizontal excess, or mandibular asymmetries, although Robinson[10] described its use with a bone graft for advancement in horizontal deficiencies of the mandible.

The intraoral approach to the subcondylar osteotomy was first described by Moose in 1964.[13] He approached the condylar neck medially with a straight bur. Winstanley[14] suggested a lateral approach in 1968, but it was not until Hebert and colleagues[15] described the use of a special oscillating saw that this approach became popular.

A variation of the VSO was suggested by Wassmund in 1927[16] (Figure 57-3A), which is similar to what is now called the *inverted-L osteotomy.* Pichler and Trauner[17] later suggested inserting bone grafts into the defect created by advancement of the mandible. Caldwell and coworkers[18] further modified the inverted-L by adding a horizontal cut just above the inferior border of the mandible to create what is now called the *C osteotomy* (see Figure 57-3B). The stated advantage of the C osteotomy was that the bone cut design made the use of a bone graft unnecessary. This advantage

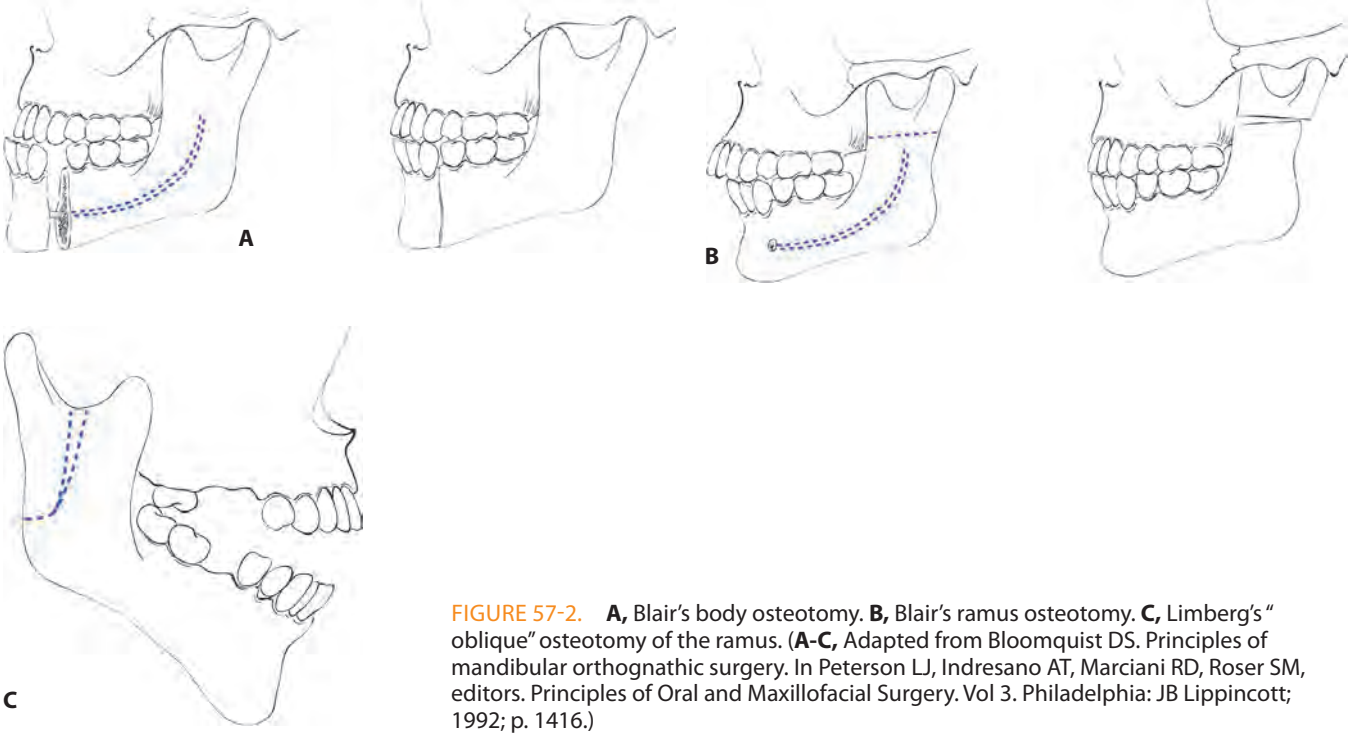


FIGURE 57-2. **A,** Blair's body osteotomy. **B,** Blair's ramus osteotomy. **C,** Limberg's "oblique" osteotomy of the ramus. (**A-C,** Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1416.)

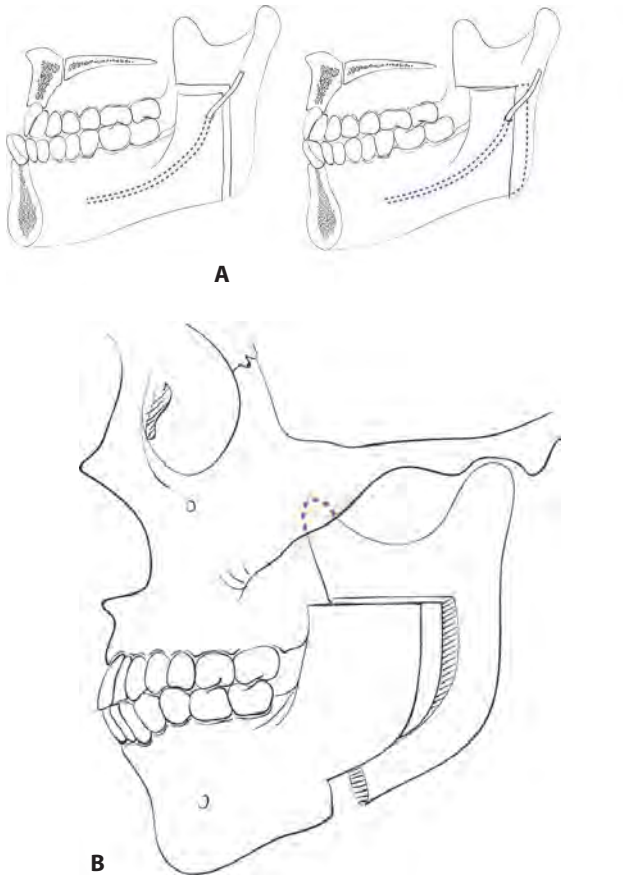


FIGURE 57-3. **A,** L osteotomy. **B,** C osteotomy. (**A** and **B,** Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1417.)

was further enhanced by the modification suggested by Hayes,[19] with the splitting of the inferior limb sagittally so that more bone contact could be achieved. A further interesting approach to this group of VROs used for horizontal mandibular deficiency is the modified-L osteotomy described by Fox and Tilson.[20] They eliminated the superior horizontal cut of the C osteotomy and, instead, extended the vertical cut to the sigmoid notch. Then the coronoid process was removed and added as a free graft into the defect resulting from the mandibular advancement.

The greatest development in osteotomies of the vertical ramus is the sagittal osteotomy, credited to Obwegeser and Trauner, but generally now used in a fashion modified from the original technique described in 1955.[21] Lane has been referred to as the developer of a form of the sagittal osteotomy, with parallel horizontal bone cuts made through the medial and lateral cortices of the vertical ramus (Figure 57-4).[22] The medial cut was made just above the lingula, with the lateral cut made just below it. This idea was expanded by Schuchardt[23] before being refined and popularized by Obwegeser and Trauner.[21] The major modifications in the osteotomy design were first made by DalPont[24] with his

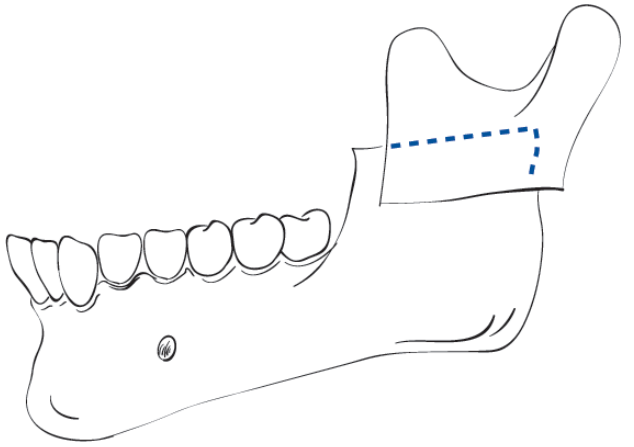


FIGURE 57-4. Lane's osteotomy. (Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1417.)

vertical cut through the lateral cortex as well as the suggestion that the medial horizontal cut be extended only to a point above the lingula and not to the posterior border (Figure 57-5). This latter technique shortens the split posteriorly, to the area of the retrolingular fossa and not to the posterior border, and as was further discussed by Hunsuck,[25] decreases the trauma to the overlying soft tissues. Many clinicians have

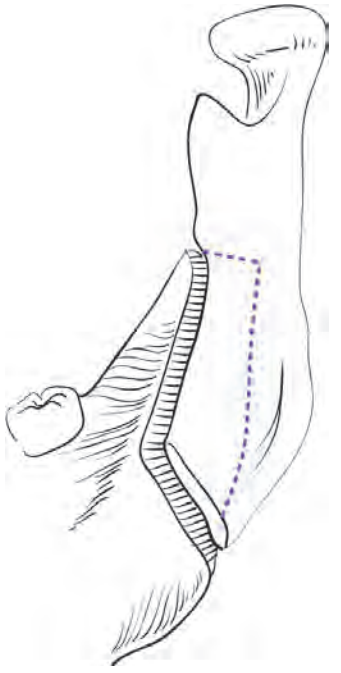


FIGURE 57-5. DalPont's modification of the sagittal osteotomy. (Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1418.)

offered suggestions for improving the sagittal osteotomy, but the only other major innovation to this technique has been the use of internal rigid fixation. Spiessl[26] suggested the use of screws for fixation of the fragments in the sagittal osteotomy. Although wire osseous fixation is still used by some surgeons, rigid internal fixation in some form (bicortical screws or monocortical plates and screws) has become the standard technique for the bilateral sagittal split osteotomy (BSSO).

Osteotomies of the mandibular body do not generally receive the same degree of attention as osteotomies of the vertical ramus, but they have undergone refinements and variations from the original anterior alveolar osteotomies of Hullihen[1] and the body osteotomies of Blair.[3] The first variation of Hullihen's procedure did not appear until 90 years after the original description, when Hofer[27] described an anterior mandibular alveolar osteotomy to advance anterior teeth in correction of mandibular dentoalveolar retrusion (Figure 57-6A). Kole[28] modified this procedure by suggesting the use of bone grafts from the mental region to the defect caused by the rotation of the anterior dentoalveolar segment (see Figure 57-6B). Clinicians now employing Hofer's osteotomy generally use some form of bone graft in the alveolar defect if significant movement of the fragment is planned. Mandibular alveolar osteotomies have expanded primarily in two ways from Hofer's original procedure. Kent and Hinds[29] initially presented the use of the single-tooth osteotomies of the mandible in 1971, and MacIntosh[30] closely followed with his description of the total mandibular alveolar osteotomy in 1974. The latter procedure continues to be popular, with minor variations recommended by other clinicians.

Osteotomies of the body of the mandible have been described in almost every conceivable form, with the most durable advancements being the step osteotomy, initially described by Von Eiselberg in 1906[31] (Figure 57-7A), and the horizontal osteotomy of the symphysis described by Hofer in 1942[32] (see Figure 57-7B). The step osteotomy was originally described for treatment of mandibular horizontal deficiency,

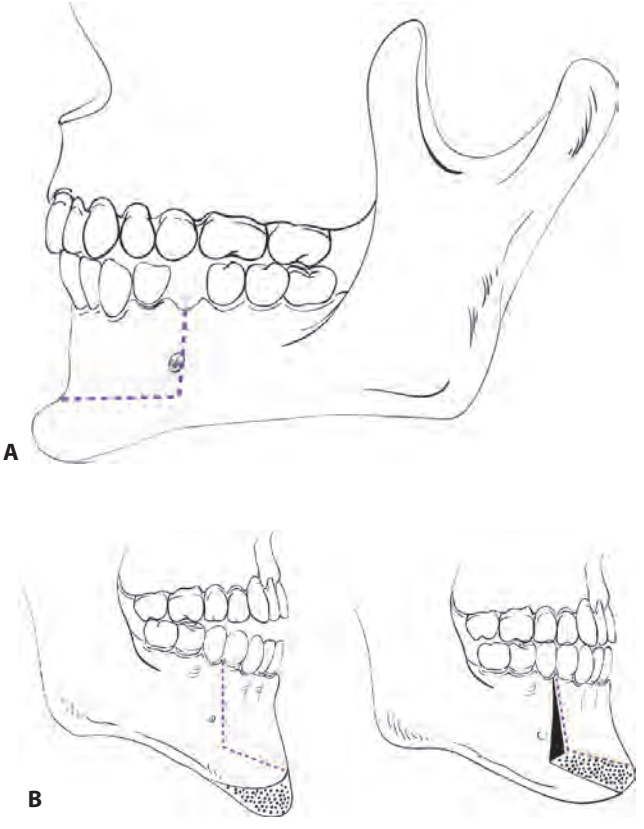


FIGURE 57-6. **A,** Hofer's subapical osteotomy. **B,** Kole's subapical osteotomy. (**A** and **B,** Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1418.)

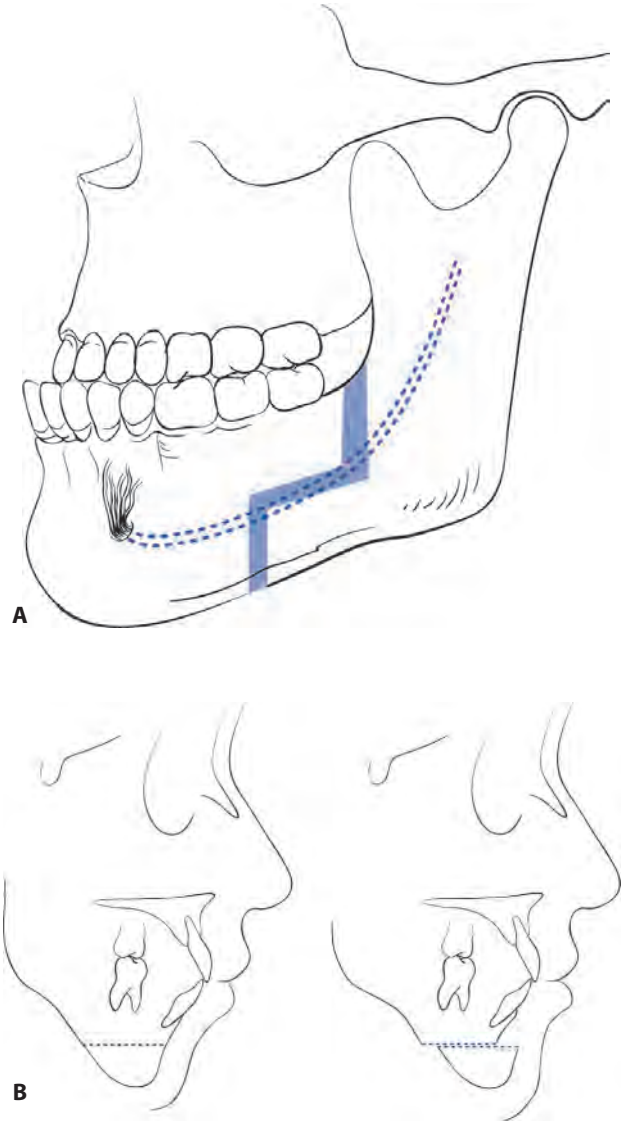


FIGURE 57-7. **A,** Von Eiselberg's step osteotomy. **B,** Hofer's horizontal osteotomy. (**A** and **B,** Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1419.)

but it has been used in various forms for mandibular horizontal excess as well as asymmetry. The horizontal osteotomy of the symphysis has also developed a large degree of versatility, with its use in various forms being suggested for almost any skeletal deformity of the bony chin.

ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS OF MANDIBULAR SURGERIES

Vascular Supply

A major concern with surgery of the facial skeleton is the vascular supply to the bony segments. This was demonstrated dramatically by the explosion of orthognathic surgery in the United States, after Bell and Levy's studies of vascular effects of the osteotomies.[33] Although all of the techniques they evaluated had been previously used in patients, there had not been any experimental evaluations of the physiologic basis for many of the procedures. Bell and Levy's work demonstrated that blood flow through the mandibular periosteum could easily maintain a sufficient blood supply to the teeth of a mobile segment, even when the labial periosteum was degloved. Blood flow from the periosteum was considered to be centripetal, to distinguish it from the blood flowing from endosteal vessels outward (centrifugal) that was associated with long bones. Previously, clinicians believed that the inferior alveolar artery had a primary role in nourishing the mandible, but Bell and Levy[33] demonstrated that there is also a sufficient blood supply from the surrounding soft tissues, even if the inferior alveolar artery was compromised. More recent work in animals suggests that the blood supply to the body of the mandible under normal conditions comes almost entirely from the inferior alveolar artery.[34] However, when this source is obstructed, the peripheral blood vessels quickly take over for the anterior mandible. The posterior mandibular dentoalveolus, however, does not benefit from this kind of collateral blood supply, which calls into question the safety of posterior mandibular segmental alveolar osteotomies. Zisser and Gattinger[35] showed pulpal necrosis in the molars of horizontal osteotomies done above the IAN in the body of the mandible of dogs.

The safety of combined mandibular osteotomies, such as ramal procedures and body osteotomies, has been a concern because of the predominant role of the inferior alveolar artery.[36] The fragility of the vascular supply to the mandibular alveolus engenders some concern over the common use of subapical osteotomies. Although their relative safety has been demonstrated by both animal studies and substantial clinical experience, subapical osteotomies need to be carefully planned to ensure as large a vascular pedicle as possible.[33,37] Complications, such as pulpal necrosis, soft tissue defects, and loss of teeth and bone, have demonstrated the delicate nature of the blood supply, especially when attempts to move small dentoalveolar fragments are made. The effect of aging on the vascular supply to the mandibular body is an area about which very little information is known, particularly whether aging causes a switch from the centrifugal to the centripetal blood supply. Bradley[38] has demonstrated an apparent decreasing capacity of the inferior alveolar vessels that occurs with aging, but the impact of this effect on mandibular osteotomies is unknown. Subapical osteotomies are not routinely performed today.

Osteotomy designs of the vertical ramus have profited from studies of the effects of surgery on vascular supply. The proximal segment of the vertical subsigmoid osteotomy maintains its blood supply through the temporomandibular joint (TMJ) capsule and the attachment of the lateral pterygoid muscle. However, the inferior tip of this fragment may undergo vascular necrosis based upon prior experimental studies.[39] This observation led to the suggestion that fewer problems may occur if the cut is made above the angle of the mandible.[39]

The importance of the periosteal blood supply as well as the endosteal supply in the vertical ramus has been explored by animal research.[40–43] When the medial pterygoid and masseter muscles are stripped, both blood flow and blood supply studies have demonstrated the possibility of avascular necrosis in the proximal segment. Comparisons of extensive muscle stripping of the vertical ramus versus preservation of the masseter attachment have demonstrated a significant difference in the vascularity of the inferior portion of the proximal fragment. These studies of blood supply of the vertical ramus may be of value in predicting the vascular effects of the C or L osteotomies. However, resorption of the proximal fragment has not been reported in these particular bone cuts, possibly because of the rarity of their use. However, given the available research, it is prudent to minimize the periosteal and muscle attachment stripping on the medial surface of the proximal fragment with either the C or the L osteotomy, or any of their variations.

The last unanswered question concerning vascular supply in mandibular orthognathic surgery is the determination of a safe distance away from the apex of the teeth to make horizontal bone cuts. Much of the data addressing this question are based upon research about the maxilla.[44] From these early animal studies, the pulpal blood supply of a tooth should not be affected if a cut is made at least 5 mm away from the apex of the tooth root. Zisser and Gattinger,[35] however, saw pulpal changes in dogs with some horizontal cuts that were made 10 mm away from the apex. Whether these distances have any relevance to humans is unknown. Clinically, the incidence of tooth devitalization from horizontal subapical osteotomies is extremely low and it can be assumed that, for the most part, 5 mm is an adequate guideline. A cut made 10 mm from the apices, although allowing a greater safety margin, is often impractical because of other anatomic limitations. The greater distance from the apices of the teeth not only minimizes direct pulpal injury but also increases the vascular pedicle to the mobilized segment.

Nerves

The surgeon working around the face must be constantly aware of the nerve network that exists in this area.

Fortunately, in considering the mandible, these concerns can be narrowed down to essentially two major nerves: the marginal mandibular branch of the seventh cranial nerve and the third division of the trigeminal nerve, and most frequently, one of its branches, the IAN. The marginal mandibular branch is usually at risk only during extraoral procedures. Although trauma to this nerve has been reported to occur during an intraoral approach, it is rare and, for the most part, appears to be preventable. Avoiding damage to this nerve during extraoral approaches to the mandible is a major surgical goal; in most cases of orthognathic surgery, it is achieved because soft tissue anatomy in patients undergoing the surgery has not been disturbed by disease or trauma. The techniques of these approaches, as well as the methods for minimizing the risks of damage to the marginal mandibular branch, are covered elsewhere in this text. Damage to the third division of the trigeminal nerve is, however, a much-discussed issue in mandibular surgery. The course of the IAN into the vertical ramus and then through the body of the mandible makes it extremely susceptible to damage from almost every mandibular surgical procedure. In most cases, the surgeon's main goal relative to this nerve is to minimize the trauma, because its avoidance is almost impossible. In the past, surgeons stressed the importance of looking for and sometimes freeing up the nerve as it either entered or left the mandible before making osteotomies in the areas of the foramina. However, there is a trend toward avoiding this step, unless it is absolutely necessary to make the osteotomy as close to the nerve as possible. The simple act of exposing the nerve seems to increase the chance for postoperative sensory deficiency.

Often, the debates regarding whether one mandibular osteotomy is preferable to another are primarily based on the potential of damaging the IAN. This has resulted in many clinicians trivializing the damage found after certain technique. Well-defined standards for both long- and short-term follow-up of nerve damage during mandibular procedures have been discussed[45]; however, in most papers, these have not been used to evaluate sensory deficits. In addition, very few controlled studies have been published comparing procedures, so, as a result, not much can be concluded regarding any of the various attempts to minimize nerve damage.

Studies evaluating the loss of tooth sensibility from horizontal osteotomies below the dental apices, however, have been quite consistent. Most authors found a relatively high loss of response to pulp testing immediately after osteotomies, especially when teeth are close to a vertical osteotomy.[46–48] However, this loss may not correlate with actual loss of tooth vitality and, thus, either tooth loss secondary to an osteotomy or the need for endodontic therapy is very low.

Muscles

Orthognathic surgery affects muscles in primarily in one of two ways: it may change the length of a muscle or it may change the direction of muscle function. Effects of these changes are still not understood, although various authors have emphasized the importance of recognizing and controlling muscular alterations of orthognathic surgery. The muscles commonly discussed in orthognathic surgery of the mandible have been the muscles of mastication and the suprahyoid group of muscles. Recent interest on the soft tissue effects of facial skeletal surgery has expanded interest to the other facial mimetic muscles. This latter group, however, has generally not been discussed relative to mandibular osteotomies, with the possible exception of the effect of anterior mandibular osteotomies on the attachment of the mentalis muscle. The muscles of mastication, however, have received considerable attention, dating back to the early vertical ramus procedures. Research interest on the effects of altering these muscles concentrated either on their effect on skeletal changes, especially relapse after mandibular osteotomies, or on the changes in function of these muscles.

Distraction of the superior segment of a horizontal osteotomy of the vertical ramus owing to the temporalis muscle influence after surgery was noted early by surgeons who used this technique.[49] Evaluation of this procedure after correction of prognathism found a superior movement of the mandible in the gonial region as well as a downward and backward movement at the symphysis. This change, which was attributed to the forces of the pterygomasseteric sling, has received considerable attention, not only in mandibular setbacks done with osteotomies through the vertical ramus but also in mandibular advancements.[50–55] The apparent shortening of the vertical ramus has been noted in a number of studies, and in some, a correlation has been demonstrated between this change and the posterior movement of the symphysis. The exact reason for the change in the gonion has not been clearly demonstrated. Kohn[54] demonstrated the movement of this point inferoanteriorly in mandibular advancements by way of a measurement he termed the "gonial arc" (Figure 57-8). Most investigators believe this represents distraction of the condyle from the fossa, and this hypothesis was further supported by the migration of the gonion back during the postoperative period.[54,55] The long-term postoperative decrease in gonial arc was generally believed to be due to remodeling, especially resorption that occurred at the mandibular angle.[40,43] Especially in early studies, resorption could have accounted for this change because of the accepted technique of completely stripping muscle attachments from the proximal segment. However, in more recent studies in which minimal muscle stripping was done, a similar result has been noted.[56,57] Will and associates[58] noted condylar distraction followed by an "overshoot" in the resettling of the condyle in the fossa. This change in condylar position may be due to either displacement of the disk within the TMJ or compression of the soft tissues of the joint by increased pressures secondary to the muscles of the pterygomasseteric sling.

The rotational change in the proximal segment of a mandibular osteotomy has been implicated in relapse by multiple clinicians who believe that the muscles of the pterygomasseteric sling reassert themselves after the surgery.[59,60]

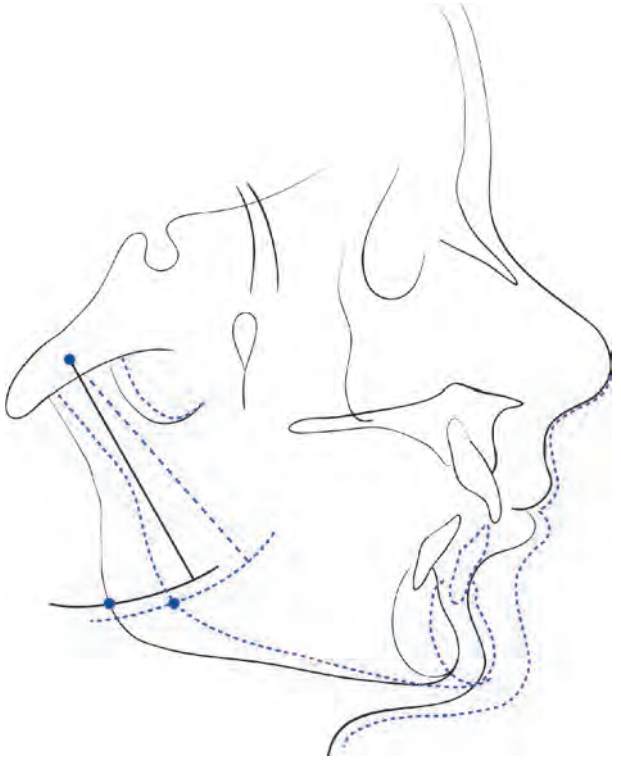


FIGURE 57-8. Gonial arc used for showing condylar position change with a mandibular osteotomy. (Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1422.)

Therefore, there has been an emphasis on carefully repositioning the proximal segment as close as possible to its preoperative position, which is a difficult task because there are many variable influences acting on condylar position that are different when records are obtained in an upright and awake position as opposed to supine under general anesthesia. Unfortunately, a correlation between mandibular ramus positioning and relapse in the case of mandibular advancements has never been demonstrated. A few studies have shown a relation between relapse of mandibular setback surgeries and the position of the vertical ramus.[61] It has been noted in these surgeries that the degree of clockwise rotation of the proximal fragment in a sagittal osteotomy seems to relate to the amount of forward relapse of the distal segment. Franco and colleagues[61] theorized that both a stretching of the medial pterygoid muscle as well as the elongation of the anterior fibers of the masseter and temporalis muscles from the clockwise rotation of the proximal segment can contribute to relapse in lengthening the muscles of the pterygomasseteric sling. This can result in a change in mandibular position as has been documented by Yellich and coworkers.[62] The degree to which this is active in orthognathic surgery remains unclear.

The contribution of the suprahyoid muscles to relapse in mandibular advancement surgery is equivocal, with many clinicians supporting an existence of this relationship. Ellis and Carlson[63] demonstrated in monkeys that relieving the suprahyoid muscles from the symphysis of the mandible decreased the amount of relapse when the mandibles were advanced. Clinical studies, however, have failed to show a relation between suprahyoid myotomies and relapse.[64,65] Animal studies have also demonstrated that adaptive changes occur in the connective tissues at the muscle-tendon and tendon-bone interfaces but only with large advancements.[66]

The belief, however unsubstantiated, that muscle pull in some way does affect the stability of mandibular osteotomies has led to a variety of recommendations. Historically, the most common method advocated is the attempt at minimizing the change in muscle position and length. The cutting of muscle attachments, such as has been recommended for the suprahyoid group, has the potential for increasing morbidity. Without significant evidence that this is of much value, this additional surgery cannot be justified. However, there has been recognition that muscles and their attachments seem to adapt fairly quickly if the bone is held rigidly for a long enough time.[66,67] It is important to recognize that intermaxillary fixation (IMF) does not provide a completely stable method of bone fixation, especially if the teeth have been under active orthodontic movement. In addition, the greatest amount of relapse of mandibular osteotomies seems to occur in the first 3 to 6 weeks after surgery. Whatever the causes of the instability during this time, several techniques have designed to provide increased stability for this initial period, to improve the stability of mandibular osteotomies. Primarily two techniques have been attempted: external supporting mechanisms and internal rigid fixation. The only external technique that has been of much value has been the wiring technique termed *skeletal fixation.* With this procedure, the bony skeletons are tied to one another, circumventing the periodontal ligaments of the teeth. This has been used with IMF, maintaining mandibular immobilization for 6 to 8 weeks (Figure 57-9).[58,67,68] The alternative procedures of internal rigid fixation techniques using plates or screws are discussed in the next section.

MANDIBULAR OSTEOTOMY TECHNIQUES

Mandibular Ramus Osteotomies

Osteotomies in the vertical ramus have been the preferred technique for correcting developmental deformities of the mandible. This preference has increased with closer cooperation between orthodontists and surgeons inztreating dentofacial deformities. Most of the time, the dental arch discrepancies can be corrected orthodontically, leaving to the surgeon the responsibility for moving the coordinated dental arch into its appropriate position, as dictated by functional and aesthetic demands. Operations in the vertical ramus, therefore, are considered primarily when the dental arch as a unit has to be moved. As previously noted, there are

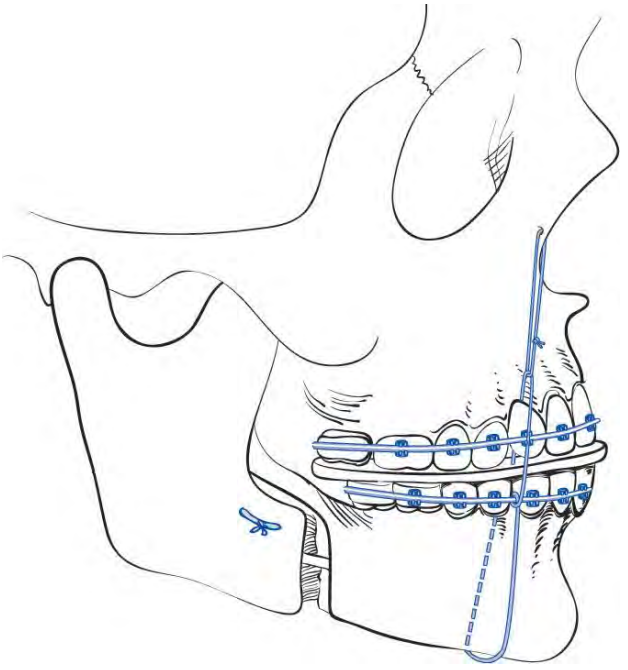


FIGURE 57-9. Skeletal fixation used with maxillomandibular fixation or mandibular advancement. (Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1423.)

numerous techniques for osteotomies of the ramus, but essentially three different procedures, with minor variations, are currently accepted techniques.

Vertical Ramus Osteotomy

Osteotomies extending from the sigmoid notch vertically behind the IAN foramen to the inferior border or angle have had several different names, but generally, the VRO seems to describe the procedure best (Figure 57-10). This osteotomy was initially done through an extraoral approach, but with the development of small offset oscillating blades with a long shaft, and adequate retraction, the intraoral route has become preferred.

Indications

The VRO has been limited to deformities requiring the mandible to be set back for mandibular horizontal excess or to be rotated for mandibular asymmetry. Robinson and Lytle[69] stated that this osteotomy can be used for mandibular advancement, but generally this recommendation was not accepted owing to questionable stability. Hall and McKenna[70] later revived this indication for minor (2–3 mm) advancements.

Technique

When preparing for an intraoral VRO, one needs to closely evaluate the panoramic and lateral head films for the position

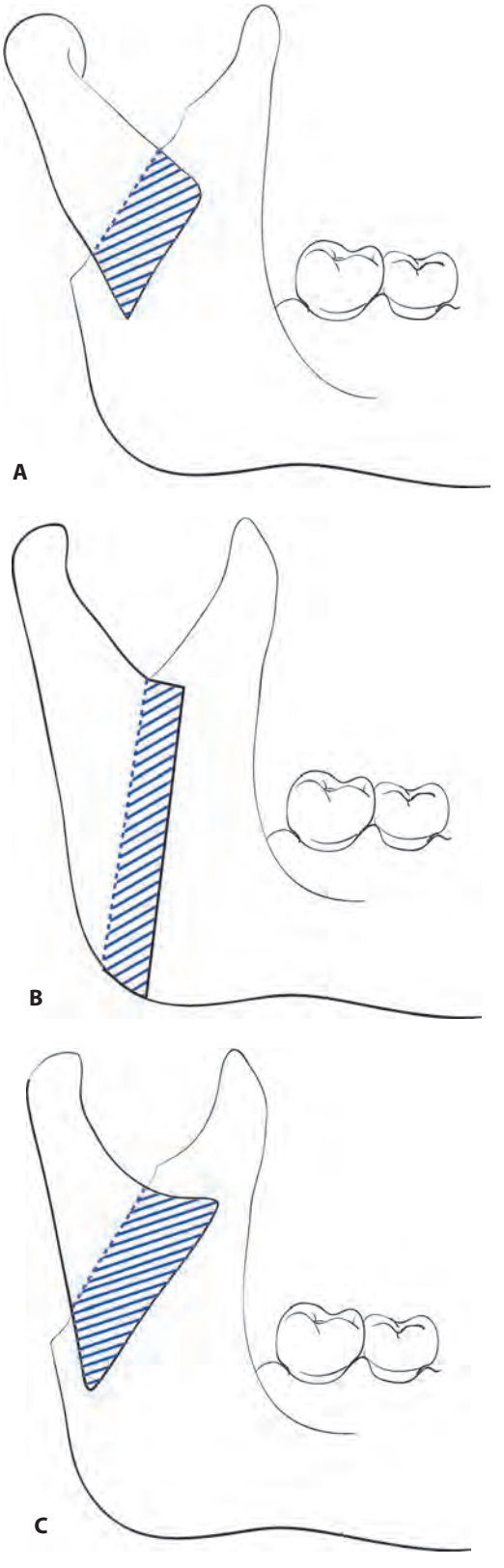
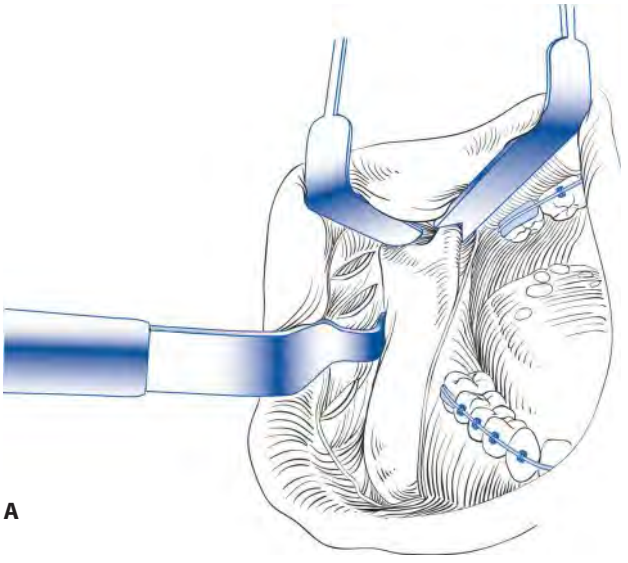
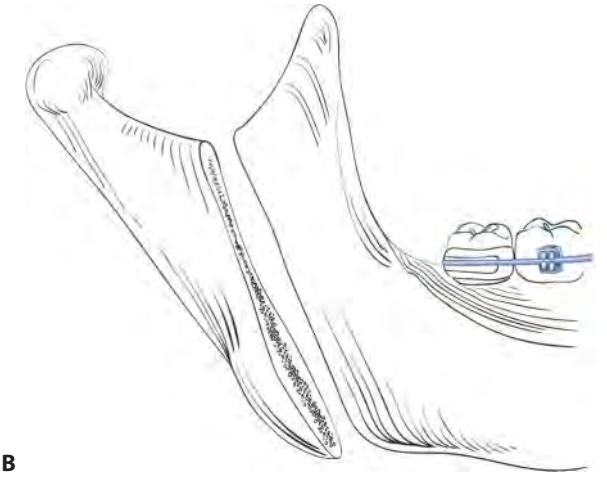


FIGURE 57-10. Different lengths of the osteotomy in the vertical subcondylar osteotomies (VSOs). (Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1423.)

of the inferior alveolar foramen relative to the posterior border of the mandible. The incision is made in the mucosa from midway up the anterior border of the ramus to the first molar area. The periosteum is reflected laterally to expose the entire ramus, with the exception of the condyle neck and coronoid tip. The posterior and inferior borders can be cleared of periosteum; but muscle attachments at the angle are generally difficult to elevate and should be left to ensure blood supply to this area. A special retractor, Merrill-Levaseur, fits around the posterior border and retracts tissue laterally, so that an oscillating saw can be used (Figure 57-11A). Also, Bauer retractors, left and right, can be used superiorly in the sigmoid notch and inferiorly in the antegonial notch for additional retraction and visualization.

The saw chosen should have a rounded blade that is set at an obtuse angle to the long shaft of the drill to facilitate the osteotomy. The blade should be used first to score the proposed osteotomy line on the lateral cortex. This line is then closely checked for its position relative to the sigmoid notch, posterior border, and angle. The use of the so-called antilingula has been proposed as the landmark for the mandibular foramen, but has generally fallen into disfavor, because of both the difficulties with its identification and its lack of predictable relation to the foramen.[12,71,72] The cut should be made no more than 5 to 7 mm anterior to the posterior border at the anticipated level of the foramen, using the retractor as a guide to the posterior border.[73] The cut is carried through the medial cortex, starting in the middle of the ramus. It is carried superiorly to the sigmoid notch and then finished at the inferior border (see Figure 57-11B). In considering the three locations of the osteotomy cut, the vascular hazards should be recognized, including the maxillary artery superiorly, the inferior alveolar artery in the middle, and the facial artery inferiorly. Therefore, the surgical ability to control a bleed from one of these three sources should factor into the decision regarding where to begin and where to end the osteotomy. As the cut is completed, anterolateral tension is maintained on the Merril-Levaseur retractor so that the proximal fragment will be brought out laterally and prevent medial displacement. A straight clamp can be used to rotate the segment laterally after the cut is made and then to stabilize it while periosteum and muscle are stripped from the medial cortex down to the angle (see Figure 57-11C). Again, a small attachment is left at the angle to ensure a blood supply. This





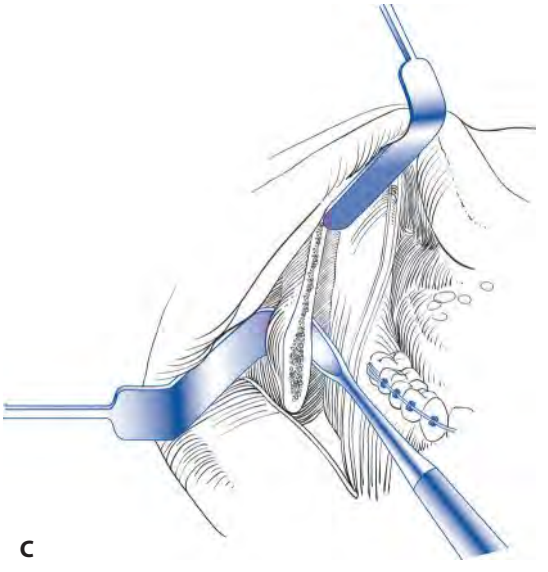


FIGURE 57-11. The intraoral VSO. **A,** Exposure. **B,** Vertical ramus osteotomy. **C,** Proximal fragment displaced laterally. (**A-C,** Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1424.)

proximal fragment can be held forward and laterally by a small gauze pack while the opposite side is being completed. If the proximal fragment is lost medially, it usually can be brought into the field with the help of a small periosteal elevator that is inserted posteromedially at the level of the sigmoid notch while the distal fragment is being pulled forward. The mandibular dentition is brought into its new position after the completion of both osteotomies, as established by a preformed occlusal splint and stabilized with maxillomandibular fixation.

Attention is directed back into the wound and toward placement, or reduction, and stabilization of the proximal fragment. Wire osseous fixation is generally not needed, although advocated by some surgeons. Most important is achieving as broad a bone contact area as possible, without displacing or rotating the condyle. Adjustment of the lateral cortex of the distal fragment may be performed with a straight fissure bur or small acrylic bur to permit the proximal fragment to lie as flat as possible against the vertical ramus. Care should be taken to ensure that the long axis of the proximal fragment does not differ considerably from its preoperative position. After a thorough irrigation of the wound, the mucosa is closed with a running stitch, using a resorbable suture. No drains or external dressings are placed, and IMF is used for 4 to 6 weeks, depending upon patient age and other factors. Postsurgical radiographs should be taken as soon as possible to confirm that the condyles have not been displaced or dislocated. A small amount of forward and downward position of the condyle is common, and this generally resolves during the period of IMF (Figure 57-12).

Preoperative submentovertex radiographs have been recommended to identify divergence of the posterior border. It has been suggested that an angle smaller than 130 degrees produces such a significant surgical problem that in this type of patient, a VRO procedure should be avoided and another technique used. The use of this radiograph as a criterion for choosing this technique has been questioned, but some still believe that it can be used to identify the more difficult cases.[73,74]

A large number of variations of the VRO technique have come in the osteotomy design. Oblique versions, with the cut ending above the angle, have been described by many clinicians, with the only apparent benefit being the relative ease in performing the technique. Theoretically, there should also be less chance of damaging the IAN, but there has been no study to confirm this benefit. Interestingly, the one potential drawback, that of decreased skeletal stability, also appears to be difficult to demonstrate.[75] In contrast with this shorter cut, surgeons have recommended that a larger portion of the inferior border be left with the proximal fragment, especially in the larger mandibular setbacks.[70] This permits a good attachment of the medial pterygoid muscle to be maintained at the mandibular angle, which has been claimed to help seat the condyle in the fossa as the patient wakens from anesthesia. This variation, including the use of 8 weeks of IMF, is claimed to decrease one of the problems of the intraoral VRO, specifically that of condylar sag and the resulting open

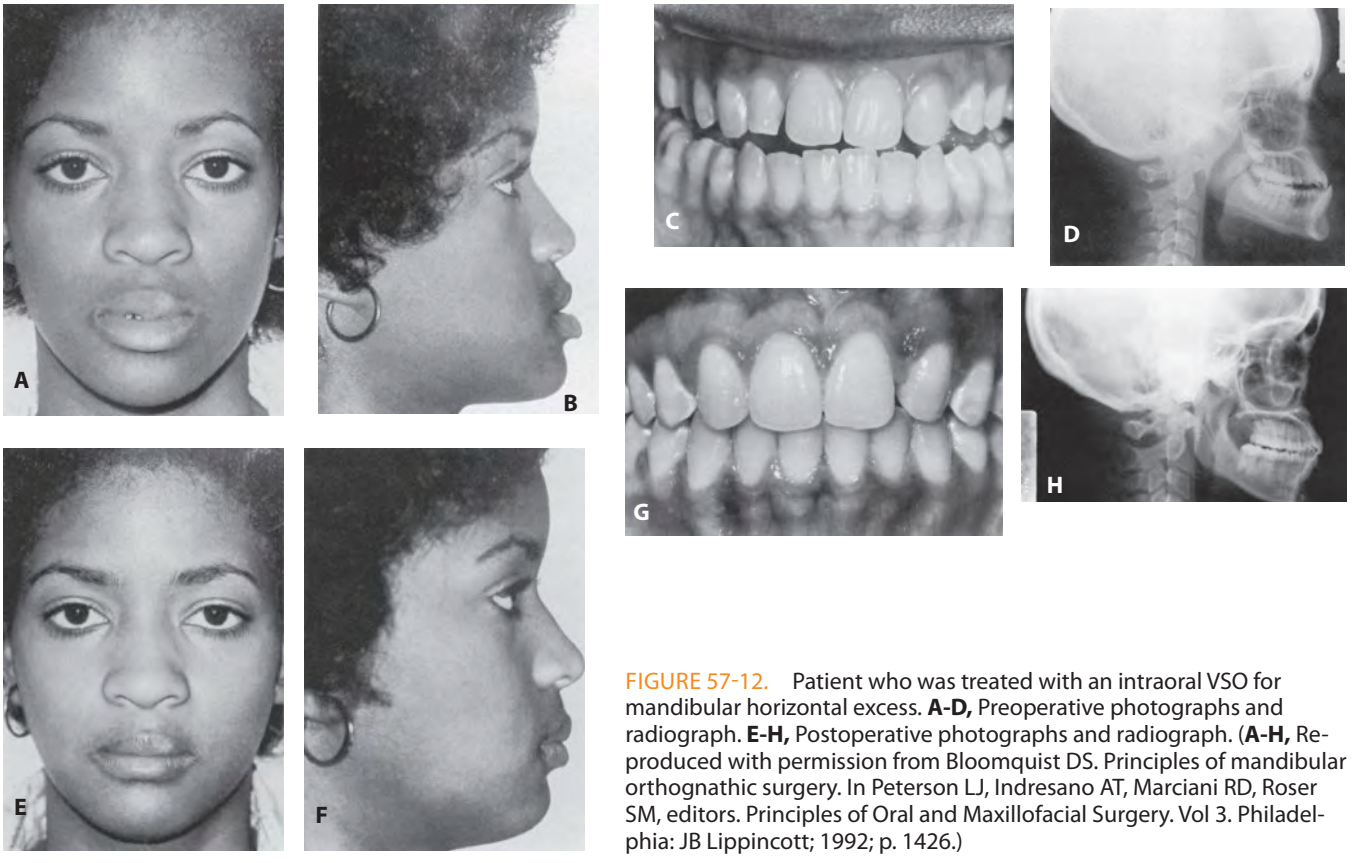


FIGURE 57-12. Patient who was treated with an intraoral VSO for mandibular horizontal excess. **A-D,** Preoperative photographs and radiograph. **E-H,** Postoperative photographs and radiograph. (**A-H,** Reproduced with permission from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1426.)

bite that can occur on the release of fixation. Unfortunately, no clinical data have been reported that support this claim. The use of osseous wire fixation has been advocated to ensure the seating of the condyle. Again, no study comparing wire osseous fixation with no fixation has shown any advantage for the use of the wire.[76–78] This may be explained in the intraoral procedures by the technical difficulties of wire placement. However, Ritzau and associates[77] showed in an excellent prospective study that even from an extraoral approach, the position of the condyle in the fossa is not improved with the use of wire osseous fixation.

The effect of the temporalis pull on relapse has led to other recommendations that include either stripping the temporalis attachment completely off of the coronoid process or coronoidotomy. The use of coronoidotomy has been recommended by some clinicians for large setbacks, with a few using this modification routinely. The advantage of the coronoidotomy relative to prevention of relapse has not been studied, but the stability of the intraoral vertical subcondylar ramus osteotomy (IVRO) with coronoidotomy, compared with the sagittal split osteotomy (SSO) of the vertical ramus in mandibular setbacks, has been investigated and the IVRO seemed to be more stable.[79]

The use of the inverted-L osteotomy is another method used to neutralize the temporalis influence. This modification of the IVRO requires stripping of the medial periosteum to identify the lingula so that a horizontal cut can be made without increasing the risk of damaging the IAN. A further modification of the inverted-L osteotomy has been the use of rigid internal fixation. Although technically a difficult surgery, it permits the early release from IMF. Unfortunately, there are no long-term studies that evaluate stability of any of the inverted-L techniques.

Alternative Techniques

The major variation of the described VRO technique is the use of an extraoral approach. The soft tissue incision is similar to that commonly used for an external approach to a fracture of the mandibular angle, with an approximately 4-cm incision made 2 cm below the angle and the inferior border of the mandible (Figure 57-13A). A combination of sharp and blunt dissection is used to access the inferior border of the mandible. Care is taken to avoid damaging the marginal mandibular branch of the facial nerve. After incising through the periosteum, the bone cuts are similar to those that have been described (see Figure 57-13B).

The external approach has been advocated for large mandibular setbacks of greater than 10 mm, difficult asymmetries, or large vertical moves in patients with unusual facial structure. Except for the risk of the scar, the risks of this technique have been reported as being comparable with the intraoral technique.[50,80]

Complications

Stability

Postoperative change in skeletal and dental position after the use of VRO in the treatment of mandibular horizontal excess

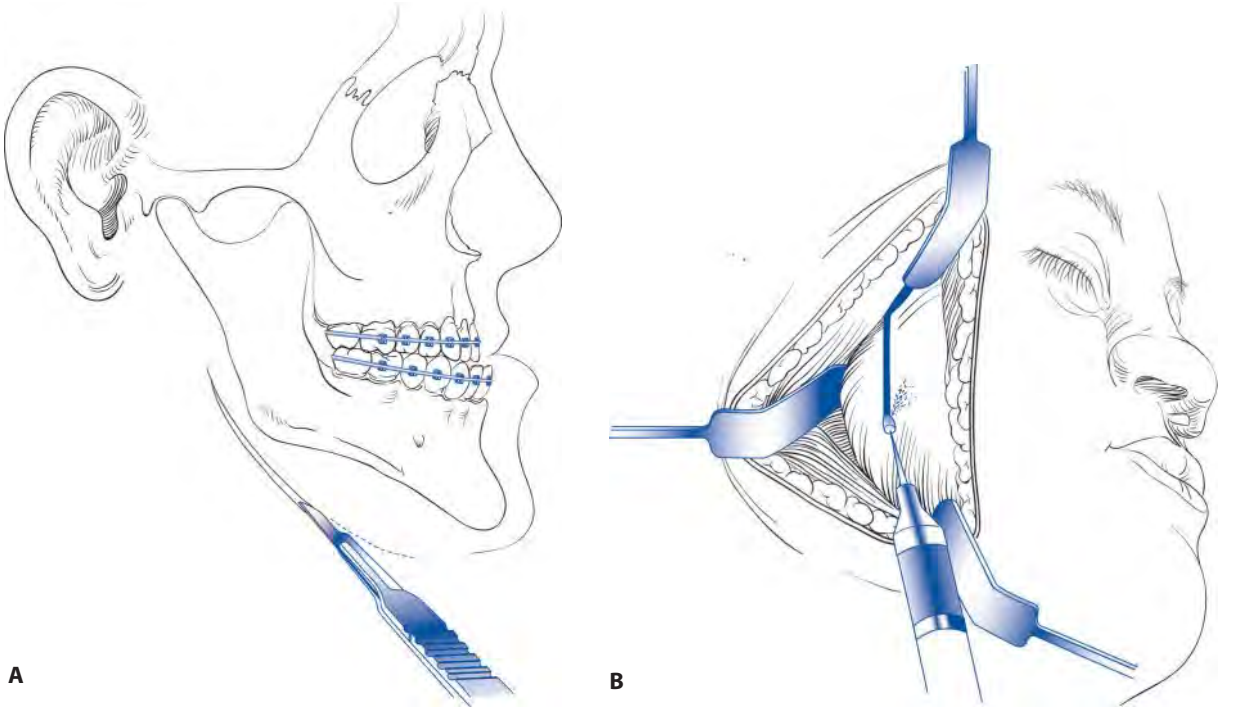


FIGURE 57-13. **A** and **B,** The extraoral VSO. (**A** and **B,** Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1428.)

has received much attention. Goldstein[81] was the first to use serial cephalograms to evaluate the postoperative change of the mandible after surgical correction of the mandibular prognathism. He noted the anterior relapse that has now been well documented. Poulton and colleagues[82] recommended overcorrecting the mandibular setback by 2 mm to provide for the relapse that they noted. This amount of average relapse has, surprisingly, remained fairly consistent throughout the history of this technique, even though technical changes in procedures have been made.[80,83] Stella and coworkers[84] noted that the variation in the amount of relapse in mandibular setbacks was large and attempts at identifying controllable variables should be made. They suggested that the proximal fragment rotation affects the short-term pogonion changes, although they did not present any corroborative research. The finding that the pogonion tends to move posteroinferiorly during IMF has been well documented.[52] This movement, later followed by the anterosuperior "relapse" that occurs after skeletal fixation wires, does seem to stabilize the initial movement but does not affect the long-term relapse.[50,51,56]

Vertical instability of the VRO develops soon after the release of IMF in many patients. This problem was initially attributed to the "condylar sag" seen on x-ray films taken soon after surgery.[73] Although condylar sag may be one cause of relapse, a major contributor seems to be insufficient time of IMF. The VRO is generally not considered an appropriate surgery for correction of anterior open bite. In Scandinavian countries, however, this surgical procedure has been successfully used for patients with mandibular horizontal excess and anterior open bite.[85]

Nerve Damage

The chance of damaging the marginal mandibular branch of the facial nerve is one of the reasons given by several surgeons for avoiding the extraoral approach to the VRO. This concern, however, seems to be unsubstantiated, in that almost all of the clinicians reporting on the results of this approach have noted very little, if any, motor nerve damage. Damage to the IAN, however, is a concern in using a VRO. The incidence of trauma to the IAN at the time of surgery has been reported to vary from being "rare" to occurring 36% of the time.[70] Long-term sensory defects have also been reported to vary from none to 35%.[45,86] These apparent discrepancies can be explained by the differences in the sensitivity of the measurement techniques; in addition, there is a wide variation in the time after surgery during which the patients were tested. Other variables, such as whether the osteotomy was approached intraorally or extraorally as well as variations in the length of the cut, theoretically could affect the incidence of sensory problems, but comparison studies have not been done. From studies that have been done, the incidence of damage to the IAN is low with the VRO compared with the SSO.[86,87] The patient, however, should be warned that short-term sensory loss is a definite risk and permanent paresthesia is possible.

TMJ Dysfunction

Interesting literature has been published on changes in TMJ function after a VRO. These have included a number of radiographic studies documenting positional change of the condyle relative to the fossa. Radiographically, there is an initial downward and forward movement of the condyle, with a subsequent tendency to return to its preoperative position.[77,78,82] Sometimes, a double contour of the condyle appears approximately 6 months postoperatively, which has been attributed to condylar remodeling after the surgery.[88] Remodeling of the glenoid fossa has also been documented.[78]

In one early review of 100 cases, 6 patients were reported to have TMJ problems at 1 year after surgery.[89] A form of the VRO has been used to treat patients with TMJ pain and dysfunction. It appears that the VRO does not put the TMJ at any significant risk, and it may in fact be salutary for patients with preexisting TMJ dysfunction.[90–92]

Other Complications

Among the other reported complications of the VRO, vascular necrosis of the proximal segment seems to be the most potentially devastating. The maintenance of some muscle attachment to the angles makes this possibility unlikely.

Inverted-L and C Ramus Osteotomies

Osteotomy designs in the vertical ramus that include both the condyle and the coronoid in the same segment have varied from Blair's simple horizontal osteotomy to the modified C osteotomy of Hayes. The horizontal osteotomy of the vertical ramus has generally fallen into disuse because of the substantial relapse potential, but many of the remaining suggested variations continue to have treatment value. The two procedures that seem to be the most popular are the inverted-L and the C osteotomites. Both are generally approached extraorally, although intraoral variants are possible.[93] Clinical studies of either technique are rare, but those that exist seem to demonstrate reasonable success in correcting skeletal deformities with minimal complications.

Indications

The C osteotomy is generally reserved for treatment of horizontal mandibular deficiency, with some authors suggesting that it can be used to close an anterior open bite. The inverted-L, however, has been used for the correction of most types of mandibular horizontal discrepancies, including an anterior open bite. Generally, advancements of the distal segment with either technique require bone grafting to ensure adequate bone union.

Techniques

The basic techniques for an extraoral approach to perform a C and an inverted-L are the same, with the only modification being the inferior horizontal cut in the C osteotomy. For that reason, the inverted-L is described first, with various modifications of the C discussed later. The patient is prepared and

draped, such that access to both the mouth and the submandibular incision area can occur without contamination of the skin wound by oral organisms. This can be accomplished in a variety of ways, but most surgeons use a plastic drape with adhesive on one edge to separate the two areas. The external drapes should be arranged so that they allow turning of the head for access to the submandibular wounds as well as access to the mouth.

The submandibular incision is made 2 cm below the angle and inferior border of the mandible. The posterior portion is curved superiorly to follow the cervical skin line as well as to improve the access to the entire vertical ramus. Generally, the incision is approximately 6 cm in length. Sharp dissection is used down through the platysma, and then blunt dissection is begun to minimize risk to the marginal mandibular branch of the facial nerve. The incision through the pterygomandibular sling and periosteum is made along the inferior border and is carried around the angle and up the posterior border about 2 cm. Periosteum and attachments for the masseter are completely stripped off the lateral cortex of the vertical ramus up to the level of the sigmoid notch. Very little periosteum is stripped off the medial side, especially at the angle, to retain as much blood supply as possible to the proximal fragment. The posterior vertical osteotomy is made at least 7 mm in front of the posterior border and extends to a point of the inferior border just in front of the angle. The horizontal cut is made above the anticipated position of the inferior alveolar foramen (Figure 57-14A). As mentioned previously with the VRO, it is wise to have a good radiographic view of the ramus so that the position of this foramen can be more accurately located. It is helpful to review the study by Reitzik and associates[72] of the position of the foramen relative to the lateral landmarks to lessen trauma to the neurovascular bundle.

Once the cuts are made, the medial periosteum may have to be elevated from some of the distal fragment to allow its advancement. Moist gauze is placed in the wound, and a similar procedure is done on the opposite side. After completion of the contralateral side, drapes are pulled back and shifted such that the oral cavity can be entered to place the mandibular teeth into the new occlusal position and secured with IMF. The surgeons who are involved in the intraoral fixation should change gloves and surgical gowns before the drapes are replaced so that the skin incisions can again be approached. The next step varies depending on the type of mandibular movement that occurred; however, the importance of maintaining the proximal fragment close to its preoperative position remains. If the distal segment is set back, the proximal segment has to be overlapped laterally (see Figure 57-14B). As described with the VRO, some adjustments of the lateral cortex of the distal segment may be necessary to permit passive position of the proximal fragment as well as to provide a good area of bone contact. The use of some form of fixation is generally recommended, although the use of no interosseous fixation has been suggested.[93] The type of osseous fixation varies widely; however, rigid internal fixation with metal plates or mesh secured with screws has become more popular.[93] After irrigation, the wound is closed in layers by whatever suturing method and material the surgeon prefers. Care should be taken to ensure hemostasis as the wound is closed. If there is any concern about hematoma

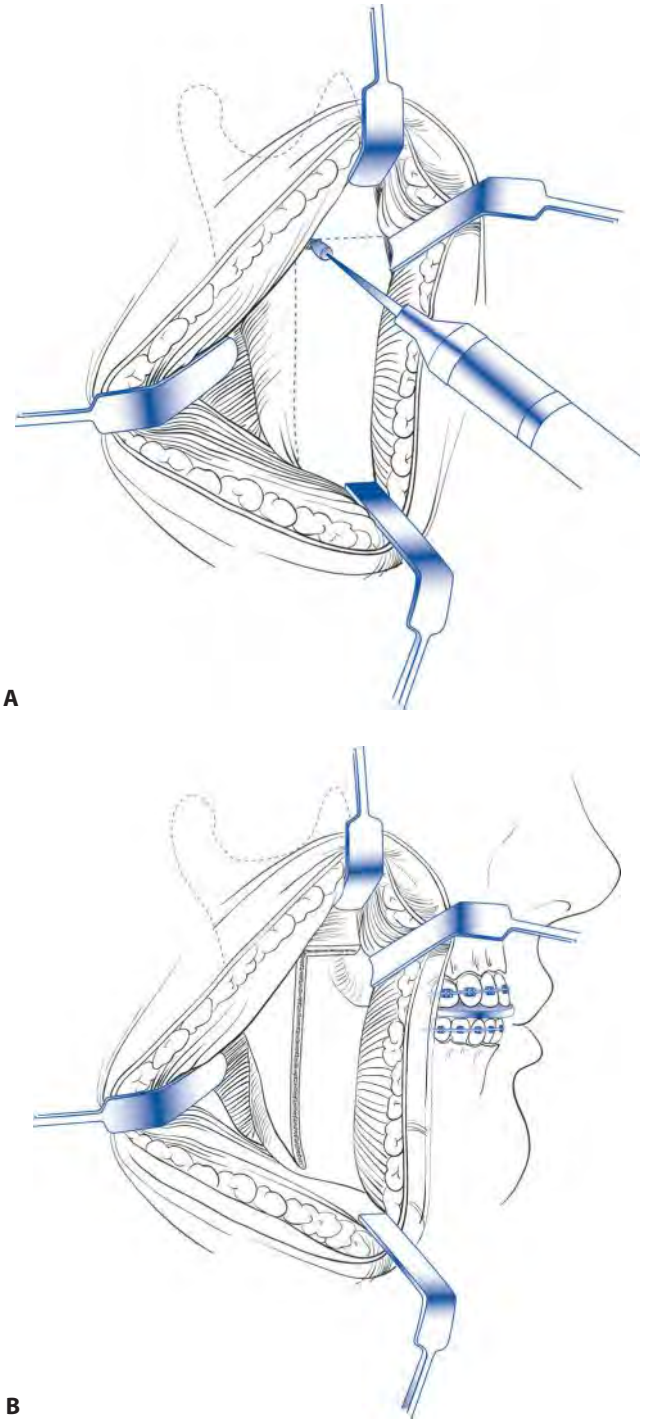


FIGURE 57-14. A and **B,** The extraoral inverted L osteotomy. (**A** and **B,** Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1430.)

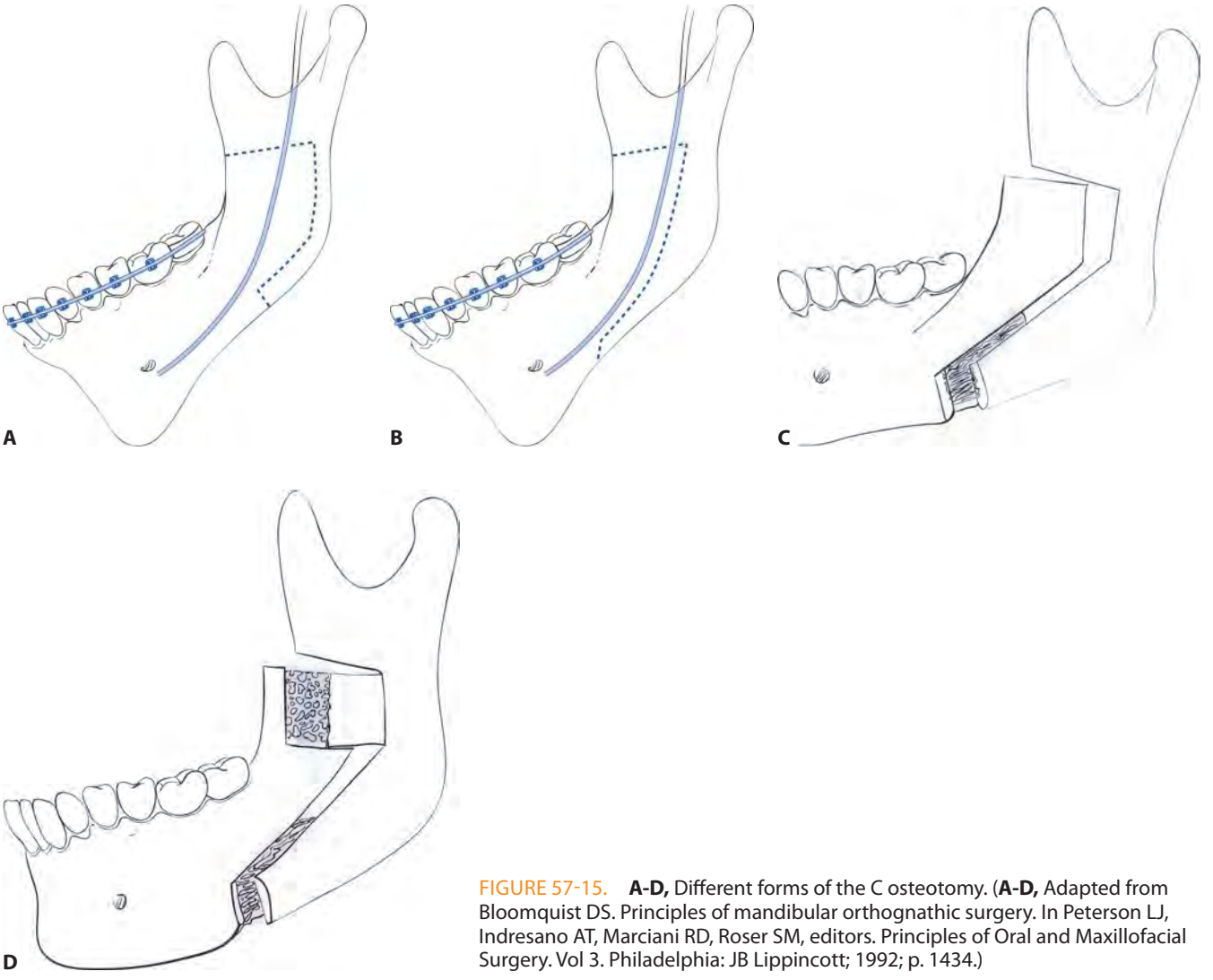


FIGURE 57-15. **A-D,** Different forms of the C osteotomy. (**A-D,** Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1434.)

formation, a small drain should be placed. External pressure dressings are maintained for 24 to 48 hours. When the bone has been stabilized with wire fixation, IMF is used for at least 6 weeks and preferably 8 weeks.

Alternative Techniques

The most commonly used variation of the previously mentioned technique is the C osteotomy. This technique was first described jointly by Caldwell and coworkers[18] in an article reviewing their experiences with what they called a *vertical-L osteotomy.* They described a variation of their basic vertical-L with the addition of a horizontal cut that extended forward from the vertical cut below the inferior alveolar canal (Figure 57-15A). This permitted a larger amount of bone contact when the mandible was advanced. They also realized the problems caused by advancing the coronoid process and recommended either cutting the coronoid loose (coronoidotomy) or including it with the proximal segment (C osteotomy). Arcing the inferior cut was suggested to permit increased bone contact as the distal segment was advanced (see Figure 57-15B).[94] Unfortunately, the proposed arc cannot always be made because the position of the neurovascular bundle may interfere. Sagittal splitting of the inferior limb of the C osteotomy was proposed both to increase the bone contact area when the mandible was advanced and to decrease the problem of "notching" of the inferior border (see Figure 57-15C).[19] This latter problem, which is noticeable in some patients, is caused by the defect along the inferior border resulting from the advanced distal segment of the mandible. A further variation used to improve bone healing includes a bone graft taken from the lateral cortex of the distal segment and transferred back into the gap of the midramus area (see Figure 57-15D). The coronoid process has also been recommended as a free graft in to this defect.[20]

A further major modification of the described techniques is the use of rigid internal fixation.[95–97] The use of vitalium mesh with two screws in each fragment has been demonstrated as being effective, but any rigid plate with screws can be used to permit the early release of maxillomandibular dental fixation.

Complications

The skeletal stability of the inverted-L and its modifications, unlike the VRO, seems to be technique-sensitive to the type of fixation used. Like stability studies for almost all aspects of orthognathic surgery, controlled clinical studies are nonexistent, and the comparison of techniques by a single institution, if reported, lacks sufficient numbers of patients to make valid conclusions. Farrell and Kent[93] evaluated inverted-L and C osteotomies and reported skeletal relapse similar to what had been reported for the BSSO. However, because there were different criteria for the use of these two types of osteotomies, comparisons between them are questionable. Greebe and Tuinzing[98] compared the stability of mandibular advancement by way of an inverted-L or a BSSO. With only a few patients, they did not show any difference between the groups, but they did claim that skeletal relapse was dependent on the ratio of posterior facial height to anterior facial height. The largest studies of the stability of the inverted L included the use of rigid internal fixation in patients who had the mandible advanced. These seem to demonstrate that the use of rigid fixation for this type of procedure is more stable than simple IMF.[96,97] However, statements relative to stability of mandibular setbacks or the closure of open bites cannot be made, although a few authors seem to advocate these skeletal deformities as indications for the use of the inverted-L osteotomy.[96,99]

The incidence of facial nerve damage has not been mentioned in any review of these techniques, although it can be inferred to be quite low, given the reports for the external approach to the VRO. The incidence of damage to the IAN should be higher than the extraoral VRO because of the horizontal portions of the osteotomy, but Reitzik and colleagues[96] reported only a 6% incidence of permanent anesthesia with inverted-L osteotomies. The incidence of unsightly scars, which many clinicians claim deters them from using this approach, is unknown with this group of osteotomies.

Bilateral Sagittal Split Osteotomy

The BSSO of the vertical ramus has in a relatively short time become the predominant orthognathic procedure of the mandible. Schuchardt[23] is credited for the use of an intraoral approach to what was called the "step" osteotomy of the vertical ramus. Specifically, he described parallel horizontal cuts through the cortex of the vertical ramus, the medial cut located above the lingual, and the lateral cut made about 1 cm below that level. A split was then made between these two cortices, and the distal segment could then be advanced or set back. Lane evidently described a very similar procedure earlier, but it probably was done extraorally (see Figure 57-4).[22] The singular credit for improving on this osteotomy, as well as being its strongest advocate, belongs to Obwegeser, who together with Trauner in 1955, described a sagittal split of the vertical ramus.[21] This intraoral technique included the medial horizontal cut above the lingula, but the lateral horizontal cut was lower than that of Schuchardt and extended to a point just above the angle, at least 25 mm below the lingual cortical cut (Figure 57-16A). A wide-splitting osteotome was then used to obtain a split between the cortices, with care taken to preserve the IAN and vascular bundle. This procedure was later slightly modified by Obwegeser by angling the lateral cut more toward the inferior border of the mandible (see Figure 57-16B). The major modifications still in use today were suggested by DalPont.[24] The change commonly attributed to DalPont is the forward extension of the vertical cut through the lateral cortex just behind the second molar. But he also suggested the use of a medial cut that extends just past the lingula so that the posterior split would occur in the mylohyoid groove instead at the inferior border. Multiple other modifications have been suggested, but surprisingly, the present-day osteotomy remains very similar to that initially described by Obwegeser and DalPont. Also in use today is the description by Hunsuck[25] to terminate the medial horizontal cut in the retrolingular fossa, and not attempt to cut through the posterior border; this would ensure that the induced fracture would occur posterior to lingula, in the thin bone, rather than in the thick cortical bone of the posterior border of the ramus.

Indications

The BSSO has been advocated for all types of mandibular deformities and for each possible mandibular movement that includes the entire horizontal ramus of the mandible.

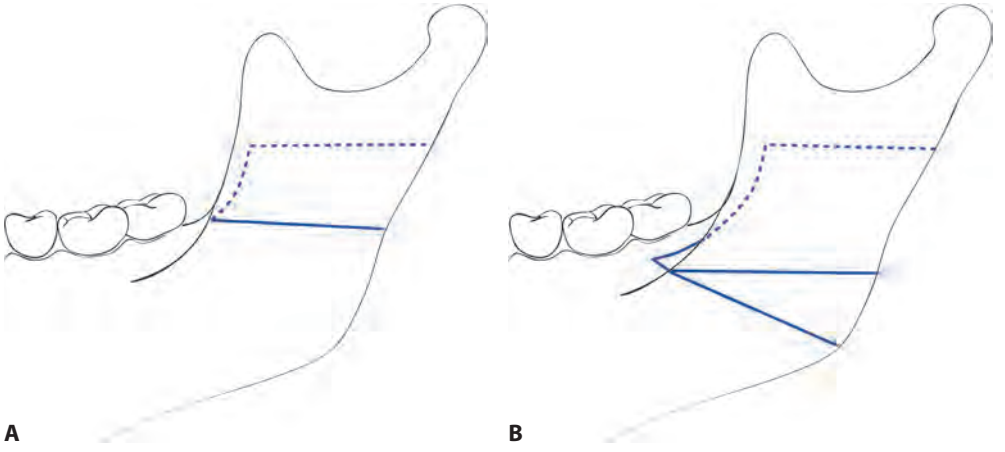


FIGURE 57-16. Obwegeser's osteotomies of the ramus. **A,** Original cortical cuts. **B,** Later lateral cortex cuts. (**A** and **B,** Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1435.)

Technique

This osteotomy has had multiple variations described, as would be expected for such a popular procedure, but many of them are based upon one surgeon's individual preferences, and the effect of these alterations of the osteotomy on the outcome of the procedure is questionable. Therefore, only the basic procedures are outlined, as well as the significant modifications that have been shown to affect the outcome or seem to have a solid theoretical foundation.

The incision is made on the anterior portion of the vertical ramus, midway between the occlusal planes. It is carried downward through the middle of the retromolar fossa to a point about 5 mm behind the second molar. Then the incisions wind laterally and forward to a point distal just to the first molar (Figure 57-17A). The incision should be kept lateral enough to allow easy closure of the wound later in the procedure when the teeth are in IMF. The periosteum is reflected to expose the lateral cortex of the mandible down to the inferior border for the vertical cut only. The exposure should be limited posteriorly to maximize the blood supply to the proximal fragment; this usually means the exposure ends at about the antegonial notch, and the masseter muscle is reflected minimally. A lateral channel retractor, or Minnesota retractor, can be placed at this time to assist in retraction as the periosteum is elevated from the retromolar area up the anterior border of the vertical ramus. Periosteal elevators are used during this portion of the surgery. The attachment of the temporalis muscle can be tenacious, but it may be reflected off of the coronoid process to the level of the sigmoid notch to ensure adequate access for the medial horizontal cut. Most times, this means stripping approximately 1 cm of the temporalis attachment off the anterior border of the coronoid process. The periosteum is then elevated from the medial surface of the vertical ramus, starting at about the level of the sigmoid notch and extending back to the medial flare at the start of the condylar neck. Inferiorly, the medial cortex is exposed to the lingula, with care being taken to minimize trauma to the IAN just below this area. The periosteal elevation can be extended inferoanteriorly along the internal oblique line to the distal of the second molar to allow better exposure of the osteotomy site, with care taken to protect the lingual nerve. A variety of retractors are available for the protection of the medial soft tissues and IAN, but it is wise to choose one that permits as much visualization as possible while at the same time protecting the soft tissue (see Figure 57-17B). Excessive medial retraction should not be done in order to minimize nerve damage. It should be noted that, most commonly, it is not necessary to carry the medial exposure to the posterior border of the vertical ramus.

The osteotomy is begun by making a horizontal bone cut through the medial cortex of the vertical ramus that extends from a point just posterior to and above the lingula to the anterior border of the ramus. Anteriorly, the cut is made about halfway through the ramus, but in the concavity above and behind the lingula, it should be shallow to allow the posterior medial split to initiate in the mylohyoid groove (see Figure 57-17C). Sometimes, it is helpful to use a large round or acrylic bur to remove bone from the internal oblique ridge so that the depth of this concavity can be visualized. Occasionally, at the level of this horizontal cut, there is no significant cancellous bone to delineate the cortices. Here, the use of a half thickness of the ramus is the most practical guideline for judging the appropriate depth of this cut.

The vertical cut through the buccal cortex is generally made just distal to the second molar and extends from the inferior border superiorly to the external oblique ridge. Sometimes, the mandible is thin and the external oblique ridge ends at the distobuccal aspect of the second molar. In this case, the superior aspect of the vertical cut should be posterolateral enough so that the roots of the second molar are not placed at risk. The cut should be as close to perpendicular to the inferior border as possible and extended just into cancellous bone. Caution must be used such that the cut is not made any deeper because the IAN may be just medial to the buccal cortex in this region.

The vertical and horizontal cortical cuts are connected, starting superiorly at the anterior border of vertical ramus and continuing down just inside the external oblique ridge to the vertical cut (see Figure 57-17D). Again, the cut is made into cancellous bone, when at all possible, with the superior part of this connection being as deep as possible, especially if no cancellous bone is present. This will minimize the chance of an inadvertent fracture of the medial cortex. Some surgeons recommend that the corners between the horizontal and the connecting cuts, as well as the vertical and the connecting cuts, be rounded again to minimize a fracture. Difficulty is encountered often and can occur if a third molar is present and has been planned to be removed at the time of surgery. It is generally prudent to plan for the mandibular third molars to be removed well in advance of the SSO because they can make the surgery more difficult. Preferably, these third molars should be removed at least 9 to 12 months in advance of the SSO. Even experienced surgeons who remove third molars at the same time as the osteotomy may have difficulty in obtaining a successful split, but in general, the presence of third molars is a significant risk factor for bad splits, such as inadvertent buccal or lingual cortical plate fractures. Also, the bone defect left by removal of the third molar can make the use rigid fixation with bicortical screws more difficult and also weaken the bone distal to the second molar, resulting in less support.

Techniques vary widely regarding how the actual bone split is accomplished. The method described here is an attempt to be as universal as possible. First, steps are taken to ensure that the limits of the split occur as defined by the horizontal and vertical bone cuts. A narrow (4-mm) thin osteotome may be driven along the horizontal cut and directed so that it cuts through the medial cortex above and behind the lingula. It is also used to ensure that the split at the base of the vertical cut is started through the midpoint of the inferior border. Many surgeons also use this type of thin osteotome to "step" along the connecting cut to help ensure

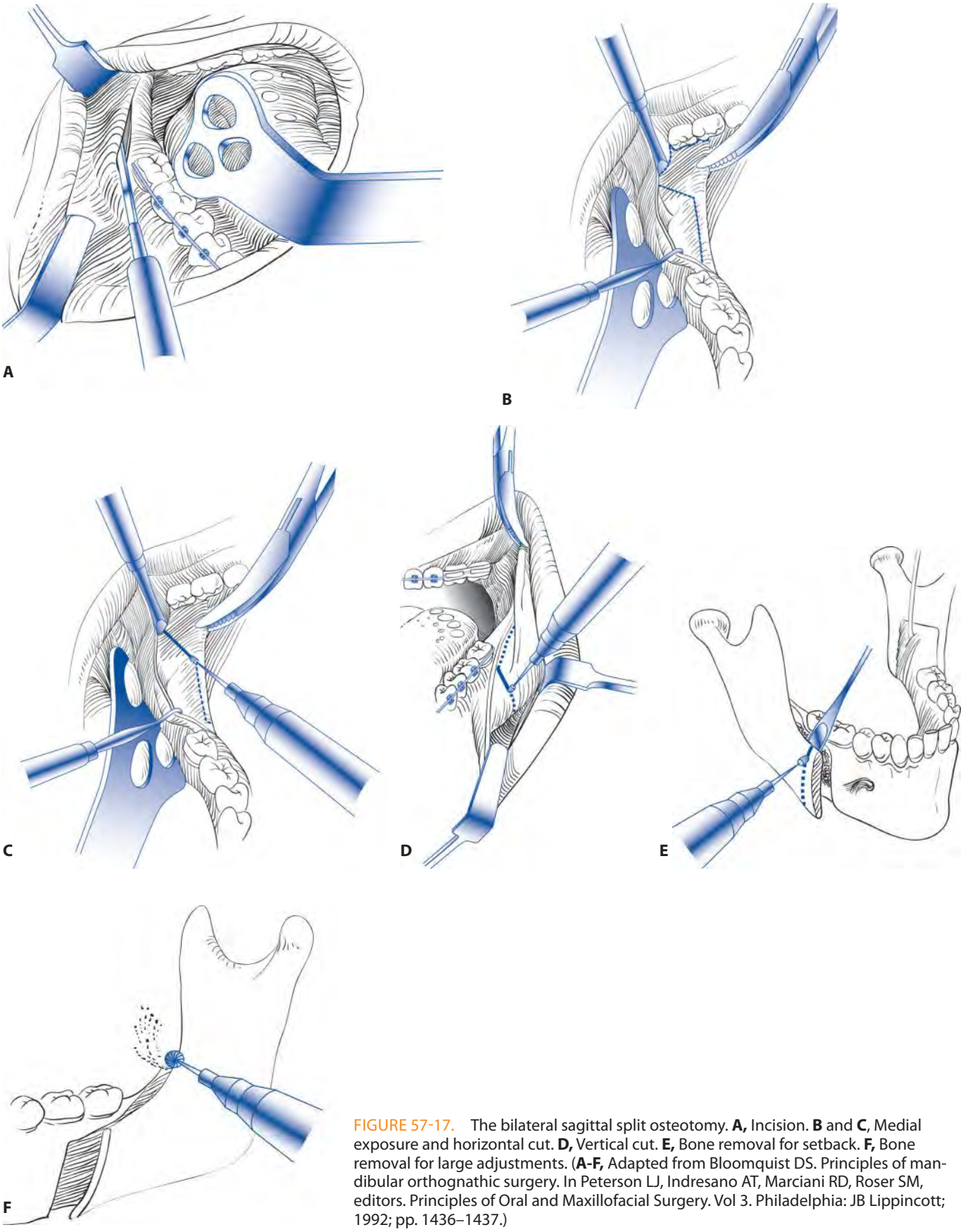


FIGURE 57-17. The bilateral sagittal split osteotomy. **A,** Incision. **B** and **C**, Medial exposure and horizontal cut. **D,** Vertical cut. **E,** Bone removal for setback. **F,** Bone removal for large adjustments. (**A-F,** Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; pp. 1436–1437.)

that the split stays close to the lateral bone cortex. Traditionally, wide-wedging osteotomes have been used to slowly complete the split. More often today, a special spreading instrument, a Smith spreader, is used along with a smaller osteotome to allow more control of the split. Generally, the movement is initiated along the vertical cut and carefully extended posteriorly. The fine osteotome can be used to keep the split close to the lateral cortex. If the IAN is encountered, it is carefully separated from the proximal fragment. The split along the inferior border can be difficult to control, and the judicious use of a thin osteotome will assist in this area. Finally, as the posterior split through the medial cortex is made, care should be used to prevent the split from continuing behind the mylohyoid fossa and starting up the neck of the condyle. The speed of the split often varies, depending on the elasticity of the bone. In older patients, in whom the bone is not as elastic, the split can occur very suddenly. Preventing inappropriate fractures is dependent on the care used not only in making the cortical bone cuts but also in ensuring that the splits occur as planned at the posterior aspect of the horizontal cut and along the inferior border.

The two key areas necessary to ensure that an appropriate osteotomy occurs and to minimize the chance of a bad split are the medial horizontal cut into the retrolingular fossa and the cut through the inferior border on to the lingual aspect of the mandible. Attention should be directed to the area of the inferior border osteotomy to assist if a bad split is noted, and control in this area could prevent propagation of an inadvertent osteotomy.

The medial pterygoid attachments are stripped off the proximal fragment to permit freedom of movement between the two fragments. If the mandibular teeth are planned to be retropositioned, either unilaterally to correct an asymmetry or bilaterally for correction of horizontal mandibular excess, an appropriate amount of bone is removed at this time from the anterior portion of the proximal fragment. The amount of bone removal may be based upon model surgery or the prediction tracings. With large setbacks or advancements, bone will need to be removed from the anterior edge of the vertical ramus. When setting the mandible back, this needs to leave space between the vertical ramus and the mandibular second molar to prevent this area from interfering with the patient's ability to clean the mandibular second molars (see Figure 57-17E). Conversely, in large advancements, bone sometimes has to be removed from the remaining portion of the anterior border of the vertical ramus of the distal segment just anterior to the lingula to prevent encroachment of the segment against the maxillary tuberosity (see Figure 57-17F). After the opposite side is split, the mandible is moved into its new position and stabilized with IMF. It may be preferable to use an interocclusal splint to ensure accurate positioning of the mandible relative to the maxilla, based on the presurgical model surgery. It is unusual for teeth to interdigitate accurately enough to obviate the use of a splint, although, if there is a full dentition and the occlusion is deemed stable, it is acceptable to apply IMF without the use of an interocclusal splint.

The placement of osseous fixation is performed at this point. Because multiple techniques are possible, different options are described in the following section on "Alternative Techniques." For the SSO, rigid fixation techniques generally include bicortical screws or monocortical plate and screw fixation. After placement of osseous fixation, if rigid fixation is used, the IMF is released, allowing the occlusion to be evaluated. The wounds are thoroughly irrigated and closed with the use of a resorbable running suture. No drains or external dressings are generally necessary, and guiding elastic intermaxillary traction may be used postoperatively (Figure 57-18).

Alternative Techniques

There are many variations to the technique described here, and in this section only the major variations are discussed. The design of the osteotomy itself has received much attention, with each variation representing an attempt to decrease the incidence of complications, although there is very little supportive research for any of the modifications. Obwegeser was responsible for two variations, the first being his original design, in which the buccal cut was horizontal and parallel to the lingual cut through the cortex of the vertical ramus (see Figure 57-16A). This original technique was modified by making the lateral cortical cut at an angle to the medial cut so that the posterior portion of the osteotomy ended just above the angle (see Figure 57-16B).[100]

A popular modification of DalPont's vertical osteotomy is the continuation of this cut completely through the inferior border, which, according to surgeon advocates, made the split easier to perform, and decreased the chance of a bad split.[59] This technique, however, makes the use of rigid fixation with bicortical screws difficult.[101] A few modifications have been suggested for the connecting cut, with the primary goal of allowing better control of the proximal fragment. These modifications appear to be primarily personal preferences because there is no evidence that these changes improve the outcome of the procedure.

A major area of variation for the SSO is in the use, or nonuse, of osseous fixation.[21,25,102,103] The original Obwegeser technique[104] used wire through the superior lateral and medial cortices. This technique, with minor variation, became the standard for the SSO until rigid fixation became popular.[59] The use of circumandibular wires and inferior border wires have also been suggested as improved methods to control the proximal fragment.[105] No evidence exists that any of these wiring techniques have an advantage for minimizing complications.[26]

Spiessl[26] introduced the concept of using screws for "rigid internal fixation" of the SSO. Following its introduction in 1974, there was a slow acceptance of this method of osseous fixation. Currently, there is little debate on the advantages of using rigid internal fixation; however, a wide variety of



FIGURE 57-18. Patient who was treated with bilateral sagittal split osteotomies (for mandibular horizontal deficiency). **A-E,** Preoperative photographs and head radiograph. **F,** Presurgery head radiograph. **G-L,** Postoperative photographs and radiographs. (**A-L,** Courtesy of Dr. S. Lake.) *(Continued)*









FIGURE 57-18. *(Continued)*

methods and materials are used. Initially, the use of three 2.7-mm "lag" screws on each side was advocated. A lag screw is placed by drilling a "guiding" hole with a larger drill through the lateral cortex, followed by a smaller hole through the medial cortex that is threaded with the use of a tap. Lag screws are then used to fix the proximal fragment tightly to the distal fragment. Compression across the osteotomy site was believed to be important to speed the healing of the osteotomy as well as to ensure the stability of the mandible. Concern developed among surgeons that compression may cause increased IAN damage and displacement of the condyles, with subsequent TMJ dysfunction.[106,107] An alternative technique, the positional screw, or bicortical screw, in which both cortices are drilled of equal diameter, has been advocated for stabilizing the osteotomy segments.[100] This technique permits maintenance of the gaps that may occur between the proximal and the distal fragments due to minor bony interferences, with no effort being made to compress the two segments together that may torque the condyle or compress the IAN. Standardization of techniques does not exist in rigid fixation of the SSO because there are many differences in screw sizes, number of screws, materials used, and whether plates are used across the osteotomy sites. Most of the research in the United States has centered on the use of three screws that are 2.0 mm in diameter. The use of monocortical screws and plates has become more widely used and a large multicenter study has provided clinical evidence to support this as routine practice. Direct comparisons of the various rigid fixation techniques in the literature are scarce and do not demonstrate that any one technique has any advantages.[109-110] One exception was noted by Fujioka and coworkers,[111] who found that there was more rotation through the osteotomy sites in patients with monocortical plate and screw fixation, perhaps justifying the need to consider additional fixation with counterclockwise rotation movements.

The use of resorbable screws has been a recent addition to fixation schemes. First attempted in Finland, the screws are made from polyglycolic acid using different manufacturing techniques and formulas. Development of these self-reinforced polylactic acid/polyglycolic acid copolymers that have reliable strength to withstand forces of mastication has made their use applicable in orthognathic surgery. The obvious advantage of resorbable fixation is to obviate future hardware removal, which is rarely necessary in the United States, but it has become an issue with patient concerns of risks of any permanent implant. The key features that are crucial in its application in orthognathic surgery are the rigidity and strength of the material, with the ability to resorb in a timely fashion. A few small studies have shown apparent stability of these resorbable implants comparable to metallic fixation; however, some inherent problems with material handling and early fixation failures have been reported.[112–114]

Suuronen and associates[112] reported the use of poly-L-lactic acid (PLLA) screws for fixation in SSO, with no apparent malocclusion or skeletal relapse. Harada and Enomoto[113] and, later, Ferretti and Reyneke[114] compared titanium and resorbable screws and noted no difference in healing between two groups, and no statistically significant difference in skeletal relapse. However, Harada and Enomoto's patients used IMF for a period ranging from 9 to 14 days after surgery.[113] Kallela and colleagues[115] used self-reinforced poly-L-lactic acid (SR-PLLA) screws for mandibular osteotomies and no IMF postoperatively. The mean advancement was 4.57 mm at B point, and the mean relapse was 17%. In their 2-year follow-up study in 1999, the authors reported osteolytic changes seen around the resorbable screws in 27% of cases, and the screw canals remained as radiolucent shadows without complete bony fill.[115] Turvey and coworkers[116] reported experience with resorbable fixation for 194 osteotomies of the maxilla and/or mandible. Forty-three of the patients had SSOs with 2-mm resorbable screws placed on each side for fixation. They reported only 1 infection at the SSO site and 1 patient who exhibited excessive masticatory forces that resulted in loosening of the fixation. More recently, the use of resorbable plates with monocortical screws has been advocated. A large group of 272 patients were evaluated for complications other than neurosensory changes. Titanium plates were used in 152 patients and resorbable plates, a copolymer of L-lactic acid and D-lactic acid, were used in 120 patients. The authors conclude that complications, including relapse, which was 18.3% for resorbable plates and 8.6% for titanium plates, must be questioned owing to the large number of variables in the study. However, the apparent successful treatment of that number of patients is impressive.[117]

Acceptance of the routine use of resorbable fixation with the SSO will require long-term evaluation with well-designed studies comparing these materials with the metal hardware. The major potential risk of permanent metal fixation is the possibility of bone remodeling, causing the hardware to become noticeable and possibly irritating to the patient. Although rarely reported in the literature, oral and maxillofacial surgeons have had experience with patients returning to have plates and screws removed simply because these implants have become noticeable or palpable. This problem must be weighed against the still unknown side effects of the resorbable materials. A prime example of these unknowns is the precise time required for total resorption and degradation of poly-L-lactic acid (PLLA) and poly-D,L-lactic acid (PDLLA) in human tissues, which is reported to range anywhere from 90 days to 5 years. Surgeons must closely follow the literature to determine the practicality of these new materials.

An interesting suggested modification in the SSO technique is the purposeful changing of the rotational position of the proximal fragment to control the direction of mandibular growth. It has generally been recommended that the proximal segment be maintained as close as possible to its preoperative position. O'Ryan and Epker[118] have further suggested that rotating the proximal fragment in a growing patient can change the vector of condylar growth and, thereby, influence the final mandibular position. Studies have failed to support this contention.[119] There is also concern with an SSO in growing children, especially those requiring significant advancement. Huang and Ross[120] demonstrated what they believed to be a cessation of mandibular growth and condylar resorption in growing patients in whom SSO procedures were performed. Whether this was a problem with the techniques used or a problem with performing surgery in such a young group of patients is unknown, but this effect has not been noted in any further literature.

A final modification of the SSO is the concomitant use of a midsymphyseal osteotomy to allow for correction of width discrepancies in the dental arch. For example, this could be used to narrow the mandibular arch form in lieu of a two-piece maxillary osteotomy, and this may be a more stable procedure. Although this procedure may be used with any of the ramal osteotomies, it has only been described with the SSO. First mentioned by Bell,[121] it has become more practical with the use of rigid internal fixation. A single four-hole plate across the bone cut between the mandibular central incisors, along with an intact orthodontic arch wire, provides sufficient postoperative stability.[122] Concerns regarding adverse effects on the TMJ and periodontium have been shown to be insignificant.[123]

Complications

Stability

The stability of the SSO of the vertical ramus is arguably the most studied complication in orthognathic surgery. Because the relapse patterns differ between mandibular advancements and setbacks, their particular etiologies most likely differ as well; however, many of the important principles for preventing relapse in each scenario may be the same. Whereas much of the research on mandibular advancement has been done in the United States, mandibular prognathism has generally received the greatest interest in the Scandinavian countries and the Far East. This highlights one of the major problems for surgeons attempting to decide on which techniques to use to minimize undesirable postoperative skeletal change. There are large variations in research techniques as well as surgical approaches that exist not only between surgical centers in different countries but also within the same country. Fortunately, there is enough corroboration in the literature that some general statements and conclusions can be made.

One of the most important findings made regarding the stability of mandibular osteotomies was that IMF does not prevent postoperative skeletal change. Intermaxillary elastics may have a minor influence on bony remodeling at the osteotomy sites, but the rigid fixation used may be difficult to overcome with elastic traction. More likely, the elastics provide some dental changes (extrusive movements), and possibly some condylar remodeling, that may serve to correct minor postoperative malocclusions. Although the influence of postoperative elastic traction on occlusion applies to any type of maxillary or mandibular osteotomy, it was first

recognized in the evaluation of mandibular SSO. It is generally believed that paramandibular connective tissue pressures and muscle vectors are the major factors influencing relapse, especially in cases of mandibular advancement.[52,53,123] However, early attempts at minimizing these effects, such as suprahyoid myotomies and external supportive devices, have not been shown to be effective.[64] Internal support techniques, however, have been shown to be effective. Prior to rigid fixation screws and plates, a type of internal support called *skeletal fixation* was shown to be effective in decreasing the downward and backward relapse pattern of mandibular advancements.[64] This fixation was used in addition to IMF and consists of wires placed from the piriform rim to circumandibular wires placed in the cuspid or molar regions (see Figure 57-9). Interestingly, Van Sickels[124] noted a decrease in relapse in patients with large advancements when skeletal fixation was combined with rigid internal screw fixation.

Other possible causes of relapse include patient age, preoperative mandibular plane angle, rotational position of the proximal fragment, amount of distal segment advancement, and displacement of condyle within the fossa. The effect of mandibular plane angle is controversial because of conflicting reported in the literature, most of which evaluated patients who had wire osseous fixation with IMF. Mobarak and associates[125] clearly found decreased stability in patients with steep mandibular plane angles when rigid internal fixation was used. Of the remaining variables evaluated, only the last two appear to be definitively supported by clinical studies. In their multicenter study, Schendel and Epker[64] found that displacement/dislocation of the condyle from the glenoid fossa was a significant predictor of relapse. This was further confirmed by Lake and coworkers,[55] who showed a correlation between the amount of advancement and the amount of relapse.

The final area that must considered in relapse in both mandibular advancement and setback is the rigidity of the fixation across the osteotomy site. Early investigators thought that fixation across the osteotomy site may hinder the natural repositioning of the condyle in the fossa that occurs during IMF.[103] Later studies showed that wire osteosynthesis was superior to no osseous fixation in fragment positioning, and the incidence of relapse was decreased.[102] However, the search for an improved osseous wiring technique that would decrease relapse was not successful, with most studies on osseous wiring techniques consistently reporting a mean relapse of approximately 30%. The large range of individual relapses that does exist has led many surgeons to believe multiple factors influence relapse.

The use of rigid internal fixation techniques has become the preferred method for minimizing skeletal relapse in the SSO. Schmoker and colleagues[126] discussed the advantages of lag screws in SSO. Their initial study of skeletal stability evaluated mandibular setbacks, comparing the use of three 2.7-mm screws versus wire fixation and one or two screws. It was not until 1985 that Van Sickels and Flanary[127] showed evidence that a similar rigid fixation technique provided increased stability in mandibular advancements. Furthermore, larger and longer studies have clearly shown the stability of using three bicortical screws for fixation of the SSO.[128–131] These studies have reported a mean relapse rate between 0.0% and 8.0%. The majority of studies comparing rigid internal fixation techniques versus the use of wire osseous fixation have also confirmed a significant difference in stability between the techniques.[132–136] Interestingly, the only clinical study that evaluated the stability of osseous wire fixation versus rigid screw fixation on patients treated at the same center seemed to show a comparable long-term stability.[137] This study, however, reported an unusually high stability of the wire osseous fixation group and did nothing to dispute the stability of the screw fixation. Dolce and associates[136] found, in their long-term follow-up of the multicenter study in which the patients were randomly assigned between internal rigid fixation with screws and wire osseous fixation, that at 5 years, the rigidly fixed mandibles were skeletally more stable, and there was no significant difference between the two groups in anterior overbite and overjet. This particular effect is difficult to explain except for some instability in the orthodontics. The number of screws may very well influence the stability, although only Spiessl's original study[26] demonstrated a difference. Knaup and associates[131] tried to demonstrate some difference in stability on large advancements in evaluating three versus four bicortical screws per side. They found no difference in horizontal changes but did show more stability with four screws when there was counterclockwise rotation of the distal fragment. This finding then led to an early attempt to reevaluate the possibility that anterior open bites can be successfully closed with BSSO stabilized with rigid screw fixation. Recently, the possibility of closing open bites with mandibular osteotomies in a stable fashion has been supported by other studies.[138]

Finally, several biomechanical studies have shown that the placement of three bicortical positional screws to fixate an SSO is more stable in an L configuration (two near the superior border, one near the inferior border) than in a linear configuration (three placed near the superior border), although there is concern that the inferior screw may iatrogenically injure the IAN or not engage the lingual aspect of the distal segment, resulting in less stability.

Another common method for rigid internal fixation of the SSO is the use of miniplates with monocortical screw fixation. Generally, 2.0-mm systems are used with two monocortical screws placed on either side of the osteotomy to secure one four-hole plate.[139–142] Questions remain over the minimum number of screws necessary to prevent relapse and whether more or larger screws, or plates, will improve stability in longer advancements. It has been shown that a single 2.0-mm screw does not seem to increase mandibular stability over wire osseous fixation, and therefore, it could be argued, in light of the previously reviewed research, that an increase in number or size of screws, and/or plates, may result in more osseous stability.[131,143]

With the exception of Schmoker and colleagues' original studies[126] on the stability of using rigid internal fixation in the correction of mandibular prognathism, a majority of the clinical research on the use of these fixation techniques has focused on mandibular advancements using SSOs. Proffit and associates[79] evaluated stability of using rigid internal fixation with screws in patients with mandibular setbacks. They compared these patients with patients who were treated VRO as well as patients who had wire IMF with SSO. Interestingly, they found that the rigid internal fixation seemed to be the less stable, with almost 50% of patients having more than 4.0 mm of skeletal relapse. In one of the largest retrospective studies of mandibular setbacks, Chou found a large skeletal relapse of 21% at 1 year after surgery.[144] This relatively large amount of skeletal relapse with rigid internal fixation has been verified by a comparison of bicortical positional screws with plate and monocortical screw fixation by Chung,[145] as well as an excellent systematic review by Joss and Vassalli.[146] It is clear from the literature that mandibular setback using rigid internal fixation is relatively unstable and that, unlike mandibular advancement, consideration for overcorrection is warranted.

Nerve Damage

The possibility of damage to the IAN during SSO has been well known since the technique was first described, but surprisingly, the issue was minimized early on. Kole[147] first mentioned a high incidence of neurosensory problems immediately after surgery for patients with SSOs, but most clinicians claimed a very low incidence of long-term neurosensory problems.[148] In contrast, Walter and Gregg,[45] in an objective study of sensory problems, noted a large incidence of long-term problems. Since this first definitive study, there has continued to be a variety of reported instances of both immediate postsurgical as well as chronic sensory disturbances.

Westermark and coworkers[149] evaluated 496 SSOs for possible correlations between neurosensory dysfunction and other variables, such as age of the patient, mandibular movement, type of split technique and fixation, degree of intraoperative nerve encounter, and surgeon skill. Nerve dysfunction developed in 40% of cases. Age had a significant influence on the recovery of the neurosensory function as well as the severity of neurosensory disturbance. Intraoperative nerve encounter and nerve manipulation as well as surgical experience were also reported to have an effect on nerve dysfunction. Other variables had no significant effects on the incidence of neurosensory dysfunction. Ylikontiola and colleagues[150] also found a statistically significant positive correlation between subjective neurosensory loss and patient age and, in addition, magnitude of mandibular movement and degree of manipulation of the IAN intraoperatively.

Other clinical research has noted a significant relation between patient age and nerve recovery. This finding was noted early on by MacIntosh,[151] who emphasized that this osteotomy should not be used for patients older than 40 years. Another interesting correlation was made by Van Sickels and associates[152] who reported that patients with a concurrent genioplasty had a greater loss of IAN sensation initially. Unfortunately, the wide variation in neurosensory testing protocols makes comparisons of these various reports difficult and makes the evaluation of techniques that have been suggested to decrease the incidence of nerve damage difficult as well.

White and colleagues[153] indicated that damage to the IAN most likely occurs either during the medial retraction of the soft tissues for the horizontal osteotomy in the vicinity of the IAN as it enters the mandibular foramen or during the vertical bone cut anteriorly where the nerve may be close to the buccal cortex. Guernsey and DeChamplain[154] believed that IAN damage occurred during the actual splitting of the mandible and reported that segments of the neurovascular bundle may remain in the proximal fragment after the split. This has led some surgeons to recommend the use of a small flat (spatula) osteotome during the split, instead of the wide-splitting osteotome, although this remains controversial.[155] The fine osteotome is malleted carefully along the lateral cortex and cancellous bone junction but may also place the nerve at risk for iatrogenic injury because its actual position in three dimensions is unknown in each patient. It has been reported that the nerve is exposed less often during the split by this technique, and consequently, it has been assumed that this results in less sensory disturbances.[156,157] Unfortunately, this technique has not been directly compared with any other, and the comparison of the occurrence of sensory disturbance between reports is not possible. Yoshida and coworkers[157] and, later, Yamamoto and associates[158] found that nerves that were close to the lateral cortex, as determined by radiographs, were more likely to have severe sensory alteration after surgery. The deficits were also more likely to be present 1 year after surgery when the marrow space between the mandibular canal and the external cortical bone was 0.8 mm or less. Some authors believe that by making the vertical cut in the lateral cortex more posterior, a lower incidence of sensory problems occurs.[157, 159] This has not been substantiated in comparison studies, and in fact, the IAN may be closer to the buccal cortex in the third molar region than further anteriorly.

Another possible cause of sensory loss to the IAN may be due to sharp medullary bone irregularities of the proximal or distal fragments or to compression of the IAN when the proximal fragment is positioned and fixated, especially if there is a "lag screw" component to the fixation.[160, 161] A round or acrylic bur can be used cautiously to remove any bone spicules as well as to widen and deepen the canal in the proximal fragment to prevent this nerve compression effect. Care must be taken when working around the nerve so that instruments used during the osteotomy do not themselves cause direct iatrogenic nerve damage. This concern has been heightened with the use of screw fixation. Paulis and Steinhauser[162] noted slightly higher incidences of long-term sensory loss in patients with rigid screw fixation compared with simple osseous fixation, but statistics were not used and the significance of their numbers is questionable. Nishioka and

colleagues[163] performed a comprehensive study involving sensory loss after the use of screw fixation and found the incidence of IAN sensory loss to be high but within the range of sensory loss reported by other well-designed objective studies. Subsequently, the effect of type of fixation on neurosensory functional outcome was extensively studied by a number of authors using different methods of clinical neurosensory testing.[164–167] Brushstroke directional discrimination was diminished to a greater extent in the rigid fixation group compared with the wire fixation group from 8 weeks through 2 years postoperatively; however, monofilament contact detectional discrimination did not show significant difference between types of fixation throughout the 2-year follow-up.[164] Despite a high number of studies on neurosensory disturbance after orthognathic surgery, the severity of IAN injury is difficult to compare across different studies because there is lack of standardization regarding which neurosensory tests were used, methods in which the tests were performed, and how the results were interpreted. Certain neurosensory tests are more sensitive in detecting sensory nerve deficit than others. Tests that evaluate patients' abilities to discriminate direction have been shown to be more sensitive indicators of trigeminal neurosensory impairment than other tests such as light touch detection. Westermark and coworkers[165] used a visual analogue scale (VAS), light touch perception, and temperature testing and concluded that there is a positive correlation with nerve dysfunction. Alternatively, Chen and associates[166] compared three methods of assessing neurosensory disturbance after SSO: two-point discrimination, pressure-pain thresholds, and perceived sensation changes in specific facial regions. The two-point discrimination test was consistent with patients' self-ratings of neurosensory problems using facial maps, but the pressure-pain test was the least sensitive to neurosensory changes. The frequency of IAN disturbance ranged from 10.0% to 94.0% depending upon the testing method and the testing site used. In a well-controlled study by Nakagawa and colleagues,[167] the occurrence of a long-lasting postoperative trigeminal sensory hypoesthesia was found to be dependent on the nerve involvement at the bone split interface, the manner of fixation, or the intraoperative handling of the tissue surrounding the nerve. Essick demonstrated improved return of sensation and less patient concerns following the SSO with the use of biofeedback sensory retraining exercises.[168] The biofeedback sessions, which occurred 1 week and 1 and 3 months after surgery, resulted in significantly less pain, "unusual feeling," and numbness than the control group. This training seems to be the only postoperative variable, other than time from surgery, that can influence spontaneous neurosensory recovery.

Although the neurosensory function of the IAN after SSO has received a great deal of attention, few studies have documented the incidence of lingual nerve dysfunction, mostly related to bicortical screw overpenetration of the lingual cortex. Jacks and associates[169] retrospectively reviewed the patient-reported incidence, duration, and perceived deficit associated with lingual nerve function. In the SSO patients, 19% reported lingual nerve sensory changes, of whom 69% reported a resolution of symptoms within 1 year and 88% reported altered activities of daily living. When compared with the IAN, lingual nerve sensory changes occurred much less frequently and resolved more frequently and sooner, but they were associated with greater perceived deficits in patients' daily activities. Zuniga and colleagues[170] were the first to report on studies performed to assess the effect of lingual nerve injury and repair on human taste perception. Gent and coworkers[171] examined perceived taste intensity and taste quality identification on localized regions of the tongue after orthognathic surgery. Lingual nerve function in taste perception was diminished at 1 to 2 months after surgery, likely due to impaired chorda tympani nerve function, but it improved by 6 to 9 months after surgery.

TMJ Dysfunction

The incidence of TMJ dysfunction is considered in two ways: first, the incidence of TMJ symptoms that are present after surgery compared with preoperative findings; and second, the change in mandibular range of motion. The latter may obviously not be related to TMJ dysfunction; conversely, it must be considered when evaluating the effects of the surgery on the TMJ. Unfortunately, few authors related these two areas when they reported on the effect of the SSO. Another factor that must be taken into account in evaluating the effects on the TMJ from orthognathic surgery is the possible contribution from presurgical orthodontics. There is certainly debate regarding the influence of orthodontics on TMJ dysfunction, in both the short and the long term.

Similar to neurosensory loss, the potential of the SSO to cause TMJ dysfunction was recognized early.[24] Reporting on the incidence of TMJ problems, however, has been highly variable, with most authors recording only the postoperative complaint without any reference to the preoperative condition. Some of the first reviews that did look at pre- and postoperative TMJ symptoms seemed to imply an increase in joint noise, but not in pain, after the SSO.[56,172,173] The use of rigid screw fixation was believed by some to cause an increase in TMJ problems by fixating the condyle into a position that may not be natural or adaptable after surgery.[174] This concern was highlighted by radiographic findings of condylar changes that occurred with rigid screw fixation. Kundert and Hadjianghelou[106] demonstrated that this tendency occurred with both wires and screws but was greater with rigid fixation than with a wire osseous fixation technique. In neither study was there a discussion of whether these changes had any clinical consequences. Hackney and associates[175] found that with bicortical screw fixation for SSO used for mandibular advancement, little change in condylar position occurred and there was no significant effect of the surgery on TMJ symptoms. Paulis and Steinhauser[162] compared preoperative and postoperative TMJ symptoms in two large groups who had either rigid screw fixation or wire osseous fixation of SSO. They found no difference in postoperative incidence of TMD between the two groups and, in fact, found a notable

decrease in TMJ pain in both groups. The possibility that the SSO may benefit patients with TMJ symptoms was suggested by Martis[176] and Karabouta and Martis.[177] They reported that only 11% of the patients who had TMJ symptoms before surgery had any symptoms after surgery, whereas about 4% of the asymptomatic patients had TMJ problems after surgery. These results were better than, but consistent with, a study of all types of orthognathic surgery patients, which showed an improvement in a large percentage of patients, with relatively small risk for the asymptomatic patient.[178]

There is, surprisingly, a body of evidence in the literature that suggests that the SSO may have a beneficial effect on preexisting TMJ dysfunction. It is generally believed that TMJ dysfunction (internal derangement, anterior disk displacement) is found at a higher incidence in class II patients compared with patients with classes I and III malocclusions. The use of the SSO as an alternative to the mandibular condylotomy to treat patients with painful TMJ dysfunction has been proposed by some authors.[179] They suggest repositioning the proximal segment and increasing the joint space, both of which are thought to have an unloading effect on the highly innervated neurovascular retrodiskal tissues. However, this is controversial and there has not been adequate research to confirm this impression. Other studies have indicated the trend that class II patients who undergo SSO advancement may have improvement in TMJ symptoms, whereas class III patients who undergo SSO mandibular setback are less likely to improve or more likely to develop worsening TMJ symptomatology. Each patient must be treated individually based upon their age, dentoskeletal deformity, and preexisting TMJ symptoms.

Debate on rigid versus wire fixation relative to the effect on the TMJ has led to a number of studies that have shown no significant difference in the incidence of TMJ symptoms between patients who received rigid fixation versus wire osteosynthesis during SSO.[180] Feinerman and Piecuch[181] compared the TMJ outcomes of the miniplate with monocortical screw group versus the superior border wire fixation with IMF group. They found no demonstrable long-term differences between the two groups with respect to mandibular vertical opening, crepitus, and TMJ pain. In fact, masticatory muscle pain and TMJ clicking improved with rigid fixation and worsened with nonrigid wire fixation. One of the reported findings of a large prospective study comparing rigid screw fixation with wire IMF was that, although large mandibular advancements along with counterclockwise rotation of the distal segment resulted in more muscle as well as TMJ symptoms soon after surgery, at 2 years, there was significant decrease in both myofascial and TMJ symptoms.

The only negative report on the effects of SSO on the TMJ was by Wolford and colleagues,[182] who evaluated changes in TMJ dysfunction in patients with presurgical TMJ internal derangement as well as the long-term stability of orthognathic surgery. Unlike other clinicians, they noted the development of new TMJ symptoms, or an aggravation of existing TMJ symptoms, in a group of patients at 14 months postsurgery. Therefore, the authors recommended that surgical correction of preexisting TMJ pathology with formal TMJ surgery be considered, either preceding or concurrently with the orthognathic procedure. In summary, it appears that there is a low risk of worsening TMJ symptoms in patients who have preexisting TMJ dysfunction with the SSO procedure. In addition, conversely, the SSO may result in improved TMJ symptoms in a greater number of symptomatic patients owing to changes in the orientation of the TMJ apparatus and subsequent TMJ adaptation. Unfortunately, standardized methods of predicting TMJ outcome in individual patients after SSOs have not been developed.

Mechanical displacement of the condyle out of its correct position has been implicated as a significant factor in postsurgical skeletal relapse after SSO. For this reason, as well as in an attempt to minimize TMJ problems, a great deal of interest has been focused by early investigators on the issues of condylar position after SSO.[183] There have been a myriad of technical notes on how to maintain the preoperative condylar position and use of different condylar repositioning devices, based on anecdotal reports of individual surgeon experiences. Harris and coworkers[184] examined factors influencing condylar position after SSO fixated with rigid fixation. The amount of advancement did not correlate with condylar displacement. Condyle angulation and superoinferior movement did correlate somewhat with the amount of advancement. In addition, Van Sickels and associates[185] found that the condylar position was slightly different with rigid fixation versus wire osteosynthesis beyond 8 weeks postoperatively, but the ultimate position of the condyle was not significantly different. They found, as have many others, that the final condylar position was posterior and superior to its preoperative location after mandibular advancement. Renzi and colleagues[186] specifically examined class III patients without preoperative TMJ dysfunction; half of the patient population was treated with a condylar positioning device and the other half was treated with manual intraoperative control of condylar position. The condylar repositioning device did not prevent the changes in condyle positions in all cases. Neither group had any skeletal or occlusal relapse or postsurgical TMJ dysfunction. The value of using condylar positioning devices was reviewed by Costa[187], who concluded that there was no scientific evidence until 2008 to support the routine use of these devices. However, some surgeons continue to use some form of positioning devices and to attempt to demonstrate benefit in the extra time required for these methods. magnetic resonance imaging (MRI) was used by Saka for evaluation of not only condylar position but also position of the disk in this prospective study comparing a condylar repositioning device versus regular manual positioning.[188] Postoperatively, they found a significant difference between these two groups, with anterior disk displacement significantly more common without the repositioning device. The condylar position after surgery, however, was found to be similar. Unfortunately, there was no mention

of postoperative TMJ symptoms, although there was an assumption that problems would occur in those patients with a displaced disk. However, it is important to understand that the incidence of new-onset TMJ dysfunction in healthy individuals after orthognathic surgery is known to be low as previously mentioned. The assumption of many surgeons that changes found with TMJ imaging have clinical consequences may be unfounded, and this study included only patients without preoperative TMJ symptoms. Therefore, it is not surprising that the patients did not develop any postsurgical TMJ dysfunction. Also, the clinical implication of condylar and disk position in the healthy versus preexisting TMJ dysfunction groups may be different; therefore, the true clinical significance of condylar and disk position in exacerbation of TMJ symptoms remains unknown.

Computed tomography (CT) has enabled clinicians to assess and quantify condylar position changes in three planes of space. Alder and colleagues[189] reported that changes in condylar position occurred in all planes of space, but the most common postoperative condylar position was more lateral with increased condylar angle, a higher coronoid process, and a condyle located more superior and posterior in the fossa. Rebellato and coworkers[190] found an increased superior postsurgical movement of the condyle with increasing magnitudes of surgical advancement of the mandible.

MRI has revolutionized the examination of the TMJ, in that it not only allows the evaluation of condylar position but also provides information on disk position and morphology. Gaggl and associates[191] reported clinical and MRI findings of the TMJ in class II patients, preoperatively and 3 months postoperatively. Clinically, patients had improvements in joint pain and abnormal joint sounds such as clicking. The MRI showed displacement of the disk in 38 of the 50 joints preoperatively and in 28 postoperatively. No correlation was made between the change in disk position and improvement in TMJ symptoms, which is consistent with other MRI studies. Ueki and colleagues[192] made interesting comparisons of the condylar and disk positions after SSO and IVRO and correlated these findings with TMJ symptoms postoperatively. Fewer or no TMJ symptoms were reported by 88% of patients who underwent IVRO and by 66% of patients who underwent SSO. MRI studies showed no change in anterior disk displacement after BSSO; however, improvement was seen in 44% of patients who underwent IVRO, at least in the early postsurgical period.

The effect of SSO on mandibular range of motion has been studied extensively. Whereas Stacy[193] found that patients who underwent mandibular setbacks with IMF generally returned to presurgical function within 9 months after surgery without any physical therapy, other authors have found very different results. Storum and Bell[194] found that without active physical therapy after the release of IMF, there was a decrease in the patient's ability to achieve preoperative opening when compared with patients who had an active rehabilitation program. This latter study is consistent with most clinicians' experience, and some form of active physical therapy is recommended after release from IMF. However, there is some research and intuitive evidence that rigid internal fixation that permits mandibular movement soon after surgery may result in a more rapid return to preoperative mandibular movement without active physiotherapy.[195] However, the literature remains inconclusive.[195,196] Nishimura and coworkers[196] found that final postoperative mouth opening was not significantly influenced by the type of fixation. Hatch[197] also found in a large prospective study of wire versus rigid internal fixation that at 5 years, that there was no significant difference between the two groups. Interestingly, they also noted that neither group returned to completely normal preoperative mouth opening levels.

Another poorly understood TMJ complication is idiopathic condylar resorption, or spontaneous resorption of the condyles, after SSO.[198] This process is different from standard orthognathic relapse with the abnormal resorption being seen primarily in a specific group of patient; specifically young female patients who have had a history of TMJ dysfunction before surgery, with a steep mandibular plane angle, and who have undergone a large mandibular advancement. Some remodeling of the condyles is expected to occur after SSO, but fortunately, only rarely does this condylar resorption result in significant clinical changes with pain and apertognathia. Cutbirth and colleagues[199] evaluated long-term condylar resorption after mandibular advancement stabilized with bicortical screws. Large advancements and preoperative TMJ symptoms significantly correlated with long-term postoperative condylar resorption at the mean follow-up of 3 years. The amount of vertical resorption did not directly correlate with the amount of relapse seen between 6 to 8 weeks or in the long term. Surprisingly, there was an improvement in TMJ symptoms for the group as a whole and even among the group who developed condylar resorption. It should be noted, however, that it is often difficult to distinguish between normal condylar remodeling and significant condylar resorption. In the Cutbirth and colleagues study,[190] the authors arbitrarily established a parameter of less than 10% loss of condylar height to be considered as "normal remodeling." Attempts to delineate the normal versus pathologic processes are difficult and may lead to an underestimation of the actual number of clinically significant condylar resorption cases that may occur.

Hoppenreijs and coworkers[200] evaluated the long-term treatment results of 26 patients (23 women and 3 men) who developed progressive condylar resorption after orthognathic surgery. The preoperative condylar configuration was noted in patients with deep bites to have more resorption on the superior aspect of the condyle, whereas patients with anterior open bites had resorption on the superior and anterior surfaces of the condyle. Thirteen patients were managed without surgery after the diagnosis of condylar resorption, and only 3 patients had class I occlusion at the end of treatment. Thirteen patients underwent a second surgical correction, with 7 patients having satisfactory occlusal results. Four of the patients had relapse with a stable occlusion not requiring

further treatment, and 2 patients had complete relapse requiring a third surgical procedure. It was suggested that without surgical intervention after condylar resorption, further resorption ceased after approximately 2 years. The authors speculated that either the mechanical loading during or after SSO and/or the impediment of blood flow to the condylar segment and the TMJ capsule may play a role in condylar resorption. However, the etiology of this process is still unclear, but it does seem to be self-limiting and the resulting dental skeletal deformity can usually be successfully treated with further mandibular surgery.

Miscellaneous Complications

A wide variety of other complications have been reported after SSO. Early reviews of complications from this procedure noted excessive blood loss, postoperative airway compromise, aseptic necrosis of bone segments, and facial nerve damage. Better experience and improved instrumentation seem to have dramatically decreased the incidence of these problems. Bleeding is generally easily managed by direct or indirect pressure over the bleeding soft tissue and vessels. Lanigan and associates,[201] reporting on a questionnaire sent to a large number of oral and maxillofacial surgeons, found only 21 cases of significant bleeding after mandibular osteotomies. Suspected sources of bleeding included the inferior alveolar artery, facial artery, maxillary artery, and retromandibular vein. Management primarily included direct pressure packing or ligation of the vessel at the point of severance through the open wound. Extraoral approaches to gain access to the facial or external carotid artery can be ineffective owing to the collateral circulation. Angiography with embolization is considered appropriate in cases of acute persistent postoperative arterial bleeding of more than 0.5 mL/min.

A group of problems that seems to persist is the inadvertent fracture in the anterior buccal aspect of the proximal segment or the posterior lingual aspect of the distal segment. Good surgical technique with extension of the osteotomies into the marrow space minimizes these problems, and care used during the split is worth the effort because correcting a "bad" split can be difficult. Fortunately, the use of screws and plates does improve the chance of obtaining a satisfactory result, in light of an unexpected fracture, with minimum further morbidity to the patient.[202] It has been believed that one of the major risk factors predisposing to buccal cortex fracture is the presence of impacted third molars. Precious and colleagues[203] retrospectively reviewed two groups of patients: one group with retained impacted third molars removed during BSSO and the other group with third molars having been removed at least 6 months before BSSO. There was a 1.9% incidence of unfavorable fractures, and the majority of fractures occurred with the group who had the third molars removed at least 6 months before the BSSO. Mehra and coworkers[204] reported 2.2% unfavorable fractures in 500 procedures. They noted a larger percentage of unfavorable fractures in the patients with retained third molars (3.2% vs. 1.2%). This finding is consistent with that of Reyneke and associates[205] who found that the presence of unerupted third molars increased the degree of difficulty of SSO, and in all 4 out of 139 patients, unfavorable fractures occurred in patients with unerupted third molars present at the time of surgery. Ideally, third molars should be extracted at least 9 to 12 months or more before the SSO, both to minimize unfavorable fractures and to allow optimal bony healing, especially when planning on the use of internal rigid fixation.

Airway patency and induction of iatrogenic obstructive sleep apnea (OSA) has become an area of concern to some clinicians, especially in the cases in which the mandible is retropositioned. Riley and colleagues[206] reported two patients who were surgically treated for prognathism and later developed OSA. Kawamata and coworkers[207] studied patients with mandibular prognathism who were treated with either SSO or VRO for mandibular setback. Using three-dimensional CT imaging, they quantified the posterior airway space after surgery and found that the lateral and frontal widths of the pharyngeal airway had decreased by 23% and 11%, respectively, and this reduction in airway dimension did not resolve at 1 year after surgery. However, in the longer postoperative period, a visible recovery of pharyngeal width was seen in some cases. The finding of decreased airway dimension secondary to mandibular setback has been confirmed by other studies. Therefore, one should be cognizant of any physiologic and medical etiologic factors, including preexisting OSA, that may have contributed to the emergence of OSA symptoms, rather than simply using the measurement of the posterior airway space after mandibular osteotomy as the sole means of predicting the development of new-onset OSA.

In general, the risk of infection seems to be low with the SSO owing to the vascularity in the head and neck region. In their clinical review of 700 consecutive cases of mandibular osteotomies, Bouwman and associates[208] reported that screw removal owing to infection was performed in 2.8% of cases. Screw loosening occurred in the first postoperative week, which resulted in an occlusal discrepancy in 4 patients. Fifteen sides required one or more screws to be removed as a result of infection. In a large study of complications in orthognathic surgery, Acebal-Bianco and colleagues[209] reported 36 infections out of 802 mandibular osteotomies (0.05%), but only 5 patients had hardware removed owing to infections.

Horizontal Ramus Osteotomies

Since Blair's first description of his osteotomy of the horizontal ramus,[3] a variety of osteotomy designs have been documented. Initially, the surgeons used extraoral, or a combination of extraoral and intraoral techniques, but since the early 1950s, the advocated approaches have primarily been intraoral. It is difficult to choose a representative technique for the body osteotomies because of the wide variations described as well as the relative infrequency of these

techniques. Of the described procedures, the step osteotomy is reviewed because of its versatility and its apparent common use in some centers.

Indications

The greatest limitations of body osteotomies are that the osteotomy must be made through the dental alveolus so edentulous spaces are usually required and the IAN is at risk for injury during these procedures. Because these osteotomies are made anterior to the pterygomasseteric sling, some surgeons believe that the results are more stable and, therefore, prefer body osteotomies in the treatment of prognathism when there are edentulous spaces. Other unusual mandibular abnormalities, such as asymmetries, may also be treated more appropriately with one of these forms of osteotomy as opposed to an osteotomy of the vertical ramus.

With the step osteotomy, the surgeon must be concerned about the horizontal component of the "step," which often has to be performed between the IAN and the apices of the tooth roots. Sufficient room should, therefore, be available for this cut, unless the surgeon plans to externalize part of the IAN so that the cut can be made at the level of or below the inferior alveolar canal.

Technique

An incision is made 4 to 5 mm below the level of the attached gingiva (enough tissue is left superiorly to permit later suturing) and is carried forward at this level to the cuspid, where it can be dropped down 5 mm and extended forward to the midline (Figure 57-19A). The periosteum is elevated inferiorly until the mental foramen is located and then the remainder of the periosteum is stripped to expose the area of the osteotomy. The attached gingiva is also carefully elevated in the area of the dental alveolar cut so that it can be protected during the osteotomy.

The vertical cut through the alveolus is made with either a saw or a bur. A finger should be kept on the lingual aspect of the mandible to prevent the power instrument from penetrating the mucosa. Because the lingual mucosa is very thin, some surgeons may find it helpful to place a fine ribbon retractor between the bone and the mucosa. This is done by making a horizontal incision a few millimeters lingual to the teeth and the edentulous area and then carefully elevating the mucosa down below the floor of the mouth. The retractor is then placed and supported with the surgeon's finger to ensure it is not displaced while making the cuts. When the surgical plan includes a mandibular setback, a block of bone must be removed to permit posterior positioning of the mandible. The distance between the parallel cuts necessary to remove the bone should be as close as possible to the planned setback, as determined by the model surgery. The vertical cuts are carried inferiorly to the level of the planned horizontal cut, which would be at least 5 mm below the dental apices. The inferior vertical cuts are then made, again using parallel cuts as necessary for a setback of the distal fragment. Finally, the horizontal cut is made, preferably by a saw, to minimize bone removal and endangering of the apices or the IAN (see Figure 57-19B).

The distal segment is related to the proximal segment with an occlusal splint and fixed with IMF. If the mandible is set back any significant distance, a wedge of attached tissue over

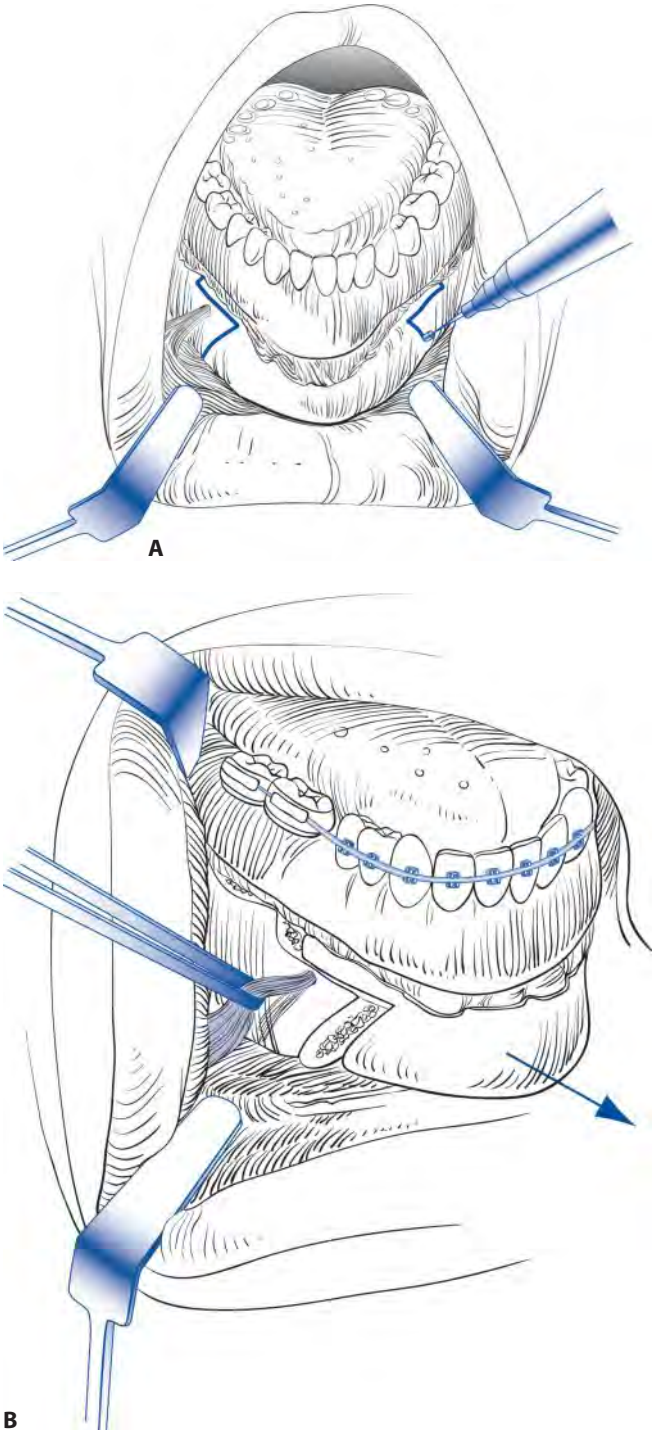


FIGURE 57-19. **A** and **B,** The step osteotomy. (**A** and **B,** Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1444.)

the alveolar vertical osteotomy must be removed to permit the setback. This wedge should be narrower than the planned movement to allow tight mucosal contact. This eliminates the need for suturing in this area, which is often difficult. Osseous wire fixation can be placed at the inferior border, or if a rigid fixation technique is preferable, straight four-holed plates with monocortical screws can be placed above and below the nerve. With a rigid fixation technique, the IMF wires can be cut and the segment stability and position checked before closure of the wound. The surgical sites are thoroughly irrigated and the mucosa is then closed with a resorbable suture. IMF, if used, is maintained for 6 to 8 weeks (Figure 57-20).

Alternative Techniques

There are multiple variations of mandibular body osteotomies. Generally, the mucosal approaches are similar, although some surgeons prefer to make a cervical incision posterior to the mental foramen and then carry it below the attached tissue in the anterior symphyseal region. This approach unfortunately presents some difficulty with wound closure, especially if IMF is used.

One of the most difficult variations in the surgical approach occurs when there is a need for visualization or exteriorization of the IAN, if an osteotomy is planned through the inferior alveolar canal. The easiest method for this is similar to that described by Epker.[210]

After the lateral surface of the mandible is exposed in the area of the planned osteotomies, parallel horizontal cortical cuts are made on either side of the anticipated route of the IAN. These cuts are extended beyond either side of the planned osteotomy, sufficient to permit adequate approach to the nerve as well as to permit enough freedom of the nerve during stretching or compression that will occur with the planned segment movements. Perpendicular cuts are made just through the cortex at approximately 1-cm intervals (Figure 57-21). Starting with the forward cuts, a thin sharp 4-mm osteotome is used to carefully start a cleavage line through the cancellous bone, preferably just below the cortex. As each individual section is broken away, the medial aspect of the fragment must be checked to ensure that a nerve is not still attached to it. After all the small lateral cortical segments have been removed, often the nerve has been exposed and judicious use of a small surgical curette is all that is needed to remove any bone spicules over the nerve as well as to carefully lift the nerve out of its canal. If cancellous bone is still covering the nerve, a medium-sized round bur can be used to carefully remove the overlying bone. A small periosteal elevator or a surgical curette can be positioned in the lateral aspect of the canal when the nerve is exposed and the thin bur used to remove the remaining overlying bone. Following the visualization or exteriorization of the IAN, the osteotomies can proceed as planned.

Although primarily used for distal fragment setback, the step osteotomy can be used for advancement. To accomplish this, a horizontal incision must be made on the lingual side of the alveolus, posterior to the vertical alveolar bone cut. The mucoperiosteum below this cut can then be elevated to allow it to be stretched with the advancement. The lateral incision is designed to permit the labial tissue to be pulled lingually, without tension, so that soft tissue closure can be done. Bone grafts are generally used in the gaps created by the advancements, especially in the alveolar area, where prostheses may later be placed.

Sagittal osteotomies of the horizontal ramus have been described with either an extraoral or an intraoral approach possible.[211,212] With the intraoral approach, the lingual exposure has to be increased to make the vertical cortical cut. This generally requires that the incision on the lingual side of the teeth be brought forward, close to the midline, so that retraction may occur without endangering the surrounding soft tissue. The advantage of this type of osteotomy is that it allows the use of rigid internal fixation with bicortical screws, and generally, a bone graft is not needed if the distal segment is advanced.

Complications

Reports of body osteotomies in the literature include mostly case reports; therefore, there are few series reporting incidence of complications. The wide variety of techniques makes it impossible to make any definitive statements about body osteotomies. Therefore, this section is primarily limited to the listing of the reported complications, many of which can be anticipated simply from the knowledge of the anatomy of the area.

Relapse

One of the primary reasons given by surgeons for using a body osteotomy procedure is the stability of the technique. Sandor and coworkers[213] assessed relapse of the step osteotomy and found it to be very stable. Most other authors have not strictly analyzed the results but claim a good long-term stability with their particular technique.[214,215]

Neural Complications

Those studies that include reports of the incidence or duration of neurosensory damage report a high spontaneous recovery rate.[214,215] Unfortunately, these studies are of questionable value because of the inadequate testing methods used. It can be expected, however, that immediate postoperative sensory loss will be present after any of the body osteotomies, especially those that require visualization or exteriorization of the IAN. The incidence of devitalization of teeth on either side of the osteotomy or of those teeth above the horizontal cut of the step osteotomy is unknown, although it is probably similar to those reported with mandibular subapical osteotomies.

The increased potential of nonunion in body osteotomies has been discussed, but the incidence is unknown. The possibility of this occurring is very low if, as has been suggested, care is used in osteotomy design to ensure sufficient bone contact as well as in the provision of adequate fixation.

The possibility of periodontal defects does exist for osteotomies made close to the teeth where the surrounding soft

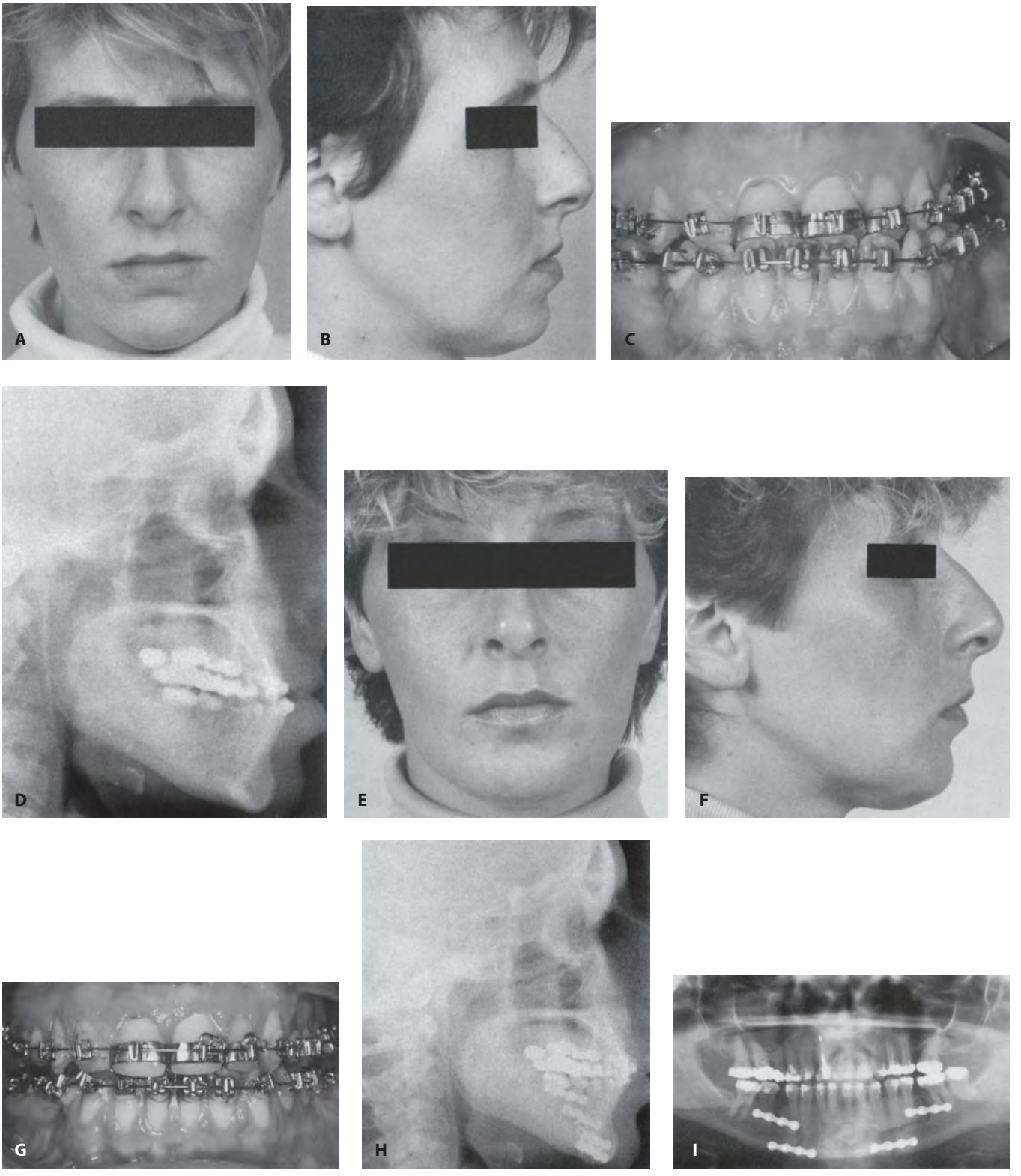


FIGURE 57-20. Patient who was treated with a step osteotomy for mandibular horizontal excess. **A-D,** Preoperative photographs and radiograph. **E-I,** Postoperative photographs and radiographs. (**A-I,** Reproduced with permission from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; pp. 1446–1447.)

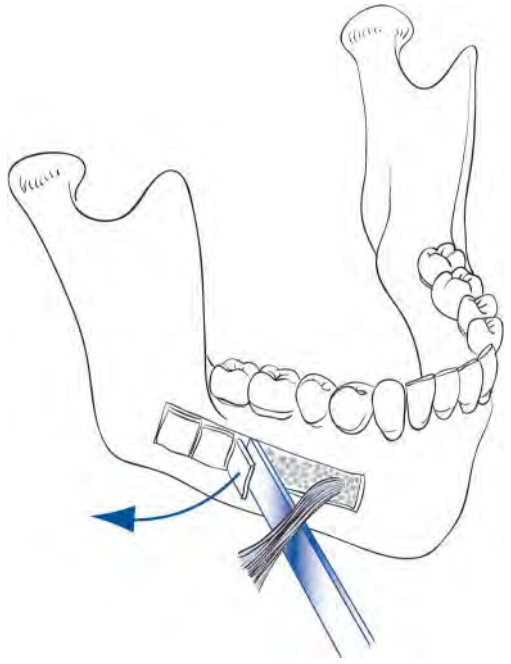


FIGURE 57-21. Decortication for exposure of the inferior alveolar nerve and vessels. (Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1448.)

tissue pedicle may be injured. As has been noted earlier, the possibility of this occurring in midline osteotomies is low, but whether this can be related to other parts of the dental alveolus is doubtful.[216]

Subapical Osteotomies

There are essentially three types of mandibular subapical osteotomies: the anterior subapical, the posterior subapical, and the total alveolar osteotomy. Whereas these techniques have fallen out of favor as primary treatment options, each has a small place in orthognathic surgery; therefore, the indications and techniques of each are described individually, and because the complications are similar, these are discussed together as well.

Anterior Subapical Osteotomy

Indications

The subapical osteotomy has historically been popular because of its versatility to move the anterior mandibular teeth and alveolus in almost every possible direction, although it is not commonly performed today. The greatest concern in this procedure is the potential for damaging teeth, and therefore, space must be present or created to permit safe vertical cuts in the dental alveolus.

Procedure

If necessary, teeth are removed to permit osteotomies or to provide space for the planned alveolar movement. The incision begins about 1 cm behind the planned vertical osteotomy and is carried forward about 4 to 5 mm below the attached tissue until reaching the cuspid, at which time, it can be positioned inferiorly and carried to the midline to connect with a contralateral incision. The periosteum is elevated, exposing the lateral cortex of the mandible, with care around the mental foramen as well as attention paid to leaving soft tissue attachments at the inferior border to ensure stability of the soft tissue–chin morphology. The attached tissue at the planned vertical osteotomy site must be elevated, and if posterior movement of the segment is anticipated, some of this tissue may have to be removed. As mentioned with the step osteotomy, the width of the tissue removed should be less than the planned posterior movement to ensure adequate soft tissue contact and coverage.

The vertical osteotomies are made using parallel cuts when the posterior movement of the segment is planned. Adequate preoperative radiologic evaluation and planning will minimize the chance of damage to the IAN. Most anterior subapical osteotomies are designed to include the cuspids and the incisors, which generally place the vertical cuts anterior to the mental foramen. Difficulties arise if the planned osteotomy includes extraction of the first bicuspid or if the cut is planned behind this area. The importance of being able to make the horizontal cut at least 5 mm below the tooth apices cannot be overemphasized. Not only is the vitality of the teeth potentially compromised, but the entire dentoosseous segment may be affected by the level of the horizontal cut. If parallel horizontal cuts are planned to move the anterior segment apically, the superior cut is made first. The inferior cut is then made, and the segment of bone is removed without overmanipulation of the dental alveolar segment that might increase the likelihood of injuring the soft tissue pedicle (Figure 57-22A). Beveling the cut from anterior to posteroinferior will minimize the amount of bone to be removed and increase the size of the lingual soft tissue pedicle. Usually, when attempting to reposition the mobile dentoalveolar segment to the rest of the mandible, further bony interferences are encountered. These exist primarily on the lingual cortex of the vertical cuts, and care must be used in the rotation of the mobile segment to access this cortex. As mentioned earlier for the step osteotomy, when possible, a retractor should be placed between the bone and the thin lingual mucosa to minimize iatrogenic soft tissue trauma.

After ensuring an adequate seating of the teeth into the surgical splint, the segment is stabilized by either wiring the splint to the teeth individually (see Figure 57-22B) or placing circumferential mandibular wires that can be combined with IMF. Osseous wires or plates with monocortical screws are rarely needed for stability, but can be used if desired. Plates and monocortical screws can be used with the splint but, generally, do not provide sufficient stability by themselves. Bone gaps caused by movement of the segment, especially by vertical movement necessary for the closure of an anterior open bite, should be grafted. The use of cortical bone from the symphysis, as advocated by Kole,[28] has been popular

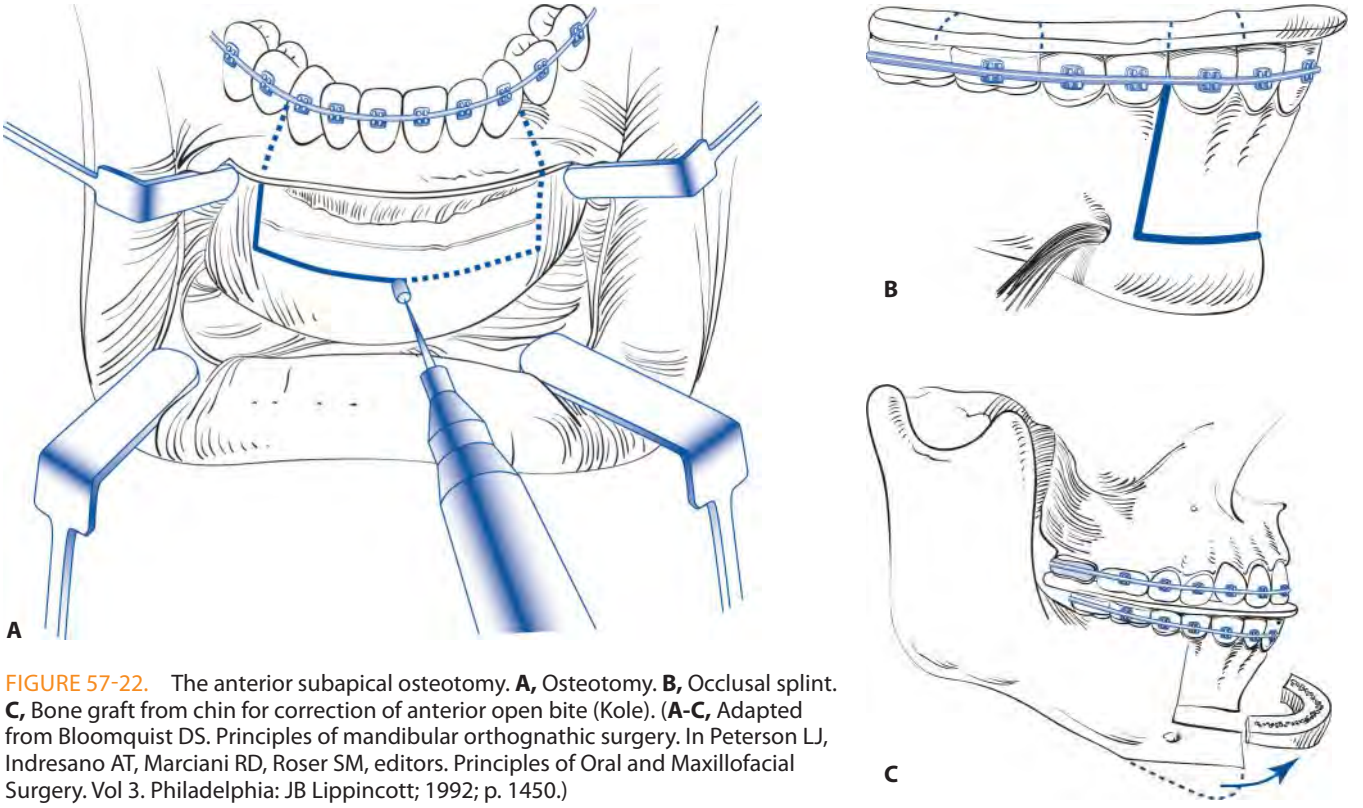


FIGURE 57-22. The anterior subapical osteotomy. **A,** Osteotomy. **B,** Occlusal splint. **C,** Bone graft from chin for correction of anterior open bite (Kole). (**A-C,** Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1450.)

because many patients with an anterior open bite have the long anterior face, which can be improved by removal of a segment of the genial bone (see Figure 57-22C). The surgical site is then irrigated thoroughly and closed with resorbable sutures.

Posterior Subapical Osteotomy

Indications

The posterior subapical osteotomy has few indications, especially if orthodontics are an option. Primarily, it can be used as an aid in the correction of supereruption of posterior mandibular teeth or ankylosis of one or more posterior teeth. Abnormal buccal or lingual positioning of these teeth can also be improved upon with this technique, when orthodontics is not available or feasible.[217]

Technique

The technique of Peterson, who first described this osteotomy,[208] can be done with local anesthesia and sedation or with general anesthesia. An incision begins 3 to 4 mm lateral to the attached gingiva, beginning at the anterior border of the vertical ramus. This incision is made down to bone and carried forward to the cuspid region. The periosteum is stripped superiorly and inferiorly sufficiently to expose the lateral cortex for the planned osteotomies (Figure 57-23). The osteotomy is outlined with a bur, based on the preoperative radiographic analysis of the length of the roots and the position of the inferior alveolar canal. The vertical cuts

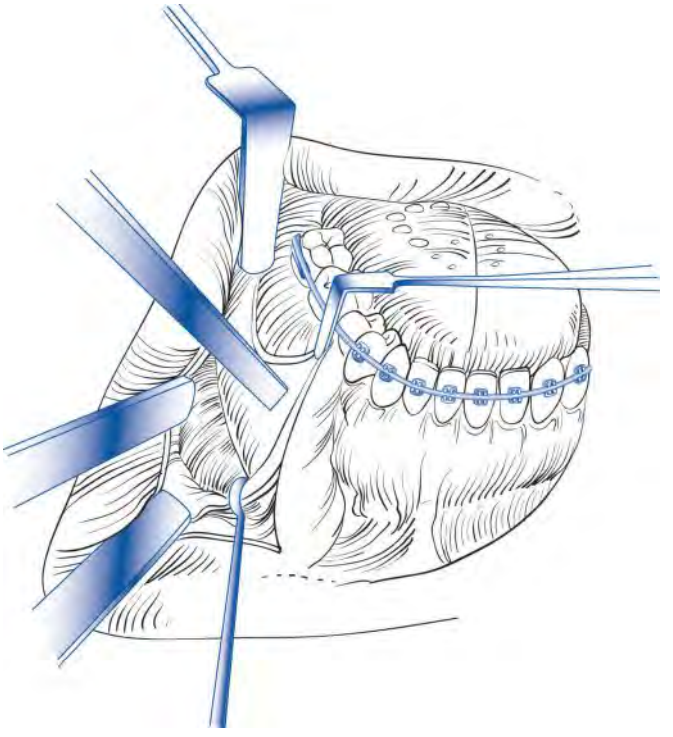


FIGURE 57-23. The posterior subapical osteotomy. (Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott Company; 1992; p. 1451.)

are made first through both cortices with a fine osteotome or thin saws. The horizontal cut is carried only through the buccal cortex, and a thick splitting osteotome is used to complete the osteotomy. Care is taken to ensure that the IAN is not caught in the mobile segment and that appropriate bony adjustments are made to permit the planned movement. The segment is positioned and stabilized with an acrylic splint and wire. Grafting is used if a bone gap is significant, and the mucosa is closed with a running resorbable suture.

Alternative Techniques

Major modification of this technique would be appropriate if insufficient distance exists between the dental apices and the inferior alveolar canal. In such a situation, the IAN may be externalized, as described previously, and a horizontal cut made through the inferior alveolar canal. Periapical radiographs taken intraoperatively after the buccal horizontal cut will ensure that the osteotomies are made at a safe distance from the teeth and canal. This latter technique has been found to be useful because adequate visualization is difficult with the presence of posterior teeth. Also, with either of these techniques, the horizontal cut can be made safely through the lingual cortex, which eliminates the lack of predictability of the induced lingual cortical fracture.

Total Mandibular Subapical Alveolar Osteotomy

Indications

The total mandibular subapical alveolar osteotomy, first described by MacIntosh and Carlotti,[218] has limited application today, but can prove valuable in isolated mandibular dentoalveolar protrusion or retrusion. It has also been advocated for closure of an anterior open bite when used with a bone graft.

Technique

An incision begins on the external oblique ridge of the base of the vertical ramus. The incision is carried down to bone and extends forward 4 to 5 mm below the attached gingiva. The incision can be positioned inferiorly in the region of the canine and forward where it meets the contralateral incision at the midline. The periosteum is elevated to expose the lateral cortex, with care taken around the mental nerve, as well as leaving some attachment at the inferior border of the symphysis for the soft tissue chin. The vertical cut posterior to the terminal molar is made first and carried down to the level of the planned horizontal osteotomy. As with the step osteotomies, the horizontal cut needs to be placed appropriately, based upon preoperative periapical radiographs. If this cut cannot be made safely between the dental apices and the inferior alveolar canal, the IAN needs to be exteriorized or the cut placed below the canal (Figure 57-24A). The horizontal cut can then be placed low enough to be at a safe distance from the dental root apices, as well as maintaining an adequate vascular pedicle to the dental alveolus. The angle of the horizontal cut should be made to facilitate segment movement; for instance, a flat cut paralleling the maxillary occlusal plane permits the straight advancement of the segment without significantly changing mandibular vertical height while at the same time maintaining a large area of bone contact.

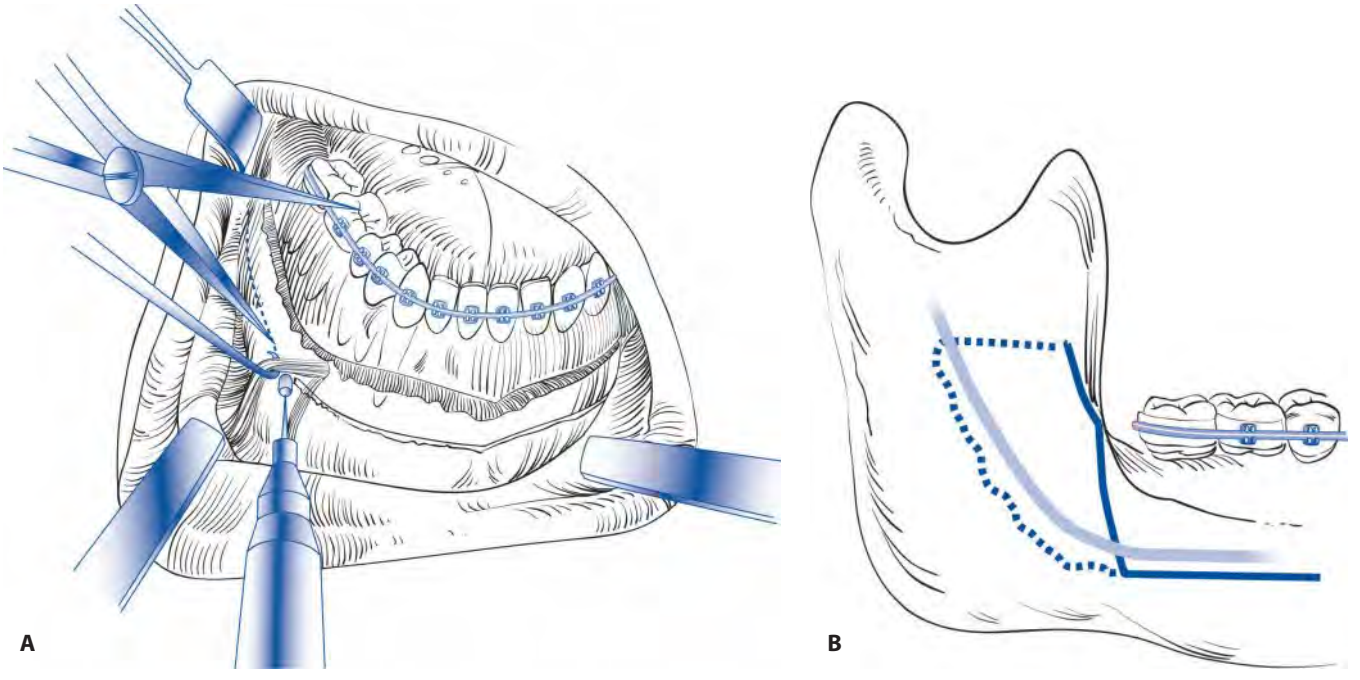


FIGURE 57-24. **A** and **B,** Variations of the total mandibular alveolar osteotomy. (**A** and **B,** Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1451.)

The mobile segment is related to the maxilla with an acrylic interocclusal splint and IMF. Osseous fixation can be achieved either with the lateral cortical wires placed in the first bicuspid area along with maintaining IMF or by rigid internal fixation with monocortical plates and screws. As with correction of an anterior open bite, any significant bone gaps created may be filled with graft material. The wound is thoroughly irrigated and closed with resorbable suture.

Technique Variations

Booth and associates[219] suggested a variation of the total mandibular subapical osteotomy that combines the SSO of the vertical ramus with the total mandibular alveolar osteotomy (see Figure 57-24B). This modification has a number of advantages over the original technique. First, the osteotomy is made below the inferior alveolar canal, thereby decreasing the risk of damaging the IAN and tooth roots, at the same time preserving the vascular supply to the mobilized segment. Also, the sagittal part of the osteotomy allows a larger bone contact area to assist in bony healing.

The total mandibular alveolar osteotomy can also be divided into interdental segments to correct axial inclination of teeth or close edentulous sites. These modifications are not easily performed with Booth's osteotomy, but can be valuable as a variation of the original procedure.

Complications

The complications of all mandibular subapical alveolar osteotomies are considered together owing to their similarities. Stability is often mentioned as one of the advantages of the alveolar osteotomies because of the minimal soft tissue forces acting on these areas. Unfortunately, there have been very few studies to document this claim, and the technique has limited use. Available studies question the stability of these segmental osteotomies. Theisen and Guernsey[220] evaluated six patients who had anterior subapical osteotomies. At 1 year after surgery, an average of 1.0 mm of movement of the incisors was noted on lateral cephalograms. In contrast, Kloosterman[48] evaluated a much larger group and found a 30% recurrence of an open bite after anterior maxillary osteotomies. Pangrazio-Kulbersh and MacIntosh[221] compared the hard and soft tissue changes in a retrospective study comparing total subapical osteotomies with BSSOs for treatment of mandibular retrognathia. They found no significant difference in skeletal changes at 1 year. Interestingly, the major difference between the two groups was at soft tissue "B" point, with the subapical osteotomy resulting in a significant decrease in the labiomental angle.

Unlike segment stability, neurologic and vascular complications of segmental osteotomies have received a great deal of attention. Much of the research has focused on pulpal changes and not on peripheral IAN sensory loss or avascular necrosis with bone loss. Both of these latter problems are recognized complications of mandibular subapical osteotomies, but there are no clinical studies noting their exact incidence.[28,36] Many authors report some incidence of sensory change of the lower lip and chin but claim that there have been no permanent problems with the return of normal sensation in about 3 months.[218,229,221]

Clinical and animal studies on the effect of mandibular osteotomies on pulpal blood flow are numerous.[35,37,39,40] An early animal study did not note many vascular changes when a lingual vascular pedicle was maintained.[33] However, all subsequent studies have noted a significant decrease in blood flow, especially to the dental pulp.[222] Histologic studies of dental pulp after subapical osteotomies reveal some pulpal necrosis in most teeth.[35,223] Whether this is of clinical importance is questionable, because there were relatively few teeth in clinical series that required endodontics or extraction.[47,48] It is likely that some pulpal necrosis occurs in greater numbers of teeth than are clinically evident.[224] The only way that pulpal changes have been assessed clinically is with "vitality testing" and laser Doppler flow studies. A change in pulpal nerve sensation does not necessarily correlate with a decrease in pulpal vascularity. However, the rate of recovery of sensory loss seems to give some measure of the iatrogenic trauma to the teeth. Early clinical studies seem to show that teeth in mandibular alveolar osteotomies fare better than their maxillary counterparts.[225] More recent and larger studies, however, have demonstrated the reverse.[47,48] One report found that approximately 40% of the teeth in the repositioned mandibular segment remain unreactive to vitality testing at 1 year.[47] This high incidence of pulpal trauma was attributed to technical errors during surgeries. The incidence of teeth requiring endodontics range from 1.5% to 10%. The teeth at greatest risk for damage are those in the mobile segment directly adjacent to the vertical osteotomies. The teeth immediately posterior to the vertical cut are approximately equal in risk to the teeth in the center of the mobile segment.

Periodontal problems have been mentioned by authors reviewing mandibular alveolar osteotomies.[48,220] The incidence and quantitative evaluations of soft and hard tissue loss have not been done, although individual cases of significant interdental bone loss have been noted. Periodontal problems are seen less frequently when the vertical cuts are made in extraction sites than when the cuts are attempted between teeth without extraction.[48]

Horizontal Osteotomy of the Symphysis (Genioplasty)

The horizontal osteotomy of the symphysis, or genioplasty procedure, differs very little from that originally described by Hofer,[32] except that the procedure is routinely performed intraorally. The versatility of this procedure for correction of skeletal deformities of the chin is impressive, and the results are superior to those of synthetic implant placement in most cases.

Indications

This osteotomy of the anterior inferior border of the mandible, with minor variations, can be used to improve almost

every conceivable skeletal abnormality of the chin. The technique is primarily used only for aesthetic reasons, and therefore, its use depends upon the patient's concern about appearance of this area of the face, more commonly in syndromic cases. Often, the surgeon has to bring the genioplasty to the patient's attention when other facial osteotomies are planned because of the impact that these osteotomies will have on chin prominence and aesthetics. The indications, therefore, are often made apparent by comprehensive presurgical treatment planning.

Technique

The horizontal osteotomy of the symphysis is often done in conjunction with other major osteotomies and, thus, is frequently accomplished under general anesthesia. However, it can be performed as a separate procedure on an outpatient basis with sedation and local anesthesia.

The mucosal incision is made on the labial side of the vestibule at approximately 1 cm above its depth and extends posteriorly to the first bicuspid region. This incision is carried just below the mucosa to the depth of the vestibule and then angled directly to the labial cortex through the mentalis muscle (Figure 57-25A) in an "apron" fashion with the posterior limbs closer to the attached gingiva to avoid the terminal branches of the mental nerve exiting from the mental foramen. The periosteum is elevated inferiorly to a point just below the intended level of the osteotomy. Laterally, the periosteum is elevated to the mental foramen and then extended posteroinferiorly to the mandibular inferior border below the mental foramen bilaterally. The extent of the posterior cortical exposure is generally determined by the position of the mental foramen and the vertical height of the mandible in this area. In most cases, the exposure terminates in about the first molar area. Caution must be taken if the genioplasty is performed at the same time as a BSSO procedure, because not only with the incisions closely approximate one another but also the osteotomies nearly overlap. In general, no attempt is made to expose the mental nerve by releasing the soft tissue around it, primarily because the nerve can be small and friable, making inadvertent severing a possibility. Therefore, "skeletonization" of the mental nerve should be avoided in all cases. The periosteal elevation posterior to the mental foramen is minimized to only what is necessary for placement of a narrow channel retractor and the reciprocating saw blade or bur.

It is helpful at this point to inscribe a vertical mark (or two or three marks) into the bone across the planned osteotomy site in the anterior mandible so that the transverse position of the inferior fragment can be more easily reoriented after completion of the osteotomy. The osteotomy cut is then made with a reciprocating saw, preferably because this technique minimizes soft tissue trauma on the lingual aspect of the mandible (see Figure 57-25B). The length and angle of the horizontal cut may have profound effects on positioning of the mobilized segment as well as the final postsurgical results. Further osteotomies or osteoplasties of the chin are made after mobilization of the inferior segment. The stabilization of the segment in its new position can be done with cortical wires, circumandibular wires, or plates and screws; prebent chin plates are available with varying degrees of advancement in millimeters, and these may be used in a reverse fashion for setback procedures of the chin. For vertical augmentation or reduction, standard four-hole plates with screws may be used, possibly using a bone graft, if necessary. The wound is irrigated and closed in two layers, mentalis muscle and mucosa, with resorbable suture. Care should be taken to reapproximate the mentalis muscle formally in order to prevent chin ptosis ("witch's chin") postoperatively. Elastoplast tape, or similar tape, should be placed across the lip and chin to support and maintain mentalis position, and it is continued for 2 to 5 days to minimize hematoma formation and to support the suture repair. Patients should be instructed

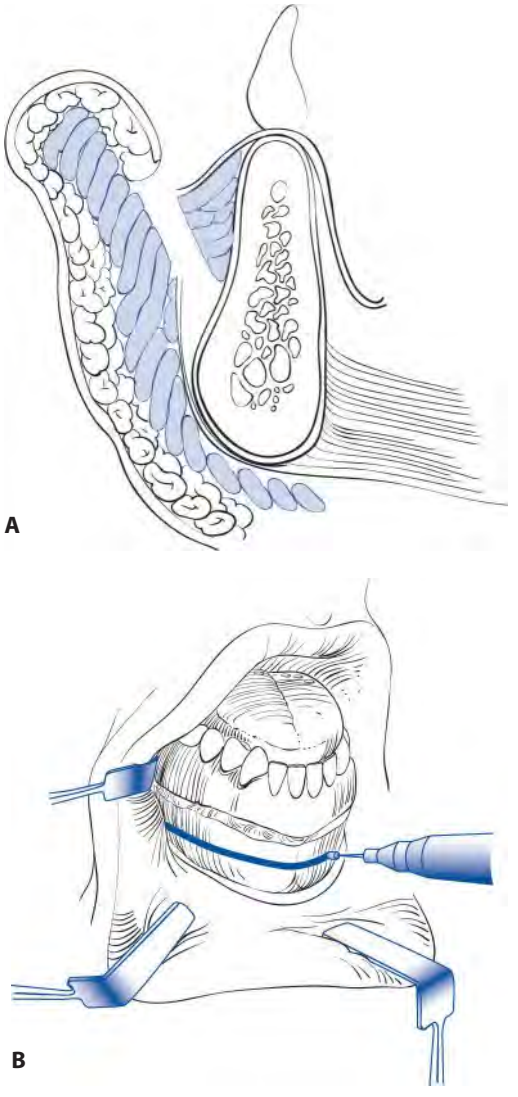


FIGURE 57-25. The surgical approach for the horizontal osteotomy. **A,** Soft tissue approach. **B,** Osteotomy. (**A** and **B,** Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1453.)

not to pull on their lip in order to minimize the possibility of wound dehiscence.

Alternative Techniques

The primary technical alterations in the horizontal osteotomy are based upon variations in the osteotomy design; and these design differences depend upon the preexisting symphyseal deformity that is being corrected with genioplasty. Obwegeser[100] concentrated on correction of horizontal deficiency of the chin when he described the basic procedure (Figure 57-26A). He suggested that a midsagittal osteotomy of the inferior fragment may be helpful in preventing the prominence of the posterior ends of the fragment, relative to the body of the mandible, as the fragment is advanced (see Figure 57-26B). A narrower chin point can also be obtained by taking a wedge of bone out from the lingual aspect of this cut (see Figure 57-26C). The length of the cut posteriorly has important aesthetic consequences, and anegonial notching can be unaesthetic when it occurs at the junction of the distal portion of the advanced genial segment and native mandible and subsequent bony remodeling that occurs in that region. Most notably, larger advancements require a larger cut to the first or second molar region. This permits a smoother line to the inferior border of the mandible. Overlapping an advanced inferior fragment on the lateral cortex of the symphysis allows both an increase in horizontal prominence as well as a decrease in the anterior mandibular vertical height (see Figure 57-26D).[226] Larger advancements of the inferior fragment can be obtained by double or triple osteotomies, rotation of the fragment combined with a graft at the posterior gap, and bone graft between the symphysis and the fragment (see Figure 57-26E-G).[227,228]

Horizontal chin excess is traditionally treated by positioning the inferior segment posteriorly.[229] Depending on the angle of the cut, this may also increase anterior facial height. Sometimes when this is done, it is necessary to remove the posterior ends of the inferior fragment to prevent unsightly protrusions from the inferior border of the mandible (see Figure 57-26H). When a patient has a normal facial height, the plane of the osteotomy should parallel the Frankfort horizontal or natural head position (NHP), if at all possible. The anterior chin projection may be reduced by using parallel, or V-shaped, osteotomies in a more vertical plane, with the middle segment removed (see Figure 57-26I).

Vertical symphyseal excess can be reduced by removing the middle segment of bone when the plane of two parallel osteotomies is more horizontal (see Figure 57-26J). These cuts, however, do not always need to be parallel and, in fact, should be designed to fit the particular structural problem. This design also permits the correction of a mild horizontal deficiency that is combined with a mild vertical excess (see Figure 57-26K). This skeletal problem can also be corrected with a single osteotomy placed more vertically and by repositioning the segment anteriorly (see Figure 57-26L). Vertical symphyseal deficiency can be addressed only with an interpositional graft, with either bone or other implant material (see Figure 57-26M). The use of plates and screws alone to hold the segment in an inferior position has been suggested as well (Figures 57-27 and 57-28).

The use of wires, pins, screws, or plate and screws for the fixation of the inferior segment is common. Precious and associates[230] evaluated the changes that occur as the bone remodels after a horizontal osteotomy. They recommended that the fixation take into account these changes, especially the positioning of rigid fixation devices. Plates placed along the superior border of the inferior fragment may become noticeable to the patient as the bone remodels.

Finally, a horizontal osteotomy can be used in combination with a midline osteotomy and BSSO to allow narrowing of the mandibular arch form and at the same time advancing or setting back the chin. In this situation, the inferior segment acts as a plate and with two screws placed on either side of the vertical cut in order to stabilize the segments (Figure 57-29).

Complications

The incidence of postoperative problems after a horizontal osteotomy of the symphysis is rarely mentioned. This may be because genioplasties are frequently done in conjunction with other osteotomies, which makes the designation of various complications to one particular portion of the surgery difficult. Van Sickels evaluated sensory loss from SSOs and the effect of other factors, including genioplasty.[152] He found that there was greater initial sensory loss with genioplasty in all age groups, but prolonged neurosensory dysfunction persisted only in older patients. It is intuitive that a BSSO combined with a genioplasty would have a higher incidence of IAN dysfunction than either procedure performed alone, and the literature confirms this suspicion. The possibility of sensory loss from genioplasty alone has not been adequately evaluated, although in one study, it was noted that postsurgical sensory loss was found in all patients, but it was temporary, with normal sensation returning within 12 months.[231] Another study reported a 3.5% long-term incidence of sensory deficits after genioplasties.

Most of the literature concerning genioplasties concentrates on the soft tissue response to the skeletal movement. Reports of relapse after genioplasty are few and conflicting. Some clinicians report that there is essentially no relapse after a genioplasty, noting only a spontaneous remodeling of the sharp corners of the advanced segment occurring with time.[232–235] These studies, however, follow patients for only up to 1 year. Two other studies with follow-up of at least 1 year do seem to show some instability of the skeletal chin advancement.[236,237] The mean relapse with a genial advancement varies widely (from 2.6% to 30%), and the one consistent finding is that most of the skeletal relapse occurs within the first year. As with stability studies of other osteotomies, there exists a large variation in individual relapse, and no attempt has been made to identify the specific etiologies. However, there are probably many factors involved, including patient age and the magnitude of advancement. There have been no studies on the stability of this surgery in correction

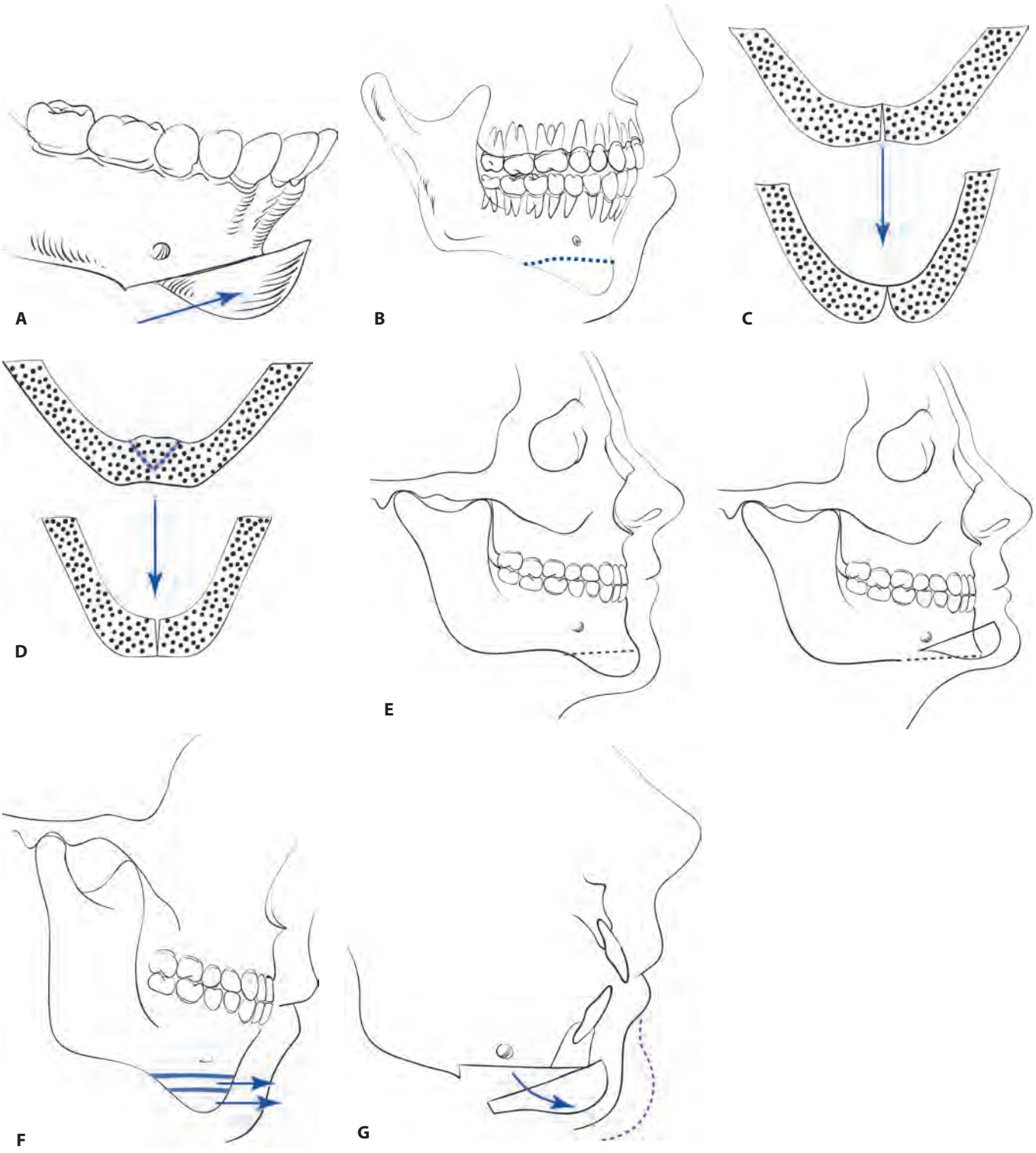


FIGURE 57-26. **A-G,** Various forms of the horizontal osteotomy. **H-M,** Various forms of the horizontal osteotomy. (**A-M,** Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; pp. 1454–1455.) *(Continued)*

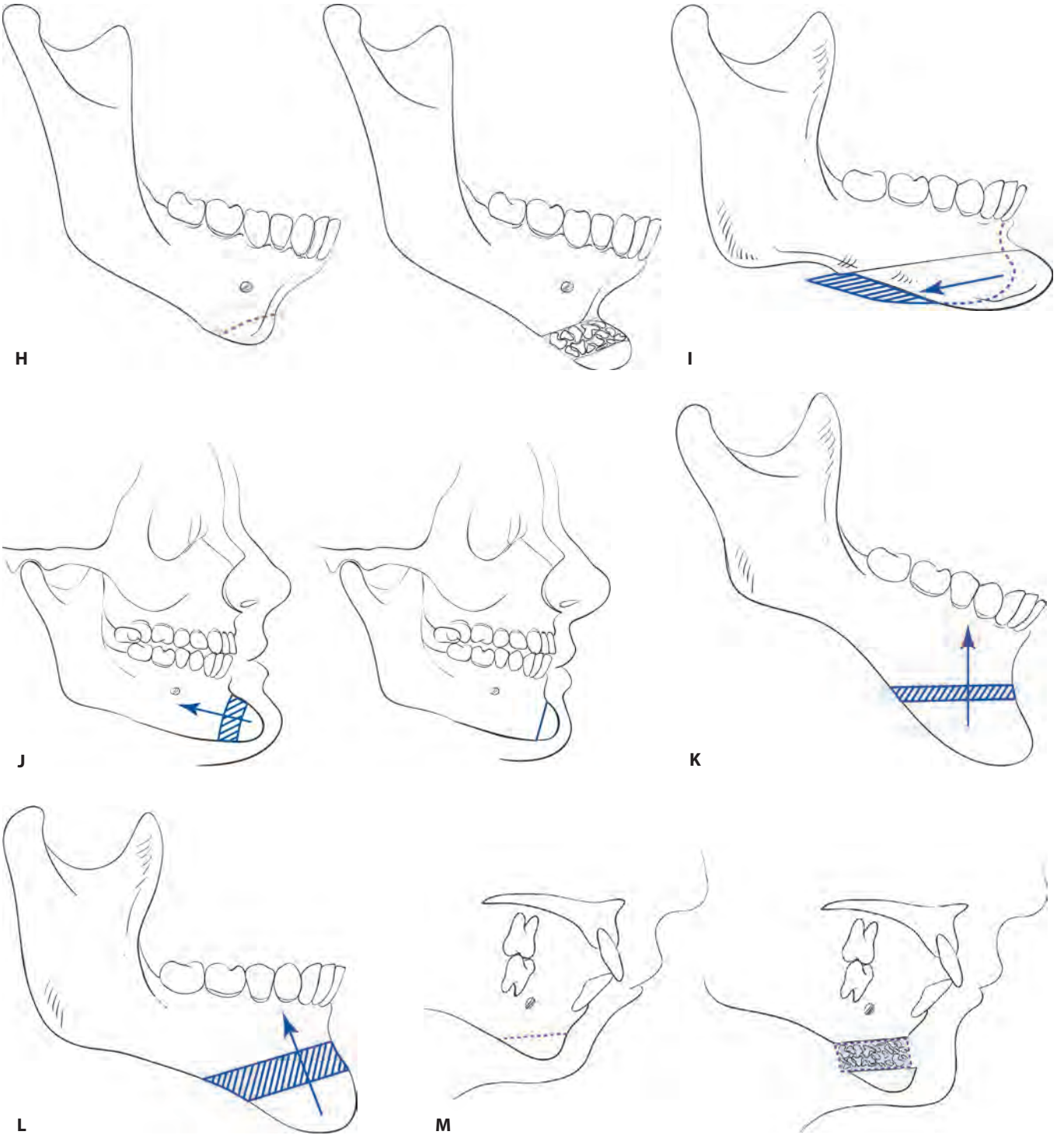


FIGURE 57-26. *(Continued)*

of other symphyseal deformities. Martinez and colleagues[234] found that regeneration of the cortical thickness of the symphysis was significantly better in patients younger than 15 years of age. They suggested that this may be beneficial if further surgical advancement of the chin is to be considered. Other complications, such as bone loss and infection, have been reported, but small samples preclude any definitive conclusions regarding precise incidence.[238]

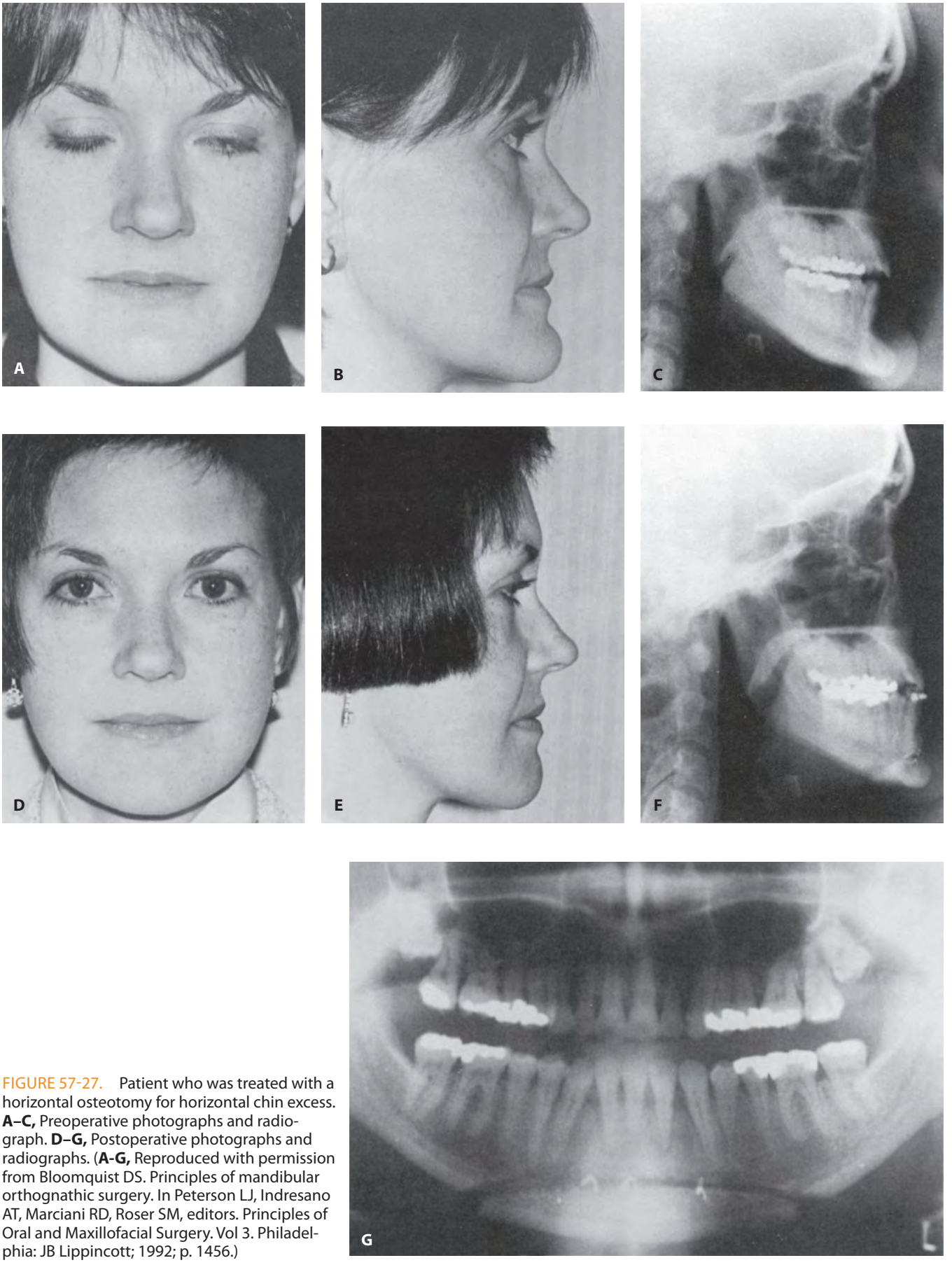


FIGURE 57-27. Patient who was treated with a horizontal osteotomy for horizontal chin excess. **A–C,** Preoperative photographs and radiograph. **D–G,** Postoperative photographs and radiographs. (**A-G,** Reproduced with permission from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1456.)

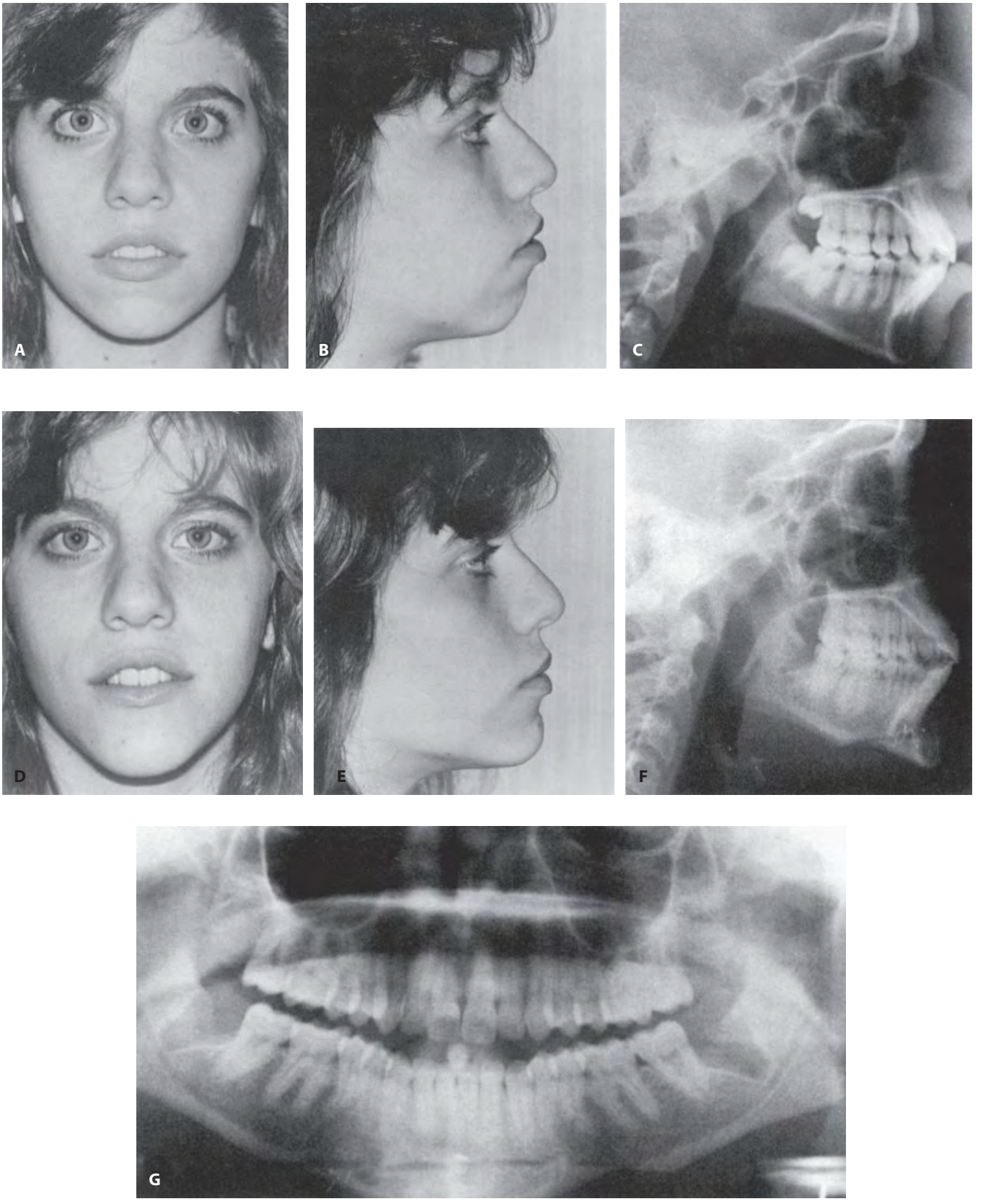
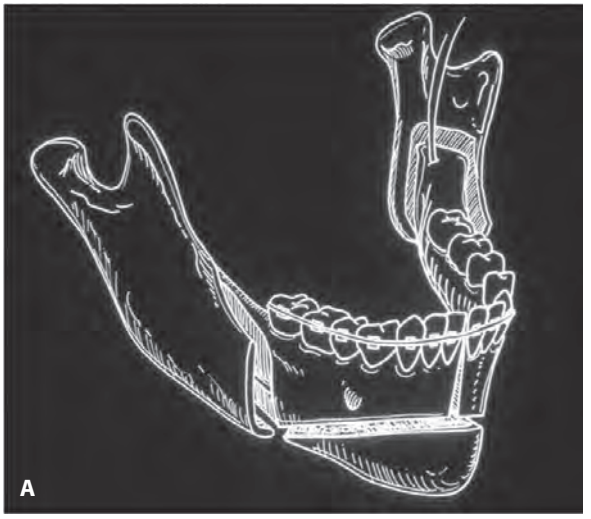
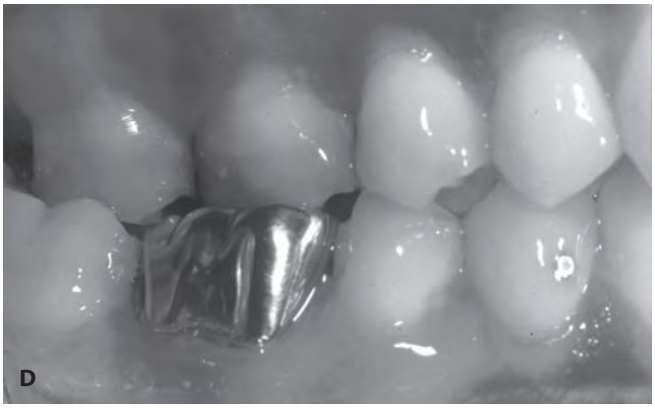


FIGURE 57-28. Patient was treated with a horizontal osteotomy for horizontal chin deficiency. **A–C,** Preoperative photographs and radiograph. **D–G,** Postoperative photographs and radiographs. (**A-G,** Reproduced with permission from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1457.)

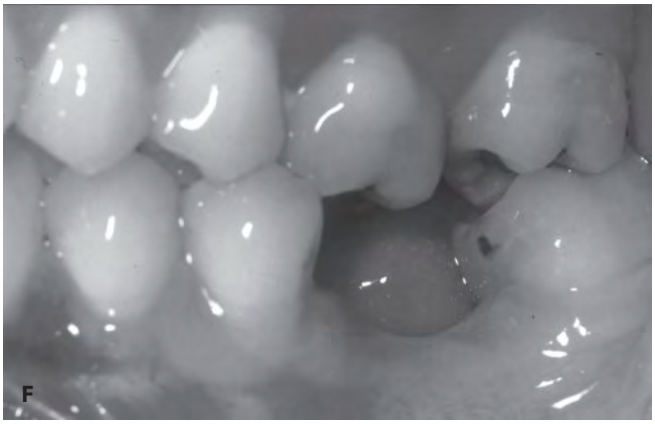












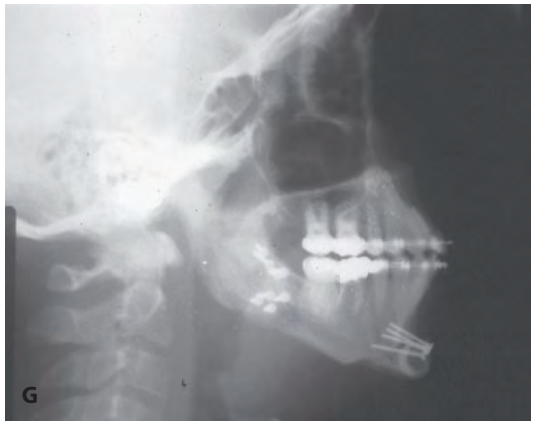




FIGURE 57-29. BSSO combined with midline mandibular osteotomy for mandibular narrowing, and genioplasty. **A,** Osteotomy designs. **B-F,** Pretreatment records. **G-M**, Postoperative records. *(Continued)*

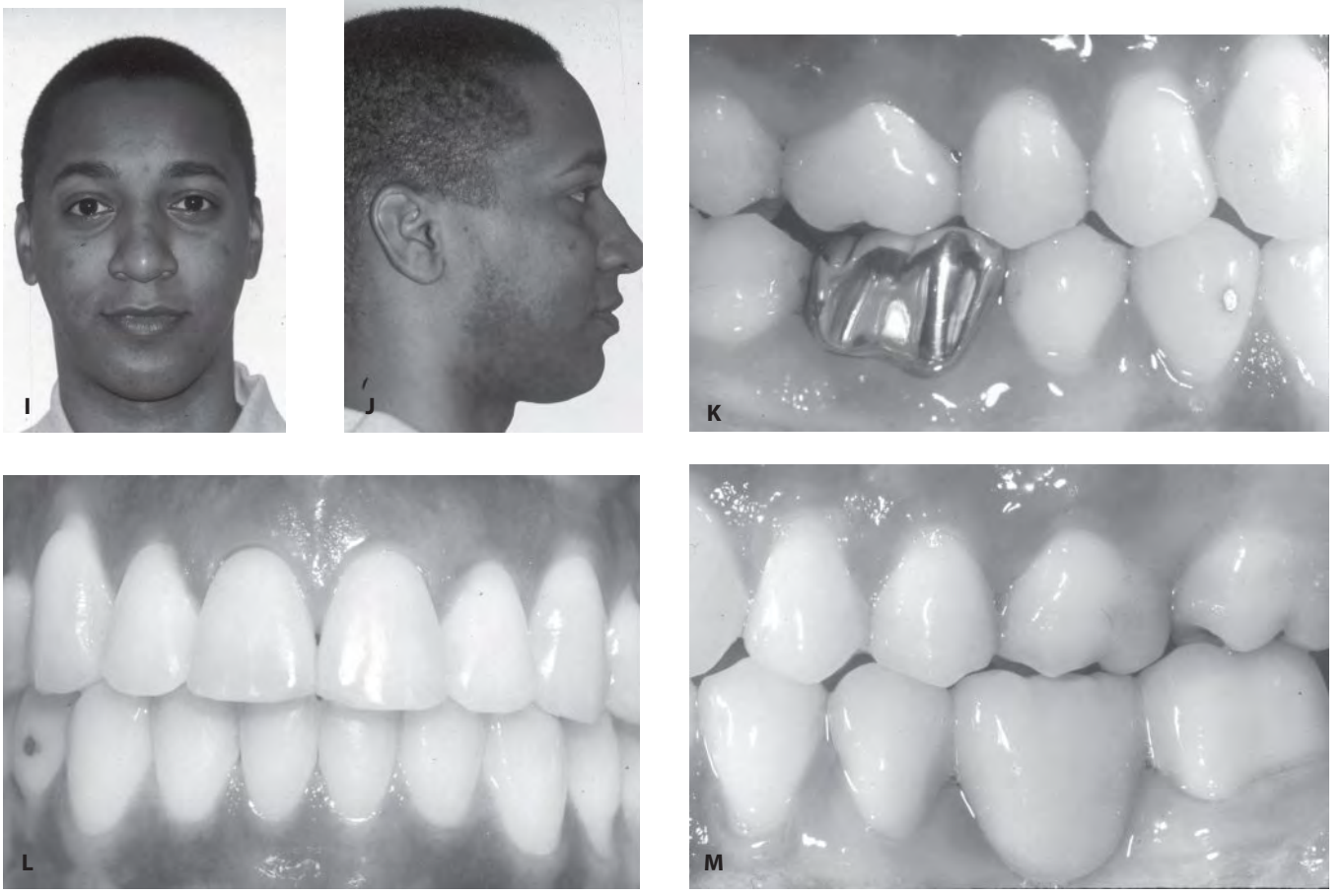


FIGURE 57-29. *(Continued)*

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CHAPTER

Principles of Maxillary Orthognathic Surgery

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HISTORICAL PERSPECTIVES

Orthognathic surgery involving the maxilla was first described in 1859 by von Langenbeck[1] to provide surgical access for the removal of nasopharyngeal polyps. The first report of a maxillary osteotomy in the United States was by Cheever in 1867[2] for the treatment of complete nasal obstruction secondary to recurrent epistaxis for which a right hemimaxillary down-fracture was performed. Throughout the next century, numerous authors described various osteotomy designs and techniques that included mobilization of the entire maxilla, mostly for access and treatment of pathologic processes. In 1901, Rene Le Fort[3] published his classic and vivid descriptions of the natural planes of maxillary fracture by applying blunt forces to cadaver head specimens. In 1927, Wassmund[4] first described the Le Fort I osteotomy for the correction of midface deformities. However, complete mobilization of the maxilla with immediate repositioning was not performed until 1934 by Axhausen.[5] Separation of the pterygomaxillary junction was advocated by Schuchardt in 1942.[6] Moore and Ward in 1949[7] recommended that a horizontal transsection of the pterygoid plates is necessary for maxillary advancement. Willmar[8] reported on over 40 patients treated in this manner with a horizontal osteotomy through the pterygoid plates and described severe bleeding in most cases likely due to laceration of one of the pterygoid branches of the maxillary artery; this technique was abandoned in favor of a vertical separation of the maxilla from the pterygoid plates at the pterygomaxillary suture or junction. Most of the early technical descriptions simply mobilized the maxilla by releasing at least some bony attachments and then placing orthopedic forces with elastic traction on the maxilla to achieve the desired movement, in a sort of unintentional distraction osteogenesis procedure. As expected, owing to soft tissue restriction, uncontrolled distraction forces, and the lack of wire or rigid fixation of the maxilla, these techniques were associated with a high degree of bony relapse. In 1965, Obwegeser[9] recommended complete mobilization of the maxilla so that repositioning could be accomplished without soft tissue or bony resistance, and this notion proved to be a major advancement in the concept of maxillary stability, as documented later by Hogeman and Willmar,[10] and Perko.[11]

Due to early concerns regarding vascular supply to the maxilla, and the belief that complete mobilization would result in complete loss of vascularity to the entire maxilla, anterior segmentalization of the maxilla was advocated in the early descriptions of maxillary surgery in order to preserve blood supply to the posterior segments, including reports by Cohn-Stock,[12] Wassmund,[4] and Spanier.[13] Because complete mobilization of the maxilla was not performed, instability and relapse occurred commonly. In addition, because surgical access was performed through palatal incisions, vascularity to the anterior maxillary segment was severely compromised. Cupar,[14,15] Kole,[16] and Wunderer[17] reported a more direct surgical access to the anterior maxilla via vestibular incisions with improved mobilization of the maxilla and maintenance of blood supply to the anterior maxillary segment. Posterior segmentalization of the maxilla was used by Schuchardt,[18] but owing to incomplete mobilization, it had limited stability with hard and soft tissue relapse. Kufner[19] improved on this posterior segmental osteotomy technique in attempt to decrease relapse by complete mobilization of the osteotomized segments before surgical repositioning. The next logical step in the evolution of maxillary surgery was to combine the techniques of anterior and posterior segmental osteotomies in order to accomplish a total maxillary alveolar osteotomy (TMAO) for segmental manipulation and surgical

repositioning simultaneously.[20,21] Several forms of TMAOs were described by Converse and Shapiro,[22] Cupar,[14] and Kole.[23] Willmar[8] further established the stability achieved by performing a complete Le Fort I osteotomy, and Bell[24] documented the overall superiority of the total down-fracture Le Fort I osteotomy for segmental and one-piece maxillary osteotomies. In addition, in 1975, Bell[24] documented that the vascular supply to the down-fractured, completely mobilized maxilla is preserved via the buccal and palatal soft tissues and that the descending palatine vessels may be sacrificed without vascular compromise to the surgically repositioned maxilla.

In further consideration of maxillary stability before rigid fixation techniques, block bone grafting to improve stability was advocated by Gillies and Rowe,[25] Cupar,[14,15] and Obwegeser[26] who first advocated grafting in the pterygomaxillary fissure. Throughout the years, clinicians have advocated block grafting, using autogenous iliac crest blocks, allogeneic block grafts, or synthetic materials (e.g., block hydroxylapatite) in the bony gaps created by maxillary osteotomy but specifically placed in the pterygomaxillary buttress or piriform rims. The use of block grafting to improve stability remained a controversial procedure despite the promulgation of certain guidelines for grafting (i.e., >6 mm of maxillary advancement, or cleft lip and palate cases), and interestingly, Willmar[8] did not find a difference in stability with and without bone grafting in a series of noncleft maxillary osteotomies. Early descriptions of rigid fixation of maxillary osteotomies were published by Michelet in 1973,[27] Horster in 1980,[28] Drommer and Luhr in 1981,[29] and Luyk and Ward-Booth in 1985.[30] Since that time, a wide variety of methods have been advocated for rigid internal fixation of maxillary osteotomies, including stainless steel or titanium bone plates and screws, metallic mesh, pins, rigid adjustable pin (RAP) system and resorbable (polyglycolic acid [PGLA], poly-L-lactic acid [PLLA] polymers) fixation.[31–33] Despite these descriptions of various surgical techniques throughout the past century, there remains a lack of consensus among surgeons regarding the specifics of the surgical technique utilized for maxillary mobilization, the considerations for maxillary distraction osteogenesis versus one-stage Le Fort surgery, the need for adjunctive bone grafting, the specific "rigid fixation" devices used (e.g., number of plates needed, appropriate plate and screw thickness and diameter), and the use of postoperative intermaxillary fixation (IMF).

BASIC PRINCIPLES OF MAXILLARY SURGERY

Maxillary deformities may manifest in any of the three planes of space: sagittal, axial, and coronal. Patients display abnormal facial anatomy and exhibit elements of maxillary and mandibular deformities, and therefore, the clinician must be able to recognize and treat a variety of maxillary and midface deformities. Subjectively, patients with maxillary deformities often describe their dentofacial problem in terms of the relative mandibular appearance, such that maxillary hypoplasia may be attributed to mandibular hyperplasia ("pseudoprognathism"). Patient expectations clearly demonstrate the importance of the position of the mandible and the chin in patient aesthetic satisfaction.[34] This perceptual preoccupation with apparent mandibular excess or deficiency in the absence of a significant absolute mandibular abnormality may require additional patient education and guidance from the surgeon in order to assist the patient in recognizing the contribution of the midface and maxilla to her or his overall facial appearance. In a similar fashion, the patient may attribute a convex facial profile to an abnormal nasal prominence in describing the chief complaint, which may be due to maxillary anteroposterior (AP) excess and/or mandibular AP deficiency.

Therefore, the clinical data base should include a comprehensive history and physical examination, dental model analysis and model surgery, and cephalometric analysis with prediction tracings in order to determine the list of treatment options. These important diagnostic and treatment planning modalities are discussed extensively in Chapter 56; however, model surgery may be the most valuable tool in preparing for orthognathic surgical correction of skeletal facial deformities. Whereas model surgery is essential for immediate preoperative surgical simulation and splint construction, it may be even more important in the early phases of treatment planning. Before initiating orthodontic or surgical treatment, model surgery is the best method to determine the final postoperative position of the mandible as well as the maxilla. No cephalometric prediction (computer-generated or hand-drawn) or photographic image manipulation can detail all of the three-dimensional and occlusal information obtained from accurate model surgery. In the pretreatment stages, the teeth may not fit together perfectly during preliminary model surgery, but orthodontic tooth movement can be simulated to permit an accurate prediction of the specific movements required in the maxilla and mandible to achieve the desired final surgical result. The model surgery measurements made at the time of this pretreatment setup should be exactly the same as those used for the actual preoperative model surgery. Pretreatment model surgery is essential when contemplating maxillary surgery alone and is also very useful when planning two-jaw surgery. Pretreatment model surgery permits three-dimensional evaluation of the maxilla and the mandible, independent of whether autorotation or osteotomy of the mandible is part of the final surgical treatment plan. It should be mentioned that the entire process of presurgical assessment and treatment planning for orthognathic surgery is undergoing major advances with three-dimensional technology, which may improve efficiency and accuracy for severe, syndromic, and/or asymmetrical skeletal discrepancies.

MODEL SURGERY

Model surgery may be useful in mandibular surgery from an academic standpoint, although the final mandibular position is determined by the occlusion in all three planes of space,

but it is particularly important in maxillary, and two-jaw, orthognathic surgical planning. The information obtained from precise model surgery permits accurate surgical planning, reveals the three-dimensional changes that are planned in the surgery, and facilitates the exact positioning of the jaws. Model surgery is critical for two-jaw surgery and fabrication of intermediate and final surgical splints, and when applied to isolated maxillary surgery, model analysis may reveal that, in fact, maxillary surgery alone is not sufficient to most appropriately treat the skeletal deformity. The principles of model surgery for orthognathic surgery, including both one- and two-jaw surgery, are covered in Chapter 56.

SURGICAL ANATOMY

Osseous Structures

The body of the maxilla contains the maxillary sinus in its entirety, except rarely when the apex of the sinus extends into the zygomatic bone.[35] The anterior, or facial, surface of the maxilla is composed of the anterolateral wall of the sinus. The infraorbital foramen is located at variable distances between the inferior orbital rim and the maxillary alveolus. The alveolar jugae, or root prominences, visible through the facial surface of the maxillary alveolar process are more prominent anteriorly, and the longest root in the maxilla of the canine tooth, form the canine fossa just lateral to the root juga. The anterior aspect of the alveolar process of each side of the maxilla surrounds the piriform aperture, or rim, and the maxillary suture in the anterior midline unites to form the anterior nasal spine (ANS). This bony prominence forms the most anterior and inferior, or caudal, support for the flexible anterior cartilaginous nasal septum. Proceeding posteriorly from the ANS, the nasal crest of the maxilla rises sharply to form a crest at the junction of the anterior and nasal surfaces of the maxilla, which forms the nasal floor and results in an inclination of this structure superiorly, or cranially, at the piriform aperture. The body of the maxilla and its frontal process form the superolateral boundary of the piriform aperture as a thin edge of bone. The piriform rim itself represents a vertical pillar of midfacial support (Figure 58-1).

In the midline between the nasal cavities, the nasal crest of the maxilla articulates with the septal, or quadrangular, cartilage, the vomer, and the perpendicular plate of the ethmoid bone.[36,37] The nasal septal cartilage rests in a central groove of the nasomaxillary crest, which extends posterior from the ANS to the posterior nasal spine (PNS). This bony-cartilagenous articulation is flexible, but it is stabilized by the perichondrial-periosteal continuity and interposed connective tissue. In the midline, at the junction of the premaxilla and the maxilla, or the primary and secondary palate, is the incisive foramen or fossa, which typically contains the openings of four canals through which the nasopalatine arteries and nerves pass.

The hard palate is formed by the midline fusion of the palatine processes of the two hemimaxillae and the horizontal lamina of the palatine bones.[38] The transverse suture

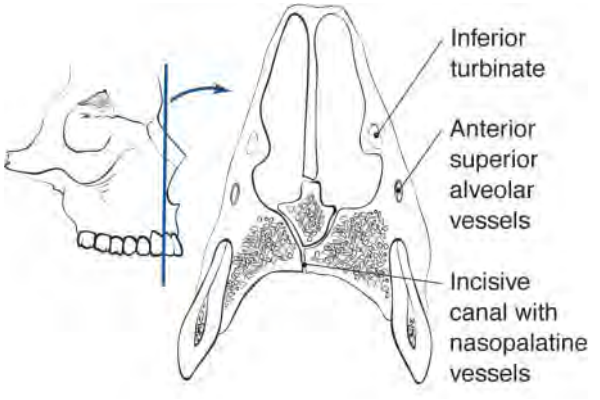


FIGURE 58-1. Cross-sectional anatomy of the maxilla at the piriform rim.

between the maxilla and the palatine bones is located approximately 1 cm anterior to the posterior margin of the hard palate or the junction of the hard and soft palate. At its most lateral extension, this suture widens into the greater and lesser palatine foramina, which are located approximately 1 cm posteromedially to the second molar (Figure 58-2). The greater palatine canal superiorly, or cranially, is also formed by the perpendicular laminae of the palatine and maxillary bones, which continue cranially to form the inferior lateral nasal wall. The inferior nasal turbinate, an isolated bony structure, articulates with the maxillary and palatine components of the lateral nasal wall in this region, and the superior and middle turbinates are part of the ethmoid bone. Posterolaterally, the maxillary tuberosity is located behind the third molar tooth, when present. Superior to the tuberosity is the posterosuperior alveolar foramina through which the posterosuperior alveolar nerves and vessels emerge. The pyramidal processes of the palatine bones unite the medial and lateral pterygoid plates of the sphenoid bone with each other, as well as to the maxilla. The pterygomaxillary junction, formed by the palatine bone, ends superiorly in the pterygomaxillary fissure leading into the pterygopalatine

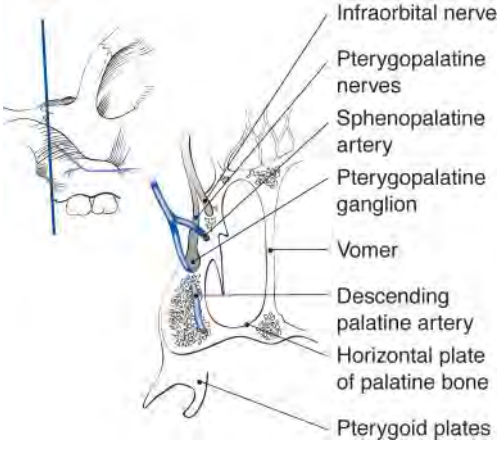


FIGURE 58-2. Cross-sectional anatomy at the pterygomaxillary junction. Note the position of the greater palatine foramen and the perpendicular plate of the palatine bone.

fossa.[39,40] The foramen rotundum, containing the second division of the trigeminal nerve, opens into the posterior wall of the pterygopalatine fossa. In addition, the pterygoid (vidian) canal also communicates into the pterygopalatine fossa. From this point extending medially, the sphenopalatine foramen leads to the lateral nasal cavity beginning posterior to the middle nasal turbinate of the ethmoid bone. Anteriorly, the infraorbital and zygomatic nerves and infraorbital vessels traverse the infraorbital canal; inferiorly, the descending palatine artery and greater palatine nerves course within the greater palatine canal.

Vascular Structures

The vascular supply to the maxilla is extensive, and it originates from large and small vessels as well as soft tissue perfusion. The normal vascular supply is derived from the terminal branches of the maxillary artery, which traverses the pterygopalatine fossa approximately 20 mm superior to the pterygomaxillary suture. The pterygoid and pterygomaxillary divisions of the maxillary artery supply various portions of the maxilla via a complex network of tributary vessels and collateral circulation. Additional perfusion is supplied via the anastomosis of the greater palatine artery with the lesser palatine artery. The lesser palatine connects with the ascending pharyngeal artery off of the external carotid artery and the ascending palatine artery off of the facial artery. This three-way junction of the lesser, greater, and descending palatine arteries at the opening of the greater palatine foramen is a critical crossroads in the blood supply to the maxilla after down-fracture. The venous outflow tracts mimic the vascular anatomy with a confluence of veins located posteriorly as the pterygoid venous plexus.

Although numerous texts concerning head and neck anatomy describe the detailed vascular anatomy of the intact maxilla, several aspects of maxillary blood flow during and after maxillary osteotomy remain controversial or unknown. The Le Fort I osteotomy had been performed for over 100 years before Bell[41,42] first identified the exact nature of the blood supply in the osteotomized maxilla, which provided detailed information regarding the viability of the pedicled maxilla. It was obvious that even though the direct blood supply to the maxillary teeth and periodontal tissues was interrupted, collateral circulation existed to provide perfusion of the dental pulp and surrounding structures[43] (Figures 58-3 and 58-4). This same circulation is also responsible for the survival of the remainder of the maxilla; however, the exact nature of the various factors affecting maxillary perfusion is still not well documented or understood. During the classic down-fracture maxillary osteotomy, the entire blood supply to the maxilla is severed except for posterior remaining buccal pedicle and that heading for the greater palatine artery distal to the greater palatine foramen. If the greater palatine artery is disrupted anterior to this junction, severe ischemia will result unless an anterior pedicle has been retained. Bell[41,42] has shown that preservation or sacrifice of the

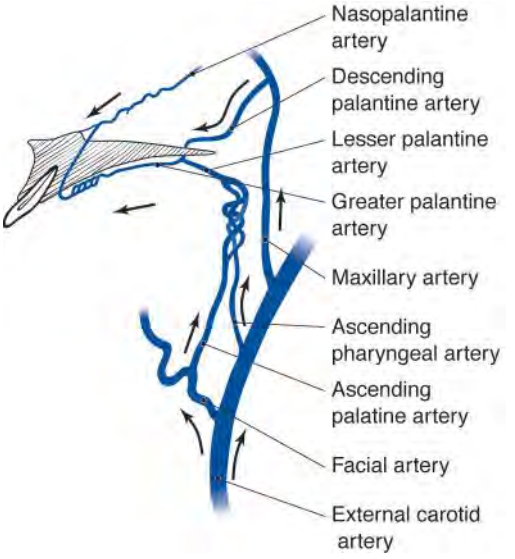


FIGURE 58-3. Pathway of the ascending palatine, ascending pharyngeal, and descending palatine arteries as they continue into the greater palatine arteries.

descending palatine artery is inconsequential and can be ligated, thereby shifting the perfusion of the greater palatine artery via the lesser palatine artery. However, the pressure head from the lesser palatine artery to the greater palatine artery will be possible only if the descending palatine artery has been ligated and not lacerated. Laceration of the descending palatine artery will not only increase uncontrolled hemorrhage but also reduce the perfusion distally through the greater palatine artery to the maxilla.

Bell[42] also verified the revascularization process after anterior maxillary osteotomy using the microangiographic technique. Brusati and Bottoli[44] performed revascularization studies similar to those of Bell and found quite different results in that the tunneling technique to access the anterior maxillary bone[16] was superior to the anterior maxillary labial pedicle technique in maintaining the blood supply, especially

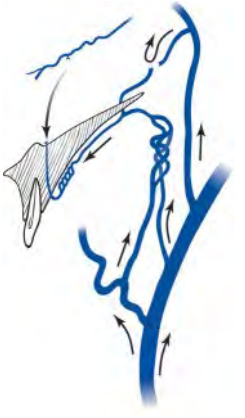


FIGURE 58-4. Soft palatal, ascending pharyngeal, and ascending palatal vessels anastomose with the greater palatine artery. Major vessels have been sectioned and tied. The *arrows* signify direction of blood flow.

to the pulpal tissues.[17] One possible explanation for this discrepancy is that Bell used a monkey model whereas Brusati and Bottoli used the dog model, which they claimed possess a more similar maxillary vasculature to that of the human.[44] The clinical significance of these differences remains unclear, and in none of these studies were the maxillae moved to a new position, which may, in fact, represent the greatest insult to the blood supply at the time of actual maxillary osteotomy surgery. It should be remembered that revascularization does not necessarily represent the reestablishment of adequate blood flow, and therefore, Nelson and coworkers[45,46] used a radioactive microsphere technique to evaluate maxillary blood flow. Unfortunately, several variables were present in this study that make interpretation difficult, including the fact that severance of the descending palatine vessels was inadvertent, and no ligation was performed, which allowed bleeding to occur through the lacerated vessels and prevented a pressure head from developing that would serve to maintain distal blood flow to the anterior maxillary segment. In other studies involving anterior maxillary osteotomies, Nelson and coworkers[46] found no significant differences among three different techniques, including the labial pedicle, tunnel technique (as described by Brusati and Bottoli)[44], plus a third procedure using only a palatal pedicle. Although no statistical difference was seen, the palatal flap seemed to be slightly superior to the others, and owing to the presence of multiple confounding variables, clinical significance could not be determined.

The Soft Tissue Envelope of the Maxilla

The midfacial superficial fascia, or subcutaneous tissues, contain variable amounts of adipose tissue as well as the muscles of facial expression within the deep fascial layer. This fascia is tightly bound to bone in most locations in the midface, except directly adjacent to the buccal fat pad and in the lower eyelids. In *Anatomy for Surgeons: The Head and Neck*, Hollinshead[39] divides the mimetic or facial muscles into five chief groups: mouth, nose, orbit, ear, and scalp. The muscle groups of the mouth and nose, which are innervated at their posteroinferior aspect by the facial nerve, are of greatest concern with regards to maxillary orthognathic surgery. These muscle groups insert into the skin and mostly arise from periosteum surrounding the midfacial facial skeleton. The upper oral group of muscles radiate from their insertions near the corner of the labial commissure (modiolus region). From a horizontal to vertical, and inferior to superior, orientation the risorius, zygomaticus major and minor, and levators (levator labii superioris alaeque nasi) insert and blend with the skin and orbicularis oris muscle. The risorius does not arise from bone but originates from the superficial fascia over the parotid gland. The risorius, zygomaticus major, and zygomaticus minor elevate and retract the corner of the mouth and upper lip laterally. The superficial levator muscles and a third deeper muscle, the levator anguli oris, elevate the lateral portion of the upper lip. In addition, the levator labii superioris alaeque nasi muscle attaches to the skin and greater alar cartilage of the nose, thus lifting the ala and widening the naris.

The orbicularis oris muscle is composed of many multidirectional fiber groups that blend with other surrounding facial muscles, encircle the mouth, originate from periosteum covering the roots of the canine teeth, insert laterally at the comer of the mouth, and pass at right angles to the encircling sphincter fillers connecting skin to labial mucosa. This diverse muscle draws the lips together, purses the lips, presses the lips against the teeth, and pulls the corners of the lips inward. The buccinator muscle arises from the mandible and maxilla and the pterygomandibular raphe, which separates it from the superior pharyngeal constrictor muscle. The fibers pass forward and slightly inferiorly to blend with the orbicularis oris and attach to the mucosa and skin of the labial region. The buccinator muscle acts to flatten the cheek against the teeth and alveolus.

Lightoller[47] and Nairn[48] place emphasis on the modiolus, which is the point at the lateral aspect and just superior to the corner of the mouth where muscles of the oral group of the mimetic muscles converge. The orbicularis oris and buccinator muscles join at the modiolus region to form a continuous muscular sheet on either side of the midline. The zygomaticus major, levator anguli oris, and depressor anguli oris (this group is referred to as the "modiolar stays") immobilize the modiolus in any position. In addition the marginal and peripheral parts of the orbicularis oris muscle are distinguishable by the fact that the peripheral aspect of the muscle lies parallel to the inner labial mucosal surface and the marginal part curls outward following the vermilion surface. As tension is expressed in the orbicularis oris, the marginal aspect of the muscle is thought to straighten and decrease vermilion exposure, thereby pulling the upper and lower lips toward each other and against the facial surfaces of the dentition.

When contemplating maxillary orthognathic surgery, consideration must be given to the nasal group of facial muscles that act to both dilate and compress the nares and whose length and function may be affected by Le Fort surgery. The nasalis muscle arises from the anterior aspect of the maxilla in a position lateral and inferior to the ala. The transverse portion of the nasalis muscle unites with the contralateral nasalis muscle over the dorsum of the nose. The alar portion of the nasalis muscle inserts into the greater alar cartilage. Thus, the two parts of the nasalis compress and dilate the nasal apertures, respectively. The depressor septi muscle lies beneath the orbicularis oris and attaches to the base of the columella and posterior ala; it functions to narrow the naris. The posterior and anterior dilator muscles are intrinsic muscles of the nose that course from the alar cartilages to the margin of the alar fat pads. The nasal mucoperiosteum is firmly fixated to the elevated piriform rim above the floor of the nose, to the lateral margin of the nasal aperture, and to the ANS. The premaxillary wings that flare laterally from the anterior midline nasal crest provide an irregular attachment of the mucoperiosteum along the inferoanterior nasal floor.

The hard palate is covered by mucosa firmly adherent to the periosteum and containing mucous minor salivary glands. The mucosa is thin in the midline region of the palate and thickens toward the alveolar process. The palatine crest is a transverse elevation at the posterior border of the horizontal plate of the palatine bone that gives attachment to the tensor veli palatini muscle. The larger, lateral pterygoid plate is the origin of the inferior head of the lateral pterygoid and the medial pterygoid muscles. A small part of the medial pterygoid muscle also arises from the maxillary tuberosity. The tensor veli palatini muscle that functions during speech and swallowing curves around the hamulus, which is the caudal, or inferior, termination of the medial pterygoid plate. From the hamulus, the tensor muscle of the palate enters the soft palatal tissues. The tensor aponeurosis is an adherent connective tissue sheath continuous with the periosteum, which covers the posterior hard palate attaching laterally to the submucosal layer of the pharynx and the tensor veli palatini tendon.

SURGICAL TECHNIQUES

Incisions, Dissection, and Exposure of the Maxilla

Exposure of the anterior, lateral, and pterygomaxillary regions of the maxilla is most commonly achieved via a circumvestibular incision horizontally through the mucoperiosteum above the attached gingival margin at the level of the maxillary teeth apices (Figure 58-5A). The circumvestibular incision typically extends from the first molar to the contralateral first molar region (see Figure 58-5B). Care is taken to avoid the parotid papilla of Stensen's duct at approximately the maxillary second molar region superior to the occlusal plane. The incision may be made with electrocautery or a scalpel, and there are no studies evaluating the two techniques, A full mucoperiosteal incision is made to the anterior wall of the maxillary sinus, and care should be taken in the anterior region to keep the incision below the ANS and avoid a superiorly placed incision that may perforate the nasal mucosa and enter the nasal cavity. The tissues above the incision are reflected superiorly in a subperiosteal plane first at the area of the piriform rims, using a periosteal elevator (#9) (see Figure 58-5C). Further superior exposure lateral to the nasal aperture will lead to exposure of the infraorbital nerve exiting from the infraorbital foramen. This subperiosteal dissection also elevates the insertions of the muscles mentioned previously, including the upper lip muscles, nasal muscles, and zygomatic muscles. Further posterior subperiosteal tissue reflection from the infraorbital foramen will reveal the zygomaticomaxillary suture, the zygomatic buttress, and the most anterior aspect of the zygomatic arch. Continuing posteriorly and inferiorly with subperiosteal tunneling, the lateral aspect of the maxillary tuberosity and its junction with pterygoid plates of the sphenoid bone are exposed. Care should be taken to avoid vascular structures by directing the subperiosteal dissection posteroinferiorly, more toward the mucogingival junction, as dissection progresses toward the pterygomaxillary fissure. Meticulous maintenance of the subperiosteal plane of dissection may prevent troublesome exposure of buccal fat pad tissue, which impairs visualization and retraction of soft tissue during the subsequent osseous surgery. A reverse-angled Obwegeser retractor ("toe-out" or "up-turned") is placed in the pterygomaxillary junction to facilitate bony exposure. Close attention should be paid to the placement of this retractor because it can be responsible for iatrogenic laceration of the periosteum and/or exposure of the buccal fat pad. The attached and unattached mucogingival tissues inferior to the incision are elevated minimally, if at all. In areas of planned interdental osteotomies for segmentalization of the maxillary arch, the attached gingiva and periosteum are elevated conservatively using a Woodson elevator, while retraction can be provided, if necessary (Figure 58-6). Because the alveolar osteotomy will be accomplished with a thin osteotome, osseous exposure requirements at the alveolar crest level are minimal and minimal soft tissue dissection is usually required. Infrequently, the intersegment osteotomy may be larger, and more exposure may be necessary to prevent tearing of the gingival papilla. In addition, a wider exposure of alveolar bone is frequently needed when the osteotomy is planned in an edentulous site or in a simultaneous extraction space. In these rare situations, a vertical releasing mucosal incision at the line angle one tooth away from the ostectomy site will facilitate

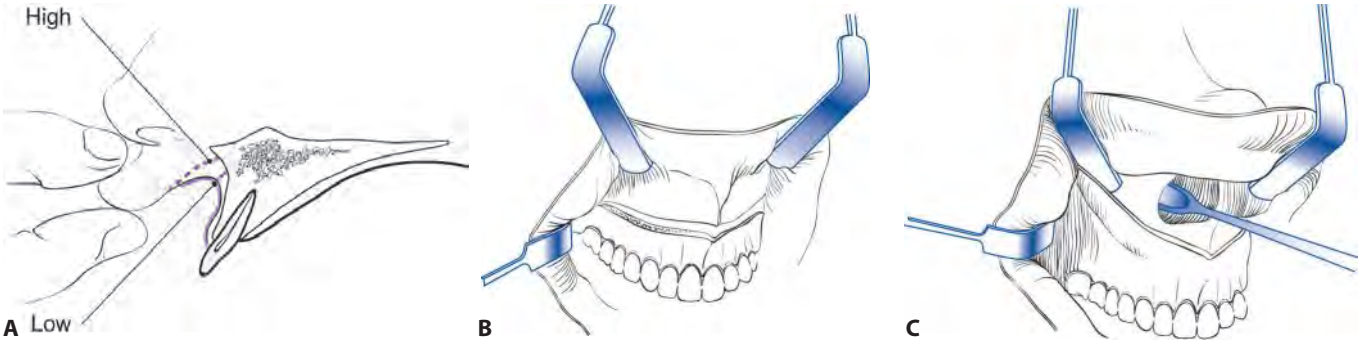


FIGURE 58-5. **A,** The soft tissue incision for maxillary surgery. **B,** The circumvestibular incision extends from the area of the first molar to the same location on the opposite side. **C,** The nasal mucosa is elevated beginning on the superolateral surface of the piriform rim.

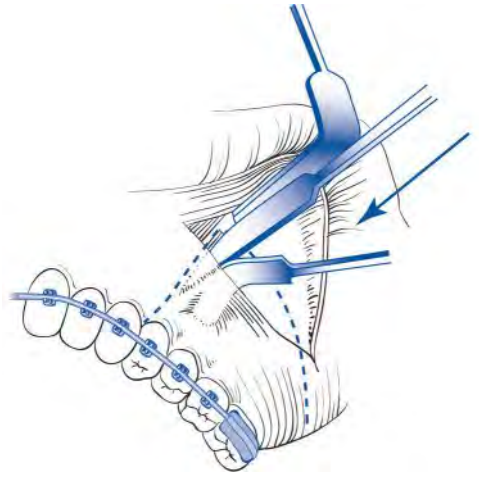


FIGURE 58-6. If segmentalization is necessary, it is best to perform interdental osteotomies before horizontal osteotomies and down-fracture. Use minimal exposure technique for interdental osteotomies.

wider exposure for the osseous procedure. This ancillary vertical incision should be used only when an anterior labial pedicle can be maintained to maximize the existing vascular pedicle during multisegment osteotomy procedures (Figure 58-7). As mentioned, for one-, two-, and most routine three-piece maxillary osteotomies, a circumvestibular incision, with minimal interdental exposure, is preferred. For three-piece maxillary osteotomies involving wide expansion or other extreme changes, four-piece maxillary osteotomies, and osteotomies in cleft maxillas, soft tissue incisions can be isolated from second molar to first premolar to maintain a wide anterior labial soft tissue and vascular pedicle to the anterior maxillary segment(s) (see Figure 58-7). In some cases, a midline vertical incision is utilized to gain access to the anterior maxillary segments, if necessary.

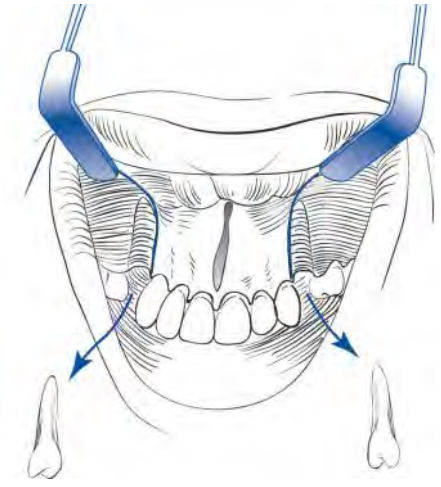


FIGURE 58-7. Modified incision for cases in which anterior perfusion is questionable. Bilateral vestibular incisions are made from the first premolar to the second molar; shown with a midline vertical incision.

Once the labial incision(s) and soft tissue and muscle dissection are completed, the nasal mucoperiosteum is elevated to complete soft tissue exposure of the osseous surgical site (see Figure 58-5C). A meticulous initial subperiosteal plane of dissection is imperative for complete elevation of the nasal mucosa without disruption of mucoperiosteal integrity, leading to persistent nasal bleeding both intraoperatively and postoperatively. Because the nasal cavity is more voluminous inside the piriform rim than at the entrance to the piriform aperture, the periosteal elevator should be held at an oblique angle to the surrounding maxillary bone adjacent to the nasal aperture; alternatively a Woodson elevator may be used to initiate the dissection at the piriform rim. While maintaining the elevator tip against the bone within the nasal cavity, the mucoperiosteum is reflected from the nasal floor, lateral nasal wall, and nasal crest of the maxilla. The dissection should continue superiorly 1 cm along both lateral nasal walls and both sides of the nasal septum to prevent nasal mucosal tearing during the nasal septal osteotomy and the lateral nasal osteotomies or during the down-fracture of the maxilla. The AP depth of this nasal mucosal dissection is approximately 15 to 20 mm. The remaining posterior soft tissue of the nasal floor is reflected more precisely after the initial down-fracture of the maxilla. At this point, in preparation for osseous surgery, there should be two reverse-angle ("toe-out") Obwegeser retractors, one on each side posteriorly at the pterygomaxillary junction; two Obwegeser retractors ("toe-in") elevating the soft tissues, upper lip muscles, nasal muscles, and zygomatic muscles and protecting the infraorbital neurovascular bundles superiorly; and two periosteal elevators, one in the inferolateral aspect of the piriform rims bilaterally protecting the lateral nasal mucosa.

Osseous Surgery

Before osseous surgery, vertical reference points must be recorded, and these may be made either inside or outside of the wound. The internal reference points are small holes made with a #701 bur in the piriform rim and the zygomatic buttress, well above the osteotomy. Measurements are made from the reference points to the gingival crest corresponding to the point-to-point measurements made at the time of model surgery using a caliper as described in Chapter 56. External reference using a Kirshner wire or Steinmann pin may also be used. An external reference is created by using a wire or Steinmann pin placed in the nasal bones, and then using a measuring device (e.g.. Perkins device) to make reproducible vertical measurements to the central incisor bracket or other inferior reference that will not be obscured if an interocclusal splint is used later during surgery. In addition, a horizontal measurement can be made using the same Perkins device with a millimeter calibrated rod placed in the lower portion of the device. After making the initial vertical and horizontal measurements, the device can be set to the planned movements and used subsequently during osseous surgery to confirm that the planned movements have been

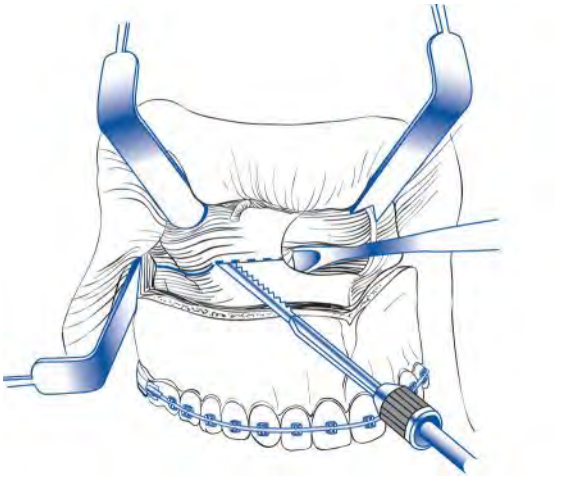


FIGURE 58-8. Lateral wall osteotomy is begun at greatest convexity of the buttress and brought forward to the piriform rim with a periosteal elevator protecting the nasal mucosa and the endotracheal tube.

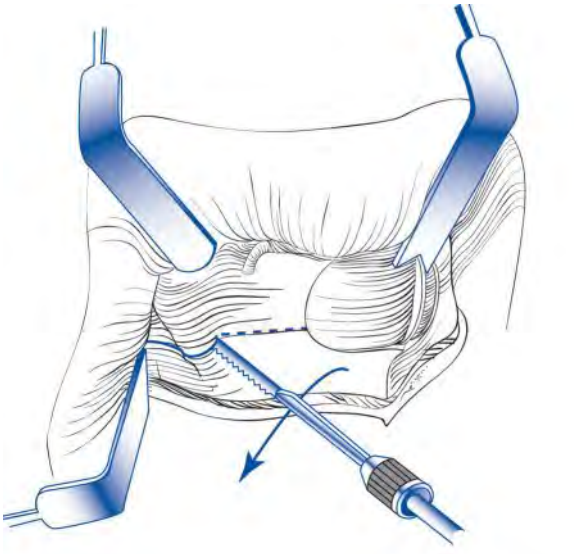


FIGURE 58-9. The saw is then turned inside out and the osteotomy from the buttress to the pterygomaxillary junction is made angling downward as it goes posteriorly.

achieved. Many studies have shown that external reference points are more predictable and reproducible than traditional scribed lines, and, similar to model surgery, the external technique (and the technique described in Chapter 56) utilize reference points that are located at a distance from the osteotomy site. This minimizes parallax error, especially with a significant maxillary advancement. It also prevents obliteration of landmarks inappropriately placed adjacent to the osteotomy, especially with a significant maxillary impaction procedure.

The design of the osteotomy will depend upon the maxillary movement desired. The initial lateral maxillary osteotomy (Figure 58-8) is generally begun at the greatest convexity of the zygomatic buttress because this area provides a convex surface for the reciprocating saw. The saw is advanced anteriorly through the lateral piriform rim below the inferior nasal turbinate while the nasal mucoperiosteum is protected with the periosteal elevator. For a standard maxillary osteotomy, the horizontal bony cut is made parallel to the maxillary arch wire and maxillary occlusal plane, in an attempt to simulate the cut performed previously during model surgery. Any modifications to the standard osteotomy are taken into consideration at this point and altered as necessary (e.g., a Z or a step osteotomy). After the anterior osteotomy is completed, the bony cut is continued posteriorly by tapering inferiorly toward the pterygomaxillary junction, using the reciprocating saw in an "inside-out" fashion (Figure 58-9).

After completion of the osteotomy of the anterior and lateral wall of the maxillary sinus, a bayonet or double-guarded nasal septal osteotome is directed slightly downward and posterior (Figure 58-10), beginning just above the ANS while the anterior nasal mucoperiosteum is retracted. Proceeding posteriorly, the osteotome is carefully maintained in the midline along the nasal crest of the maxilla. There is a tendency toward superior deviation of the osteotome while separating the cartilaginous and vomerine septum from the nasal crest of the maxilla; this necessitates slight downward inclination of the nasal septal osteotome. A finger placed at the PNS region near the soft palate confirms that the osteotome has been carried sufficiently posterior. Next, the lateral nasal walls are osteotomized using a thin spatula or single guarded nasal osteotome directed posteriorly and inferiorly if necessary, while medial retraction of the nasal mucoperiosteum is accomplished with a periosteal elevator. The osteotome is gently malleted posteriorly for a distance of approximately 20 mm to avoid premature injury to the descending palatine neurovascular bundle that resides in the lateral posterior nasal wall. In many cases, the use of these osteotomes will initiate the down-fracture of the maxilla, and some surgeons may choose to perform this portion of the osteotomy after all other osteotomies have been completed, including the pterygoid plate osteotomy, in which the pterygoid plates are separated from the maxillary tuberosity (Figure 58-11) using a small, sharp, curved osteotome (pterygoid

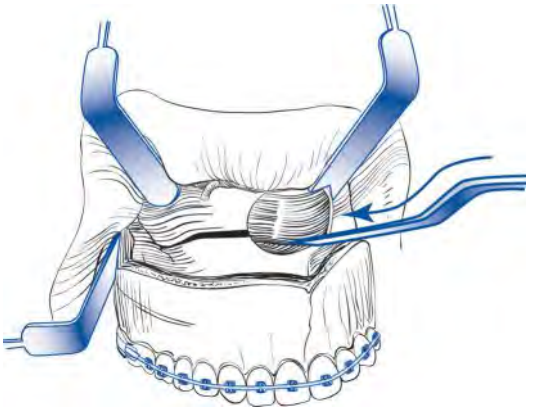


FIGURE 58-10. Separation of the nasal septum from the septal crest of the maxilla with a special osteotome.

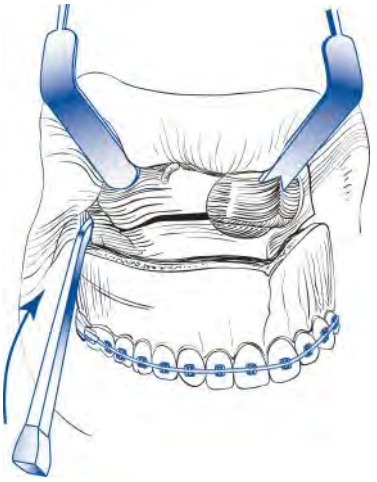


FIGURE 58-11. Pterygomaxillary separation with a small, sharp, curved osteotome directed medially.

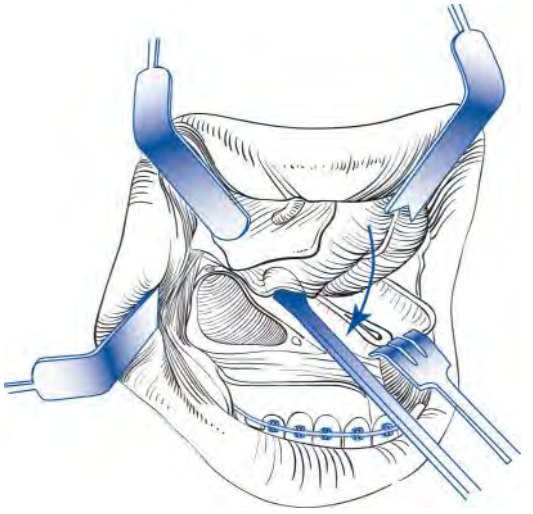


FIGURE 58-12. Down-fracture is accomplished with a sharp-toothed Senn retractor, with simultaneous elevation of nasal mucoperiosteum.

osteotome). This instrument is preferred over the traditional large thick pterygomaxillary osteotome (swan-neck osteotome) because the thin cutting edge and more gentle curve limit the extent of fracture[49] and promotes precise division of this bony junction, although studies indicate that the pterygoid osteotomy may occur in a variety of erroneous locations, including the alveolus, through the plates horizontally, and close to the skull base, as well as other locations. The tip of the curved pterygoid osteotome is directed as anteriorly, inferiorly, and medially as the tunneled buccal soft tissue pedicle will allow. A finger placed on the palatal side of the maxilla and posterior to the maxillary tuberosity will help verify complete separation of the maxilla from the pterygoid plates while avoiding trauma to the palatal vascular pedicle. Because the maxillary artery has been shown to lie in the pterygomaxillary fossa approximately 20 to 25 mm above the pterygomaxillary fissure, the use of a 10-mm pterygoid chisel will allow a 10- to 15-mm margin of safety during performance of the osteotomy. At this point, all osteotomies have been completed, and downward pressure is placed on the anterior maxilla using digital manipulation or the sharp hooks of a Senn retractor to facilitate initial down-fracture of the maxilla (Figure 58-12). If moderate pressure does not result in mobilization of the maxilla, an evaluation and verification of all osteotomies should be performed, and typically, the pterygoid disjunction is found to be incomplete. The curved pterygoid osteotome is again placed into the pterygomaxillary junction, malleted gently, and then torqued to attempt to mobilize the maxilla. If no significant movement is achieved, the pterygoid osteotome may be carefully placed further superiorly in the pterygomaxillary fissure, directed anteriorly, and malleted again until the separation is complete. Another area of possible incomplete osteotomy is the lateral nasal wall, which can be checked with a guarded osteotome and completed if necessary.

When mobilization occurs, the nasal mucoperiosteum is elevated in a progressive fashion posteriorly until the posterior edge of the hard palate is encountered (see Figure 58-12). Any significant tears in the nasal mucosa should be repaired at this point with resorbable suture to prevent persistent nasal bleeding. Portions of the pterygoid plates or perpendicular process of the palatine bone that resist fracture may be completely separated from the maxilla using a carefully placed double-action rongeur under direct visualization (Figure 58-13). After initial mobilization of the maxilla, the descending palatine neurovascular bundle may be identified, isolated, and either protected or ligated with hemoclips and divided. If the descending palatine vessels are maintained, care must be taken to avoid inadvertent laceration or the presence of sharp bony edges that may contribute to intraoperative bleeding during maxillary repositioning or postoperative

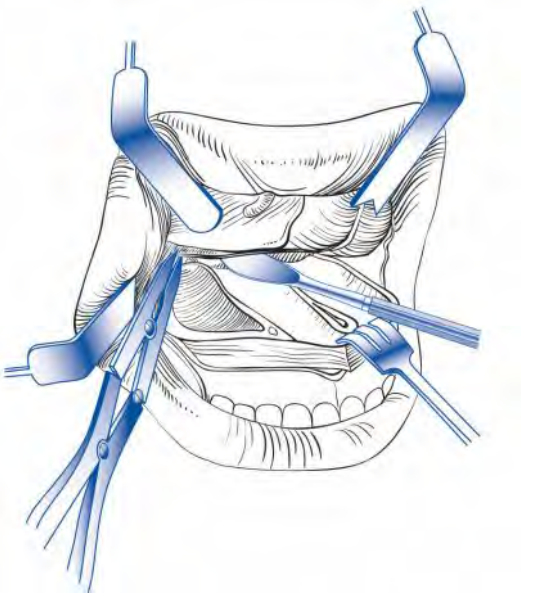


FIGURE 58-13. Complete removal of bone around the perpendicular plate of palatine bone. The descending palatine artery is isolated, ligated, and divided.

hemorrhage, as documented by Lanigan and West.[50] Significant movement of the posterior maxilla can cause tensile forces and disruption of the descending palatine vessels. Superior repositioning of the maxilla may also compress the exposed vessels between the superior and the inferior osseous segments. Severe postoperative bleeding after Le Fort I maxillary osteotomy has been reported,[51–54] and attempts to preserve the neurovascular bundle may increase this complication. Ligation and division of the descending palatine vessels has been shown to have no deleterious effects on maxillary perfusion or neurosensory recovery.[55,56] The bone of the perpendicular plate of the palatine bone surrounding the neurovascular bundle is carefully removed using a Woodson elevator and a rongeur (preferable to rotary instruments) that facilitates isolation of the neurovascular bundle (see Figure 58-13). Other clinicians argue against the routine ligation of the descending palatine vessels if they can be protected adequately, especially during segmental maxillary procedures. After maxillary down-fracture, if additional mobilization of the maxilla is needed, a J stripper, typically used during mandibular sagittal osteotomies, may be placed over the posterior border of the midline of the nasal floor at the PNS region (Figure 58-14), and anterolateral pressure is exerted to progressively mobilize the maxilla. A Tessier retromaxillary lever or similar instrument may also be placed posterior to the tuberosity region with anterior pressure. Finally, with extreme caution, two Rowe disimpaction forceps may be used with the straight arm of the forceps placed on one side of the nasal floor and the curved arm placed on the palatal mucosa. With two of these forceps, significant torque may be placed on the maxilla to achieve mobility, but significant pressure on the palatal vascular pedicle may compromise blood flow to the anterior maxilla if used inappropriately or for an extended period of time, especially during segmental maxillary surgery and their use has been discouraged as a potential cause of aseptic necrosis of the maxilla.[57] The goal of all of these maneuvers is to move the maxilla into the approximate final position with only gentle digital pressure and a minimum of soft and hard tissue restriction. After down-fracture, significant, unexpected sinus pathology may be débrided and submitted for histopathologic evaluation, if indicated. After mobilization of the maxilla from the cranial base is completed, a reassessment of the surgical movement should be performed. Based upon the planned movement all potential bony interferences posterior to the second molar should be eliminated before the application of IMF or maxillomandibular fixation (MMF). Next, MMF is applied, either with or without the interocclusal splint fabricated during model surgery. On occasion, with a full dentition and good interocclusal contacts, the splint may not be used. However, even in cases such as this, an interocclusal splint provides a more definitive seating of the teeth than tooth-to-tooth contact. If there is instability in the occlusion, a planned open bite posteriorly, or a tripod plan for the occlusion to be corrected orthodontically after surgery, the interocclusal splint should be used. Also, the splint may or may not be left in place, typically wired to the maxilla, after surgery.

Condylar positioning during rotation of the maxillomandibular complex and application of rigid fixation to the maxilla are paramount to success, and some surgeons prefer complete paralysis of the patient during this process. The physiologic position of the condyle is thought to be in a superoanterior orientation relative to the glenoid fossae against the posterior slopes of the articular eminences, with the disk interposed between the condyle and the fossa, and the surgeon should position the condyles appropriately before autorotation (Figure 58-15). The importance of this stage of the surgery cannot be overstated, and the most likely points of unrecognized bony interferences are in the areas of

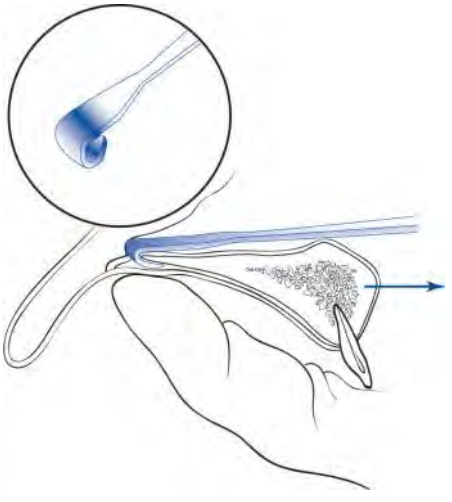


FIGURE 58-14. Mobilization of the maxilla with a J stripper.

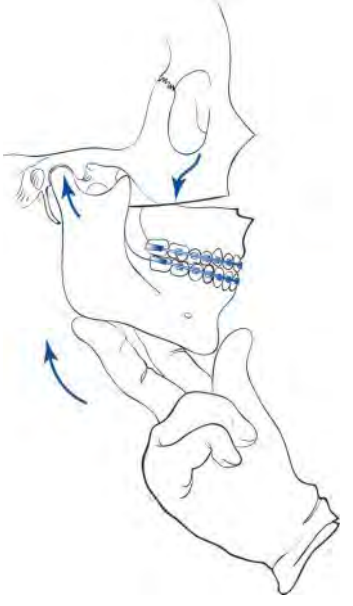


FIGURE 58-15. Manual positioning of the maxillomandibular complex with condyles seated. Note the posterior pivot point that must be removed.

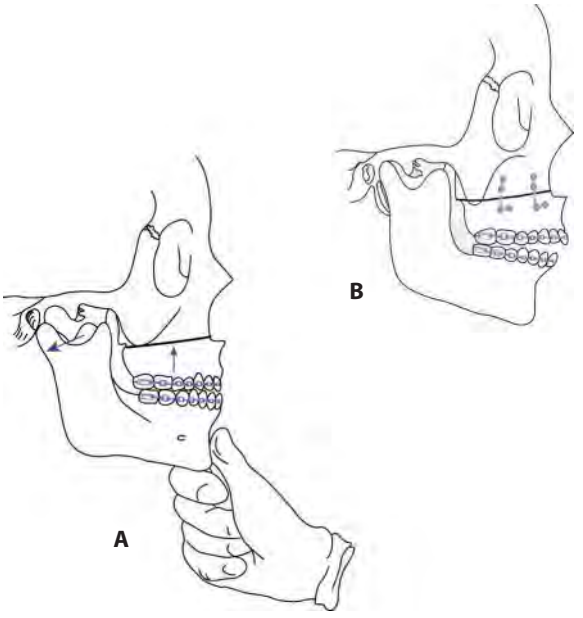


FIGURE 58-16. Inappropriate positioning of the condyles around posterior pivot points **(A)** will result in open bite **(B)** after release from maxillomandibular fixation.

the pterygoid plates, the maxillary tuberosities, and the perpendicular plate of the palatine bone. It is possible to rotate the maxillomandibular complex while being unaware of a premature pivot point in these posterior bony areas (Figure 58-16A). This will result in immediate malpositioning and a class II open bite discrepancy upon release of MMF. If a significant period of MMF or training elastics is used postoperatively, this discrepancy may not become apparent for weeks or months (see Figure 58-16B). Once these posterior interferences have been removed, the surgeon continues to rotate the entire complex around the temporomandibular joints until the appropriate vertical relationship is achieved. The cartilaginous septum and vomer as well as the nasal crest of the maxilla are reduced in height equal to the planned movement of the maxilla. This may require a submucous resection of the cartilaginous nasal septum to prevent buckling of the septum from pressure as the maxilla is repositioned. A groove can be fashioned in the midline of the nasal floor to accommodate the recontoured septum and prevent nasal septal deviation postoperatively. A portion of the inferior edge of the cartilaginous septum should also be removed, and the tendency is to not remove enough because there may be unequal areas of contact between septum and maxilla. Even if the maxilla is inferiorly repositioned, buckling of the septum may occur because the cartilaginous septum extends anterior and inferior to the ANS and, therefore, may be buckled as the maxilla moves forward even with some downward movement (Figure 58-17). This is the reason that the surgeon should err on overreduction, rather than underreduction, of the nasal septum and nasomaxillary crest. As mentioned, the maxillary movements have been predetermined by model surgery, and splint fabrication dictates the horizontal and transverse maxillary movements but not the vertical position. At the time of surgery, as the maxilla is rotated around the condyles, bony interferences are removed only at the points of contact, not as a full wedge reduction of bone planned upon the movement,[58] because "telescoping" of the bony edges may occur without the need for bony reduction (Figure 58-18). Selective bony reduction of interferences only results in improved bone-to-bone contact and avoids large gaps between bony segments. Once the desired vertical relationship has been achieved based on the measurements and reference points as described in Chapter 56, the maxilla should be fixated in position with internal rigid fixation, typically using four 2.0-mm plates with 16 self-drilling screws, placed at the piriform rim and zygomaticomaxillary buttress regions, because these represent the structural pillars of the midface. Next, MMF is removed and the mandible is rotated based upon the condylar position into the interocclusal splint while the splint is held against the maxillary dentition. This step is

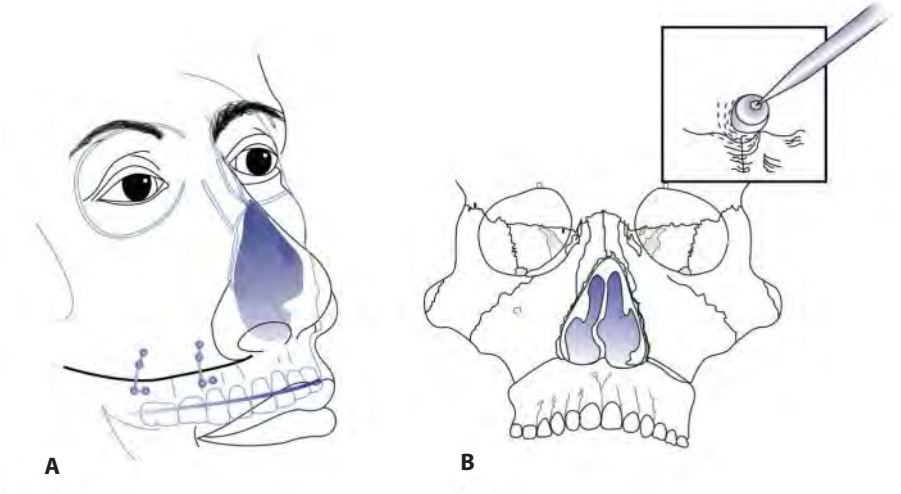


FIGURE 58-17. **A,** Anterior aspect of the cartilaginous nasal septum extends anteroinferiorly to the anterior nasal spine. **B,** Pure horizontal advancement of the maxilla will buckle the septum unless adequate bony and cartilaginous relief is provided.

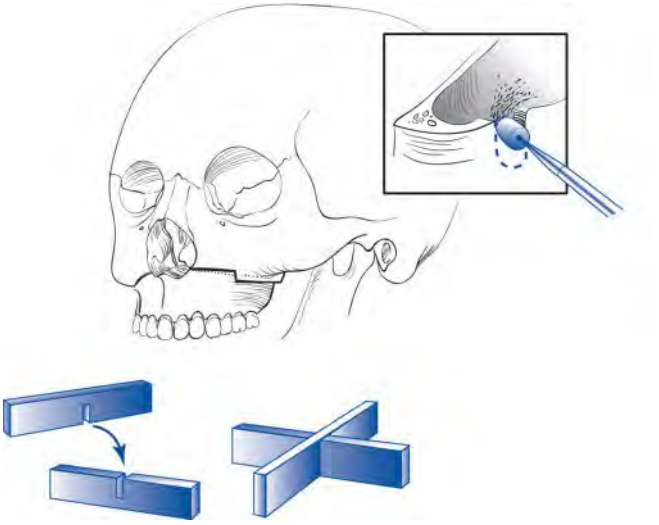
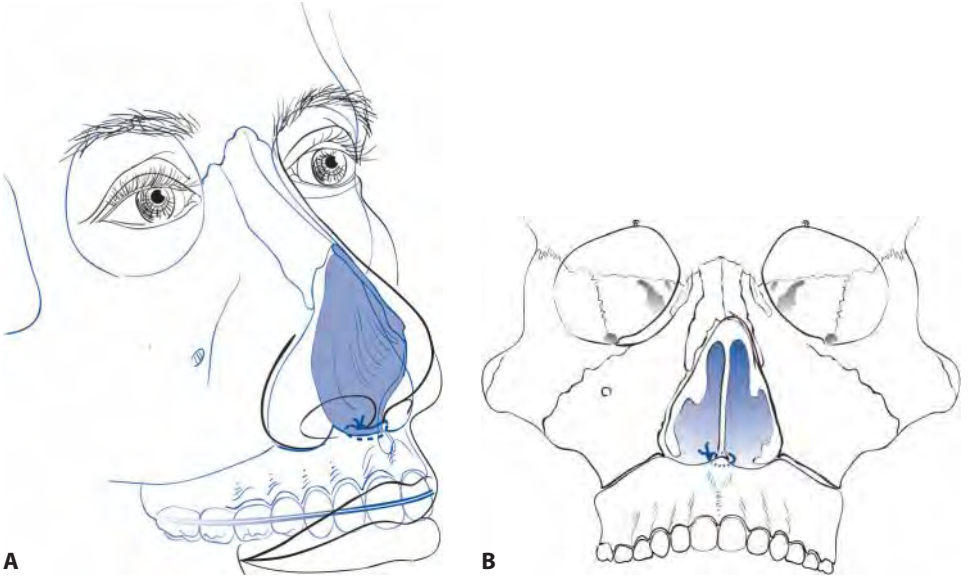


FIGURE 58-18. Removal of bony walls and slotting of the contact points.

critical to determine whether the condyles were adequately seated during fixation of the maxilla. An immediate anterior open bite or significant malocclusion at this point necessitates removal of rigid fixation and inspection and reduction of bony interferences to allow correct condylar positioning in order to achieve the appropriate maxillary movements. After release of MMF and verification of the correct, stable and repeatable occlusion, the surgical splint is usually removed unless the planned postoperative surgical occlusion is unstable, as mentioned previously. Variations in the previously discussed basic osteotomy design may enhance osseous contact, facilitate bone graft placement, or assist in the application of rigid fixation devices and result in improved stability of the repositioned maxilla. These variations are described later as they apply to specific maxillary movements. To prevent nasal septal deviation, despite adequate bone and cartilage removal, it is desirable to suture the nasal septum to the ANS in the midline. This is done by drilling a hole through the ANS and passing a 2-0 resorbable or nonresorbable suture through the hole and then through the cartilaginous nasal septum (Figure 58-19). This will also contribute to prevention of postoperative displacement of the septum during nasal extubation. The use of alar base cinch sutures to control alar base widening using 2-0 nonresorbable suture and V-Y or double V-Y closure to preserve vermilion display using 4-0 chromic gut sutures are discussed in Chapter 60. Recontouring of the bony inferior piriform rim and reduction of the ANS are often useful in preventing widening of the alar base and preventing overrotation with elevation of the nasal tip. After copious irrigation, wound closure is performed by suturing the periosteum using a polyglycolic acid suture followed by mucosal closure with chromic gut. Alternatively, a single 3-0 chromic gut suture may be used to close the mucosa and periosteum from the superior tissue to the inferior alveolar unattached periosteum and mucosa.

Segmental Maxillary Procedures

A variety of surgical options exist when segmental maxillary surgery is required. Whereas there are regional variations and specific orthodontic preferences for preparing the dentition for surgery, the need for segmentation of the maxilla occurs in up to 30% of cases. Whereas two-piece maxillary surgery is frequently required for a surgically assisted maxillary expansion procedure, the three-piece maxillary osteotomy is perhaps performed most commonly when segmentation of the maxilla is required. Once again, the decision-making process regarding which of the options will be utilized in each case is determined by pretreatment and preoperative assessment and model surgery simulation. The need for dental extractions, most commonly of the premolar teeth with the segmental osteotomies performed through the extraction sites, is also determined at this stage of treatment planning. If no extractions are required, the interdental osteotomies



A B

FIGURE 58-19. **A** and **B,** To avoid septal deviation, the cartilaginous septum should be sutured to the anterior nasal spine.

may be made reliably between the parallel or divergent roots of the canine and the lateral incisors or posteriorly between the canine and the premolar teeth. When possible, the osteotomy should be performed between the canine and the premolar teeth to preserve more hard and soft tissue with the associated vascular pedicle to the anterior maxillary segment. If the anterior six maxillary teeth interdigitate well with the lower anterior teeth, the interdental osteotomy is performed between the canine and the premolar teeth. This places a potential periodontal defect at the interdental osteotomy site more posterior in the oral cavity, removed from the aesthetic zone. If extractions are required based upon by the coordinated orthodontic and surgical treatment plans, the teeth may be removed early during presurgical orthodontic treatment or during the orthognathic surgical procedure. The indications and considerations that influence this decision are covered in Chapter 55. Regarding the specific surgical technique, the interdental osteotomy is created with gentle soft tissue retraction and the use of a thin cement spatula osteotome while palpating the palatal mucosa in the area of the osteotomy. As described, the standard circumvestibular incision can be performed, and a conservative tunneling technique can be created from the circumvestibular incision inferiorly to the alveolar crest on the facial aspect of the alveolus. The osteotome is malleted through the interdental alveolus until the tip is just palpable under the palatal mucosa (see Figure 58-6). With care, the osteotomy can be carried superiorly to the level of the planned horizontal maxillary osteotomy and medially to the horizontal surface of the hard palate. This should be performed before any of the other maxillary osteotomies are done because the maxilla must be stable at the time to withstand the forces of the mallet. If teeth are to be extracted at the time of the interdental osteotomy, an alternative flap design is recommended in order to maintain an anterior soft tissue pedicle for blood supply to the anterior segment (see Figure 58-7). It should be remembered that segmentalization with a standard, or modified, interdental osteotomy may be more difficult when alternative Le Fort osteotomy designs are used, such as high Le Fort I, II, or III designs, because access and ability to fully complete the segmental osteotomies may be compromised; for example, when Z osteotomies are used, interdental cuts between canines and premolars are more difficult than between canines and lateral incisors.

After complete maxillary down-fracture and full mobilization of the maxilla, the remainder of the segmental osteotomies can be completed. The palatal soft tissue is very thin in the midline and the bone is very thick, but the opposite is true in the area between 5 and 10 mm lateral to the midline. For that reason, two parasagittal osteotomies are frequently utilized along the floor of the nose using a bur with a rounded tip, such as a Steiger bur, or 1703 fissure bur (Figure 58-20). The use of two parasagittal, or paramedian, osteotomies creates a bony island in the midline to avoid perforation of the thin palatal mucosa in the midline region; this creates a separate "island" of bone in the midline of the palate. The

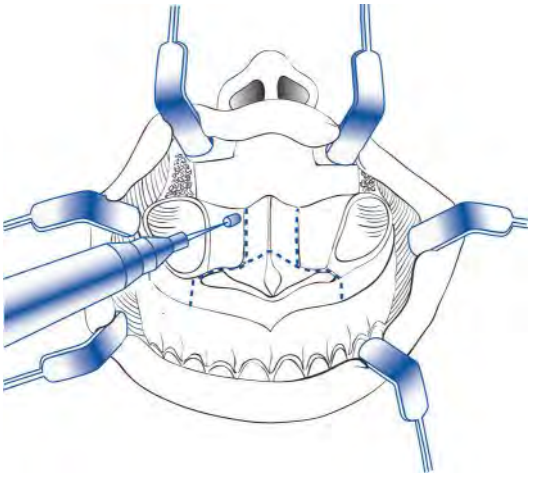


FIGURE 58-20. Following down-fracture, the maxilla is segmentalized with a rounded-end cutting bur such as a Steiger bur by making two parasagittal cuts that join across the midline and connect with the interdental osteotomies.

parasagittal cuts are joined with the interdental cuts in order to perform the three-piece maxillary osteotomy and release the three dentoalveolar segments. If significant torquing of the anterior maxillary segment is planned, the two parasagittal cuts must be joined across the midline so that there are three dentoalveolar segments and one midpalatal bony fragment (i.e., a total of four bony segments). In two-piece maxillary osteotomy designs, the two parasagittal cuts are joined with one midline interdental cut between the central incisors at the incisive canal (i.e., three bony segments). It must be remembered that the orthodontic arch wire must be sectioned at the interdental osteotomy sites in order to ensure complete segment mobilization and allow any necessary recontouring in the osteotomy site. Finally, if large interdental bone removal is necessary to close an extraction space, surgical access may be needed from the palate, especially in the midline (Figure 58-21). In this case, it is recommended that an anterior labial mucoperiosteal pedicle is maintained with a small midline vertical incision in order to access the ANS region (see Figure 58-7). This technique allows for a midline palatal incision and the use of conservative circumdental incisions to access the palate for bone removal. The individual segments are then wired to the final surgical splint The splint should have some palatal coverage for segmental maxillary surgery, with relief so that pressure is not placed on the palatal vascular pedicle. If bone grafting is required on the palate, for example, with a significant transverse expansion, this must be done before the maxilla is repositioned and stabilized vertically. In contrast, any interdental and/or buttress bone grafting, if necessary, can be performed just before closure of the soft tissue wounds owing to access from the facial aspect of the maxilla. After splint fixation, the orthodontic arch wire can be luted together with rapid-curing acrylic or a new orthodontic arch wire can be placed. The length of time the splint is left in place depends upon the magnitude and direction of segment movement. Typically,

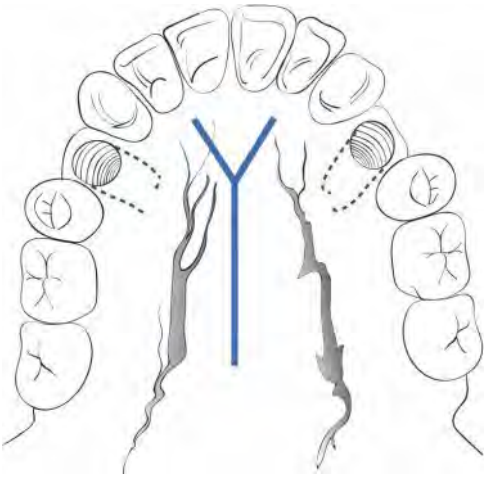


FIGURE 58-21. A midline palatal incision with a Y at the anterior aspect can be used with caution to access the palate for interdental bone removal in closing large extraction spaces.

splints are left in place for segmental cases for approximately 3 weeks for smaller movements and up to 8 weeks for greater degrees of expansive movements. After splint removal, the patient returns to the orthodontist for fabrication of the appropriate retention devices and completion of postsurgical orthodontic treatment.

Superior Maxillary Repositioning

When considering maxillary impaction surgery, historical descriptions suggested a lateral maxillary wedge ostectomy based upon the amount of the planned superior maxillary movement. However, this should be avoided because it often results in large gaps in the bony interfaces as the maxilla is moved superiorly because the contour of the maxilla is not consistent and has several bends and turns that may result in unpredictable superior movement of the maxilla, with some areas having premature contacts that require reduction and others exhibiting a "telescoping" effect of the bones with desirable overlap.[58] Furthermore, any shifting, tilting, or advancement of the maxilla may further reduce bone-to-bone contact, making premature bony removal unnecessary and potentially deleterious. Instead, sequential intermittent bony reduction of premature osseous contacts avoids unnecessary loss of bone and ensures a more predictable final contact between the maxilla and the cranial base (see Figure 58-18). Therefore, the standard horizontal osteotomy is performed, and no bone is removed until the splint is in place with IMF, and the maxillomandibular complex is rotated superiorly, with the condyles seated appropriately, to determine the specific location of bony interferences (see Figures 58-15 and 58-16). The areas of premature bone contact can now be determined as the maxilla is positioned superiorly, and bone is removed minimally at the contact points to permit the planned superior repositioning based upon the reference landmarks. In many cases, isolated interferences of the anterior wall of the maxillary sinus may be managed with the creation of slots, or grooves, in the zygomatic buttress region or anywhere necessary along the lateral maxillary wall (see Figure 58-18). One must be careful that the grooves do not inhibit the free rotational movement of the maxillomandibular complex and cause displacement of the maxilla with condylar malpositioning. This technique is particularly valuable when the maxilla is shifted laterally or torqued in a transverse direction, which would make prediction of a predetermined ostectomy difficult. In most cases of maxillary superior movement, significant bony reduction is required on the superior aspect of the maxilla as well, especially in the posterior regions. This bony reduction can be performed using a rongeur initially followed by a large round, or pineapple-shaped, bur while protecting the nasal tissues and the descending palatine vessels. If these vessels were not sacrificed previously, they may require ligation at this point if significant bony interferences are present in these areas. Once the maxillary vertical position is ideal based upon the reference points, the maxilla is rigidly fixated, the MMF is released, and the occlusion is assessed for accuracy, stability, and repeatability.

Anterior Maxillary Repositioning

The traditional standard Le Fort I osteotomy is ideally horizontal in angulation parallel to the maxillary occlusal dentition. However, in an attempt to avoid the long maxillary canine root, there may be a tendency to incline the angle of the osteotomy in a superior direction from posterior to anterior into the piriform rim. Such an inclination in the osteotomy design may not coincide with the desired forward maxillary movement, and if the angulations of the left and right sides are not coincident, an asymmetry may be created with the maxillary movement.

For this reason, a variety of straight, stepped, and Z osteotomies have been described in the literature and may be individually designed to accommodate the planned maxillary movements[59–64] (Figures 58-22 to 58-25). If bone grafting

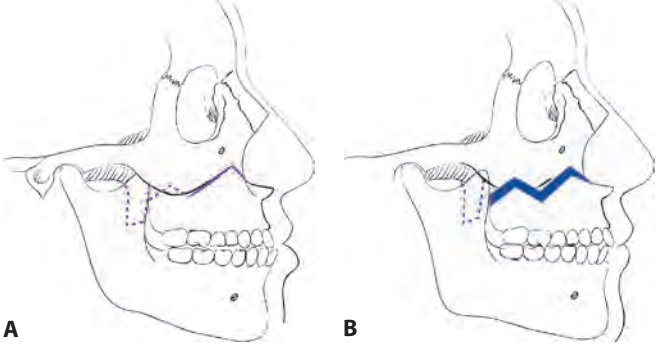


FIGURE 58-22. A Z-shaped osteotomy can be designed in the lateral walls of the pirifom rims and the buttresses **(A)** so that the maxilla may be moved downward and forward **(B)** without loss of all bony contact.

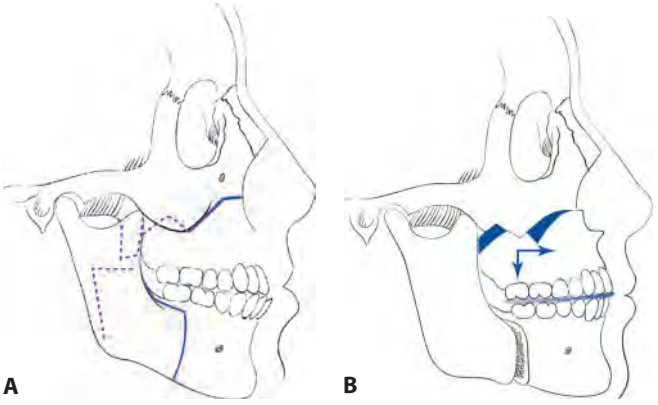


FIGURE 58-23. A Z osteotomy with the posterior cut steeper than the anterior one to increase posterior facial height **(A)** and to rotate the maxilla downward and forward with adjustment to the occlusal plane **(B)**.

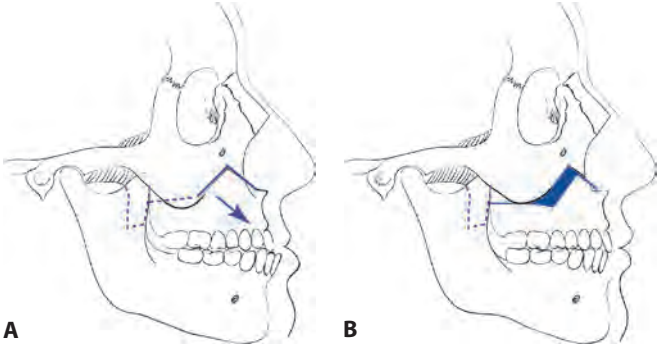


FIGURE 58-24. A Z osteotomy with the posterior cut shallower than the anterior one to increase anterior facial height **(A)** and to rotate the maxilla down in the front and adjust the occlusal plane to a steeper angle **(B)**.

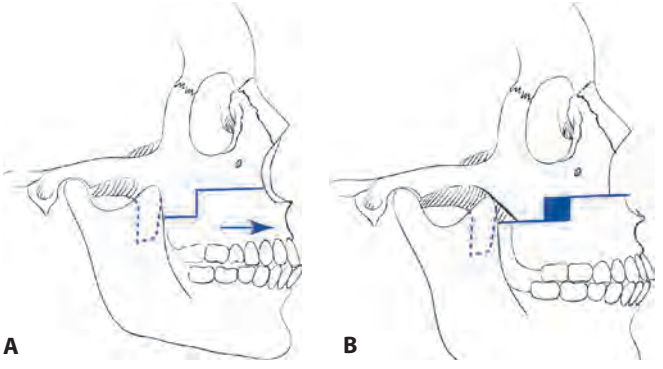


FIGURE 58-25. An alternative method for advancement is to create a step **(A)** in the buttress and place a bone graft **(B)** in the step after repositioning.

(Figure 58-26) is planned for stability with a large maxillary advancement, the vertical steps in the osteotomy design, or Z osteotomies, can provide additional, and improved, sites for placement of the bone grafts. In many cases of AP maxillary deficiency, there is an additional component of

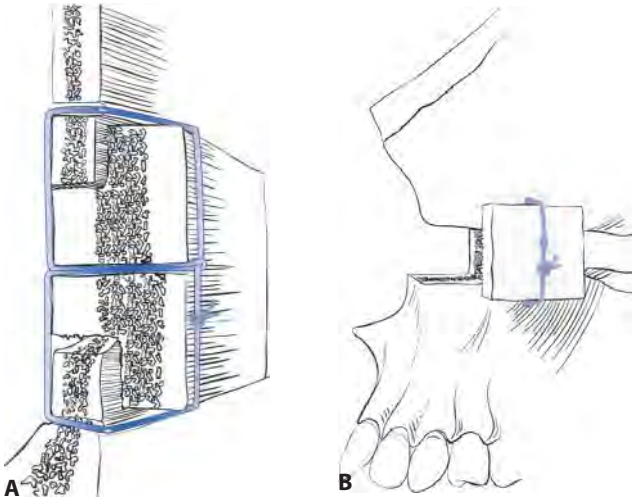


FIGURE 58-26. **A,** A single hole is placed in the middle of the bone graft and a loop of 28-gauge stainless steel wire is placed through the hole from inside out. The two ends are divided, with one placed through the superior cranial base wall and the other through the inferior maxillary segment. Finally, one end is passed through the loop and twisted to the other, much like an Ivy loop. **B,** Bone graft shown in place.

midface, or malar, hypoplasia, and various osteotomy designs have been advocated to simultaneously attempt to advance the zygomatic bone and or infraorbital rim (e.g., high Le Fort I, or Le Fort II, or modified Le Fort III, zygoma osteotomies, zygomatic arch osteotomies, quadrangular osteotomies, or double-step osteotomies of the maxilla and infraorbital rim). Unfortunately, many of these techniques fail to improve the malar hypoplasia and result in a worsening of the facial profile, such that a "dish-face" deformity may result (Obwegeser). Perhaps the most predictable method by which to address malar hypoplasia is to consider prosthetic malar augmentation using stock or custom implants (e.g., high-molecular-weight porous polyethylene). This option may be used at the time of the Le Fort advancement surgery or in a delayed fashion to determine whether the maxillary surgery itself had a significant enough positive impact on the malar hypoplasia to result in the patient declining any future surgery for aesthetic reasons. These prosthetic implants may also be placed in the paranasal regions for augmentation in this area, if necessary. Finally, severe maxillary hypoplasia, due to a cleft lip and palate or other syndrome or etiology, may be managed with distraction osteogenesis, which is covered in Chapters 62 and 63.

Inferior Maxillary Repositioning

Inferior repositioning of the maxilla presents a unique challenge in orthognathic surgery owing to the increased relapse potential resulting from impingement of the maxilla on the pterygomandibular sling of the medial pterygoid and masseter muscles.[65–68] In the pre–rigid fixation era, various techniques were recommended for additional stabilization and

fixation of the maxilla after inferior repositioning, including skeletal suspension wires (circumzygomatic, frontozygomatic suture, and piriform rim wiring), interosseous wires, Steinmann pins, Wessberg pins, and RAPs, until the introduction of rigid fixation plates and screws in the United States in the early 1980s.[32,60,63,69–71] Whereas bone grafting has been used for stabilization of the inferiorly repositioned maxilla, it may not be required if a series of slanted Z, or step, osteotomies are used[59,63,64,72] (see Figure 58-22). The specific angulations of these osteotomies are planned so that the maxilla will slide down the inclined plane of the cuts while maintaining bony contact as it is repositioned anteriorly and inferiorly. Based upon the inclination of the anterior portion and the posterior portion of the osteotomies, the maxilla may be positioned more anteriorly or more inferiorly (see Figures 58-22 to 58-24). Most surgeons use bone grafts and rigid fixation to stabilize the maxilla that has been inferiorly repositioned with a resultant lack of bone-to-bone contact. Bone grafts may be secured with bone screws or plates if sufficient bone is available, or with stainless steel wires (see Figure 58-26). Patients who are candidates for inferior maxillary repositioning often have paper-thin maxillary bone due to the vertical skeletal growth pattern or lack of occlusal forces. Although rigid internal fixation with plates and screws is the stabilization method of choice, occasionally insufficient bone is available adjacent to the osteotomy sites for plate placement, and in these cases, the RAP system can be a useful alternative.[63]

Posterior Maxillary Repositioning

Posteriorly repositioning, or setback, of the maxilla must be considered carefully because there will be a resultant loss of upper lip support as well as paranasal osseous support for the overlying soft tissues. At the pterygomaxillary disjunction of the Le Fort osteotomy, bone must be removed from either the pterygoid plates (with great caution) or the maxillary tuberosity, which extends into the alveolar process. An alternative technique is to intentionally direct the osteotomy through the maxillary tuberosity or alveolus just posterior to the second molar to ensure a predictable osteotomy and guide the position where bone will be removed.[73,74] Also, this technique will leave tuberosity bone attached to the pterygoid plates, which may then be more safely removed. A possible complication of this technique is damage to the greater palatine artery distal to its anastomosis with the lesser palatine vessel. Alternatively, maxillary horizontal excess may also be addressed with an anterior maxillary osteotomy, especially when extractions are planned, or if edentulous sites are present; this is discussed later in this chapter.

Rigid Internal Fixation for Maxillary Osteotomy

Rigid internal fixation with bone plates and screws is the current standard for maxillary stabilization after Le Fort surgery. Although this technique has eliminated many of the concerns with postoperative stability and relapse from wire fixation or no fixation, the technique is less forgiving than wire fixation and, therefore, precise intraoperative positioning is even more critical. A wide variety of plating systems and sizes are available, and there is no consensus on the number of plates and screws necessary or the thickness of the plates or diameter of the screws required for stable fixation. Typical midface plating sets have 1.5- and 2.0-mm plates and screws that work well for maxillary fixation, and most surgeons prefer to use four 2.0-mm plates in the pillar regions of the midface at the piriform rims and zygomaticomaxillary buttress regions (Figure 58-27). Preformed plates are available with specific bends for specific maxillary advancements, and computer planning may allow fabrication of customized fixation devices in the future. Whereas resorbable plates and screws are available for midface applications, most surgeons do not use these on a routine basis and reserve these for the pediatric age group, when indicated. The use of rigid internal fixation for maxillary surgery has also decreased the need for adjunctive bone grafting for large maxillary advancements and has essentially eliminated the need for skeletal suspension wiring techniques, because relapse is no longer a significant concern with the current fixation techniques.

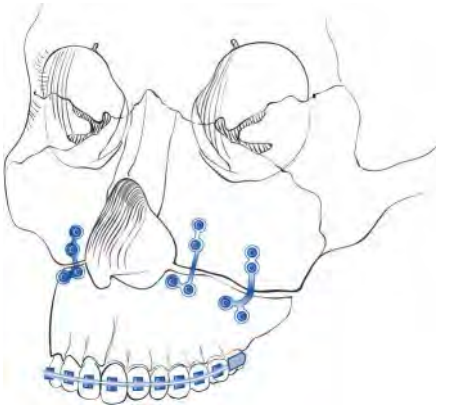


FIGURE 58-27. Bone plating encompasses a wide variety of plates and screws, ranging from very rigid to very malleable. Generally, 2.0-mm plates are used in the piriform rims and either 2.0- or 1.5-mm plates in the buttresses.

SPECIFIC PROCEDURES

Anterior Maxillary Osteotomy

Numerous surgical techniques have been advocated to perform the anterior maxillary osteotomy, and the three main options involve the use of one of three vascular pedicles: labial (Figure 58-28), palatal (Figure 58-29), or a combination of these, with the use of vertical incisions in both (Figure 58-30). The anterior segmental maxillary osteotomy can be performed successfully and, when done properly with attention to detail and respect for the hard and soft tissues, generally has few complications. However, if there is compromise

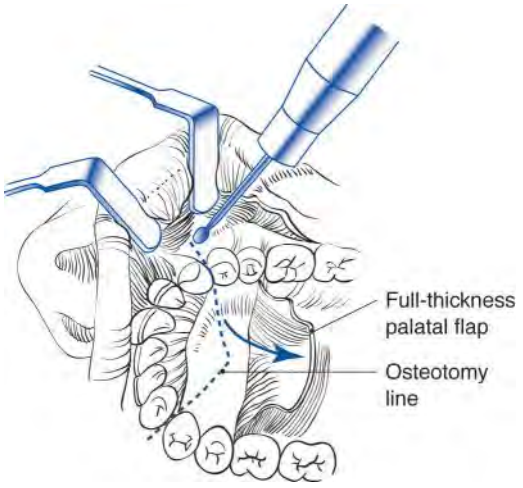


FIGURE 58-28. Labial pedicle for anterior maxillary osteotomy; the palate is flapped open.

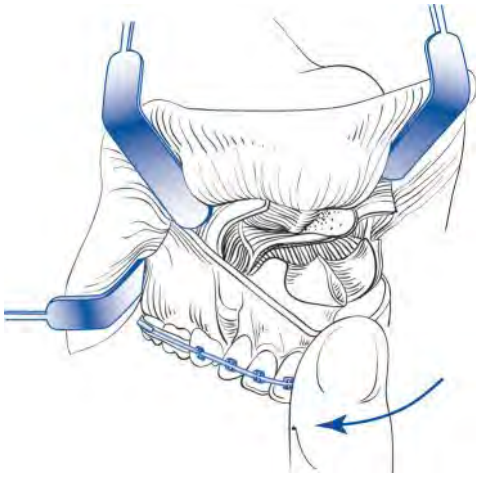


FIGURE 58-29. Palatal pedicle for anterior maxillary osteotomy is created with a horizontal labial incision.

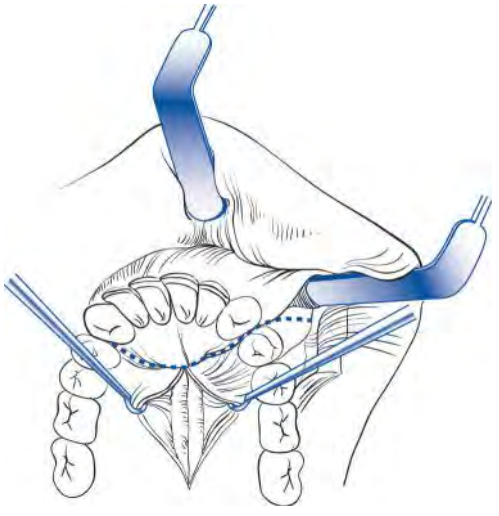


FIGURE 58-30. A combination of labial and palatal pedicles can be used for an anterior maxillary osteotomy without extractions.

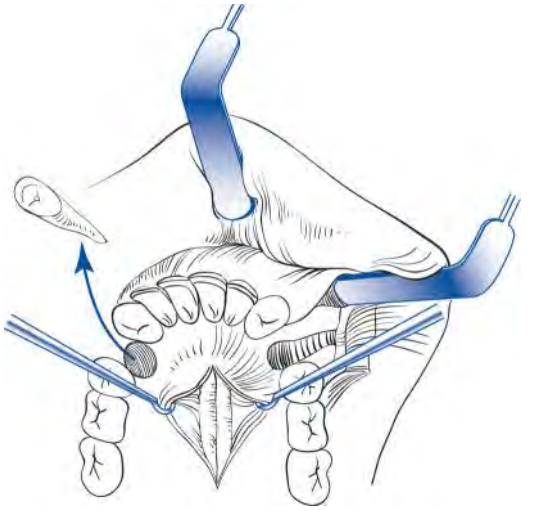


FIGURE 58-31. A midline palatal incision can be used with caution to access the palate for interdental bone removal in closing large extraction spaces.

of the vascular pedicle to the anterior maxillary segment, the complications can range from minor periodontal defects, which can be devastating in the aesthetic zone, to loss of teeth, bone, or the entire anterior maxillary segment due to avascular necrosis. Anterior maxillary osteotomies are generally used to treat horizontal maxillary excess when the posterior occlusion is ideal or if the posterior occlusion can be corrected with mandibular surgery. Commonly, an anterior maxillary osteotomy, with simultaneous premolar extractions, is used for bimaxillary protrusion in which both the anterior maxilla and the anterior mandible require retraction (Figure 58-31). These procedures can also be used for correction of an anterior open bite occlusal relationship. On occasion, an anterior maxillary osteotomy may be combined with a mandibular advancement and anterior mandibular segmental osteotomy in cases planned for correction of a severe curve of Spee. The specific surgical sequence must be considered carefully when both jaws are involved because the surgical procedures must be done systematically so that the surgeon does not lose orientation. There are two possible scenarios: (1) the posterior occlusion will not change because the posterior maxillary and mandibular teeth will not be moved, or (2) mandibular surgery will be performed to correct the posterior occlusion. This a critical difference because, if the posterior occlusion is not going to be changed by surgery, the models must be mounted in centric occlusion, not centric relation, for model surgery. If the posterior occlusion will be altered by mandibular surgery, a new centric relation will be established by the surgery and model surgery can be done as usual. In the first scenario, the maxillary anterior model is cut and repositioned to the best relationship against the uncut mandible in centric occlusion and the remaining maxillary dentition, and then a splint is constructed. In the second scenario, if mandibular surgery is planned, two mandibular models are mounted, with one mandibular model cut, and the other left intact to preserve the intermediate phase

and fabricate the intermediate splint. Then, the anterior maxilla and the mandible models are cut and repositioned to the final occlusal position and the final splint is fabricated. The cut maxilla can then be articulated with the uncut mandible to establish the intermediate position, and a second (intermediate) splint is made. Typically, the final splint will be wired to the maxilla for a postoperative period, so there must be a separate intermediate splint that articulates with the final splint and the mandibular teeth (a splint within a splint technique). Particularly with segmental surgery, the model surgery should simulate the actual surgery to provide a clear understanding of the three-dimensional movements necessary to perform the surgical procedure. Measurements and reference marks should be made at the level of the interproximal spaces and the root tips, perhaps correlated to the radiographs to determine precise root position. Also, reference marks should be made on the palate at the root tips and the maxillary midline. If maxillary widening is planned, transpalatal reference marks should also be used. The use of intermediate splints in segmental cases is different from total arch cases because the posterior maxilla is not mobilized and the anterior maxillary positioning is more difficult to determine precisely. For example, the anterior maxilla may fit into the splint and appear ideal until the mandible is rotated into occlusion, and if the mandible does not fit ideally, it is possible that the anterior maxilla can be rotated or tipped superiorly or inferiorly within the splint, and this must be adjusted before fixation. For this reason, the mandible should not be wired into MMF but left to freely rotate into the maxillary splint. Also, at surgery if the mandible is held into MMF during the fixation process for the anterior maxilla, it is possible for the condyles to be pulled out of the fossae resulting in an immediate postoperative malocclusion (anterior open bite). Therefore, the splint must be ligated to the posterior maxilla first, and then the anterior maxilla is brought into the splint and ligated. If the mandible rotates freely into the desired occlusion, the maxilla is in the correct position, and rigid fixation may be applied. Next, if mandibular surgery is planned, it may be initiated at this time.

In cases of segmental maxillary surgery, the choice of surgical technique depends upon surgical access to the areas that will be most difficult to visualize intraoperatively. For example, in the case of an anterior open bite in which no teeth are to be extracted, the anterior segment will be rotated clockwise and downward after the interdental osteotomies. Access to the interdental area, the midline of the palate, and the ANS is not as critical as it is with other surgical movements. This procedure can be done with a circumvestibular incision, or with bilateral horizontal incisions, in the canine-molar regions, and a vertical incision in the midline between the central incisors. Conversely, if first premolars have been extracted, or are planned to be extracted, and the anterior maxilla is planned for retraction, access to the midpalatal region is essential. The original description of the Wunderer technique, in which a transverse palatal incision allows the palatal soft tissues to be elevated posteriorly, provides excellent access to the hard palate but care must be taken to preserve the labial soft tissue pedicle, and most surgeons are hesitant to transect the palatal soft tissue pedicle with segmental maxillary surgery.[16] However, if superior repositioning of the anterior maxillary segment is planned, access to the junction of the ANS and the nasal septum is limited, and a vertical incision may be made over the ANS, but because this labial flap represents the complete blood supply to the anterior maxilla, it is not recommended routinely. An option for most anterior maxillary osteotomies is a "hybrid" procedure that avoids many of the potential complications of segmental surgery and allows adequate access to all regions (see Figure 58-30). The labial incisions are performed laterally, with a vertical midline incision to allow access to the ANS–nasal septum region, however, instead of a full palatal flap (Wunderer). Circumdental incisions are made around the necks of the teeth on either side of the interdental osteotomies with a midline incision over the midpalatal suture, with a small anterior Y incision, if necessary. This Y incision extension should be performed anterior to the interdental osteotomy cut and should be as conservative as possible.

Fixation techniques for anterior maxillary osteotomies are as varied as the surgical techniques themselves.[75] Orthodontic arch wires and cast splints represent the two extremes for fixation; however, orthodontic appliances are the simplest and easiest to use, at least as a part of the fixation scheme, and supplemental fixation may be required. An occlusal splint with a skeletal suspension wire from the ANS or piriform rim region is helpful in such cases, especially if there is any significant tension on the free maxillary segment. Interosseous wires or smaller profile plates and screws can be carefully used to fixate the segment, perhaps in the 1.0- to 1.5-mm range rather than 2.0-mm plates and screws used for standard Le Fort surgery. Although Erich arch bars have been used for additional fixation and in certain cases may be appropriate, a lower level of precision can be expected owing to torque of the maxillary segment from the circumdental wires. The most critical factor with segmental maxillary surgery is that, at the time of surgery, the anterior maxillary segment must be mobile enough that it does not require any significant pressure to place it in the desired final position. Then, fixation can be applied by one of the methods discussed that will hold the segment in the proper position throughout the healing period, and MMF is not commonly used.

Posterior Maxillary Osteotomy

The posterior maxillary osteotomy (PMO) and its modifications are rarely indicated today[17,20,76–81] because open bite correction or transverse maxillary expansion is performed more easily and predictably with Le Fort I surgery. PMO is usually indicated for preprosthetic reasons to correct hypereruption of a posterior maxillary dentoalveolar segment. Meticulous model surgery is essential to visualize the three-dimensional movements and anticipate osseous interferences

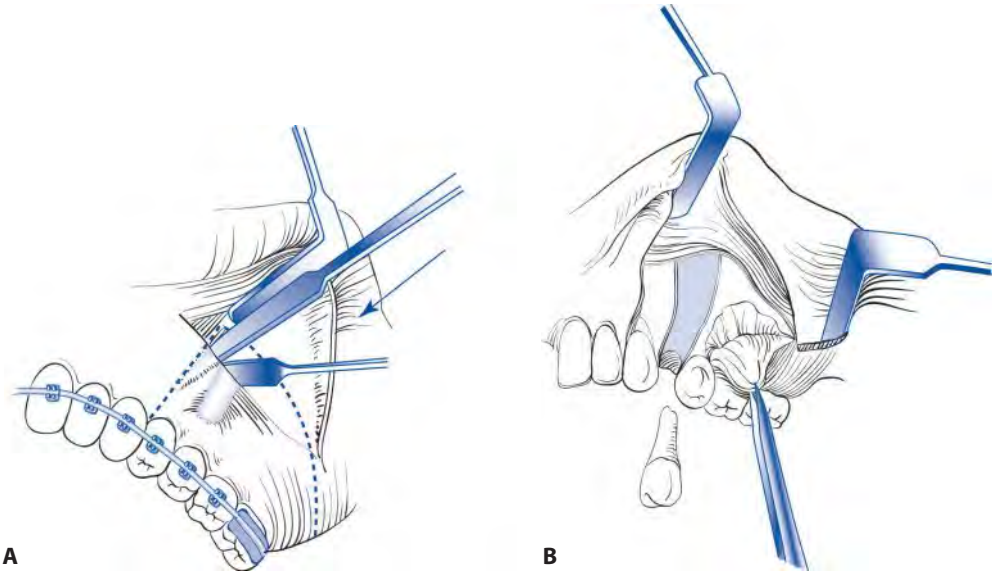


FIGURE 58-32. **A,** Posterior maxillary osteotomy. Horizontal vestibular incision with tunneling access to the interdental papilla. The *dashed line* marks horizontal and interdental osteotomies. **B,** An alternative soft tissue flap design can add a vertical releasing incision from the horizontal incision inferiorly through the gingiva at the mesial line angle of the canine tooth.

of the individual segments. Periapical radiographs are useful for evaluating interdental and subapical osteotomy sites. Once again, the dental models should be mounted on the articulator in the centric occlusion relationship, not centric relation, unless the mandible is also planned for surgery. In general, outpatient intravenous anesthesia with airway protection can be used for isolated posterior segmental procedures. A high palatal vault allows access to the palatal osteotomy via a transantral approach beneath the nasal floor. The soft tissue incision is made horizontally in the maxillary buccal vestibule from the anticipated anterior interdental osteotomy site to the second molar region (Figure 58-32A). A mucoperiosteal dissection beneath the superior aspect of the incision exposes the lateral maxilla, and the pterygomaxillary region is exposed via soft tissue retraction in a tunneling technique. At the anterior interdental osteotomy site, conservative tunneling of the periosteum exposes the full vertical extent of the dentoalveolar segments. After retraction of the soft tissue with skin hooks and right-angle retractors, the facial interdental osteotomy may be outlined with a small fissure bur, or it can be completed directly with a thin cement-spatula osteotome. An alternative soft tissue flap design can add a vertical releasing incision from the horizontal incision inferiorly through the gingiva at the mesial line angle of the canine tooth (see Figure 58-32B). A horizontal osteotomy is performed approximately 5 mm above the roots of the teeth and joined with the anterior interdental osteotomy (see Figure 58-32A). The vertical interdental osteotomy should be completed first so that the segment is not mobile while using interdental osteotomes. The palatal osteotomy is accomplished with a small, sharp, curved osteotome directed at the junction of the vertical alveolus and the horizontal palatal shelf. The surgeon may place a finger on the palatal mucosa to detect complete osseous sectioning while minimizing palatal mucosal trauma (Figure 58-33A). In patients with a high palatal vault, the transantral osteotomy is

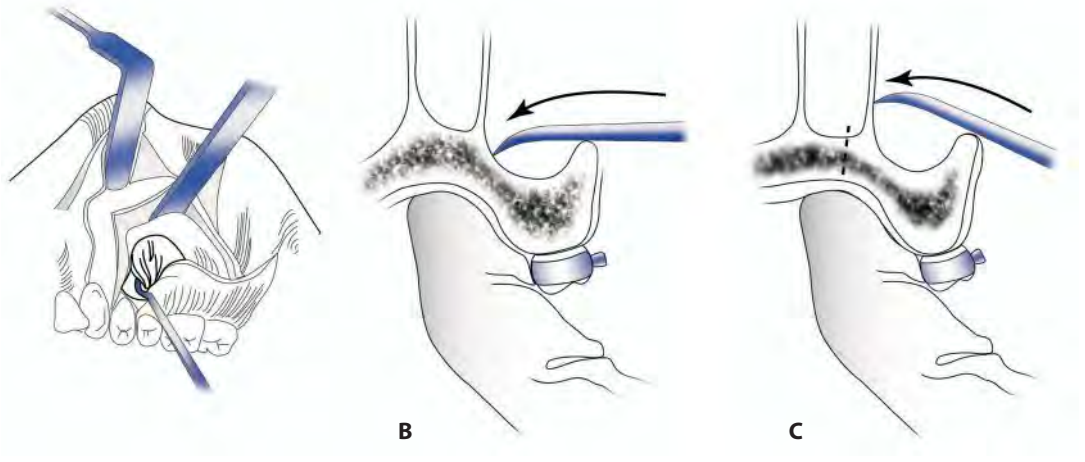


FIGURE 58-33. **A,** Transantral osteotomy is made at the junction of the horizontal palate and vertical alveolar process. **B,** Approach for deep vaulted palates. **C,** Approach for flat shallow palates.

completed along the entire AP extent of the planned palatal osteotomy (see Figure 58-33B), except in the area of the descending palatine neurovascular bundle. Next, the pterygomaxillary junction is separated with a curved osteotome using a technique similar to that for a total maxillary osteotomy. In contrast, a low, flat palatal vault can be osteotomized more easily through the nasal floor (see Figure 58-33C). The posterior dentoalveolar segment is down-fractured using digital pressure, and any osseous interferences may be removed using a bur or rongeurs. Any previously inaccessible medial and posterior walls of the mobile segment are addressed now after mobilization and displacement of the posterior segment. Bone removal at the perpendicular plate of the palatine bone and segment mobilization should continue until the segment can be repositioned with minimal soft tissue tension or bony interferences (Figure 58-34). Final contouring is accomplished while holding the splint in the stable portion of the maxilla anteriorly, and then the mandible is rotated into its correct occlusal position to ensure that no distortion of the splint has occurred. A splint modification should be considered that results is a slightly thicker splint with transpalatal acrylic or wire reinforcement that will add rigidity to prevent inadvertent distortion of the posterior extension of the splint and to support the osseous segments postsurgically. Once the segments are ligated to the splint, the repositioned posterior maxillary segment(s) may be fixated with interosseous wires, suspension wires, stable pin fixation, or bone plates and screws. Bone grafts are rarely required, and additional stability may be obtained by luting the orthodontic arch wire back together with quick-curing acrylic or by placing a new rectangular surgical arch wire across the interdental osteotomy site; MMF is usually not required. If the posterior segment is repositioned laterally or medially to a significant

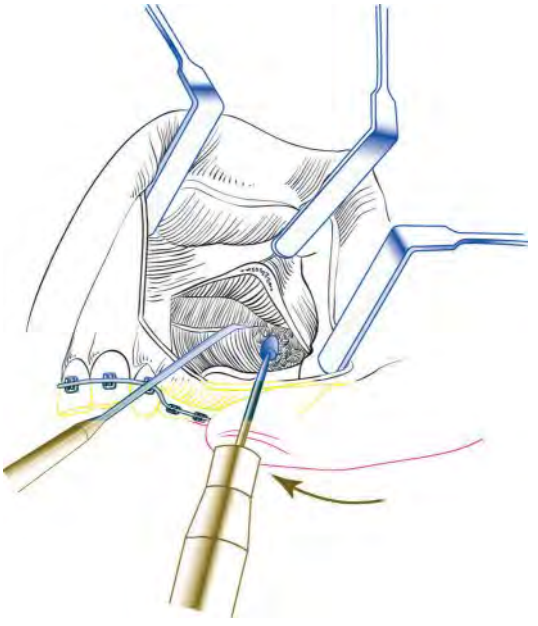


FIGURE 58-34. Bone is removed at the perpendicular plate of the palatine bone using a transantral approach.

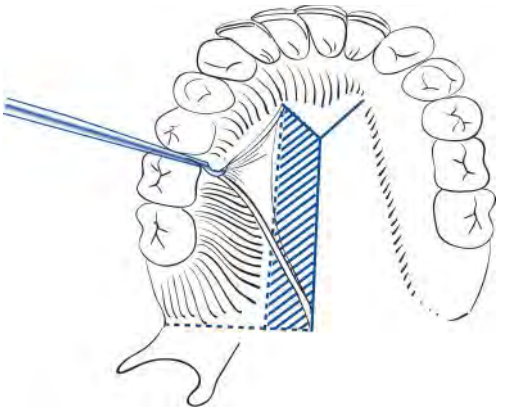


FIGURE 58-35. A midline palatal incision gives access for the removal of bone as the posterior maxillary segment is moved medially.

degree, additional access may be necessary; this can be performed via a midline palatal incision with palatal tissue reflection laterally (Figure 58-35). Meticulous surgical dissection can ensure maintenance of the integrity of the descending palatine vessels. This approach also provides access to the sinus and nasal cavity, especially if the palatal vault is high and the osteotomy is performed through the sinus as mentioned (see Figure 58-33B). If the alveolus is short and the palatal vault is flat or shallow, the osteotomy usually traverses the medial sinus wall and passes through the floor of the nose (see Figure 58-33C).

Total Maxillary Alveolar Osteotomy

The TMAO was designed to avoid some of the problems encountered with the Le Fort I down-fracture technique; however, the hypothesized advantages, including improved nasal airway, improved stability due to better bony contact, improved ability to widen the maxilla, and better maxillary perfusion, have not been realized clinically.[82–88] Throughout the past several decades, there has been very little need to consider this surgical option over the traditional standard Le Fort osteotomy, considering the increased potential risks, complications, and morbidity of the TMAO.

Surgically Assisted Rapid Palatal Expansion

History

The concept of maxillary transverse width discrepancy correction orthodontically originated in the United States in 1860 by G. H. Angell in *Dental Cosmos*,[89] where he described widening of the maxillary dental arch by opening the midpalatal suture. The use of this technique was abandoned by most American practitioners by the early 1900s, until Haas reintroduced the idea in 1961 with the introduction of rapid palatal expansion (RPE, or rapid maxillary expansion [RME]) appliances that orthopedically corrected maxillary arch width discrepancies.[90] In growing children, nonsurgical RPE resulted in fairly predictable opening of the midpalatal

suture with bony expansion, but in adults, the stability of RPE has been questioned and potentially results in dental tipping and periodontal defects rather than true bony expansion. Several authors including Timms and Moss[91] and Haas[90,92] have shown that orthodontic RPE results in alveolar bending, periodontal ligament compression, lateral tooth displacement, and tooth extrusion.[94] For these reasons, Haas advocated overexpansion to allow for some degree of relapse; however, even with 50% overexpansion, nonsurgical RPE has been associated with significant relapse and subsequent failure in the skeletally mature adult patient, although orthopedic RPE is relatively successful and stable in children and adolescents who have not yet experienced closure of the midpalatal suture. Although historically the midpalatal suture was thought to be the area of greatest resistance to expansion, Isaacson and Ingram[93] have shown that the major site of resistance to expansion is not the midpalatal suture, but all of the remaining maxillary articulations. Lines[94] and Bell and Epker[95] demonstrated that increased facial skeletal resistance to expansion was located at the zygomaticotemporal, zygomaticofrontal, and zygomaticomaxillary sutures, whereas Wertz[96] hypothesized that the resistance was attributable to the zygomatic arches. This controversy regarding the specific areas of resistance in the midfacial skeleton stimulated the development of a variety of maxillary osteotomy designs to expand the maxilla using orthodontic appliances. Lehman and coworkers[97] demonstrated that there was improved expansion with surgery and the use of an RPE orthopedic appliance. Kennedy and colleagues[98] reported a significant increase in the amount of lateral osseous movement possible in animals that underwent surgical osteotomies before orthodontic RPE. Whereas the results reported in the literature varied with the specific techniques used as well as the timing of orthopedic expansion device activation, the majority of studies show that surgically assisted rapid maxillary (or palatal) expansion (SARME or SARPE) is more stable than orthodontic expansion alone. SARPE is one of the earliest forms of distraction osteogenesis of the maxilla used to widen the maxilla in the transverse plane. The benefits of its use are gradual callous distraction that allows the soft tissues to accommodate to the bony movements thereby resulting in improved long-term stability. The role of surgery with RPE is to release the areas of resistance in the maxilla before orthopedic RPE. Whether transverse maxillary expansion will be performed alone or in conjunction with surgery will depend on the patient's age, the magnitude of expansion required (if > 5 mm is required, a SARPE may be indicated), and the condition of the midpalatal suture and will not be based solely upon the maxillomandibular relationship. Lines[95] found SARPE to be extremely valuable in young patients (growing children) exhibiting maxillary collapse, maxillary retrusion, and pseudo–class III malocclusions. When maxillary expansion and total maxillary osteotomy are required, two treatment regimens are possible: (1) SARPE followed by a one-piece maxillary osteotomy at a later date or (2) two-piece (or multiple-piece) maxillary osteotomy in one procedure. Several factors must be considered when determining which method is preferable for each individual patient.

SARPE versus Multiple-Piece Maxillary Osteotomy

There is controversy regarding the relative risks and benefits of SARPE followed by Le Fort I in two procedures as opposed to a single-stage, multiple-piece maxillary osteotomy; of the many factors involved in each of these two approaches, only the issue of transverse stability has been studied extensively. The vast majority of the data supports the fact that the transverse stability of SARPE far exceeds multipiece osteotomy.[99–106] It should be mentioned that the poorest results reported for SARPE are nearly as unstable as those reported for multiple-piece maxillary surgery; however, the surgical technique is not described.[107] In general, a SARPE will increase total arch length, permitting extraction-free orthodontic leveling and aligning and allowing a greater degree of transverse expansion with lower morbidity than segmental maxillary surgery. It has been suggested that vertical and horizontal repositioning of the maxilla are better managed in a single-stage procedure with segmental maxillary surgery,[109] and although no data to substantiate or refute this suggestion have been offered, typically when SARPE is performed before orthodontic therapy, the maxillary arch can be leveled and aligned orthodontically, within the basal bone without extrusion of teeth, which is then followed by a one-piece maxillary osteotomy to correct any vertical and horizontal discrepancies. The main reasons for this preference is that segmental maxillary surgery has a much higher incidence of both minor and major complications than SARPE and one-piece maxillary surgery, and also segmental surgery often requires premolar extractions that add another surgical procedure and additional morbidity and potential complications.[52,110] In addition, surgical splints must be left in place postoperatively for several weeks after segmental maxillary surgery, thereby increasing postoperative discomfort and negatively affecting the performance of adequate oral hygiene. Finally, issues of cost, not only from a financial standpoint but also including time off from work or school as well as the psychological impact of surgery, must be taken into consideration.[109] Therefore, SARPE followed by Le Fort I single-piece osteotomy is used most often, and the use of multisegment maxillary surgery is reserved for two specific case scenarios. The first example involves cases in which there are different occlusal planes in the maxilla that are not amenable to correction solely with an SARPE and cannot be adequately addressed with orthodontic leveling and aligning within a reasonable time period. But, with SARPE, the creation of a large midline diastema between the maxillary central incisors and the two hemimaxillae allows considerable orthodontic leveling options without tooth extrusion so that these cases requiring segmental surgery to correct variable maxillary occlusal planes are not common. The second example requiring segmental surgery includes the patient who has undergone orthodontic therapy for months or years

before referral for a surgical option and the dentition has reached a maximum limit of movement, possibly with root resorption, which would make a single-stage segmental maxillary procedure more desirable.

SARPE Technique

It should be mentioned that a variety of techniques exist for the SARPE procedure based upon where the surgeon and orthodontist believe that the major points of resistance to expansion exist. In addition, some limited osteotomy design SARPE surgeries may be performed in the outpatient setting using intravenous sedation, although if the SARPE is performed as a standard Le Fort I two-piece osteotomy without down-fracture, an operating room setting with endotracheal intubation may be preferred. A typical SARPE surgery begins with local anesthesia and bilateral mucoperiosteal incisions that extend from the piriform rim to zygomatic buttress region in the area of the maxillary first molar (Figure 58-36A). Bilateral osteotomies are then performed from the piriform rims to low in the pterygomaxillary junction (see Figure 58-36A). A simple linear AP osteotomy from the piriform rim to the pterygomaxillary junction is recommended for the SARPE.

More complicated designs[108,110,111] appear to offer advantages in theory and on paper but, in fact, are not clinically relevant when applied to the three-dimensional geometric structures such as the maxilla and the movements realized with the procedure. A sloped variation of the osteotomy of the lateral maxillary wall[110] is based upon the expectation that as the maxilla is expanded, it will "ride down" this slope laterally and inferiorly. This concept appears valid in a two-dimensional drawing; however, in three dimensions, if the osteotomy is flat from lateral to medial, as expansion occurs, the bone at the piriform region slides laterally over the flat surface lateral to it, and the bone in the buttress region slides laterally over the flat surface lateral to this area. Therefore, if the lateral maxillary wall cuts are straight and horizontal, perpendicular to the midsagittal plane, from lateral to medial, the angulation of the cut from anterior to posterior does not affect the vertical position of the segments as they are expanded. As a result, a standard horizontal osteotomy may be used for the SARPE procedure. Next, osteotomies are made in the anterior 1.5 cm of the lateral nasal walls, because this is the thickest portion of the anterior maxillary wall. Separation of the hemimaxillae is performed by driving a thin spatula osteotome between the central incisors in the

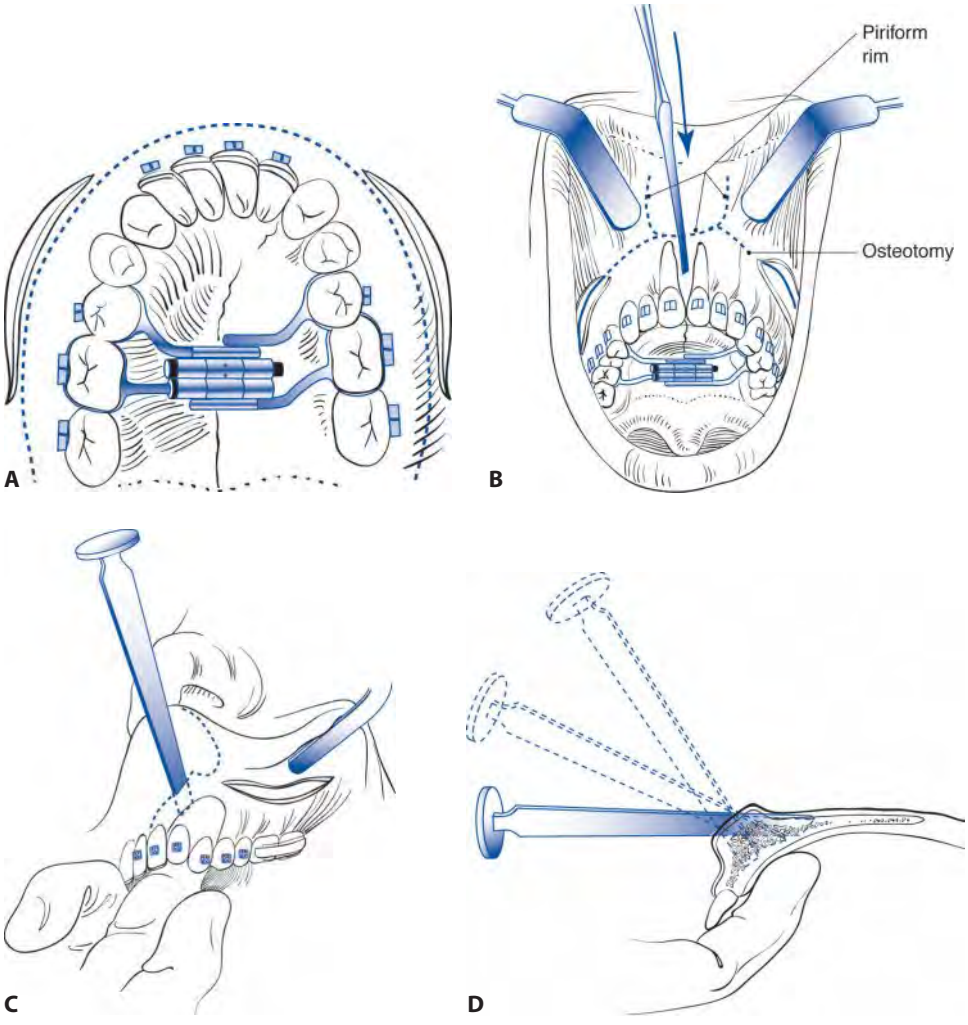


FIGURE 58-36. Surgically assisted rapid palatal expansion. **A,** Bilateral horizontal mucoperiosteal incisions are made, followed by bilateral osteotomies from the piriform rims to pterygomaxillary junctions. **B–D,** Division of the hemimaxillae is accomplished by inserting an osteotome in the midline.

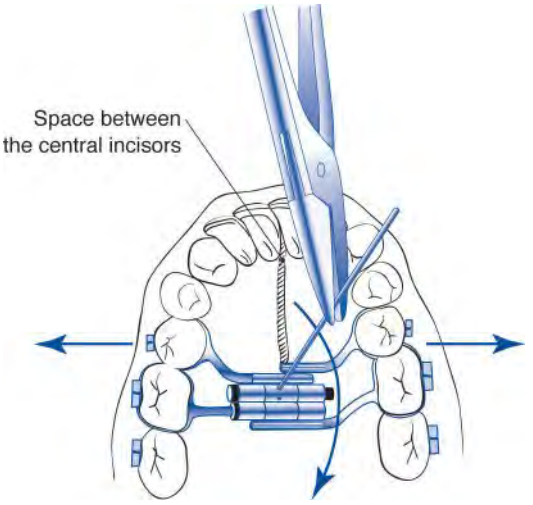


FIGURE 58-37. Surgically assisted rapid palatal expansion. Expansion device is turned to separate the hemimaxillae.

alveolus in the midline and parallel to the palate posteriorly for approximately 1 to 1.5 cm (see Figure 58-36B-D). The expansion device is turned three or four activations (¼ mm each activation, although some devices are 0.2 mm/activation) until separation is noted between the central incisor teeth (Figure 58-37). Both segments are mobilized until equal mobility is seen bilaterally in each hemimaxillary segment. Some authors recommend a subtotal Le Fort I osteotomy with a horizontal osteotomy, vertical midline osteotomy, and with pterygoid and nasal septal separation.[109,111] Shetty demonstrated with a photoelastic model that the midpalatal and pterygomaxillary articulations were the primary anatomic sites of resistance to expansion forces.[112] This study used only incomplete cuts of the lateral maxillary wall, from second bicuspid to second molar. It is unclear whether these findings would be as significant with complete cuts from the piriform to the pterygomaxillary fissure. Need for separation of the pterygomaxillary junction is, therefore, a subject of debate. Several studies have shown minimal relapse without pterygomaxillary disjunction and, therefore, do not advocate a pterygoid osteotomy for improved stability.[99,103] However, a reasonable argument for pterygomaxillary separation is that potential complications may be minimized because pterygoid separation will reduce forces transmitted to the skull base upon expansion.[111] Of course, additional sutural release will obviously contribute to improved segment mobility with an SARPE, and the potential morbidity, although significant, is typically uncommon. In addition, nasal septal separation from the nasal crest of the maxilla will avoid postoperative nasal septal deviation because the septum may remain attached to one of the two maxillary segments. Also, two sources of potential hemorrhage, via manipulation of the pterygomaxillary junction and separation of the nasal septum from the nasal crest of the maxilla, should be avoided if this procedure is performed as an office-based procedure or on an outpatient basis under intravenous sedation. Steroids are used routinely, but systemic antibiotics are not necessary. As with distraction osteogenesis, a latency period after the SARPE procedure of approximately 5 days should be used to allow some soft tissue healing and the formation of a fibrovascular bridge to support future bone formation in the distraction gap. The activation period can begin after 5 days and the expansion appliance is activated, or turned with a special "key," according to specific instructions (usually a rate and rhythm of twice-daily activations of 0.25 mm each turn), until the desired expansion is achieved based upon the orthodontic requirements. After completion of the distraction phase, a consolidation period should be allowed for bony healing with the device locked in place to prevent counterclockwise rotation, using a stainless steel wire through the activation device or composite resin, or some other material, placed to prevent device deactivation. This is usually held in place for a period of 8 to 12 weeks after completion of bony movement. An alternative use of SARPE is unilateral SARPE that can be achieved by completing a vertical interdental osteotomy between the appropriate teeth and connecting that cut with a unilateral horizontal osteotomy extending posteriorly to the pterygomaxillary junction. If the entire hemimaxilla is planned to be mobilized, the surgical procedure is performed the same as described for a standard bilateral SARPE case, with the horizontal Le Fort osteotomy performed on one side only. If widening of an isolated portion of one posterior hemimaxilla is planned, the interdental osteotomy must be completed to the midline palatal suture (Figure 58-38). The posterior maxillary segment is mobilized (Figure 58-39A) and expanded in the same manner as for the bilateral SARPE procedure (see Figure 58-39B). The long-term effects on nasal airway resistance after the SARPE procedure are unknown, although some studies have shown increased nasal airflow whereas others have not shown changes in nasal airflow in long-term follow-up.[113,114]

Zygomatic Osteotomy and Modified Le Fort Osteotomies

As discussed in the section on anterior maxillary repositioning, patients with severe midface deficiency may benefit from enhancement of the prominence of the zygoma region. From an aesthetic standpoint, prominent malar prominences are desirable, and with a growing public awareness of surgical capabilities, there is an increased demand for cosmetic procedures designed to enhance this region of the face. Numerous methods have been developed to augment the malar eminences and the paranasal regions, mostly using bone grafts or synthetic implants. Autologous grafts, from the ramus, chin, or coronoid process, are disappointing because of resorption and the donor site morbidity. Allogeneic transplants such as lyophilized cartilage have been used with some success but are prone to migration, inflammation, and resorption. At the present time, the preferred method for malar augmentation is the use of alloplastic implants, most commonly with high-molecular-weight porous polyethylene

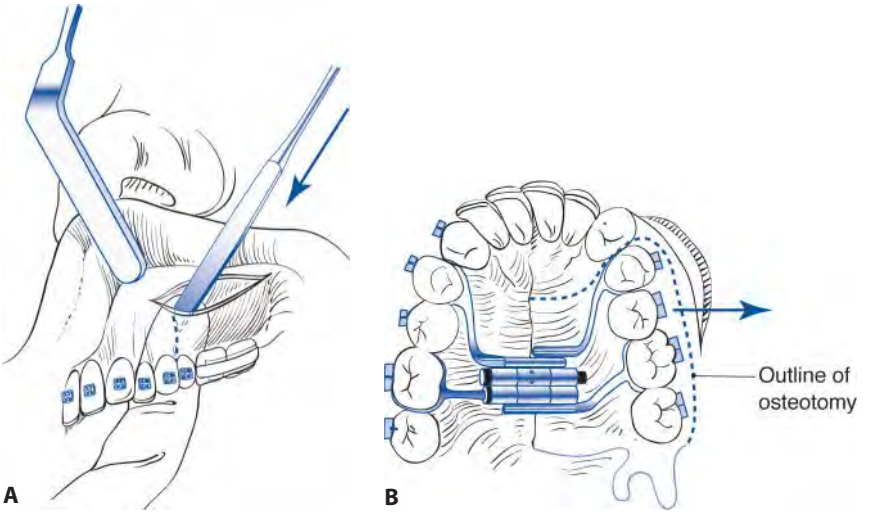


FIGURE 58-38. Unilateral surgically assisted rapid palatal expansion. **A,** The osteotomy is driven to the midpalatal suture. **B,** The horizontal osteotomy is completed in the same manner as the bilateral osteotomy.

or silicone. If alloplasts are contraindicated or declined by the patient, the zygomatic osteotomy may be a useful alternative. The zygomatic osteotomy is approached through an intraoral incision, and a reciprocating saw is used to create a parasagittal osteotomy through the zygoma immediately adjacent to the root of the zygoma (Figure 58-40A and B). This is performed as close to the lateral orbital rim as possible, and the zygoma is out-fractured gently so that an interpositional material can be placed in the gap to hold the zygoma in a lateral position. The interpositional material (bone graft or synthetic implant) can be stabilized in a traditional fashion in this area. This technique provides lateral zygomatic projection, but it does not provide anterior projection, unless the interpositional material is fashioned in such a way as to project the zygoma in a forward position (see Figure 58-40C).

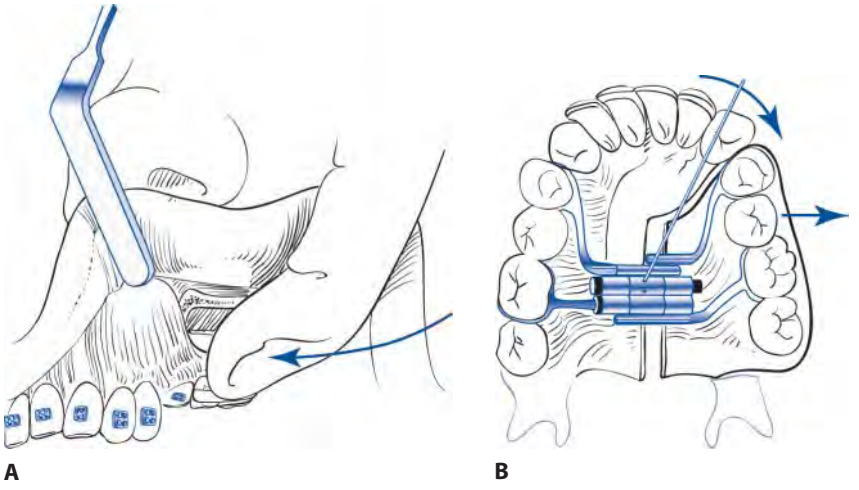


FIGURE 58-39. Unilateral surgically assisted rapid palatal expansion. **A,** The segment is mobilized. **B,** The expander is activated until the desired expansion is achieved.

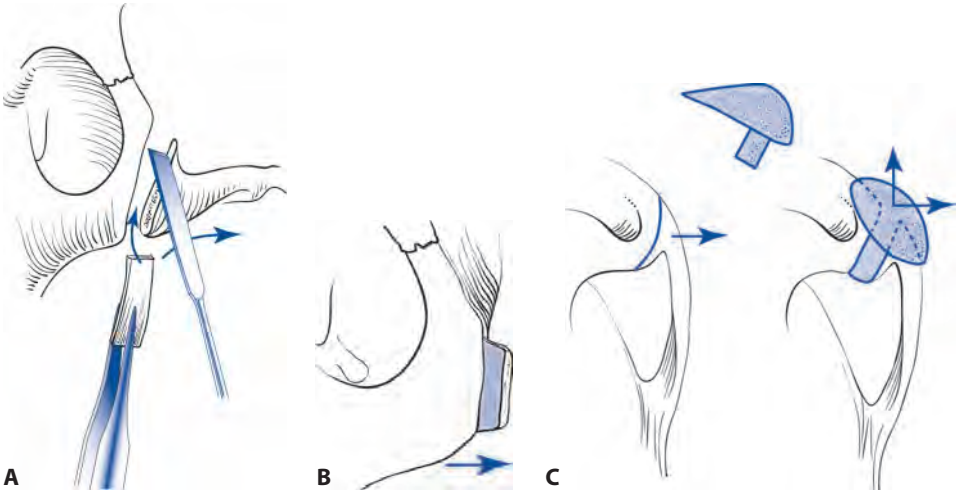


FIGURE 58-40. Zygomatic osteotomy. **A,** The parasagittal vertical osteotomy cuts through the root of the zygoma. **B,** With out-fracturing and placement of graft material for transverse augmentation of the malar eminence. **C,** Mushroom-shaped graft placement for anteroposterior and transverse augmentation of the malar eminence.

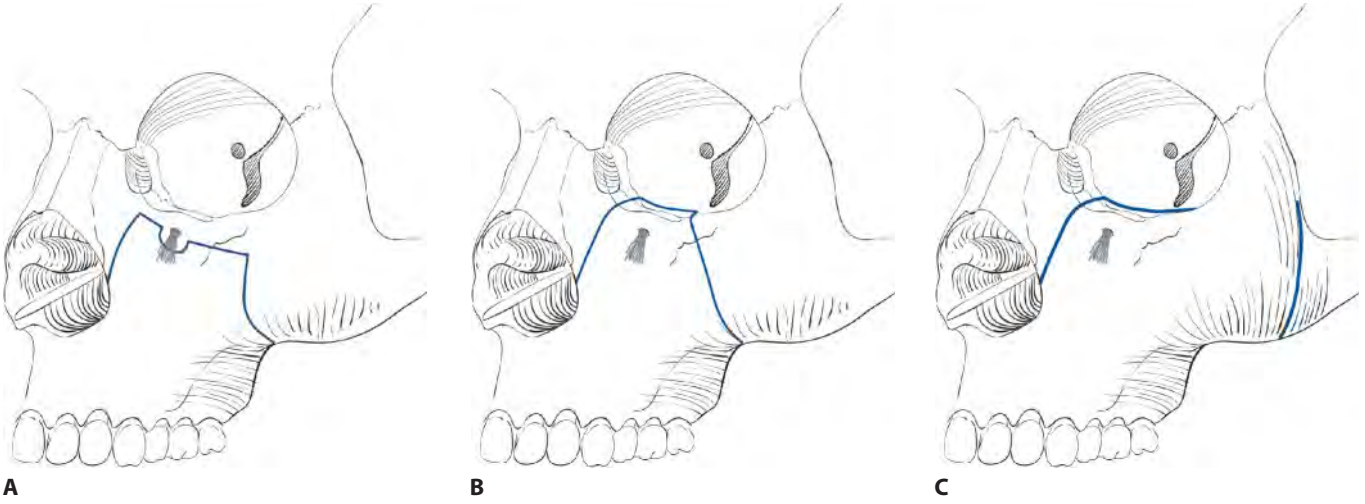


FIGURE 58-41. Modified Le Fort I osteotomy. **A,** High Le Fort I below the infraorbital rims. **B,** Quadrangular Le Fort I extending into the orbital floor. **C,** Quadrangular Le Fort I including the lateral orbital rim and zygoma.

The wide range and variety of maxillary osteotomies that represent modifications of the traditional Le Fort I procedure have been referred to by many terms including the *modified Le Fort I, II, III, high Le Fort I, pyramidal, middle, intermediary, quadrangular,* and *maxillary-malar-infraorbital osteotomies* (Figure 58-41). These techniques or modifications to these procedures have been described previously[115]; however, this group of maxillary osteotomies is limited clinically with regards to their use for maxillary expansion, rotation, and torque, or yaw, movements. Therefore, with the clinical successes of silicone and porous polyethylene and other synthetic implants to the malar, infraorbital, lateral orbital, and paranasal regions, these more invasive and potentially hazardous osteotomy designs are rarely used clinically (Figure 58-42).

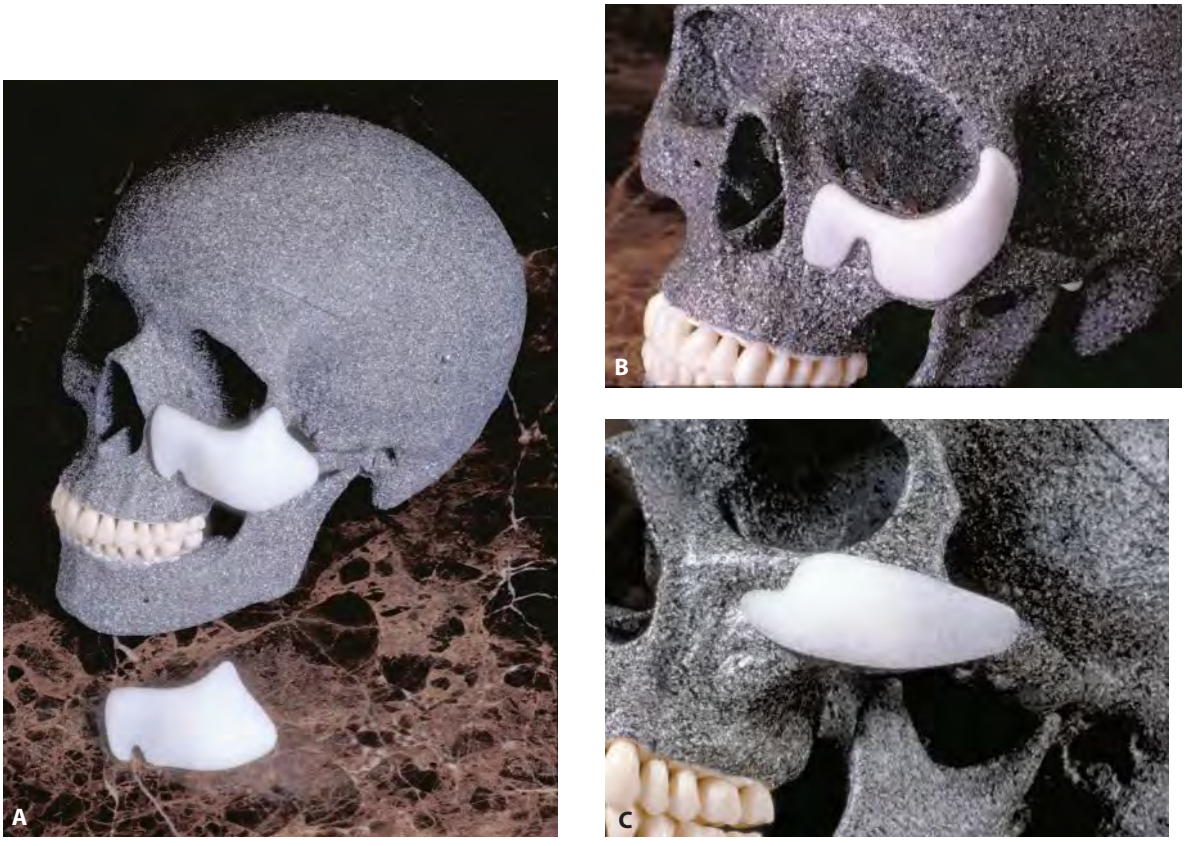


FIGURE 58-42. Porous polyethylene implants. **A,** Infraorbital augmentation with the zygoma. **B,** Infraorbital augmentation with the zygoma and lateral orbital rim. **C,** Zygoma augmentation.

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CHAPTER 59

Facial Asymmetry

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Facial asymmetries comprise a heterogeneous group of craniofacial disorders charcterized by significant alterations in dental relationships as well as facial form that are readily apparent even to the untrained individual, and these present significant psychosocial issues for patients and families. During the 1960s, surgical treatment of orthognathic deformities developed when satisfactory results were not attainable with orthodontic therapy alone. On occasion, mild cases of jaw deformities and dental malocclusions may be camouflaged by dental treatment and growth modification.[1] In contrast, severe skeletal malocclusions are often beyond the envelope of discrepancy for orthodontic treatment and growth modification; therefore, a variety of surgical procedures of the maxilla and mandible have been developed. Some severe malocclusions are beyond the scope of orthodontic therapy alone, and some orthognathic deformities are beyond single-jaw surgery. Although a one-jaw osteotomy might improve function and aesthetics, bimaxillary, two-jaw, or double-jaw, surgery is often indicated for larger-magnitude anteroposterior (AP) discrepancies, most open bite deformities, and most cases of skeletal asymmetries.[2–5] With refinements in orthognathic surgery, it has become clear that some skeletal problems are beyond the envelope of one-jaw surgery, and although the novice might assume that one-jaw surgery is indicated owing to simplicity, the final functional and aesthetic outcome is often compromised and the bony movements are unstable. In general, two-jaw surgery allows a much greater degree of flexibility with regards to a three-dimensional approach to treatment of facial asymmetries.

In the 1960s and 1970s, surgeons attempted to limit orthognathic surgery to one jaw, usually the mandible, for fear of vascular compromise with maxillary osteotomies. Mandibular ramus osteotomies performed in conjunction with maxillary osteotomies were complex, technically difficult, time-consuming, and unstable procedures and were associated with higher patient morbidity.[6] It is difficult to correct a maxillary occlusal cant in the nongrowing patient without adjustment of the mandibular plane of occlusion. Similarly, midline discrepancies resulting from unilateral tooth loss are more rapidly corrected by mild asymmetrical rotational changes in the maxilla and/or mandible rather than with a prolonged course of orthodontics with possible gingival and periodontal problems.

This chapter focuses on the unique characteristics of asymmetrical orthognathic deformities as an indication for two-jaw surgery. The most important concept to understand about bimaxillary surgery is not the ability to perform simultaneous maxillary and mandibular procedures but to know the indications for surgery as well as the sequential treatment plan and also how to maintain a stable reference during complex asymmetrical bimaxllary surgery. Although little information is available on orthognathic asymmetry as an indication for bimaxillary surgery, it is well recognized that an inappropriate diagnosis, inaccurate treatment planning, and lack of intraoperative surgical reference points and planes are common errors in the approach to these patients. Skeletal asymmetries require three-dimensional bony and soft tissue changes with complex skeletal movements that result in aesthetic facial changes, and facial symmetry has a high degree of correlation with perceived facial attractiveness. Even mild facial asymmetries may be easily perceived by the untrained individual, and higher degrees of asymmetry are correlated with clinical depression, neurosis, feelings of inferiority, poor self-esteem, and general poor quality of life health-related problems.[7]

ETIOLOGY OF FACIAL ASYMMETRY

Although there are multiple potential causes of facial asymmetry, the differential diagnosis can be grouped into three general classes: congenital, developmental, and acquired deformities.[8,9] *Congenital* anomalies are conditions acquired during in utero development and can be further subdivided

into malformations, deformities, and disruptions. Malformations are the result of an intrinsically abnormal developmental process during embryogenesis. For example, unilateral cleft lip is a type of malformation.[9] Deformities represent an abnormal form, or position, of a part of the body caused by a nondisruptive mechanical force applied during the fetal period.[9] Mandibular deformation may result from a prolonged, acute lateral flexion position of the head, with the shoulder pressed against the mandible, during late intrauterine growth. Lastly, disruptions are morphologic defects resulting from a breakdown of an otherwise normal developmental process.[9] Examples of disrution are facial clefting and limb amputation from in utero formation of amniotic banding.[9] *Developmental* anomalies are conditions arising during postuterine growth through adulthood, and *acquired* anomalies are clinicial conditions arising from either traumatic injuries or pathologic lesions. Finally, the etiology of facial asymmetry may be idiopathic without any identifiable cause.

CONGENITAL ANOMALIES

Hemifacial Microsomia

Hemifacial microsomia (HFM) is a craniofacial malformation resulting from defects in the first and second branchial arches that presents with asymmetrical unilateral, or bilateral, hypoplasia of the orbit(s), maxilla, mandible, ear, cranial nerves, and facial soft tissues (Figure 59-1).[10] Current evidence supports the theory that HFM results from a defect in the proliferation and migration of embryonic neural crest cells,[11,12] whereas another theory maintains that hemorrhage, or thrombosis, of the stapedial artery during fetal development leads to impaired unilateral facial growth.[13,14] In fact, the exact etiologic factors involved in the development of HFM still remain unknown.

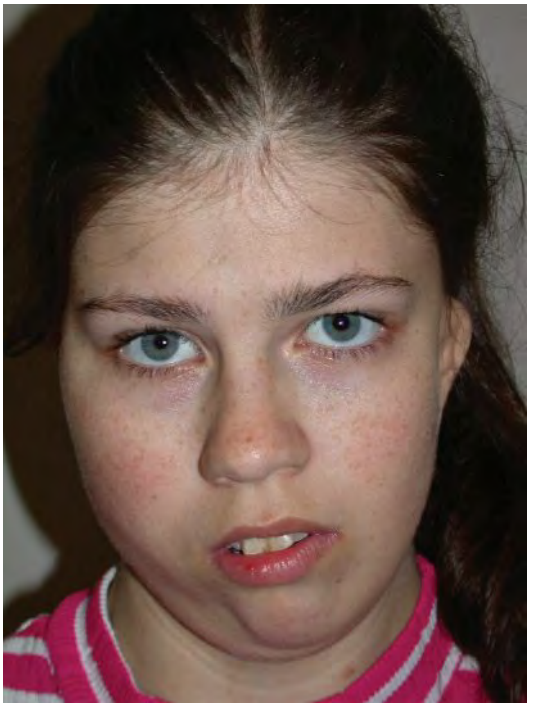


FIGURE 59-1. A 16-year-old with hemifacial microsomia (HFM) type I, with maxillary cant, dental midline deviation, and facial asymmetry.

Two important factors need to be considered in the treatment planning of the patient with HFM: (1) the facial growth potential and magnitude of growth restriction and the effect on surrounding hard and soft tissue structures and (2) the degree of hypoplasia of the glenoid fossa, mandibular condyle, and ramus unit.[15,16] Appropriate classification of the extent of the HFM deformity can provide useful information in order to determine an accurate diagnosis and ideal reconstruction options and a valid estimation of prognosis. The Pruzansky classification of HFM,[17] modified by Kaban and coworkers,[18] currently provides a clinically useful framework to help guide the treatment plan, based upon the presence or absence of critical structures of the condyle-fossa-ramus complex.[18,19] HFM type I deformity can be summarized as a generalized mild hypoplastic state involving the muscles of mastication, the glenoid fossa, and the mandibular condyle and ramus unit. The temporomandibular joint (TMJ) functions with normal rotation but restricted translation movements. Patients typically present with mild mandibular retrognathia and facial asymmetry. Because there is acceptable TMJ function and mild facial dysmorphology, surgical therapy is usually not indicated. HFM type IIA deformity involves a hypoplastic cone-shaped condylar head that is located medial and anterior to a hypoplastic glenoid fossa. TMJ function is often satisfactory so, again, surgical intervention is usually not indicated. HFM type IIB deformity involves a moderate to severe hypoplasia of the glenoid fossa, condyle, and mandibular ramus. Unlike the type IIA deformity, these patients have no functional articulation between the temporal bone and the condyle. However, manual jaw manipulation usually reveals a posterior stop of the condyle when placed in contact with posterior slope of the glenoid fossa.[20] HFM type III has a complete absence of the mandibular ramus and condyle. No manual condylar seating or posterior stop is achievable with jaw mamipulation. In contrast to types I and II, type III patients present with severe mandibular dysmorphology and often require surgical reconstruction of the fossa-condyle-ramus complex (Figure 59-2).[20]

The treatment protocols for HFM remain an area of controversy. The treatment philosophy of using interceptive orthodontic therapy, combined with early surgical treatment in growing children, is based upon the hypothesis that HFM is a progressive deformity and that early treatment can positively affect future facial growth.[15] Alternatively, a treatment protocol based upon the theory that HFM is not progressive in nature has been well described by Posnick,[16] and with this philosophy, treatment is considered primarily later in life during the adolescent period.

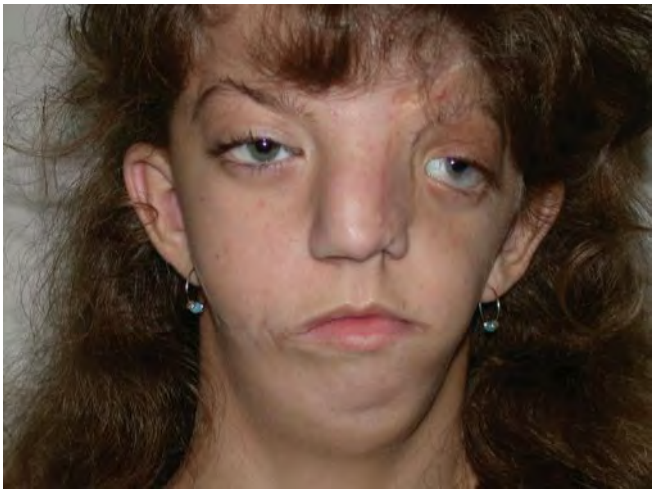


FIGURE 59-2. A 14-year-old with HFM type III, associated with facial clefting and lack of a condyle.

Cleft Lip and Cleft Palate

Patients with cleft lip and palate often present with bilateral or unilateral midface deficiency resulting in an apparent increased AP projection of the paranasal, nasal, infraorbital, and zygomatic regions as well as at the occlusal level.[21] However, the degree and location of maxillofacial growth deficiency in children with cleft lip and palate are largely dependent upon the location and type of cleft lip and cleft palate repair performed and the patient age at the time of repair.[22–24] Most studies show that children with a repaired cleft lip and palate have a decreased vertical and horizontal maxillary growth pattern and a decreased vertical growth of the mandibular ramus with a steep mandibular plane angle.[25–27] Ross[28] has shown that approximately 25% of patients with a repaired cleft lip or cleft palate have a midface deficiency and class III malocclusion that require skeletal orthognathic surgery in adolescence.

Craniosynostosis: Plagiocephaly

The term *plagiocephaly* is derived from the Greek word *plagios*, which refers to the twisted shape of the skull when viewed from a craniocaudal perspective. The etiology of this type of craniosynostosis is often a unilateral premature fusion of the coronal or lambdoid sutures. Unilateral synostosis of the coronal suture results in an asymmetrical parallelogram-shaped forehead and brow. The affected side is usually flattened, and the contralateral side may show compensatory overgrowth with frontal bulging or frontal bossing. In addition, synostosis of the coronal suture often indirectly affects the lower facial morphology, with deviation of the root of the nose toward the affected side and chin deviation to the unaffected side, opposite to the flattened forehead. The mandible generally develops normally but may exhibit secondary dysmorphology as well.[29,30]

Congenital Hemifacial Hyperplasia

Congenital hemifacial hyperplasia is a rare unilateral enlargement of the craniofacial soft and/or hard tissues. Although the term "hemihypertrophy" has commonly been used, it is inappropriate because the condition is due to tissue hyperplasia (increased number of cells) and not tissue hypertrophy (enlargement of individual cells).[31] Pollock and colleagues[32] have hypothesized that the asymmetrical facial development results from abnormal neural crest cell migration. Yoshimoto and associates[33] found increased proliferative activity of osteoblasts in a patient with congenital hemifacial hyperplasia and hypothesized that fibroblast growth factor (FGF), and its receptor signal transduction axis in osteoblasts, may be selectively involved resulting in the progression of hemifacial overgrowth.

DEVELOPMENTAL FACIAL ASYMMETRIES

Primary Growth Deformities

Facial Hemiatrophy

Facial hemiatrophy (Parry-Romberg syndrome) is characterized by a progressive unilateral facial loss of skin, associated soft tissues, cartilage, and hard tissues (Figure 59-3). The left side is more often affected than the right side. Associated clinical findings include Jacksonian epilepsy, cutaneous dyspigmentations, and ipsilateral alopecia.[31] An internet survey of 205 facial hemiatrophy patients revealed a median age



FIGURE 59-3. Right facial hemiatrophy (Parry-Romberg syndrome), after skin and bone grafting.

of onset of 10 years with a range of 1 to 50 years. In most patients (71%), the facial hemiatrophy began before age 15 years.[34-36] The etiology of facial hemiatrophy remains unknown, but there have been associations established with Lyme disease, ablation of the superior cervical sympathetic ganglia, localized scleroderma, Rasmussen's encephalitis, and systemic lupus erythematosus (SLE).[37–41] Alterations in the peripheral trophic sympathetic system is one of the more commonly accepted theories of the etiology of this disorder.[31] The various treatments for the soft tissue deficiencies of facial hemiatrophy, or hemifacial atrophy, have included silicone injections, alloplastic implants, microfat injections, and microvascular free tissue transfer.[42–44]

Hemimandibular Hyperplasia

Another developmental anomaly resulting in facial asymmetry is hemimandibular hyperplasia, which is characterized by a diffuse enlargement of the condyle, the condylar neck, and the mandibular ramus and body regions.[45] In 1986, Obwegeser and Makek[46] described the deformity as hemimandibular hyperplasia or hemimandibular elongation. In 1996, Chen and coworkers[47] proposed that all cases of hemimandibular hyperplasia actually represent a spectrum of variations in condylar overgrowth, and it was proposed that if condylar overgrowth is not arrested, it can progress into hemimandibular hyperplasia. In spite of the differences in nomenclature, no specific etiologic factor has been established. Condylar growth patterns can be evaluated by serial clinical comparisons, including serial cephalometric tracings, and bone scan techniques. However, no ideal method has been found to assess whether condylar overgrowth is active or inactive, or quiescent. Treatment for hemimandibular hyperplasia is guided by patient age and condylar growth activity; these range from orthopedic maxillary management to condylectomy. Although it is generally accepted that surgical treatment should be delayed until growth activity has ceased,[45] others believe that early surgery, performed while condylar growth is still active, may cause a surgical insult and postsurgical scarring that can beneficially halt the condylar growth. Also, in some severe cases, condylar resection during active growth of the condyle may be necessary for correction of a significantly asymmetrical, or socially unacceptable, facial appearance.

Secondary Growth Deformities

Sternocleidomastoid torticollis is a condition resulting from a birth trauma–induced hematoma of the sternocleidomastoid muscle that gradually undergoes fibrosis over time and leads to muscular contraction and torticollis. However, the precise etiologic factors of this entity are still unknown. If the condition is not corrected early enough with proper physical therapy, or surgery, for neck and sternocleidomastoid muscle mobility, malformed facial development will usually result ipsilateral to the side affected by the torticollis.[48,49] Duchenne's muscular dystrophy and cerebral palsy often result in areas of decreased muscle tone, which can affect the development of facial form by limiting the amount of bone formation at sites of normal muscle attachment and function. Consequently, facial asymmetry and dysmorphology may be a clinical finding seen in patients with Duchenne's muscular dystrophy and cerebral palsy.[50]

ACQUIRED FACIAL ASYMMETRIES

Condylar Trauma

A common cause of facial asymmetry in the growing child is trauma to the mandibular condyle, although in many cases, neither the child nor the parent can recall the traumatic event (Figures 59-4 and 59-5).[51] Trauma-induced injury to the condyle can lead to a hemarthrosis that results in intra-articular scarring and restriction of condylar translation. Proffit and Turvey[52] described this clinical scenario as a functional ankylosis, or soft tissue extracapsular ankylosis, because, in most cases, condylar function is acceptable. In addition to limitation of jaw movement caused by fibro-osseous TMJ ankylosis and coronoid process elongation and temporalis tendon scarring, complete bony ankylosis of the condyle to the temporal bone and skull base may also occur after intracapsular hemarthrosis, resulting in no rotation or any condylar movement. Consequently, trauma-induced scarring or bony ankylosis of the TMJ can also result in relative degrees of restricted skeletal growth. In other words, the greater the degree of translational restriction, the greater the resultant facial deformity. A similar clinical scenario involves whether open reduction with internal fixation (ORIF) of a condylar fracture is required to stabilize the condylar cartilaginous growth center in skeletally immature patients. Studies in skelettaly immature primates and in children have revealed that the displaced condylar segment undergoes resorption and replacement with a new condylar head with regeneration

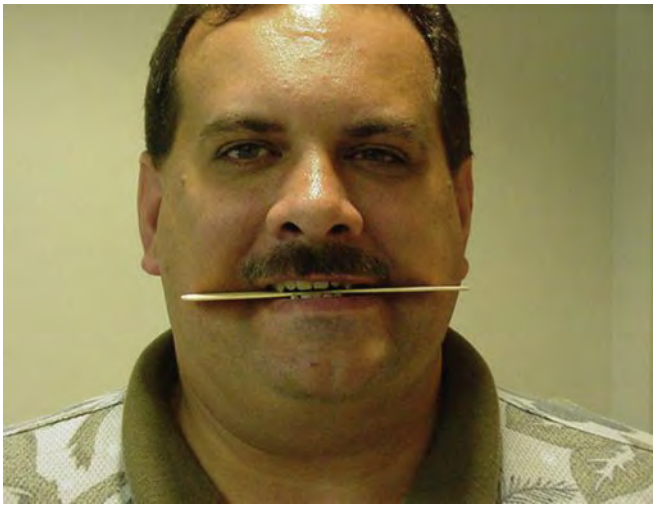


FIGURE 59-4. A 38-year-old who had a right condylar fracture during adolescence resulting in excessive growth of the right condyle and jaw deviation to the left.

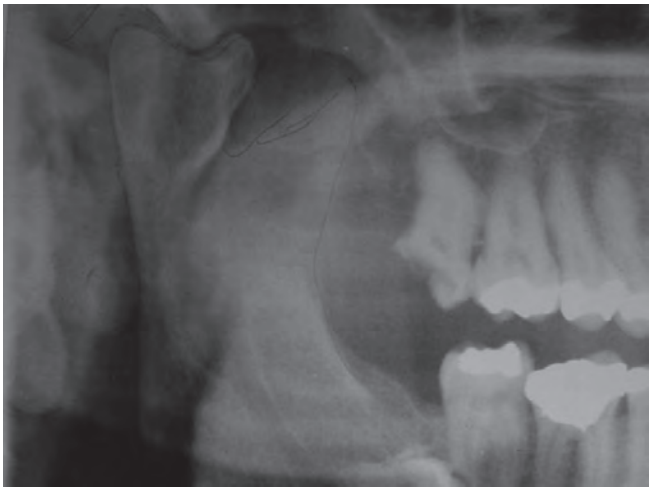


FIGURE 59-5. Panorex of the patient in Figure 59-4 shows right condylar overgrowth and asymmetry.

of the overlying cartilage based upon functional remodeling. Therefore, there is nothing intrinsically critical about the condylar head as a mandibular growth center, and because the condylar head in children is regenerated spontaneously, the necessity of ORIF of a displaced condylar segment is unnecessary. Moreover, the surgical procedure is not without potentially significant morbidity, and the resulting scar and possible soft and hard tissue restriction from the ORIF procedure may outweigh the benefits of surgical anatomic condylar reduction. Several studies have indicated that ORIF of condylar fractures in children should be avoided.[52–54]

Juvenile Idiopathic Arthritis

Facial asymmetry may be found in patients affected with juvenile idiopathic arthritis (JIA) of the TMJ.[55] JIA is a disease characterized by chronic inflammation of one or more joints, affecting children up to the age of 18 years. The TMJ is frequently involved and can lead to facial growth disturbances, including facial asymmetry.[55] TMJ involvement may be asymmetrical and very apparent or may be mild or asymptomatic and not be clinically detectable.[56,57] However, symptomatic TMJ involvement may not be associated with any evidence of facial growth disturbances; and conversely, facial growth disturbances may be present without any clinical TMJ symptoms.[56] Both polyarticular-onset and pauciarticular-onset JIA have been found to have a negative impact on the form, function, and aesthetics of the face. However, the effects are more pronounced with polyarticular JIA.[58,59] Characteristic facial features of patients with JIA include a small mandible, class II malocclusion, and anterior open bite occlusion. Patients with polyarticular JIA with TMJ involvement tend to have small, short faces with underdeveloped mandibles.[60] Currently, no effective therapeutic method is available to eliminate the progression of the disease and its effect on facial development, although methotrexate therapy has been shown to minimize TMJ destruction and craniofacial dysmorphology in patients with polyarticular JIA.[61] Corticosteroids have been used in the treatment of JIA, but their therapeutic benefit is controversial.

Degenerative Joint Disease

Degenerative joint disease (DJD) is considered an end-stage result of progressive internal derangement and osteoarthritis of the TMJ. Usually, patients have bilateral involvement of the TMJ, but unilateral involvement is not uncommon. The wear and tear effects of DJD on the TMJ results in condylar-glenoid erosion and decreased condylar ramus height with asymmetry. Clinically, patients often present with preauricular noise or crepitus, limitation of mandibular motion, preauricular pain, and an anterior open bite occlusion.[62]

CLINICAL PATIENT ASSESSMENT

The usual comprehensive methods of patient evaluation includes the chief complaint, and with additional information gained from the history of the present illness, the answers to pertinent questions regarding a history of facial trauma, arthritis involving other joints, and genetic or congenital malformations may be identified. A physical examination of the head and neck region in the patient with a facial asymmetry should include[63]

1. Visual inspection of the entire face including facial subunits for symmetry in the vertical and horizontal planes.
2. Palpation of the face to differentiate between soft and hard tissue defects.
3. Comparison of dental and facial midlines with each other and with the central facial axis.
4. Inspection for gonial angle asymmetry or differences in the degree of antegonial notching.
5. Analysis of the relationship between the upper lip and the maxillary central incisors.
6. Inspection for malocclusion, occlusal cants, excessive inclination of anterior teeth, dental crowding, open bite occlusal relationships, the maximal interincisal opening, and the presence of mandibular deviation upon opening.
7. Comprehensive examination of TMJ function including protrusion and lateral excursion movements.

Following the data gathering including the chief complaint, history of present illness, past medical history, family history, social history, and physical examination, radiographic and laboratory studies may be indicated, and articulator-mounted diagnostic dental casts are evaluated, a prioritized problem list and corresponding treatment plan options may be developed and presented to the patient and family.

Radiographic Assessment

Panoramic Radiograph

The Panorex can provide information about the relative size and shape of the mandibular condyle as well as ramus height and antegonial notching. Degenerative changes of the TMJ or

asymmetrical mandibular ramus morphology may be identified with a comparative vertical measurement of the condylar head apex (condylion) to the base of the sigmoid notch and the sigmoid notch to the mandibular angle (gonion) bilaterally.[63] Also, dental and sinus abnormalities as well as orbital and ear deformities may be detected if the field of view includes these structures.

Posteroanterior Cephalometric Radiograph

The posteroanterior (PA) cephalometric radiograph enables one to evaluate the magnitude of the facial skeletal deformity relative to the cranial base. Acetate overlay tracings soft and hard tissue features can be created and compared with true vertical and horizontal axes. These reference landmarks may not be easy to determine, especially in severe deformties or where the landmark may be missing (e.g., zygomatic bone or condylar hypoplasia or aplasia). Although the most appropriate midline reference is controversial and may include a midpoint between two lateral measurements (e.g., medial aspect of the frontozygomatic suture), a consistent midline reference is the crista galli of the ethmoid bone. In cases of ear abnormalities, the ear rod on the involved side should rest against the temporal bone in the area where the normal ear should be so that horizontal references can be compared without the need to account for an abnormal head posture. Also, owing to the overlap of several skeletal structures, there are limitations to the use of the PA cephalogram in the diagnosis of facial asymmetries, but in general, deviations of the dental and skeletal midlines, occlusal cants, orbital asymmetries, ear and external auditory canal (EAC) abnormalities, and vertical ramus discrepancies may be determined with this radiograph.[63]

Lateral Cephalometric Radiograph

A lateral cephalometric radiograph can provide an indication of vertical ramus height differences by the finding of two separate radiographic mandibular inferior border outlines. However, to determine the relative significance of the differences in dentofacial superimposition, one must know whether the EACs are level with the patient's natural head position, and as mentioned, only a single cephalometric ear rod should be used if the patient's EACs are abnormal or not located at the same level.[63]

Computed Tomography

Computed tomography (CT) can provide two-dimensional localized views of the facial skeleton, or it can be developed further into three-dimensional views that can provide excellent overall detail necessary for the proper diagnosis and treatment planning of a complex facial dysmorphology (Figure 59-6).

Stereolithographic Modeling

Three-dimensional CT scans can provide information to allow the fabrication of an actual three-dimensional skeletal model via stereolithography (SLA) or computer-aided design/computer-aided manufacturing (CAD/CAM). These models can assist the surgeon with physical three-dimensional assessment and formulation of surgical predictions; however, owing to the high cost, these models are primarily used for planning of complex dentofacial and craniofacial deformities.[64]

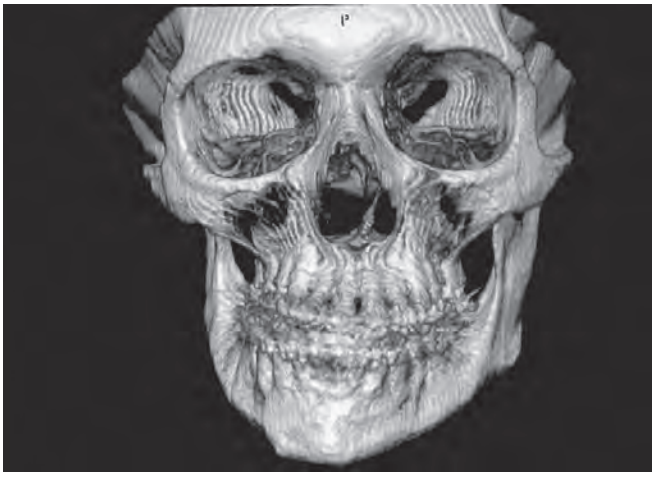


FIGURE 59-6. Three-dimensional computed tomography (CT) scan of asymmetrical craniofacial skeleton (right HFM).

Most recently, three-dimensional CT treatment planning has become a popular technique for planning three-dimensional orthognathic and skeletal jaw movements, especially in cases of severe asymmetries planned for complex rotation and transverse jaw movements, and fabrication of SLA intermediate and final occlusal splints during the treatment planning process.

Bone Scans

Radionuclide skeletal scintigraphy, including technetium 99m pertechnetate, gallium, and indium-labelling, as well as positron-emission tomography (PET) scans, have been shown to be sensitive diagnostic methods for identifying mandibular overgrowth in the patient with a facial asymmetry and to determine activity or quiescence of the increased growth. However, bone scan findings are nonspecific and may be the result of a variety of bone and soft tissue abnormalities, including soft and hard tissue carcinomas, sarcomas, metastatic disease, hematologic disease, infections, inflammatory processes, metabolic diseases, and trauma, including recent dental extractions.[65] In patients with asymmetrical mandibular overgrowth, nucleotide uptake is not imaged in a symmetrical fashion bilaterally, and in most cases, patients demonstrate an increased nucleotide uptake and activity on the affected side. However, caution must be exercised when evaluating an area of increased uptake in order not to confuse condylar overgrowth with other conditions (e.g., arthritis, TMJ disorders, trauma) that can mimic nucleotide uptake activity.[66] Additional diagnostic radiographic techniques of

fusing PET-CT images to high-resolution structural CT images have been shown to provide improved resolution and delineation of the precise area of bone activity.[67]

SURGICAL TREATMENT

Some facial asymmetries may not always be apparent to the patient and family, and therefore, appropriate treatment can begin only when a proper diagnosis has been established. The face should be evaluated in all dimensions, with a careful analysis of the vertical and horizontal proportions and the corresponding facial subunits. Failure to recognize asymmetry until after surgery is complete is generally the reason for the poor final outcome. In general, treatment planning of facial asymmetry is similar to that of orthognathic surgery, except that, depending upon the specific skeletal abnormality, more emphasis is placed on the frontal, as opposed to the profile, view during the examination. As mentioned, cephalometric analysis may be grossly inaccurate owing to inappropriate ear rod positioning during lateral and PA cephalograms, and whereas these radiographs are simple screening tools, standard and three-dimensional CT scans are much more accurate. CT data can be converted by CAD/CAM imaging into an actual acrylic SLA model that can be used for predictive model surgery, customized facial implant fabrication, and design of custom distraction osteogenesis devices. Despite the available radiologic studies, the clinical examination is the most important diagnostic tool, and it should be remembered that body posture, mannerisms, and the presence of facial hair and various hairstyles may mask a facial asymmetry and may direct the treatment plan in an erroneous direction. In one study of 495 patients with facial asymmetry, the mandible was the facial bone most often affected.[68] Upper face and midface asymmetry was found only in 5% of patients, but mandibular asymmetry with chin point deviation was present in 75% of cases. Chin deviation, usually apparent on clinical examination, is most often to the left, indicating a tendency for increased right-sided mandibular growth.

Delayed Treatment

In many cases, a delayed treatment approach may be most appropriate because the management of facial asymmetry in preadolescent children is extremely complex and the results are not always predictable owing to continued facial growth. The interpretation of studies on growth modification using functional appliances have been difficult because of a variety of treatment designs with lack of standardization of treatment and control group populations and difficulties with randomization of the groups, as well as other confounding variables in the studies. Although noninvasive techniques, such as growth modification, will not cause irreversible harm to the patient, most asymmetrical craniofacial syndromes, condylar deformities, and traumatic injuries sustained at an early age do require surgery. Bite block therapy can be helpful in reorienting the plane of occlusion in children with occlusal cants, but it rarely obviates the need for surgery, and typically, bite block therapy is mainly used as an intervention for secondary growth deformities. The functional status of the TMJ is a critical component to consider in the overall treatment planning process for the patient with facial asymmetry, and mild asymmetries in the growing patient with functional TMJs should be considered for early interceptive orthodontics only, and growth completion should be permitted to occur before consideration for surgical intervention. It is well accepted that jaw movement is critical after condylar fractures to prevent limitation of mandibular motion and the development of ankylosis and that physical therapy and rehabilitation stimulate continued condylar and mandibular growth. Lack of function usually results in an asymmetrical mandible and secondary maxillary and midface asymmetries. After condylar trauma, mandibular hypoplasia with growth retardation is seen much more frequently than mandibular hyperplasia or overgrowth. In general, mandibular hyperplasia may be accelerated after an adolescent growth spurt, whereas delayed growth with mandibular hypoplasia is more often present in the preadolescent years and childhood. Regardless of the exact etiology of the mandibular asymmetry, it is important to establish a surgical treatment plan that will achieve the most functional, stable, and aesthetic outcomes. Orthognathic asymmetries should be treated after growth completion because this surgery will require a combination of maxillary and mandibular surgery with the application of rigid internal fixation.

Orthodontic Considerations

Human facial symmetry is directly associated with facial attractiveness. It is also well documented that true symmetry is not the norm, and comparison mirror-imaging of the right sides of a face compared with the left side reveals two completely different facial forms.[69,70] For the typical orthodontic patient, minor facial asymmetries become a concern only when they present with aesthetic complaints. Severe asymmetries often result in cross-bites, malocclusions, cheek biting, poor masticatory forces, condylar dysfunction, myositis, tendinitis, and chronic head and neck pain. From a diagnostic standpoint, patients with asymmetries differ from the typical orthodontic patient in several ways, and the clinical examination and data gathering process should generate sufficient information for accurate diagnosis and formulation of the most appropriate treatment plan. The data should include photographs from multiple views, lateral and PA cephalometric radiographs, and facebow-mounted dental models.[62–71] Facial structures may be evaluated with a reference grid formed by the midsagittal plane and several perpendicular reference lines, according to the area being evaluated (e.g., interpupillary, subnasal, stomion). A tongue blade, or Fox plane, can be used to determine whether an occlusal cant is present, because unilateral vertical maxillary excess and mandibular asymmetries are usually associated with an occlusal plane cant;[63] and this is the reason why most asym-

metries cannot be effectively treated with single-jaw surgery. Typical orthodontic diagnostic records rely heavily on the profile view, because the lateral approach is derived from traditional diagnoses based upon cephalometric radiographs; however, patients are more aware of their aesthetic presentation from the frontal view. Additional diagnostic records may include a PA cephalometric radiograph, a submentovertex radiograph, and an accurate facebow transfer with casts mounted on a semiadjustable articulator.[65–73]

The orthodontic management of patients with asymmetries does not differ a great deal from the typical orthognathic surgical patient. Good communication and a team approach during all phases of treatment are essential. Once the diagnosis and treatment plan have been established, the presurgical orthodontic phase is initiated, and basic principles of presurgical orthodontics must be observed. All tooth movements that may compromise stability should be avoided, especially if the intended movement may be more easily accomplished with the surgical movements of bony segments (e.g., transverse maxillary expansion is more effectively managed with two-piece segmental maxillary surgery rather than orthodontic maxillary expansion, especially in the adult patient). Dentoalveolar decompensation in the maxillary arch must take into account the postsurgical position of the maxillary central incisor. Maxillary AP movements, as well as posterior impactions, have the greatest effect on the upper incisors with regards to AP positioning and dental torque, respectively. Dentoalveolar decompensations in the mandibular arch must observe the anatomic limits of the outlines of the bony symphysis. It must be remembered that the morphogenetic pattern in patients with maxillomandibular discrepancies results in variable abnormal bony architectural changes, and therefore, cephalometric norms cannot be applied to all asymmetries. In addition, there should not be any compromise in the presurgical orthodontic treatment plan that might severely limit the final orthodontic and surgical treatment outcomes. The need for extractions should be assessed and performed if indicated, and dental impressions and early model surgery can be helpful in confirming that the presurgical goals are appropriate and/or that the treatment objectives have been achieved.

Common orthodontic problems in the asymmetry patient include improper buccal root torque in the upper arch, improper arch coordination (especially when AP movements are planned), and a lack of sufficient incisor overjet that can limit placement of the jaws into an ideal class I canine and molar occlusal relationship. The decision to extract teeth is often difficult, and perhaps the first issue that must be addressed is whether there is significant dental crowding or a Bolton discrepancy. This determination is based primarily on the ideal planned final position of the upper and lower incisors in the basal bone of the maxilla and mandible. The relationships of the upper incisors to the sella-nasion (SN) (104 degrees), palatal plane (104 degrees), and nasion point A (NA) line (4 mm and 22 degrees) are excellent indicators of whether these teeth require decompensation. It must be kept in mind that posterior impaction of the maxilla decreases the torque, or protrusion, of the upper incisors, so the orthodontic treatment must take this into consideration and not retrocline the incisors because they will be further uprighted by the surgical movement. In addition, large unilateral vertical changes, such as maxillary impaction or down-grafting, will rotate the midline of the maxillary incisors. However, when this is anticipated, the upper incisors should be maintained in a slightly proclined angulation. As a surgical treatment objective (STO), the position of the upper incisor, and a point relative to N-perpendicular, can be used as reasonable cephalometric references. Therefore, dental crowding as well as the ideal position of the upper incisors within basal bone of the maxilla will ultimately determine the need for extractions. Upper second bicuspid extractions are preferred when minimal incisor decompensation is required anteriorly; and maxillary first bicuspids are extracted when the upper incisors require greater degrees of decompensation, such as in class III cases. If mild crowding is present (≤4 mm) and no dentoalveolar decompensation is needed, a nonextraction approach is acceptable, and some interproximal reduction (IPR) may be required. If significant incisor decompensation is required, the cephalometric correction must be factored into the assessment of the degree of crowding. For every 3 degrees of change in the angulation between the lower incisor and the mandibular plane, it is necessary to add or subtract 2.5 mm to the measured clinical crowding. In patients with class II malocclusions, cephalometric correction most often adds to the clinical crowding assessment because the lower incisor typically requires a more upright repositioning, whereas with class III malocclusions, cephalometric correction usually alleviates this crowding. In other words, cephalometric correction takes into account the goals for the lower incisor into the crowding assessment; moreover, it helps to decide which teeth should be extracted. When the measured crowding in the lower arch is moderate (5–9 mm), mandibular second bicuspids should be extracted, which will result in alignment of lower incisor angulation and complete closure of the extraction spaces. When crowding is severe (>10 mm) after cephalometric correction, mandibular first bicuspids should be extracted to allow alignment and proper positioning of the lower incisor. The rationale is that when two bicuspids are extracted, an average of 14 mm of total arch space is created. If the total crowding is 10 mm including cephalometric correction, then 4 mm of space is left. These 4 mm are used by the forward movement of the posterior teeth as lost anchorage during the alignment and retraction of the lower incisors. Maximum decompensation is often required with minimal clinical crowding, therefore, requiring first bicuspid extractions. If crowding exceeds 14 mm, extractions alone are not sufficient to alleviate the crowding and achieve an ideal position of the lower incisors. IPR may help to create another 3 to 4 mm of total arch space to accomplish these goals simultaneously.

Patients with hyperdivergent facial forms who present with an asymmetry may require differential maxillary impac-

tion; in such cases, there is a need to flatten the curve of Spee in both arches before surgery. The curve of Spee is often different from left to right, and flattening the curve allows for maximum intercuspation to be achieved between the anterior teeth and the bicuspid teeth, with the creation of minimum posterior open bites. If the posterior maxilla is intentionally overimpacted when relapse is expected, the result will be a posterior open bite, and no vertical elastics are placed distal to the bicuspids for the first 8 weeks after surgery. The goal should be to achieve maximal intercuspation, although in the patient with a facial asymmetry, the occlusion on one side may be more open than on the other; it is usually the hypoplastic side that remains slightly open, and generally teeth can be extruded with vertical elastics postsurgically. An open bite of 2 mm or less is acceptable, and occasionally desirable, because some settling and relapse occur after surgery; and in general, no orthodontic forces should be applied in the direction of the potential surgical relapse. Patients with hypodivergent asymmetrical faces typically require mandibular advancement with an increase in the lower anterior facial height. The original malocclusion is often characterized by an excessive overbite, overjet, and accentuated curve of Spee, which may be different from side to side. The maxillary curve of Spee should be flattened and the ideal position of the upper incisors achieved. No attempt should be made to level the lower curve of Spee because forward movement of the mandible to an ideal overbite and overjet will increase the lower anterior facial height and the curve of Spee is maintained. If the lower mandibular plane angle has been maintained during mandibular advancement, this may result in a posterior open bite in the bicuspid or molar region. The increased interocclusal space with this postsurgical tripod occlusion will allow leveling of the mandibular curve of Spee with minimal effort after surgery, using a flexible braided wire in the lower arch as vertical intercuspation elastics are applied to extrude the lower posterior teeth and close the posterior open bite. A class II or class III vector may be incorporated in the postsurgical period in order to achieve optimum occlusal intercuspation. The maxillary arch should be stabilized with a heavy rectangular arch wire. Postsurgical orthodontic procedures are usually completed within 6 to 8 months after surgery if all other phases of treatment have been successful. Vertical elastics may be directed by the orthodontist depending on the occlusion and the unique differences of the hyperplastic or hypoplastic side of the face. Severe orthognathic asymmetries are often difficult from an orthodontic standpoint owing to the presence of unilateral differences in a hyperplastic jaw, with a contralateral hypoplastic dental and skeletal compensation.

Surgical Options

In rare cases, facial asymmetries may be treated in a single jaw, although generally, asymmetrical growth results in compensation of the teeth, alveolus, and the other jaw and the rest of the facial skeleton and soft tissues. Furthermore, this compensation may be significantly different from side to side and will require different orthodontic mechanics. Facial asymmetry may be improved from an aesthetic standpoint without standard orthognathic surgery, using a variety of techniques including an inferior border ostectomy or recountouring procedure, inferior border augmentation, and genioplasty procedures. The aesthetic impact of an asymmetry involves both the hard and the soft tissues, and commonly, the zygoma and periorbital and nasal structures may be involved, as well as the adjacent soft tissues, such as the salivary glands, muscles, and adipose tissue, with quantitative differences from side to side. The expectations of the patient as well as the surgical possibilities should be discussed at length, because asymmetrical deformities can rarely be corrected completely. Most patients notice horizontal or transverse facial discrepancies more often than vertical asymmetries (e.g., maxillary occlusal cants are difficult to detect clinically). Horizontal asymmetries resulting in maxillary dental midline, chin, and nasal deviations are typically very obvious clinically. Asymmetries in vertical facial height are less apparent clinically, and in severe cases, such as HFM or other syndromes, soft tissue augmentation and even free vascularized tissue may be necessary for correction.

The oral and maxillofacial surgeon should make a note of even minor anatomic asymmetries such as orbital, nasal, and upper lip position; maxillary dental midline position; smile arch; amount of gingival display on each side of the midline; cheek mass and malar prominence; the presence of dimples or clefts; mandibular dental midline position; mandibular deviations on opening or protrusion; TMJ articulation, rotation, and translation; gonial angle position; and cervical and neck anatomy. All of these should be documented. The surgical procedure should be selected based upon the etiology of the asymmetry with a healthy concern for stability and relapse in these patients. For example, when correcting a maxillary cant, vertical impaction is more stable than vertical down-grafting, and often the discrepancy may be corrected by a combination of both types of vertical maxillary movements. Severe asymmetries with a short mandibular ramus height may require an extraoral inverted-L osteotomy with bone grafting and rigid internal fixation. This technique releases the pterygomasseteric sling and provides excellent access to the hypoplastic ramus for bone grafting and application of rigid fixation. Vertical changes of less than 6 to 8 mm may be treated via an intraoral approach with a bilateral sagittal split osteotomy (SSO) and rigid fixation. Rotational movements of the mandible produce proximal segment flaring on the side that the mandible rotates away from and ramus collapse on the side that the mandible is rotating toward. Proximal segment flaring may require bony modification within the SSO site to allow a more appropriate relationship of the proximal and distal segments. Some surgeons prefer a unilateral vertical oblique ramus osteotomy (VRO) (on the side that the mandible rotates toward) combined with a unilateral SSO (on the side that the mandible rotates away from). This combination surgery usually requires intermaxil-

lary fixation, which may be beneficial in asymmetrical cases. With current techniques, monocortical plate and screw fixation can be applied to a VRO, using intraoral right-angle instrumentation, a transbuccal trocar technique, or an extraoral, or endoscope-assisted, approach. Conversely, intermaxillary fixation is often necessary, and even beneficial, because alignment of the segments with rigid fixation is not always possible and the soft tissue tension will attempt to retract the mandible to the original asymmetrical position.

As mentioned previously, a treatment plan should be established with an accurate clinical examination, cephalometric analysis, and model surgery, and the surgical procedure should be executed efficiently and with minimal patient morbidity, and the issues of relapse, stability, and mode of fixation should be determined before surgery and discussed with the patient. The first, and perhaps most important, treatment plan decision the surgeon must make is the ideal position of the maxillary central incisor. This is a critical decision and will essentially determine the three-dimensional position of both the maxilla and the mandible. The surgeon must consider dental midline discrepancies, incisor proclination or retraction excesses, occlusal plane abnormalities, smile arch aesthetics, dental/gingival display at rest and upon smiling, and upper and lower lip support. An intermediate splint is useful in repositioning the maxilla, assuming that an accurate facebow transfer and model surgery have been performed. The intermediate splint is only one method used to align the maxilla and correct the asymmetry, although surgical experience and a thorough appreciation of aesthetics and facial symmetry are often more valuable tools for the surgeon. The maxilla must be placed in the proper and most symmetrical position, and then consideration should be given to the occlusion in relation to the uncut mandible; this position should be evaluated in relation to the overall treatment plan. The maxillary position can best be measured using a combination of an external landmark reference (nasal bone pin), Fox plane, upper lip position, as well as internal reference marks on the anterior maxillary wall. The intermediate splint helps to position and maintain the maxilla in the correct position during application of rigid fixation. The concept of an intermediate splint is based upon proper condylar position which is difficult to similate because functional condylar position in an awake, upright patient is not the same as it is in a supine, paralyzed patient. Furthermore, asymmetrical problems often originate from abnormal condylar (TMJ) disorders, and many patients with facial asymmetries do not have bilateral symmetrical condylar rotation and, therefore, exhibit a great deal of muscular compensation during jaw posturing. If an intermediate splint does not seem to position the maxilla properly in all three dimensions, the surgeon should consider other references to determine facial symmetry; however, no specific measurements can replace surgical experience. In bimaxillary surgery, the osteotomies are performed in the mandible first, without completion of the osteotomy until after maxillary surgery is complete and rigid internal fixation has been applied to the repositioned maxilla. This surgical sequence prevents the creation of excessive forces on the new maxillary position via vibration of the drill or saw used for the mandible with a bite block in position and the forces transmitted to the maxilla. In addition, some surgeons believe that this sequence seems to expedite the overall surgery. An alternative surgical sequence completes the mandibular surgery with rigid fixation before the maxillary surgery, with the premise that it is more appropriate to reposition a mobile structure (the mandible) to a more stable structure (the maxilla and cranium), but this plan requires the precise predetermination of both the mandibular and the maxillary positions.

Mandibular ramus surgery is often difficult in cases of asymmetry because the ramus and body of the mandible are usually deformed and may be hypoplastic with a limited range of motion. Limited surgical access and a smaller soft tissue envelope create a difficult surgical challenge. If the mandible is cut first but not split, the osteotomies are made and a moist sponge is packed into the wound for hemostasis while the maxillary surgery is completed. The mandible is later split and moved to the proper position with the maxilla and held in the proper occlusion with an occlusal wafer, or final splint, with intermaxillary fixation wires. Rigid fixation of the mandible can be achieved with either bicortical positional screws or monocortical plates and screws, assuming that condylar position is appropriate. In large horizontal rotations of the mandible, bicortical screws cannot be placed as true compression screws (lag screws) without significant torque placed on the condyles. Modifications of the lingual cortical plate, and selective grinding of the bony interferences, can be helpful to increase the bony contacts. In most cases, monocortical plates and screws provide adequate stability, without compression of the inferior alveolar nerve, or torquing of the condyles; these can be placed transorally without the need for a transbuccal trocar used with bicortical screw fixation.

Adequate surgical mobilization of the maxilla and mandible is a key step that must be performed during bimaxillary surgery. The mandible and maxilla should be mobile enough to be positioned passively without tension on the soft tissues. This can be a significant concern because jaw repositioning can create tension and restriction in the pterygomasseteric sling, and and if the mandibular segments are stretched into position under tension, long-term stability may be compromised. A balance must be achieved between adequate reflection of periosteum and loss of vascularity and maintenance of an adequate connective tissue envelope. Therefore, in severely hypoplastic mandibles with only rudimentary condyles, considerations for condylar reconstruction should include extraoral procedures of the mandibular ramus with costochondral grafting, or in some situations, distraction osteogenesis may be more appropriate in order to achieve soft tissue expansion with the gradual bony movements. Adjunctive simultaneous soft tissue procedures may be considered after bony repositioning of the maxilla and mandible and occlusal correction orthognathic surgery and internal rigid

fixation. Alignment of the chin, nose, and malar complex should be performed after the functional anatomy of the maxilla and mandible has been established. Although simultaneous hard and soft tissue surgery has multiple advantages, it may not always be feasible, and some surgeons choose to wait to see the unpredicatble soft tissues changes after bimaxillary surgery before addressing the soft tissue asymmetries.

Facial Asymmetry Case Examples

Case 1

This is a 20-year-old woman with facial asymmetry resulting from trauma who required bimaxillary surgery (Figure 59-7). She has a chief complaint of difficulty eating and chewing, with a history of chronic myofascial pain and masticatory dysfunction. She sustained a jaw fracture as a young child without treatment, and she has experienced the development of gradual facial asymmetry. She had an orthodontic consultation and it was determined that orthodontic treatment alone or single-jaw surgery would be insufficient to correct her asymmetry.

The physical examination revealed the obvious facial deformity, more pronounced from a basal view, which can often better demonstrate the deformity; also upon biting on a tongue blade, occlusal cant can be demonstrated (Figure 59-8). She was normocephalic and asymmetrical, with a short face and a jaw deviation to the right (Figure 59-9). The TMJ articulation, rotation, and tramslation function was normal without clicking or popping. The maximum incisal opening was 56 mm, with a deviation to the right. The mandibular midline was 7 mm to the right, the overjet was 5 mm, the overbite was 2 mm; there was excessive lower incisor dental display. She had a right buccal cross-bite, an anterior cross-bite, maxillary hypoplasia, a canted maxilla, and a short mandibular condyle (Figure 59-10).

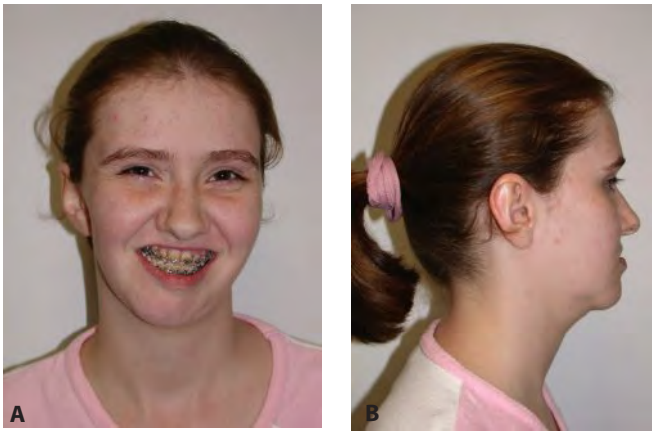


FIGURE 59-7. **A,** A 20-year-old who had an untreated jaw fracture in childhood; note facial asymmetry. **B,** Profile view shows midface deficiency and relative mandibular prognathism.

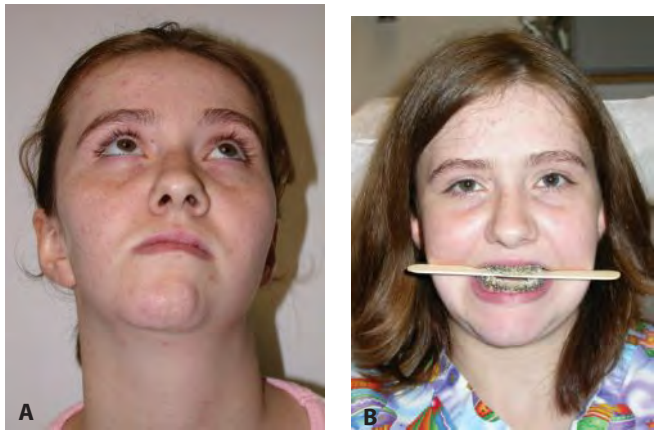


FIGURE 59-8. **A,** Worm's-eye view shows asymmetry with chin deviation to the right. **B,** Tongue blade demonstrates the occlusal cant plane relative to the normal orbital level.

The presurgical workup included a three-dimensional CT scan, cephalometric and panoramic radiographs, and facebow mounting for fabrication of an accurate intermediate splint and predictive model surgery (Figures 59-11 to 59-13). The Erickson model block was used to measure the vertical changes in the maxilla and to fabricate an intermediate splint to assist in positioning the maxilla (Figures 59-14 and 59-15). The surgical plan included a Le Fort I advancement of 4 mm with a differential maxillary down-grafting of 4 mm on the right



FIGURE 59-9. Posteroanterior (PA) cephalogram shows decreased right facial height and severe asymmetry.



FIGURE 59-10. Occlusal analysis shows asymmetry and midline discrepancy of both jaws.

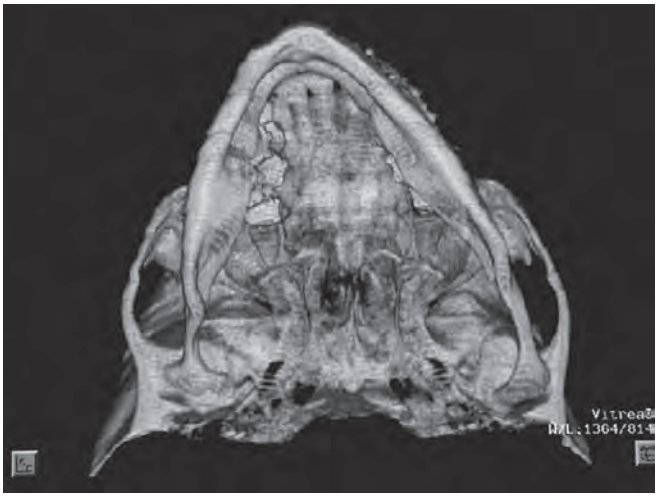


FIGURE 59-11. This three-dimensional CT clearly shows the asymmetry and deviation.



FIGURE 59-12. Lateral cephalogram shows relative mandibular prognathism despite the condylar fracture, although there is an inferior border discrepancy.

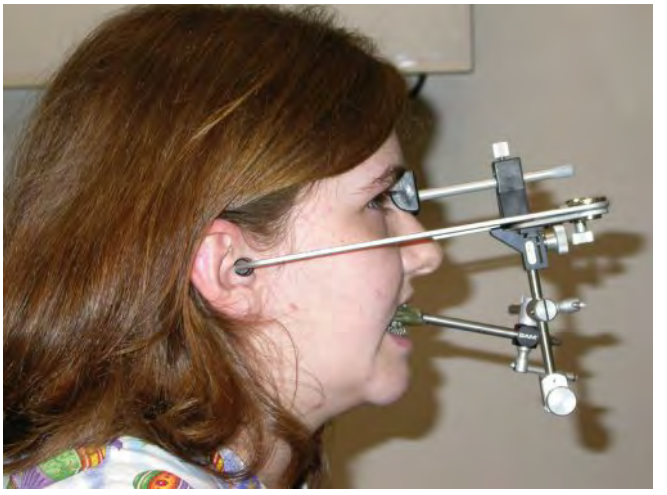


FIGURE 59-13. A facebow transfer is valuable but may be difficult in severe asymmetries with an abnormal ear and external auditory canal (EAC).



FIGURE 59-14. Mounted models on Erickson block with reference lines help determine surgical movements and splint fabrication. Note midline discrepancy in the midline reference lines on the bases of the casts.



FIGURE 59-15. Lateral view of mounted models shows the planned asymmetrical maxillary osteotomy movement.

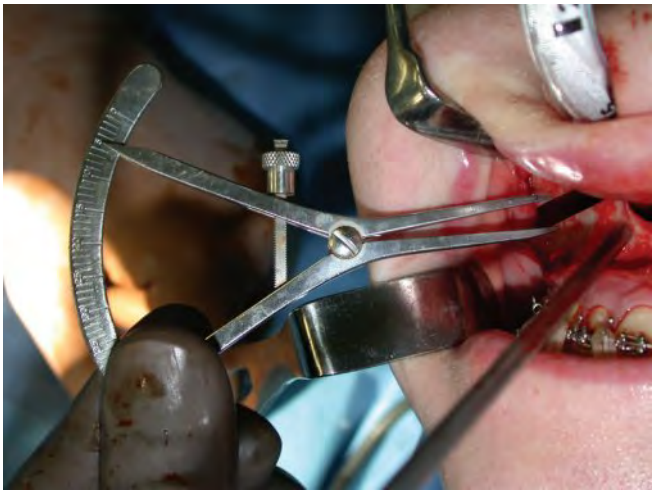


FIGURE 59-16. Intraoperative measurement confirms correct positioning with the intermediate splint; compare maxillary gap to Figure 59-15.

side (Figure 59-16). The resultant maxillary osteotomy gap was grafted with allogeneic tibial bone that was mortised in place and fixated. This surgical plan was determined with model surgery to level the cant of the maxillary occlusal plane. A Steinmann pin was placed in the nasal bones as an external vertical reference point; in addition,



FIGURE 59-17. A modified Fox plane can be used as a reference to evaluate leveling of the maxilla in reference to the infraorbital rims.



FIGURE 59-18. **A,** Postoperative lateral view with animation. **B,** Postoperative frontal view with animation.

a Fox occlusal plane was used to evaluate the level of the maxilla in relation to the infraorbital rims during surgery (Figure 59-17). Multiple methods of evaluating symmetry are helpful in achieving a good result, and intermediate splints, internal and external reference landmarks, and careful clinical inspection from different views are all valuable surgical skills for bimaxillary jaw surgery in the patient with a facial asymmetry. The unsplit, intact mandible is also a key reference for determining any changes in maxillary position. In this case, after the maxilla was correctly positioned and the occlusal plane leveled, the mandible was split and repositioned to the corrected plane of occlusion. A bilateral SSO was cut before the Le Fort procedure but was not split, and as mentioned, this is a common sequence for most surgeons. Consideration was given toward a VRO on the left, but the rotation seemed favorable and the fixation was more stable than anticipated. Most patients with facial asymmetry have horizontal as well as vertical mandibular hypoplasia and require lower facial advancement more often than reduction. A genioplasty was performed to advance the chin point 4 mm and to level the deviation of the symphysis region. After the orthognathic phase of surgery, intraoperatively the endotracheal tube was switched from a nasal to oral intubation for the perfomance of a simultaneous rhinoplasty procedure. A standard internal rhinoplasty was performed to narrow the nose, refine the tip, and reduce the nasal dorsum. Often, facial asymmetry affects multiple facial subunits, and the nose may appear to be asymmetrical or deviated in relation to the maxilla, mandible, and chin, so attention to detail is required in order to create overall midface symmetry.

At 3-month follow-up, the postoperative results were satisfactory to the patient and surgeon (Figure 59-18), the occlusion was aligned and stable, and finishing orthodontics were completed without difficulty.

Case 2

This is a 17-year-old young man with a large open bite and right laterognathism, with posterior vertical maxillary excess, dental crowding, a class III molar relationship, dentofacial asymmetry, and a mandibular prognathism (Figures 59-19 to 59-22). He complained of lip incompetence; difficulty eating, chewing, and biting food; nasal obstruction; and xerostomia. His father had a history of mandibular prognathism as well. He denied previous facial trauma, and the orbital rims were symmetrical, but the right ear was slightly lower than the left. The TMJs articulated well and there was no facial pain. The mandibular midline was 6 mm to the right of the facial midline, and the chin was excessive vertically and 6 mm to the right of the mandibular midline. Also, there is a mild transverse deficiency of the posterior maxilla and a wide posterior mandibular dimension.

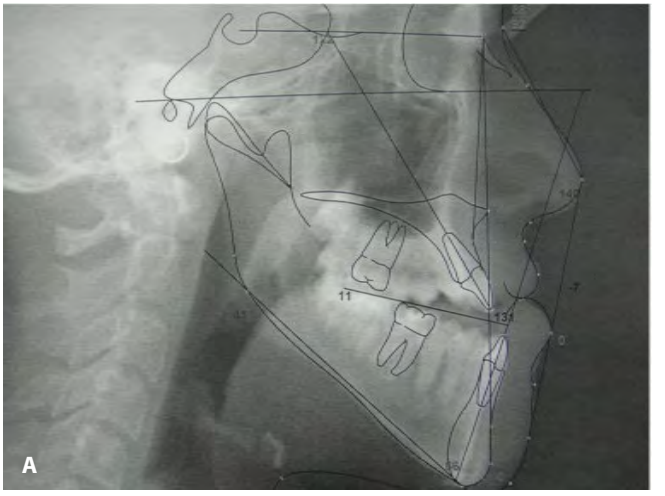
The treatment plan began with an extraction of teeth numbers 1, 16, 17, 18, 2, 4, 13, 21 and 28 before presurgical orthodontics, which was performed to level, align, and decompensate the dentition in preparation for a possible segmental Le Fort I osteotomy with posterior expansion, a bilateral SSO, genioplasty with vertical reduction and rotation, and possible midline mandibular osteotomy with mandibular narrowing. Presurgical records included



FIGURE 59-19. **A,** Initial frontal view. **B,** Initial profile view.



FIGURE 59-20. Initial occlusion.



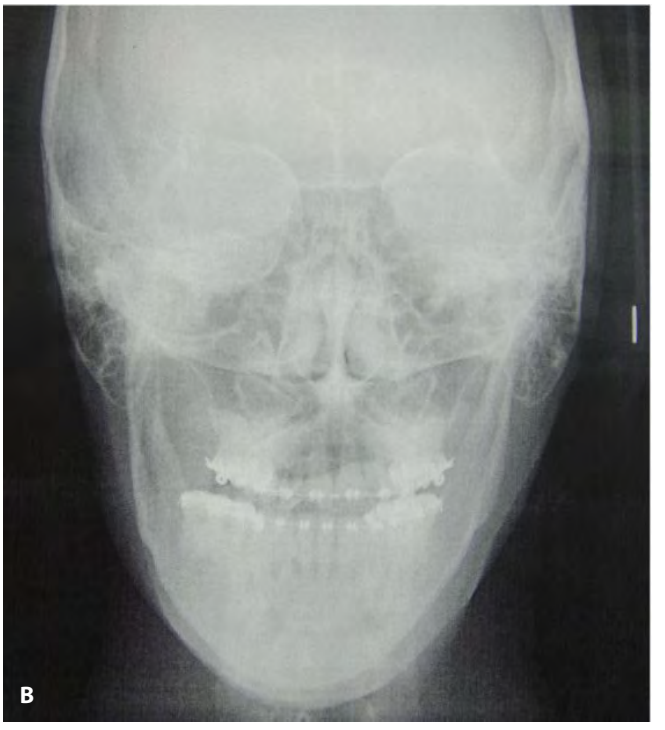


FIGURE 59-21. **A,** Presurgical cephalometric analysis. **B,** Presurgical PA cephalogram.

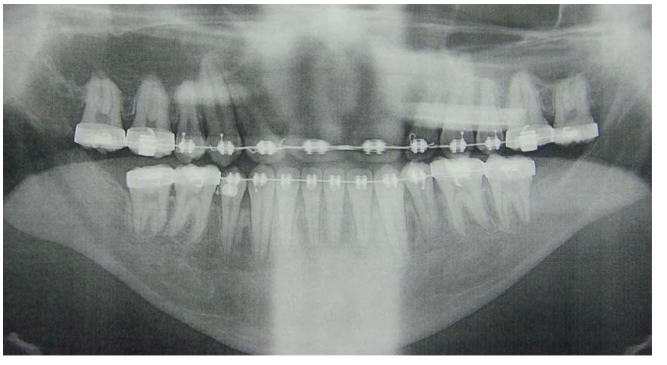


FIGURE 59-22. Initial Panorex.

mounted dental models, a facebow transfer, and detailed radiographs. A three-dimensional CT scan would have also been valuable. The surgery consisted of a bilateral SSO that was cut, but not split until after the maxilla was positioned, and the anterior midline mandibular osteotomy was performed. The maxilla was advanced 6 mm, using an intermediate splint to position the maxilla, which was fixated rigidly with a 6-mm prebent advancement plate at the piriform rims, and two additional plates in the posterior maxillary buttress regions. During surgery, both internal and external references were used to properly position the maxilla vertically, and the use of prebent plates assisted in the proper AP positioning of the maxilla. Model surgery had been performed with a midline mandibular osteotomy with plans to narrow the excessive mandibular width and correct the mandibular asymmetry. The chin was also extremely long and deviated to the right side; therefore, a vestibular incision was used and vertical reference marks were made in the symphysis bone. A low geniotomy wedge resection was performed and a midline osteotomy was created between the mandibular central incisors (Figure 59-23). The SSO segments were then separated, and the mandible was secured into the customized splint. The occlusion was placed into intermaxillary fixation. The anterior midline osteotomy was first fixated with a four-hole noncompression titanium miniplate. These four screws were placed apical to the anterior tooth roots (Figure 59-24). A 5-mm wedge of bone was then removed and the chin point was repositioned with two cross-shaped titanium plates. The inferior chin point repositioned 5 mm to the left, and then the SSO was fixated with monocortical plates bilaterally. The teeth articulated into the splint well, and then the nasal septum was sutured to the anterior nasal spine and an alar base cinch suture technique was performed; finally, the anterior maxillary vestibular incision was closed in a V-Y fashion.

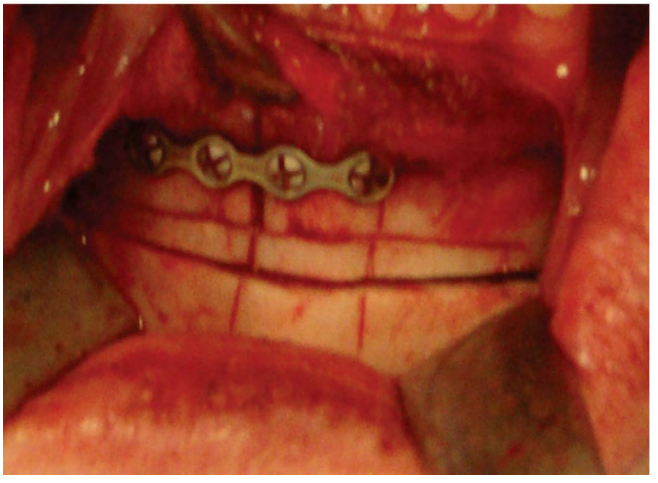


FIGURE 59-23. Genioplasty with wedge excision and midline osteotomy.

The postoperative course was without complication, and at 1 month, the splint was removed. The occlusion remained stable at 1 year follow-up, and the facial symmetry was excellent (Figures 59-25 to 59-29). This case is an example of severe developmental, nontraumatic orthognathic asymmetry, which can be challenging for the orthodontist and surgeon because the orthodontic preparation may be different for the left and right sides. If the asymmetry is mild, it is tempting to inappropriately manage the problem with single-jaw surgery. Many such facial asymmetries are often undertreated, when in fact they may require overcorrection to account for some relapse potential. In fact, each case must be treated as an individual because each asymmetry is unique and requires careful attention to detail in the diagnotic and treatment phases of management.

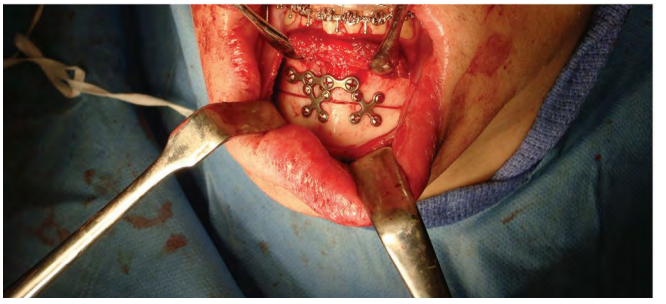


FIGURE 59-24. Genioplasty fixation with plates and screws.



FIGURE 59-25. Postsurgical profile view.

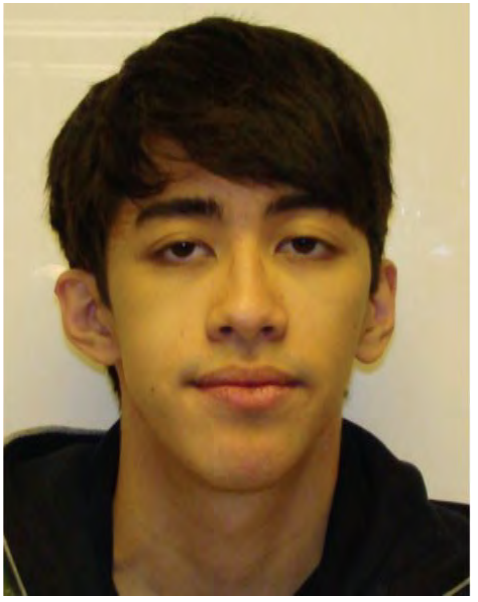


FIGURE 59-26. Postsurgical frontal view.



FIGURE 59-27. Final occlusion.

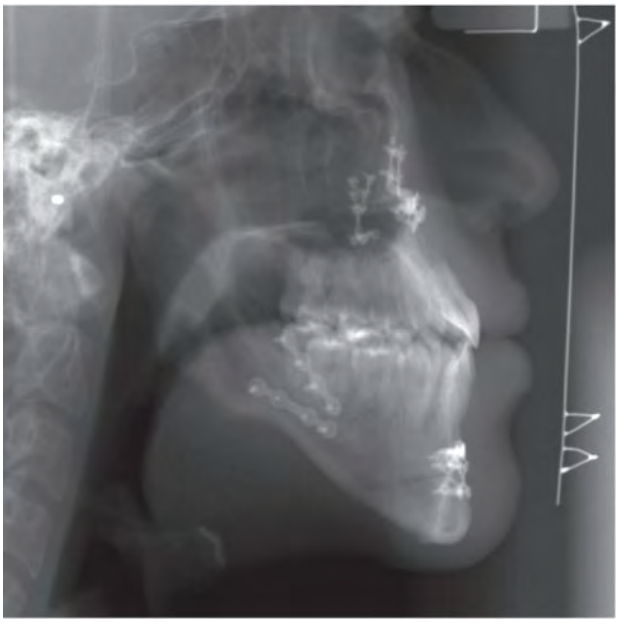


FIGURE 59-28. Final lateral cephalometric view.

CONCLUSIONS

Correction of asymmetrical orthognathic deformities often requires surgery of both the maxilla and the mandible. Combining osteotomies of the maxilla and the mandible is more complicated than single-jaw surgery and is perhaps associated with increased morbidity, but the surgical options are more extensive and the postoperative results are improved with less potential replase. Bimaxillary jaw surgery does not result in twice the morbidity of single-jaw surgery and is typically tolerated as well as single-jaw surgery with similar periods of postsurgical convalescence. The indications for bimaxillary surgery include severe facial asymmetries and deformities that cannot be adequately managed with one-jaw



FIGURE 59-29. One-year follow-up records.

surgery, skeletal deformities of both jaws, unfavorable planned surgical movements that are prone to relapse, and complex three-dimensional movements for which single-jaw surgery would represent a functional and cosmetic compromise. Skeletal dentofacial asymmetries may develop from a primary etiologic event but usually present with secondary compensations of both the hard and the soft tissues of the face; such an asymmetry is an excellent indication for bimaxillary surgery. Current surgical techniques have reduced the morbidity and hospital length of stay and have improved the overall functional and aesthetic surgical outcomes. In addition, the use of three-dimensional imaging and computerized treatment planning has recently been used to improve accuracy and predictability of the diagnosis and treatment plan; this reinforces the concept that comprehensive facial analysis and attention to detail in the treatment of facial asymmetries are critical to a successful outcome.

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CHAPTER

Soft Tissue Changes Associated with Orthognathic Surgery

Stephen Schendel, MD, DDS, Richard Jacobson, DMD, MS, and Dror Aizenbud, DDS

The analysis of facial soft tissues and the associated aesthetic outcomes in orthognathic surgery is the most challenging and unpredictable component in the orthodontic-surgical treatment planning process. There remains a need for an ideal procedure capable of assessing and predicting accurately the entire soft tissue facial envelope in a three-dimensional manner. The final aesthetic facial appearance and the establishment of a functional and stable occlusion are both equal and important goals in orthognathic surgery. Soft tissue changes related to the skeletal maxillary and/or mandibular movements have been classically studied in the lateral profile view from cephalometric evaluation and comparative ratios of soft to hard tissue movements established based upon mean values of responses and on a simple correlation of one variable with another or using linear regression equations.[1–18] In addition, these ratios have been calculated from linear changes limited to specific two-dimensional cephalometric landmarks in the midsagittal plane. This method is restrictive, and its accuracy and reliability in predicting realistic profile changes is questionable because such an approach does not take into account the more complex geometric three-dimensional interactions of highly variable individual parameters such as soft tissue tension, thickness, muscle strain, posture, and tonus. In addition, such technical parameters as surgeon expertise and soft tissue closure are unpredictable variables that are difficult to account for in the predication of final outcomes.

Traditionally, several methods for the evaluation and prediction of facial soft tissue outcomes have been described in the literature and are separately reviewed in this chapter: (1) the lateral cephalometric "line drawing" tracing prediction (manual and computer-assisted),[19–21] (2) the photographic prediction,[22,23] (3) the computerized video (digital) imaging (video-cephalometrics) prediction,[24–38] and (4) the three-dimensional computer-assisted prediction utilizing image fusion.[39–51]

LATERAL CEPHALOMETRIC PREDICTION (MANUAL AND COMPUTER-ASSISTED)

The cephalometric soft tissue and skeletal analysis for planning orthodontic treatment has been reported as early as the 1950s and rapidly became an irreplaceable tool in the evaluation and treatment planning process for patients who require orthognathic surgery.[52] The literature is replete with reports describing different methods of cephalometric analysis, planning and prediction based on specific landmarks repositioning in response to determined skeletal movements.[1–21,24–38] Three visual treatment objective methods have traditionally used: (1) the "cut-and-paste" technique, which involves manual cutting, moving, and repositioning of cephalometric acetate overlay tracings to obtain the desired aesthetic profile as well as a class I occlusal relationship,[19,21] (2) the computer-assisted technique, which generates soft tissue prediction profile tracings by using software programs from the lateral cephalometric radiograph,[20] (3) the computer-assisted photographic imaging technique, which integrates digital images of the patient with the lateral cephalogram to predict a two-dimensional image simulation of the suitable skeletal and soft tissue profile outcome.[24–38] In all three techniques, the soft tissue profile is adjusted and changes are planned according to previously reported soft tissue–to–bone ratios. Factors influencing accuracy of such a prediction tracing include understanding of profile planning, accuracy of surgery, degree of relapse, accuracy and reproducibility of tracings, soft–to–hard tissue ratio, reliability of cephalometrics

and of photography, and variation in lip posture limitation of two-dimensional planning. Authors comparing the accuracy and predictability of the manual versus the computed technique have found quite similar values in mandibular advancement planning results with only a few points differing between prediction and outcomes of cephalometric tracings.[5,7] Conversely, the hand technique has been found more accurate in the maxillary and bimaxillary prediction of soft tissue changes, particularly in the lip area.[5]

Historically, the first studies on soft tissue changes after orthognathic surgery were directed to the lower lip and chin after mandibular procedures.[53] Conversely, it was not until the mid-1970s that the first reports on soft tissue changes associated with maxillary surgery (advancement, intrusion, retrusion, or bimaxillary) were published.[2,4,11,17] This correlates with the evolution of orthognathic surgery from the lower jaw to the upper with increasing knowledge and skill.

Mandibular Surgery

The lower lip, the mentolabial fold (MLF), and the neck-chin angle are the anatomic regions most strongly influenced by the anterior or posterior surgical repositioning of the mandible.

Mandibular Advancement

The main changes affect the lower lip with a global reduction in thickness, lengthening, and straightening, as well as the chin area, with a decrease in the lower labial sulcus or MLF depth, whereas changes on the upper lip (minor anterior movement) are usually negligible, temporary, and limited only to patients with class II deep-bite deformities. The lower lip also presents the most variable soft tissue–to–bone ratios, ranging from 0.38:1 to 0.80:1, contrary to the chin ratios, which are almost invariable in a 1:1 relationship, probably due to the tight attachment of the mental soft tissue to the underlying skeletal bases[1,8,11,13,14,54] (Table 60-1).

Mandibular Setback

With mandibular horizontal retropositioning, the lower lip has been found to become more everted and shorter, with a closure of the MLF, whereas the upper lip can sometimes be displaced posteriorly with a discrete opening of the nasolabial angle. The Li:L1 (labi inferiorus:lower incisor) ratio was approximately 0.6:1, and in the chin region, Pg' (pogonion') followed Pg and the B point in an almost 1:1 ratio. These ratios have been consistently identified in several studies regardless of the magnitude of the setback movement[1,20] (Table 60-2).

Genioplasty

The most unpredictable points when planning a genioplasty are the superior and inferior vermilion borders, as well as the Pg' and Me' (menton).[1,5,15] Also, studies have shown a difference, although slight, between the sliding genioplasty and the alloplastic genioplasty (Tables 60-3 and 60-4). The relationship between the soft tissue and the bone is worst with the setback genioplasty in which about half of the bony movement is lost because of the soft tissue response, which often can lead to a less than desirable result or a flat lower lip–chin relationship lacking the normal S curve (Table 60-5). Vertical elongation of the chin also demonstrates a poor tissue-to-bone response (Table 60-6).

Maxillary Surgery

Soft tissue changes related to maxillary surgery have proved to be relatively more unpredictable than in the mandibular surgery, regardless of the type and the amount of skeletal movement produced. The nasolabial angle and the upper lip are the anatomic regions most strongly influenced and the most variable depending on the adjunctive soft tissue procedures and neuromuscular tone.

Maxillary Advancement

The main changes induced by maxillary advancement are located in the nasal region and the upper lip. The vermilion border of the upper lip (Ls) typically advances horizontally with a rotational and translational movement around the subnasale following the upper incisor (U1) in a soft tissue–to–bone ratio ranging from 0.33:1 to 0.9:1.[1,4,6–7,9,18,55] The latter high value has been reported when a V-Y technique and nasal cinch for the closure of the maxillary vestibular incision is utilized, which prevented the thinning and shortening of the upper lip and widening of the nose invariably associated with maxillary advancements.[3] The thinning of the upper lip may result in the loss of the visible vermilion border, which is aesthetically undesirable. The nasal effects imply a decreasing of the nasolabial angle, widening of the alar bases—which can be limited by using the alar base cinch suture procedure—deepening of the supratip break, and nasal tip elevation (1 mm for every 6 mm of superior movement of the U1).[56–58] In some patients, the tip elevation may produce positive aesthetic changes, such as the disappearance of the nasal hump that usually accompanies maxillary advancement or impaction[56–58] (Tables 60-7 and 60-8).

Maxillary Impaction

Surgical repositioning of the maxilla mainly affects the morphology of the nose and upper lip in both vertical and horizontal dimensions.[1,7,9,12,16,17] The decrease in the amount of the visible vermilion due to an inward rolling of the upper lip, a down-turning of the corners of the mouth, and a widening of the alar base of the nose are the main undesirable aesthetic effects associated with the decrease of the face height resulting from this procedure. Some authors have clinically observed that such changes are probably mainly related to postoperative muscular reorientation of the oronasal area rather than to the amount of skeletal movement. The ratios of vertical shortening in the upper lip related to the superior impaction of the maxilla have been reported to range from 0.2:1 to 0.4:1 by authors who did not perform an anatomic reorientation of the facial muscles around the

TABLE 60-1. **Soft Tissue Changes Associated with Mandibular Advancement**

Anatomic Structure	Landmarks	Ratio	Author(s)
Lower lip (H)	Li: Ii	0.62:1	Lines and Steinhauser[11]
		0.85:1	Talbott[72]
		0.38:1	Quast et al[73]
		0.56:1	Mommaerts and Marxer[14]
		0.43:1	Hernandez-Orsini et al[74]
		0.26:1	Dermaut and De Smit[75]
		0.66:1	Thuer et al[76]
		0.8:1	Ewing and Ross[77]
		0.6:1	Mobarak et al[78]
	ILS: B pt.	0.88:1	Keeling et al[79]
	Three-dimensional analysis	1.25:1	McCance et al[43,79]
Chin (H)	Pgs: Gn	1:1	Lines and Steinhauser[11]
	Pgs: Pg	0.94:1	Hernandez-Orsini et al[74]
		0.97:1	Quast et al[73]
		1:1	Thuer et al[76]
			Keeling et al[80]
			Ewing and Ross[77]
		1.03:1	Mommaerts and Marxer[14]
		1.04:1	Talbott[72]
		1.1:1	Dermaut and De Smit[75]
		1-1.1:1	Mobarak et al[78]
	Gns: Gn	0.95:1	Hernandez-Orsini et al[74]
		0.97:1	Quast et al[73]
	Mes: Me	0.87:1	
		0.97:1	Hernandez-Orsini et al[74]
	Three-dimensional analysis	1.25:1	McCance et al[43,79]
Chin (V)	Mes: Me	0.93:1	Mommaerts and Marxer[14]
Lower labial sulcus (H)	ILS: B pt.	0.86–0.95:1	Mobarak et al[78]
		0.88:1	Thuer et al[76]
		0.93:1	Hernandez-Orsini et al[74]
		0.97:1	Quast et al[73]
		1:1	Ewing and Ross[77]
		1.01:1	Talbott[72]
		1.06:1	Mommaerts and Marxer[14]
		1.19:1	Dermaut and De Smit[75]
Upper lip (H)	Ls: Li	–0.02:1	Hernandez-Orsini et al[74]
Menton	Mes: Me	0.92–1.04:1	Mobarak et al[78]

Gn=gonion, H=horizontal, ILS= inferior labial sulcus, Li= labi inferiorus, Ls= labi superiorus, Mes= soft tissue menton, Pgs= soft tissue pogonion, V=vertical.

oronasal area. Conversely, it was shown that when a functional orientation of the musculature is accomplished (V-Y closure of maxillary vestibular incision and alar base cinch suture),[4,100] the postsurgical lip closely approaches its preoperative morphology, presenting only a minimal and negligible shortening (10%).[15] This finding is of capital importance in planning and predicting the final suitable vertical maxillary impaction and the resultant lip-to-tooth relationship. After the posterior maxillary intrusion, the soft tissue chin (MLF and Pg') has been found to autorotate on the same arc as the bony chin on in a 1:1 ratio, with the MLF angle unfolded from the inferior labial sulcus[55] (Tables 60-9 and 60-10).

Maxillary Setback

The upper lip and the nasal soft tissues closely follow the skeletal movements posteriorly.[1,7,12] As observed in advancement and intrusion cases, there is also a widening of the alar bases with a tendency for the tip of the nose to rotate downward, with a resulting opening of the nasolabial angle.[59] This may in some patients result in a parrot's-beak nasal appearance. The same deformity can also be found after inferior repositioning of the maxilla and down graft. There is also a tendency for the upper lip to thicken after the maxillary posterior movement (see Table 60-6).

Bimaxillary Surgery

Different studies have confirmed that the soft tissue response associated with double-jaw surgery is similar to those found in single-jaw procedures, with the exception of vertical movement of the lower lip and chin.[23,27,29]

Photographic Prediction

First described by Henderson in 1974,[22] the photographic soft tissue profile planning technique has been progressively

TABLE 60-2. **Soft Tissue Changes Associated with Mandibular Setback**

Anatomic Structure	Landmarks	Ratio	Author(s)
Lower lip (H)	Li: Li	–0.75:1	Lines and Steinhauser[11]
		–0.93:1	Gjorup and Athanasiou[81]
		–1.02:1	Mobarak et al[78]
	Li: Pg	–0.5:1	Enacar et al[82]
		–0.6:1	Hershey and Smith[83]
		–0.69:1	Aaronson[84]
		–0.8:1	Gaggl et al[85]
	Three-dimensional analysis	–1:1	McCance et al[43,79]
Chin (H)	Pgs: Gn	–1:1	Lines and Steinhauser[11]
	Pgs: Pg	–0.83:1	Gaggl et al[85]
		–0.9:1	Hershey and Smith[83]
		–0.91:1	Gjorup and Athanasiou[81]
		–1:1	Robinson et al[86]
		–1.04:1	Mobarak et al[78]
	Three-dimensional analysis	–1:1	Gaggl et al[85]
Lower labial sulcus (H)	ILS: B pt.	–0.03:1	Gjorup and Athanasiou[81]
		–1:1	Robinson et al[86]
		–1.09:1	Mobarak et al[78]
	ILS: Pg	–0.93:1	Aaronson[84]
Upper lip (H)	Ls: Li	–0.2:1	Lines and Steinhauser[11]
	Ls: Pg	–0.2:1	Hershey and Smith[83]
		–0.32:1	Gaggl et al[85]

Gn=gonion, H=horizontal, ILS= inferior labial sulcus, Li= labi inferiorus, Ls= labi superiorus, Pgs= soft tissue pogonion.

abandoned owing to the difficulty in correctly obtaining a satisfactory superimposition of an enlarged photograph on the cephlometric radiograph and the evolution of more sophisticated techniques. Other techniques have used Moiré stereophotogrammetry. In general, these techniques are little used today.

TABLE 60-3. **Soft Tissue Changes Associated with Advancement Genioplasty**

Anatomic Structure	Landmarks	Ratio	Author(s)
Chin (H)	Pgs: Pg	0.57:1	Bell and Dann[87]
		0.75:1	McDonnel et al[88]
		0.67:1	Proffit and Epker[89]
		0.85:1	Bell and Gallagher[90]
		0.81:1	Gallagher et al[91]
		0.93:1	
		0.7:1	Epker and Fish[92]
		1:1	Krekmanov and Kahnberg[93]
Lip (H)	Li: Pg	0.44:1	Busquets and Sassouni[94]

H=horizontal, Li=labi inferiorus, Pgs=soft tissue pogonion.

TABLE 60-4. **Soft Tissue Changes Associated with Alloplastic Chin Implants**

Anatomic Structure	Landmarks	Ratio	Author(s)
Chin (H)	Pgs: Pg Silicone	0.60:1	Bell and Dann[87]
	Pgs: Pg Proplast	0.90:1	Dann and Epker[95]
		1:1	Proffit and Epker[89]

H=horizontal, Pgs=soft tissue pogonion.

Computerized Digital Video Imaging

Visual Treatment Objective (VTO) has been done by manual creation of simulated treatment tracings based on cephalometric radiographs. The first computer-based cephalometric systems appeared in the late 1970s.[60,61] At present, a number

TABLE 60-5. **Soft Tissue Changes Associated with Setback Genioplasty**

Anatomic Structure	Landmarks	Ratio	Author(s)
Chin (H)	Pgs: Pg	–0.33:1	Hohl and Epker[96]
		–0.50:1	Krekmanov and Kahnberg[93]
		–0.58:1	Bell[97]
	Interpositional	–0.75:1	Wessberg et al98

H=horizontal, Pgs=soft tissue pogonion.

TABLE 60-6. **Soft Tissue Changes Associated with Vertical Augmentation or Reduction Genioplasty**

Anatomic Structure	Landmarks	Ratio	Author(s)
Augmentation			
Chin (V)	Interpositional	1:1	Wessberg et al[98]
Reduction			
Chin (V)	Pgs: Pg	–0.26:1	Park et al[99]
	Mes: Me	–0.25:1	Hohl and Epker[96]
		–0.35:1	Krekmanov and Kahnberg[93]
		–0.40:1	Ewing and Ross77

Mes=soft tissue menton, Pg=pogonion, V=vertical

TABLE 60-7. **Soft Tissue Changes Associated with Maxillary Advancement with Nasolabial Muscle Reconstruction**

Anatomic Structure	Landmarks	Ratio	Author(s)
Upper lip (H)	Ls: Incisor (vertical maxillary excess)	–0.76:1	Schendel et al[17]
	Ls: Incisor (bimaxillary protrusion)	–0.66:1	
Upper lip (H)			
	Ls: Is	-0.67:1	Radney and Jacobs[16]
Upper labial sulcus (H)	SLS: Is	–0.33:1	
Nose (H)	Sn: Is	–0.33:1	
Nasolabial angle	NLA	Increase	

H=horizontal, Is=inferior stomion, Ls=labi superiorus, NLA=naso-labial angle, SLS=superior labial sulcus, SN=soft tissue nasion

of two-dimensional computer-assisted imaging systems allow image fusion of photographs, tracings, and radiographs. These computer-assisted programs permit rapid measurement and treatment planning of the skeletal and soft tissue. The validity and reliability of these systems are limited by their two-dimensional nature when dealing with a three-dimensional object.[62] The first reports on the video imaging techniques in the late 1980s dramatically changed the concept and the approach of the treatment planning and counseling in orthognathic surgery. These techniques enabled the superimposition of the lateral cephalometric radiographs and/or tracings on a two-dimensional patient's digital profile image, thus allowing the visualization as well as the manipulation of parts of the image in order to simulate the suitable aesthetic results according to the movement of the underlying maxillary bone.[25–27,29] Moreover, computer imaging provides a more realistic and tangible image of the treatment compared with the traditional "line drawing" cephalometric profile tracings of the surgical simulation. The two main advantages in using this technique are (1) the facilitation and enhancement of the communication between the orthodontist and/or the surgeon and the patient and (2) the capability of helping in the preoperative treatment planning by manipulating the images until the desired soft tissue profile is achieved. Studies have shown that 89% of surgical candidates who underwent this technique found their two-dimensional image predictions realistic and considered that the desired result was achieved.[31,33] Moreover, 83% of

TABLE 60-8. **Soft Tissue Changes Associated with Maxillary Advancement**

Anatomic Structure	Landmarks	Ratio	Author(s)
Upper lip (H)	Ls: Is	0.5:1	Dann et al[4]; Proffit and Epker[89]; Radney and Jacobs[16]; Bundgaard et al[100]
		0.62:1	Mansour et al[12]
		0.65:1	Rosenberg et al[101]
		0.74:1	Lin and Kerr[102]
		0.82:1	Rosen[103]
		0.9:1	Carlotti et al[3]
	SLS: Is	0.66:1	Hack et al[7]
		0.91:1	
	Three-dimensional analysis	1:1	McCance et al[43,79]
Upper lip (V)	Ls: Is	0.3:1	Dann et al[4]
		–0.3:1	Bundgaard et al[100]
	Ss: Is	–0.32:1	Rosen[103]
Nasolabial angle	Nasolabial angle: Is	–1.2°:1	Dann et al[4]
Nasal tip (H)	Pn: Is	0.28:1	Dann et al[4]
		0.36:1	Hack et al[7]
	Pn: Ia	0.17:1	Proffit and Epker[89]
Nasal base (H)	Sn: A pt.	0.57:1	Freihofer[104]
		0.51:1	Rosen[103]
	Sn: A pt. (thick lip)	0.3:1	Stella et al[18]
	Sn: A pt. (thin lip)	0.46:1	
	Sn: Ia	0.24:1	Mansour et al[2]
	Sn: ANS	0.60:1	Hack et al[7]
	Three-dimensional analysis	1.25:1	Ewing and Ross[77]
Upper labial sulcus (H)	SLS: Ia	0.52:1	Mansour et al[12]
	SLS: A pt	0.38:1	Hack et al[7]
		0.76:1	Lin and Kerr[102]

ANS=anterior nasal spine, H=horizontal, Ia=incisor point, Is=inferior stomion, Ls=labi superiorus, Pn=pronasale, SLS=superior labial sulcus, Sn=soft tissue nasion, Ss=stomion superius, V=vertical.

TABLE 60-9. **Soft Tissue Changes Associated with Maxillary Impaction**

Anatomic Structure	Landmarks	Ratio	Author(s)
Upper lip (V)	Ls: Is	–0.38:1	Schendel et al[17]
		–0.51:1	
		–0.3:1	Radney and Jacobs[16]
	SS: Is	–0.4:1	
	SLS: Is	–0.42:1	Mansour et al[12]
	Ls: Pr	–0.31:1	
	Ls: ANS	–0.06:1	Sakima and Sachdeva[105]
	SS: ANS	–0.41:1	
Upper lip (H)	Ls: Ia	0.89:1	Mansour et al[12]
Upper labial sulcus (V)	SLS: Is	–0.25:1	Radney and Jacobs[16]
	SLS: ANS	0.12:1	Sakima and Sachdeva[105]
Upper labial sulcus (H)	SLS: Ia	0.76:1	Mansour et al[12]
Nose (V)	Sn: Is	–0.2:1	Radney and Jacobs[16]
	Pn: Is	–0.16:1	
	Pn: Pr	–0.15:1	Mansour et al[12]
Nasal base (V)	Sn: Pr	–0.28:1	
Full Face Evaluation			
Upper lip (middle) (H)	Three-dimensional analysis	1:1	McCance et al[43,79]
Alar Base (H)	Three-dimensional analysis	1.25:1	
Nasal base (V)	Sn: ANS	0.29:1	Hack et al[7]
Upper labial sulcus (V)	SLS: A pt	0.54:1	
Upper lip (V)	SLS: Is	0.72:1	

ANS=anterior nasal spine, H=horizontal, Ia=incisor point, Is=inferior stomion, Ss= stomion superius, Ls=labi superiorus, Pn=pronasale, Pr=porion, V=vertical.

patients found that this technique was useful in deciding whether or not to under-go orthognathic surgery.[31,33] The most problematic and challenging parameter to solve is to accurately calculate the postoperative soft-to-hard tissue changes, given the variability of the soft tissue thickness, tonicity as well as translations relative to bony changes and the two-dimensionality of the system. For the moment, the currently used commercial preprogrammed algorithms are based on clinically retrospective studies of stability of the soft tissues after bone and dental movements and use default soft-to-hard tissues ratios. The utilization of such default ratios accounts for some of the variability and inaccuracy in predicting the soft tissue outcome regardless of the technique, manual versus computed. Studies revealed several specifics concerning the accuracy and reliability of predictions regardless of the software program used: (1) upper and lower lips are the least accurately predictable regions, probably owing to a high individual variability in thickness and tonicity, (2) the vertical plane prediction changes are generally more accurate than in the sagittal plane, and (3) sagittal changes are more easily perceived than vertical changes.[27,33]

TABLE 60-10. **Soft Tissue Changes Associated with Mandibular Autorotation**

Anatomic Structure	Landmarks	Ratio	Author(s)
Lower lip (H)	Li: Ii	0.75:1	Mansour et al[12]
Lower lip (V)	Si: Is	–0.93:1	
	Si: Me	–1.03:1	Sakima and Sachdeva[105]
	Li: Me	–1.48:1	
Chin (H)	Pgs: Pg	0.86:1	Gaggl et al[85]
		1:1	Radney and Jacobs[16]
	Pgs: Me	0.79:1	Sakima and Sachdeva[105]
Chin (V)	Pgs: Gn	–0.8:1	Lines and Steinhauser[11]
	Pgs: Me	–0.98:1	Sakima and Sachdeva[105]
	Mes: Me	–1.2:1	Mansour et al[12]
Lower labial sulcus (H)	ILS: B pt.	0.9:1	Mansour et al[12]
		1:1	Radney and Jacobs[16]
Inferior labial sulcus (V)	ILS: Me	–1.05:1	Sakima and Sachdeva[105]
Inferior labial sulcus (H)	ILS: Me	0.61:1	

H=horizontal, Gn=gonion, Ii= lower incisor, ILS=inferior labial sulcus, Is=inferior stomion, Li= labi inferiorus, Me=menton, Pgs=soft tissue pogonion, Si= soft tissue incisor, V= vertical.

Mandibular Advancement

Dentofacial Planner Plus and *Quick Ceph Image* software programs have been found to be relatively accurate in predicting changes in the lower third of the face associated with mandibular advancement.[24,35] Changes of reference points between the computer's predicted and post-treatment tracings in the lower lip in the vertical plane are more predictable than in the sagittal plane, thus the final preoperative computed prediction usually tends to be more protrusive than the actual value, whereas the opposite is true in the chin area, with a soft tissue pogonion (Po') that tends to be more superior.[24,35] An overall predictability of more than 80% can be attained particularly in the lip area, whereas the least predictable region is the submental area.[24,35] On the contrary, authors who tested the *Prescription Planner/ Portrait* software found that the computer-predicted lower lip was more retrusive and thinner than on the actual profile, and they found no differences between patients who underwent mandibular advancement alone versus associated with genioplasty.[36,37]

Mandibular Setback

The only available study used *Quick Ceph Image* and *Portrait Planner* software programs and showed that (1) the results were accurate enough for use in communication and patient education, (2) 30% of the sample showed significant errors greater than 2 mm and these errors were bigger in the vertical plane in the area of the lower lip, with a tendency to underestimate the amount of lip retraction, and also in the chin area, (3) the accuracy and predictability were not as good as in mandibular advancements, probably owing to the technique of image compression, and (4) both software programs showed similar results.[26]

Bimaxillary Surgery

Several studies evaluating the accuracy of computerized surgical prediction methods using the *Quick Ceph Image* software program showed that the most important discrepancies between the computer generated tracing versus the actual tracing were the following: (1) the lower lip tended to be predicted more inferior, shorter, and more protrusive than on the actual profile, (2) the upper lip tended to be shorter, with the superior labial sulcus located more posterior than on the actual profile, and (3) the soft tissue pogonion (Pg') was located more inferior than on the actual profile. These differences have been found to be probably related to the inaccuracy and variability of the default soft-to-hard tissue ratios used by the software rather than to the type of maxillary movement, association of mandibular or genioplasty procedure ,or type of closure of the Le Fort I incision (V-Y vs. non V-Y).[38]

Authors who have tested the *DentoFacial Planner* software in predicting changes in patients who underwent a Le Fort I procedure (advancement and intrusion) found that the nasal tip and the subnasale on the computer prediction were more posterior than on the actual profile.[28]

Maxillary Impaction

The utilization of *Orthognathic Treatment Planner (OPT)* and *Prescription Portrait* software programs have shown a good global accuracy and reliability to within 1.5 mm in the majority of the measurements, but better in the sagittal plane.[30]

Three-dimensional Computer-Assisted Prediction

The foremost critique, which could be addressed to the previous methods of prediction, is that the analyses were based on two-dimensional lateral cephalograms, whereas the actual soft tissue changes are of course always three-dimensional! For this reason, current efforts are all directed in developing as accurately as possible a reliable and easily usable three-dimensional computer treatment planning software to predict the facial soft tissue outcome in response to skeletal movements as close as possible to reality.

In the early 1980s, different authors, supported by the advances in three-dimensional computed tomography (CT) scanning techniques, pointed out the importance of the management of the third dimension in the evaluation and surgical planning of craniofacial surgery, inaugurating a new and exciting era.[40,45] The first rudimentary computer-assisted software programs were at the beginning elaborated only for planning osteotomies and bone relocation, without taking into account the soft tissue envelope changes.[40,45] In the mid-1990s, the need to improve the three-dimensional soft tissue representation and prediction gave rise to a tremendous impetus, which led to a plethora of more or less reliable and accurate computer systems.[39,43,46] One of the most used methods in assessing the three-dimensional details of the face is based on cone beam computed tomography (CBCT) scan data, which allows the separate visualization of the different constituents of the facial anatomy, thus enabling the surgeon to differentiate and separately manipulate the bone from the air and soft tissue surfaces.[39,41–44,46–50]

Even though the CBCT scan has the capacity of displaying both bone and soft tissue details, the three-dimensional reconstructed soft tissue images are generally incomplete and not lifelike. For this reason, some authors have recommended the simultaneous use of laser surface scanner images or photogrammetric images, which are merged with the scan images, in order to improve the quality and accuracy of the production of the three-dimensional facial morphology.[41,42]

Dolphin Imaging software can integrate and superimpose traditional two-dimensional photographs with digital two-dimensional lateral and frontal cephalometric radiographs. Two-dimensional photographs can also be imported and wrapped onto three-dimensional CBCT and CT images for presentation purposes (Figures 60-1 to 60-4). Dolphin Imaging allows the operator to use both orthogonal and perspective projection-type cephalometric renderings. Visualization filters can then be applied and transparency adjusted for viewing the soft and hard tissues. Virtual orientation of the face imaging space using the semitransparent filter allows

the operator to rotate and orient the virtual patients, optimally matching bilateral and anatomic structures. Simulation of surgical results and soft tissue movement is possible only in the two-dimensional environment. Traditionally, in photogrammetry as seen previously, separate three-dimensional data sets (each containing its own coordinate system) are generated for each viewpoint, as a range map, and are subsequently stitched together to produce a new overall three-dimensional coordinate system. Stitching multiple sides together has historically worked well for data input of "inanimate objects" in which motion is not a factor. This technique does not work well when the subjects are animate, because "stitching" separate three-dimensional images together to generate a single three-dimensional model of the patient can compromise accuracy owing to the discontinuity of surface information. There is no guarantee that two separate images taken at different points of time with the movement factor will still match and can result in a fracture of information along the midline. This compromises accuracy. The preferable way to generate a three-dimensional surface image derived from multiple viewpoints is to generate a single unified and continuous coordinate system by selecting the best quality data for any given xyz coordinate from each of the viewpoints. For this to work, the reconstructive algorithms must be able to place a value on the quality of each point generated. This is the great advantage of using active photogrammetry and very fast capture time incorporated in systems such as the 3dMD system.

The importance of three-dimensional virtual surgical planning increases with the complexity of the deformity and reconstruction needed to correct it. Three-dimensional image fusion has further increased the importance and accuracy of virtual treatment planning and several systems are now available. At the center of the three-dimensional approach is the "**P**atient-**S**pecific **A**natomic **R**econstruction" (***PSAR***), which is an anatomically accurate record in which all of the patient's three-dimensional images are superimposed into one valid three-dimensional structure and combined with the relevant biomechanical properties and is called *Image Fusion*.[61,62] There are a number of potential advantages of registering anatomically accurate three-dimensional facial surface images to CT/CBCT data sets.

- Surface images may correct for CBCT surface artifacts of the soft tissue caused by patient movement.
- Independently acquired surface images compensate for soft tissue compression from upright CBCT device stabilization aids (i.e., chin rest, forehead restraint) or from supine soft tissue draping changes.
- Surface images most notably may supplement missing anatomical data (e.g., nose, chin).
- Surface images may provide a more accurate representation of the soft tissue that reflects the patient's natural head position for diagnosis and treatment planning.

In relation to surface three-dimensional construction, a surface image has two components, the geometry of the face and the color information, or texture map, that is mathematically applied to the shape information.

Stereo photogrammetry is the optical method of obtaining an image by means of one or more stereo pairs of photographs taken simultaneously. The concept was first applied to the face as early as 1967.[63] This technique differs from the other optics-based methods in that it requires no special pattern projection and the subject can be illuminated with regular photographic flash. Once the three-dimensional geometry of researchers have reported accurate identification of facial landmarks from 0.5 to 0.2 mm.[64–67]

In the early 2000s, studies were focused mainly on the generation of real-time and mathematical realistic modeling of nonlinear three-dimensional soft tissue deformations occurring simultaneously with the underlying bone movements, such as the CASP technique (computer-assisted three-dimensional virtual reality soft tissue planning and prediction for orthognathic surgery), which is based on three-dimensional reconstructed CT images combined with surface data as follows: processing of CT data using a specific algorithm and soft tissue and bone segmentation, generation of a three-dimensional color individualizing facial model by mixing digitized color photographs with the reconstructed three-dimensional soft tissue (color facial texture-mapping technique), and utilization of two separate algorithms to study the soft tissue model deformation.[48–51] The final result is a virtual reality workbench, which allows a real-time generation of individualized predicted and animated color



FIGURE 60-1. Preoperative views. Young woman with combined maxillary and mandibular deformity and muscle hyperfunction, class II malocclusion with vertical maxillary excess, and micrognathia. **A,** Frontal. **B,** Lateral. **C,** Three-quarter view. **D,** Smiling.

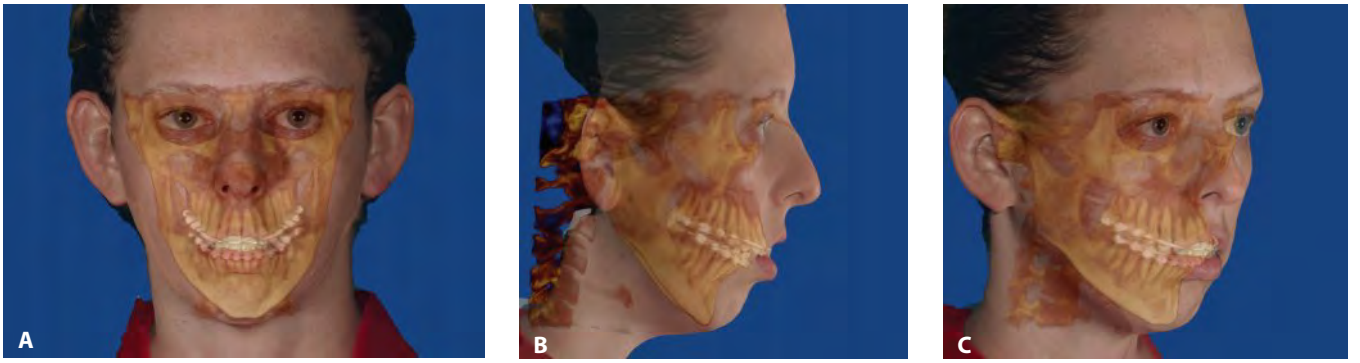


FIGURE 60-2. Preoperative dolphin views with semitransparent soft tissue. **A,** Frontal. **B,** Lateral. **C,** Three-quarter view.

facial models resulting from translation and rotation of the underlying bone.

Recent studies have described three-dimensional soft tissue prediction procedures using two different mathematical simulation models based on the finite element method (FEM) or mass spring. Studies have shown the sound superiority of the three-dimensional technique over the two-dimensional software programs in predicting the lower third of the face, especially in the lip and chin area.[41,42] This type of technique is seen in the 3dMDface,[68] Vultus System, which allow the capture of the face surface by using a multiple camera views synchronized in a single capture to produce a single three-dimensional polygon surface mesh. The surfaces images obtained with no radiation are then visualized and manipulated using a specific software platform.

Workup

Initiation of the patient *workup* requires collecting, cataloging, and archiving of all relevant three-dimensional image data sets related to one point in time, referred to as an *episode of care.* Registration of the data sets could be performed using fiducial markers, but there are considerable workflow and quality drawbacks to this method, including the additional time needed to place the markers themselves and image distortion. A more direct method is to do a best-fit scenario between the data sets. The first step in this registration process entails segmentation of the outer surface of the CBCT data to generate a separate object that represents the outer geometry keeping the original spatial relationship (*DICOM skin*). Next, the geometry of the three-dimensional optical surface image is registered to the DICOM skin, which acts as the reference object to ensure that the three-dimensional surface adopts the coordinate system of the DICOM upon completion. If the software is well designed and user-friendly, superimposition should only take less than 30 seconds to complete. After registration, numerous options allow interaction with the data sets by rendering the volumes either independently or separately including scrolling through the volume in three-dimensional or simultaneously in the coronal, sagittal, and axial planes.

Simulation refers to an imitation of a real-world process in a computer program using mathematical models to study

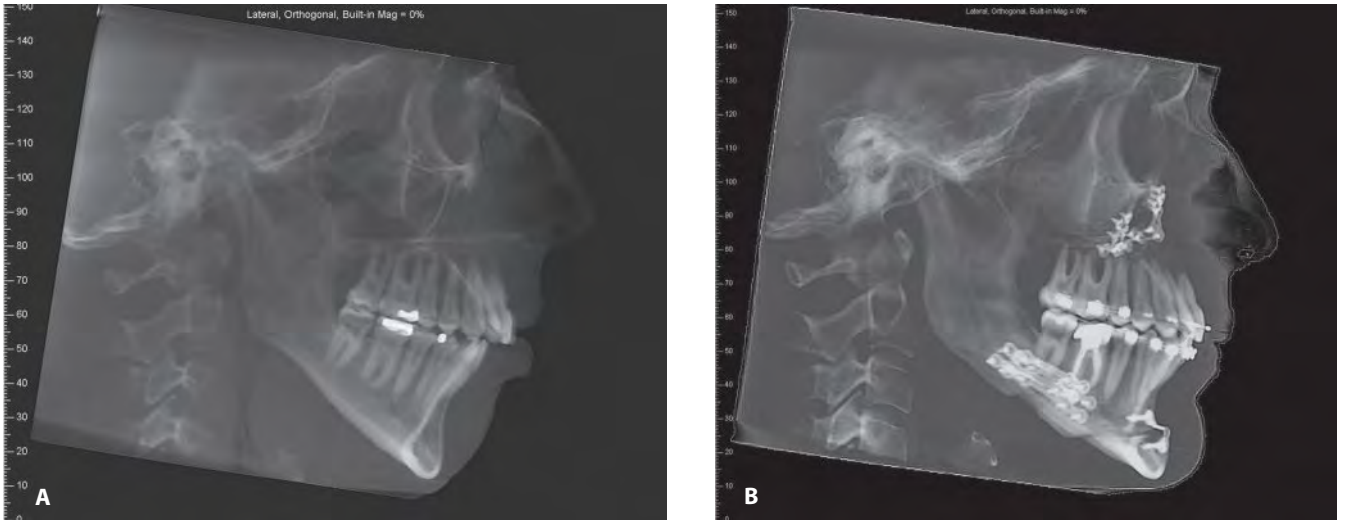


FIGURE 60-3. Cephalometric radiographs demonstrate maxillary impaction and slight advancement with larger mandibular advancement and associated chin advancement by sliding osteotomy. **A,** Preoperative. **B,** Postoperative.



FIGURE 60-4. Postoperative facial views. Perioral muscle reconstruction was done per Schendel to prevent unaesthetic nasal and lip changes.[58, 59] **A,** Frontal. **B,** Lateral. **C,** Three quarter view. **D,** Smiling.

the effects of the changing parameters and conditions to make a decision. Computer-based simulation gives the clinician the opportunity to perform virtual surgery or treatment, increasing the possibility of a successful outcome with no risk to the patient.

The Mass-Spring Model technique of simulation involves implementing a biomechanical model that defines the relationship between the hard and the soft tissue with hundreds of thousands of nonlinear connector points. This generates three-dimensional deformable tissue models that include spring-based force computations to model the physical characteristics of real tissue reactions. This mechanism simulates deformable tissues, such as the soft tissue of the face. The models employ force computations from physical laws and study of facial surgery cases and apply these forces to the three-dimensional model components. The computations modeled include tissue deformation and relaxation; external forces such as gravity; and three-dimensional collision detection with force feedback. The advantage of this approach is that computations are well within the power of available PCs and occur in real time.

Results

Use of three-dimensional imaging and computer simulation for treatment occurs as the following patient demonstrates. Figure 60-5 shows the stages in treatment planning for a young woman with mandibular retrusion. The PSAR has been created by the 3dMD Vultus system and the profile and three-quarter views are visualized in Figure 60-5. Osteotomies are simulated for the mandibular advancement by the sagittal split ramus osteotomy technique and the resultant mandibular advancement done virtually. The result is displayed and altered until the aesthetic and functional result desired is achieved. The measurements in all three planes of space are then automatically generated (Figure 60-6). The soft tissue is simultaneously reconstructed and the final aesthetic result can be seen in the virtual environment (Figure 60-7). Surgery can then proceed along these guidelines (Figure 60-8). The actual soft tissue changes occurring with surgery can be visualized in a histogram with the actual spot measurements recorded (Figure 60-9). We have found this system to be accurate for advancement surgery of either the maxilla or the mandible (Figure 60-10). Inaccuracies in the soft tissue simulation arise when vertical movements are

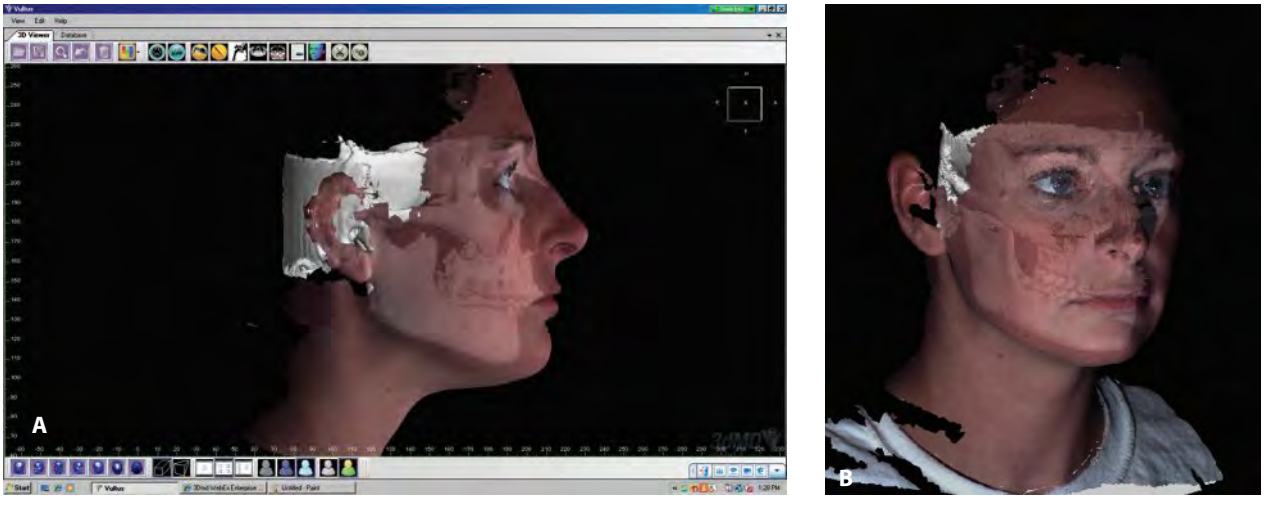


FIGURE 60-5. **A,** Patient-specific anatomic reconstruction (PSAR) of a case of a young woman with mandibular retrusion. The profile view is shown. The skin has been made semitransparent so that the underlying skeleton can be visualized. Only the bone, only the soft tissue, or a combined image with variable transparency can be visualized such as shown here. **B,** Three-quarter view.

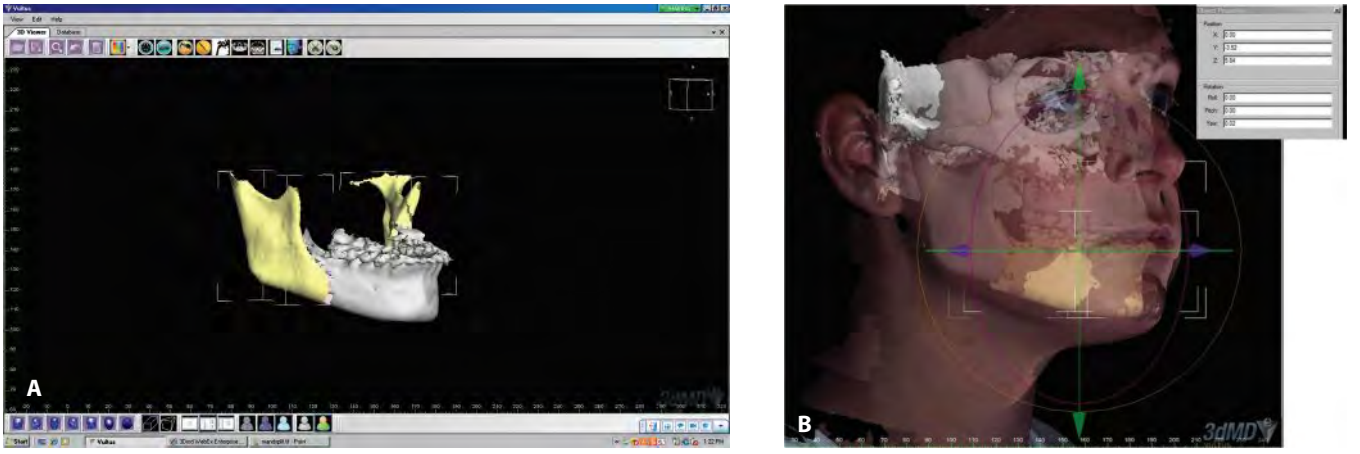


FIGURE 60-6. **A,** Osteotmies marked on the mandible only, which has been isolated. Note that the sagittal split osteotomies are anatomically correct and can be varied according to the need by the surgeon. **B,** Jaws repositioned on the PSAR of the skeleton and transparent soft tissue with actual surgical three-dimensional vectors shown.

undertaken or when there is preexisting soft tissue deformation from the device or muscle hyperfunction in the original data set. Also, soft tissue variations occur according to how each surgeon handles the soft tissues and what adjunctive techniques are performed. Because of these facts, the soft tissue simulation module should include the ability to rearrange the tissues based on the surgeon's experience. The Vultus system has such an option, and we have found it very accurate when this is done.

DISCUSSION

Correctly planning and accurately predicting surgical outcome is paramount in facial surgery. Gossett and coworkers[69] have outlined a three-fold purpose for computer-simulated predictions: (1) guide the treatment to the desired result, (2) give the patient a reasonable preview of the outcome; and (3) serve as a communication tool between orthodontist, surgeon, and patient. Advancements in computer imaging have revolutionized the treatment of dentofacial deformities and specifically orthognathic surgery. Use of this image fusion technology now permits the creation of PSARs on a routine basis, and a more comprehensive diagnosis and treatment plan can, thus, be obtained. Both the skeletal and the associated soft tissue changes thus become more accurate and predictable. At this point, no system is fully automatic but the present systems provide a great improvement over the old two-dimensional soft tissue calculations. Surgeon experience and incite are still important parts of obtaining the desired functional and aesthetic result. In the end, treatment outcomes are improved. The ideal system would predict the soft tissue behavior resulting from the movements of the underlying bone and

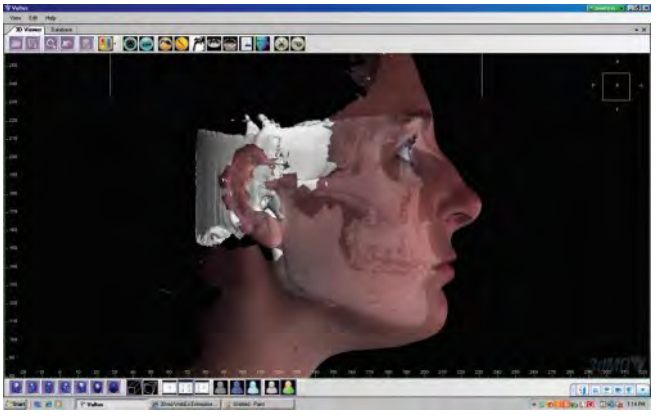


FIGURE 60-7. Reconstructed facial soft tissues in the virtual model of the profile after simulated surgery. Again, the overlying soft tissue has been made semitransparent to help with the visualization.

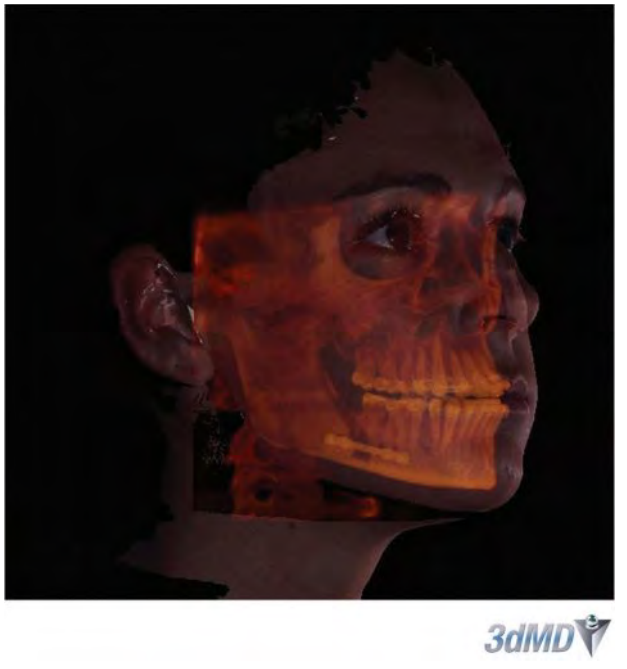


FIGURE 60-8. Actual postoperative result from mandibular advancement with transparent skin.

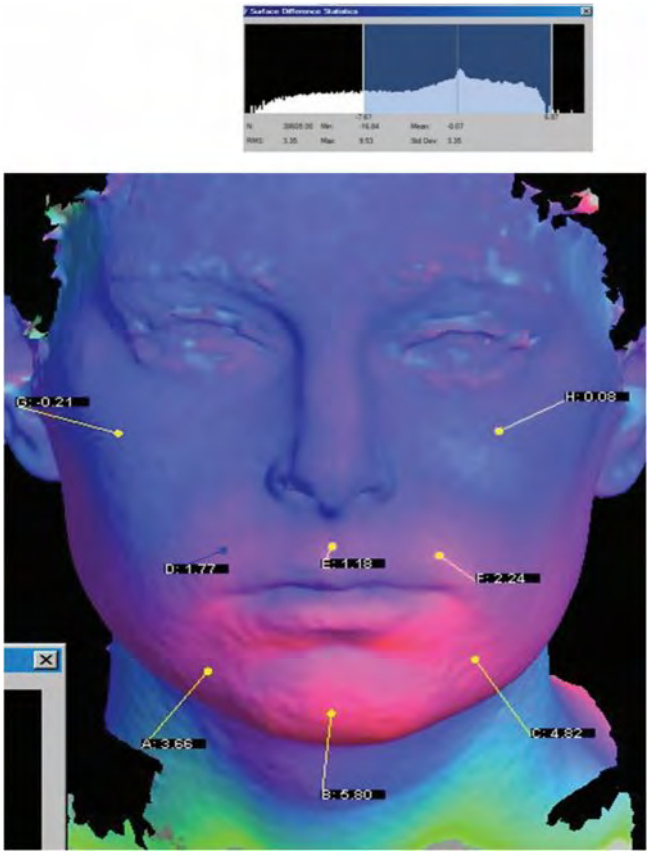


FIGURE 60-9. Histogram created shows the actual postoperative soft tissue changes. The areas with more change are indicated by *orange to red colors.* Spot measurements can be done as shown.

transfer of the ideal and customized computerized surgical plan to the operating room. In conclusion, it should be kept in mind that all the previously mentioned prediction techniques are strongly dependent on rapidly developing technology subject to daily improvements. Different studies have shown that the accuracy and reliability are far from perfect yet but are constantly improving.[70,71]



FIGURE 60-10. View of actual and simulated profiles. The actual profile is outlined in *green* for visualization. Note the good overall accuracy.

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Suggested Readings

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CHAPTER 61

Complications of Orthognathic Surgery

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Complications of elective orthognathic surgery may be minor or major, immediate or delayed, and can be divided into several broad and overlapping categories (Table 61-1). Several factors involved in the specific types of problems that may occur, including any significant patient medical history; the use of tobacco; the presence of oral habits; whether surgery includes the maxilla, mandible, or chin; the applied regional surgical anatomy; the magnitude of bony movement; the need for segmental jaw surgery; and the specific methods of fixation employed. The variey of morbidities encountered may fall into one or more of the following general areas: vascular problems, neural complications, infectious disorders, aberrant fracture of the osteotomy segments, occlusal discrepancies, temporomandibular joint (TMJ) dysfunction, dental injuries, and other miscellaneous complications. The approach to orthognathic complications addressed in this chapter is divided into two areas of clinical significance: prevention and management of these complications. In general, prevention focuses on the preoperative patient evaluation, data gathering, and treatment planning phases, whereas the intraoperative surgical management may result in complications that are not completely in the control of the surgeon and may also require immediate attention. Many of the undesirable results of orthognathic surgery are caused by errors in the preoperative clinical examination, dental model analysis and model surgery, or cephalometric analysis or computer planning for surgery. It should be remembered that the majority of patients with dentofacial deformities have altered or unusual craniofacial anatomy that may dictate the need for a departure, or variation, from usual established treatment protocols, but attention to detail is tantamount in the data gathering and treatment planning stages of care. Complications from elective surgery typically performed in young, healthy patients, even when minor in nature, may be devastating to the patient and family; therefore, a comprehensive understanding of the possible complications and prevention and management is critical for the oral and maxillofacial surgeon.

TABLE 61-1. **Complications of Orthognathic Surgery**

Diagnostic errors	Relapse
Treatment planning errors	Malocclusion
Errors in model surgery	Limited mouth opening
Vascular supply problems	Pain, edema
Bleeding	Sinus problems
Hematoma formation	Fistula formation
Nerve injuries	Lip shortening
Unfavorable osteotomies	Nasal septal deviation
Nonunion	Nasal airflow obstruction
Malunion	Alar base widening
Temporomandibular joint problems	Epiphora
Condylar resorption	Anosmia
Facial scarring	Dental injuries
Cosmetic deformities	Periodontal problems
Infection	Salivary gland injuries
Hardware failure	

VASCULAR COMPLICATIONS

Acute Maxillary Hemorrhage

Severe acute hemorrhage has been documented with both maxillary and mandibular surgery and can have both immediate and delayed effects.[1–7] In general, massive hemorrhage is rare both acutely and in the immediate postoperative

period, but it is more likely to occur with maxillary versus mandibular surgery. The blood vessels that are most at risk of iatorogenic injury during maxillary surgery include the maxillary artery, posterior superior alveolar artery, and descending palatine arteries.

Massive blood loss can rarely occur secondary to injury to the internal carotid artery and the internal jugular vein, but more likely, it occurs from injury to the major branches of these blood vessels. During Le Fort surgery when separating the pterygoid plates from the maxilla, it is possible to fracture the base of the skull by vigorously manipulating chisels or directing chisels in an aberrant direction superiorly.[8,9] In fact, there is extreme variability in the location of the posterior portion of the Le Fort osteotomy; this may not be under the control of the surgeon and may result in high fractures near the skull base or low anterior fractures through the pterygoid plates or even further forward in the maxillary alveolus. This can result in direct or indirect damage to major vessels in the region of the posterior maxilla and skull base. When considering vascular injuries from maxillary surgery, it should remembered that blood vessels may be injured directly during osteotome placement (branches of the maxillary artery) or indirectly through shattering the pterygoid plates with bone edge shearing through the regional vasculature (descending palatine arteries, ptergoid venous plexus). Meticulous efforts should be made by the surgeon to properly direct osteotomes low and medial in the pterygoid plate region and to down-fracture the maxilla only when it is deemed possible to do so without the need to apply excessive forces. If the maxilla is extremely difficult to mobilize, the posterior osteotomy may be redirected into the tuberosity region and away from the pterygoid plates to avoid the vessels in the area (Figure 61-1).[10] Patients who are undergoing a maxillary osteotomy to correct malposition of their maxilla after trauma may present special considerations. The previous midface trauma may have resulted in fractures at the base of the skull as well as unpredicatble scar formation and fusion of bony segments that do not fall along the usual natural suture lines or planes of cleavage. Therefore, excessive surgical manipulation in order to mobilize the maxilla may result in the direction of the osteotomy following the previous fracture lines and not along the usual intended path of induced fracture from Le Fort surgery.

Generally, when brisk bleeding is encountered during Le Fort surgery, the surgeon should follow the basic principles of completion of the osteotomy, with down-fracture of the maxilla, and assessment of the area for the bleeding source, at the specific site of hemorrhage. This may be difficult especially if the bleeding is brisk, so as an alternative, the region may need to be packed with gauze to limit blood loss and allow for some vasculature contraction and platelet aggregation in order to improve visualization. The surgeon should keep in mind that the best opportunity to identify and ligate a bleeding vessel is when it is first cut. Ligature clips may be applied or electrocautery may be used if the vessel is seen clearly. In the posterior aspect of the maxilla, placement of vascular suture would be difficult. When brisk hemorrhage obscures the surgical field, gauze packing, with thrombin impregnation, should be followed by attempts to directly occlude the bleeding vessel. Another option is to pack resorbable hemostatic materials, such as Surgicel (oxidized cellulose), Avitene (microfibrillar collagen), or Gelfoam (absorbable gelatin sponge), in the region under pressure, with expected tamponade of the source of bleeding.

The internal and external carotid arteries, or terminal branches, may be susceptible to both direct and indirect insult during the orthognathic surgery. Thrombosis of the internal carotid artery after orthognathic surgery may occur because of excessive neck extension with poor patient positioning.[11] Mortality associated with thrombosis of the internal carotid artery has been estimated to be 40%, with an additional 52% of patients sustaining a significant permanent neurologic deficit. Extension of the neck serves to stretch and partially fix the internal carotid artery against the cervical vertebrae

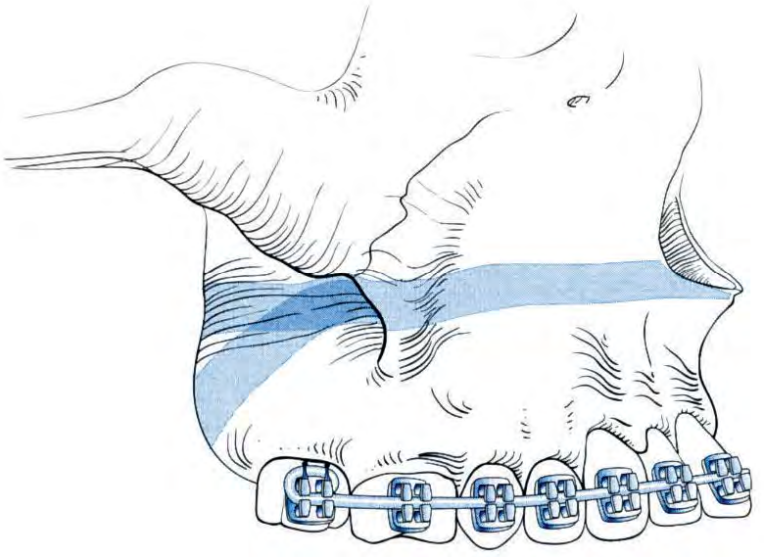


FIGURE 61-1. The posterior osteotomy is directed into the tuberosity region behind the second molar when the maxilla is difficult to mobilize.

during surgery, and contralateral rotation of the head results in further stretch of the artery. Malpositioning of the patient's head in this manner places the internal carotid artery at risk for both direct and indirect trauma and should be avoided.

Delayed Maxillary Hemorrhage

Delayed hemorrhage after a Le Fort I osteotomy may occur as early as several hours after surgery during the night of surgery to as late as 9 or 10 days postoperatively after surgery. The blood vessels most frequently involved are the descending palatine artery, the internal maxillary artery, and the pterygoid venous plexus of veins.[6] Typically, delayed hemorrhage is more likely venous in origin involving the pterygoid venous plexus of veins.

Recommendations to reduce these types of vascular injuries include careful placement and orientation of the pterygomaxillary osteotome in the pterygomaxillary suture with angulation of the osteotomy inferior from the zygomaticomaxillary crest toward the pterygoid plates.[12] The mean distance from the most inferior junction of the maxilla and the pterygoid plates to the position of the internal maxillary artery in the pterygopalatine fossa is 25 mm. With an average length of an osteotome of 15 mm, assuming normal vascular anatomy, the margin of safety in order to separate the entire pterygomaxillary junction is only 10 mm. However, as mentioned, patients with dentofacial and craniofacial anomalies may have anatomic variation from these normative values. The internal maxillary artery and its branches are most vulnerable to damage during their course through the pterygopalatine fossa and ptergomaxillary fissure, at the specific time when the maxillary tuberosity is separated from the pterygoid plates with the curved pterygomaxillary osteotome.

The posterior superior alveolar and the descending palatine arteries may be severed during the Le Fort I osteotomy procedure because they lie in close proximity to the bony walls, although the posterior superior alveolar artery does not usually present a significant concern for bleeding. It is generally recommended that the greater palatine arteries be preserved by gentle bone removal in the area of the posteromedial maxilla that surrounds these vessels. This should be performed with retraction and protection of the vessels, and a rotary instrument may be used to undermine the bone adjacent to the vessels; then a chisel could be used to fracture the bone away from the vessels (Figure 61-2). However, if bleeding is encountered, the descending palatine artery should be ligated or cauterized rather than allowing it to retract and continue to bleed, possibly resulting in delayed hemorrhage in the postoperative period. Preservation of the descending palatine vessels has been shown to have minimal effects on a single-piece Le Fort osteotomy owing to the extensive blood supply from the palatal and buccal soft tissues; however, consideration should be given toward preservation of these vessels during segmental maxillary surgery in order to maximize the blood supply to the maxillary segments and minimize neural deficits to the palatal mucosa and dentition.

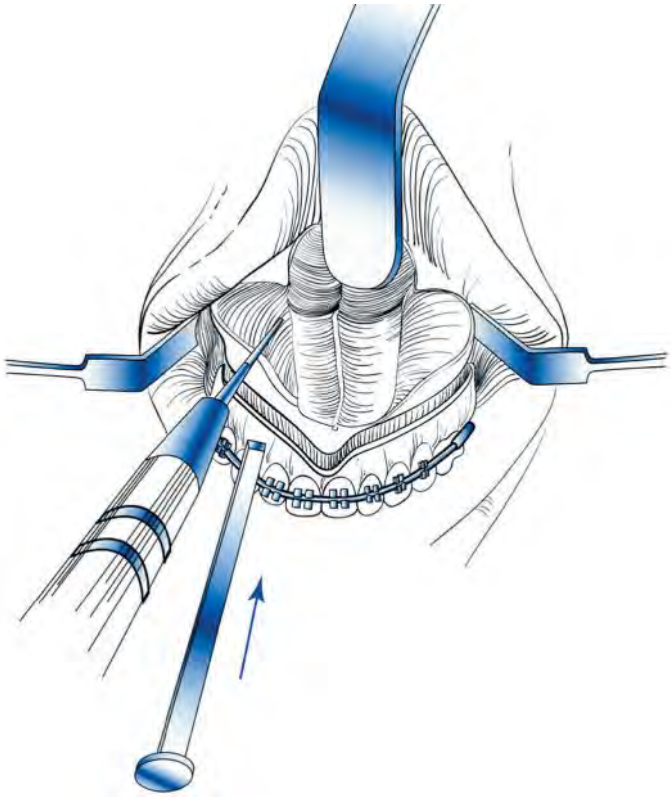


FIGURE 61-2. A drill is used to create a groove both medially and laterally to the descending palatine artery; then a chisel is used to gently fracture the bone and preserve the vessel.

In general, when large magnitude advancements of the maxilla are necessary (>8 mm), it may be difficult to preserve the palatine vessels, and they may be stretched to the point of severance, or to the point of thrombosis, so routine sacrifice may be the most prudent option.

There are several treatment options for the patient with postoperative hemorrhage after maxillary surgery, and these vary with the degree and severity of the bleeding. The most obvious sign of this type of problem is hemorrhage eminating from both nares.[3] When a patient is initially seen with postoperative bleeding, intermaxillary fixation (IMF), if present, should be released. The patient's general physical status should be assessed, high blood pressure should be controlled, and appropriate complete blood count and bleeding and coagulation studies should be ordered. Abnormal coagulation parameters warrant prompt correction and possible hematologic consultation. It should be remembered that this surgery may be the first in a young patient and may disclose a previously undiagnosed bleeding disorder. With a good light source and suction for visualization, the nasal cavity should be examined to reveal whether a bleeding site is arterial or venous in nature. Most likely, in the immediate perioperative period, significant nasal bleeding may result from tears in the nasal mucosa that may not have been addressed during surgery and may have become more active after extubation with a nasal endotracheal tube (ETT). If

adequate visualization and assessment are not possible, the nose should be anesthetized and decongested with a local anesthetic and a vasoconstrictor. Local anesthetic injections in the nose and around the palatine foramina are useful in decreasing postoperative hemorrhage, at least to allow for evaluation and definitive ligation. If the bleeding is minor in nature, it may be possible to treat the patient with bed rest and the use of anterior and posterior nasal packing for 3 to 5 days, which can also be used for recurrent bleeding or for a patient not responding to initial therapeutic measures. For a patient who does not respond to local attempts at management, or in whom the bleeding is severe or persistent, a return to the operating room for exploration of the surgical site and direct ligation or packing is recommended. As a preferred, less invasive option, angiography of the vessels in the area may be peformed promptly and with minimal morbidity. This will allow direct embolization of the vessel at the exact point of severance, providing that there is sufficent bleeding to allow visualization of contrast extravasation. Additional operative techniques may be employed depending upon the specific clinical examination or angiographic findings. These include packing of the maxillary sinus or, in extreme cases, ligation of the external carotid artery at the lingual/facial artery branch; however, with collateral circulation, persistent bleeding may still occur after the external carotid artery is ligated. In general, severe late postoperative hemorrhage is most effectively managed by an interventional radiologist with angiography and embolization.

Mandibular Hemorrhage

As with maxillary surgery, major vessels can be injured during mandibular orthognathic surgery. Occlusion of the internal carotid artery has been described after a mandibular bilateral sagittal split osteotomy (BSSO),[13] and major central nervous system morbidity may occur after this injury, as mentioned previously. The medical and surgical management of internal carotid artery thrombosis is beyond the scope of this chapter, and prevention is accomplished with appropriate patient head positioning. Typically, vascular injuries with mandibular surgery are due to indirect trauma either through forceful placement of a retractor on the lingual surface of the ramus of the mandible to retract the inferior alveolar neurovascular bundle or the use of a mallet and chisel on the medial aspect of the mandible, with severance of the inferior alveolar artery or vein. Placement of retractors and the use of chisels on the medial posterior aspect of the mandible should be performed with caution. It is preferable to limit dissection and subsequent chisel use to the area of the retrolingular fossa just distal to the area of the mandibular foramen.

Hemorrhage with Sagittal Split Osteotomy

Early reports noted numerous incidences of excessive bleeding with the sagittal split osteotomy (SSO).[14] The vessels most commonly injured are the internal maxillary artery, the facial artery, and the inferior alveolar artery. These injuries were attributed to inexperience, excessive soft tissue stripping and dissection, and lack of appropriate instrumentation for the specific procedure. With current surgical techniques and instrumentation, excessive hemorrhage during an SSO is not a major concern. In a series of 256 mandibular SSOs, the incidence of hemorrhage was 1.2% (3 cases)[15] and included 2 cases of inferior alveolar artery injury and 1 anterior facial artery injury. In a series of 1264 consecutive patients who underwent an SSO of the mandible, 15 patients (1.2%) had bleeding complications.[16] Of this group, the most common injury occurred to the retromandibular vein, and there was 1 case of bleeding from the facial artery. Four patients required reoperation owing to the development of a large expanding hematoma. It should be noted that the majority of the cases in this series were perfomed using the classic Obwegeser technique without modifications. Hemorrhage due to vascular injury on the medial or lateral aspects of the mandible during an SSO may be controlled in a number of ways. The simplest methods include gauze packing, using electrocautery, clamping with a hemostats or hemoclip if the severed vessel can be identified, or injecting epinephrine (1:100,000 or 1:50,000) into the vessel walls. Obviously, caution should be exercised when applying electrocautery in close proximity to neural tissues, such as the inferior alveolar nerve, and when indicated, bipolar cautery should be used. As with bleeding from maxillary surgery, extraoral cervical approaches to the carotid arterial system to control bleeding sources are seldom necessary, and in these rare cases, the involved vessel may be amenable to angiography and embolization.

Hemorrhage with Vertical Ramus Osteotomy

Vascular injury during a vertical ramus osteotomy (VRO) of the mandible could be divided into three regions of the ramus: superior, middle, and inferior. In the superior sigmoid notch region, the maxillary artery is at risk for injury and it should be protected with careful placement of a Bauer retractor in the sigmoid notch. In the midramus region, the inferior alveolar artery and vein enter the mandibular foramen, and surgeons use the antilingula as the anterior reference for placement of the vertical osteotomy just posterior to this landmark to avoid inferior alveolar neurovascular injury. The inferior aspect of the VRO places the facial artery and vein at risk for injury; this area should also be protected with a Bauer retractor. The posterior aspect of the VRO places the retromandibular vein at risk for injury, and careful subperiosteal dissection around the posterior border of the ramus should be followed by careful placement of a Merrill-Levaseur retractor. In consideration of these sites of potential bleeding and the need to access them surgically for control, the sequence of the VRO cuts should correspond to the surgeon's ability to access a bleeding vessel if severed after completion of the osteotomy. Note that the VRO should be "scored" through the lateral ramus first but not "completed" through to the medial ramus. Therefore, because the facial artery and

retromandibular vessels may be controlled with extraoral pressure or retraction under or behind the mandible, the inferior cut should be completed first. Because the maxillary artery can be accessed with gauze packing to tamponade any bleeding in the sigmoid notch region, this superior cut should be performed second. Finally, because the inferior alveolar artery is inaccessible from a lateral approach, the middle of the VRO should be completed third or last, because the osteotomy could then be completed, the proximal segment retracted laterally, and the inferior alveolar artery accessed immediately for packing or ligation directly through the osteotomy site. In a study of intraoral VROs, there was a low incidence of damage to the maxillary artery.[17] The masseteric artery may be injured inadvertently by placing a saw or bur cut too far superior for the horizontal osteotomy on the medial aspect of the mandible into the region of the sigmoid notch. The inferior alveolar artery may also be injured during a VRO, which is usually caused by a vertical osteotomy of the ramus placed too far anterior, owing to variability of the position of the mandibular foramen with reference to the antilingula. This may result in severance of the inferior alveolar nerovascular bundle on the medial aspect of the ramus from the oscillating saw used laterally on the ramus for the vertical osteotomy. During a VRO, access to the bleeding inferior alveolar vessel is difficult, given the lateral approach. Fortunately, in most instances, intraoperative bleeding along the ramal cut or in the sigmoid notch can be controlled by gauze pressure tamponade, until completion of the osteotomy allows improved access for vessel ligation of electrocautery. Late vascular sequelae, such as the formation of an aneurysm, may require angiography and embolization.

Vascular Compromise (Avascular Necrosis)

The morbidity of vascular compromise or ischemic complications varies between maxillary and mandibular procedures. The long-term complications of loss of blood supply resulting from orthognathic surgery range from fibrosis of pulpal tissues and minor periodontal defects and gingival papillary necrosis to major loss of teeth, alveolus, or complete bony segments, and the more severe complications increase with increasing number of bone segments. The most frequent complication associated with segmental maxillary surgery is interruption of the blood supply.[18] The major risk factors include three or more segments, advanced age, co-morbid medical history including small vessel disease, immunocompromise, or poor wound healing (e.g.. diabetes mellitus), a smoking history, previous maxillary surgery, simultaneous premolar extraction with posteriorization of the maxilla and kinking of the palatal soft tissues, the inadvertent or prolonged use of Rowe disimpaction forceps for mobilization of the maxilla with palatal pressure, prolonged intraoperative hypotensive anesthesia, a severe skeletal abnormality with significant bony movements (>10 mm), superior maxillary movement (impaction), maxillary expansion with stretching or tearing of the palatal mucosa, pressure from a palatal coverage splint, lack of segment stabilization, inadequate preoperative evaluation, and poor follow-up and lack of recognition of the early signs of the development of avascular necrosis (e.g., gray, dusky gingiva).[18] Several recommendations have been proposed to avoid vascular complications associated with segmental maxillary osteotomies. Preoperative planning is critical to ensure that adequate space exists between teeth for interdental osteotomies; careful examination of periapical radiographs before surgery should be done to confirm root divergence, and model surgery should simulate the surgical plan and minimize the amount of bone removal in the osteotomy sites. Intraoperative deails should include care during interdental osteotomies with the use of chisels and burs, or preferably saws (to prevent soft tissue damage despite contact of the cutting blade with the palatal mucosa), with copious irrigation for completion of the osteotomies. These steps will minimize the amount of heat generation and decrease the chance of creating necrotic bone or root defects. Release of soft tissues adjacent to osteotomy sites and gentle mobilization of segments to avoid tearing and cutting of flaps are essential. Splints with palatal bars used for stabilization should not impinge on the palatal pedicle that serves as the major blood supply to maxillary segments. Special consideration must be given to the patient who has had previous palatal surgery or multiple segmental procedures or has a cleft palate. In these instances, standard flap designs may not be adequate, and multiple small access incisions may be preferable to a standard bilateral circumvesticular incision. Complications such as periodontal defects, pulpal necrosis, and delayed union or nonunion are more commonly seen in the anterior region of the mandible but may be associated with any maxillary or mandibular segmental procedure. Mandibular segmental procedures require detailed planning of soft tissue incisions and careful elevation of soft tissue pedicles. In order to minimize intraoperative complications, the vascular supply to involved segments must be optimized by designing as large a soft tissue flap as possible to supply the involved segment(s), maintaining maximal soft tissue attachment to the segment(s) to be mobilized, avoiding stripping of the lingual mucoperiosteal pedicle, and making the bony osteotomies as apical as possible to include as much muscle tissue for blood supply as possible.

Avascular, or aseptic, necrosis or the major loss of soft and hard tissues of the maxilla is rare and is most often due to a compromised blood supply. As mentioned previously, this may be caused by a kinked vascular pedicle in the palatal mucosa or iatrogenic tearing of palatal flaps. Isolated cases of loss of the entire maxilla or individual segments have been reported (Figure 61-3).[18] More common and subtle complications secondary to vascular compromise range from flattening of the papilla and loss of the gingiva to periodontal defects in the areas of the osteotomies. The treatment of avascular necrosis may be divided into initial and delayed management strategies. Initially, the problem must be identified, and supportive care is provided, including irrigation, chlorhexidine rinses, and instruction on good oral hygiene practices. If



FIGURE 61-3. Avascular necrosis after a Le Fort I osteotomy.

mobility of the maxilla exists, attempts should be made to stabilize the mobile segments, with the possible need to return to the operating room. As mentioned, intermaxillary fixation should be released, if present, to allow for a comprehensive assessment. Prophylactic systemic antibiotics may be considered. The most appropriate initial management is prompt referral for hyperbaric oxygen (HBO) therapy. The problem with avascular necrosis is that by the time it is diagnosed, the damage has already occurred, and complete resolution, despite prompt HBO therapy, is unlikely. The delayed, or long-term, management of avascular necrosis includes allowing a sufficient period of time for demarcation of necrotic segments (months), then planning conservative removal of nonviable bone segments and teeth and repair of any associated oroantral and oronasal fistulae that may be present. Reconstructive options must include free flap bone graft options (fibula) with associated soft tissue transfer, owing to the compromised blood supply in the area, which will not provide an environment conducive to support healing of a standard iliac crest corticocancellous bone graft. After bone grafting, dental implant and prosthetic reconstruction may be considered.

Avascular necrosis of the proximal segment with an SSO is rare and has been attributed to excessive stripping of the segments.[19] Loss of bone secondary to aseptic necrosis has resulted in facial disfigurement with loss of the mandibular angle projection, or excessive antegonial notching. As early as 1974, Grammer and colleagues[20] noted that necrosis of large areas of bone in the proximal segment after mandibular SSO occurred secondary to wide lateral elevation of the mucoperiosteal pedicle in the animal model. They proposed that devitalized bone usually revascularized; however, when revascularization does not occur, a substantial loss of bone from the mandibular ramus can occur, especially in the gonial angle region. In 1977, Epker[21] presented modifications of the sagittal split and discussed a technique in which the amount of lateral dissection of the masseter muscle and associated periosteum was greatly decreased. Adoption of this technique has greatly minimized the incidence of avascular necrosis after a mandibular SSO. In addition, rigid fixation of the bony segments, which permits early revascularization amd prevents segment mobility, has also minimized the incidence of avascular necrosis in the mandible.

Early publications suggested that an advancement genioplasty could be done successfully by repositioning the anterior inferior border of the mandible as a free, but pedicled, bone graft. However, after a horizontal osteotomy of the inferior border of the mandible with advancement, resorption of the advanced bone segment occurs to varying degrees and may result in slight, or almost complete, loss of the advanced genial segment. Preservation of the lingual pedicle (geniohyoid and genioglossus muscles) and the buccal pedicle (platysma and depressor anguli oris and depressor labii inferioris muscles) minimizes bone resorption and results in a more predictable soft and hard tissue chin contour. Larger magnitude genial advancements may require wider soft tissue dissection and release of lingual and buccal soft tissues to achieve the desired advancement, and suprahyoid myotomies were historically performed to allow greater chin advancement before the use of rigid fixation. However, with maintenance of an adequate labial soft tissue pedicle and the use of rigid internal fixation, bone loss should be negligible.

The intraoral vertical subcondylar osteotomy is the mandibular procedure in which the proximal segment is at most risk for avascular necrosis due to the wide release of periosteal attachments. One study reported 2 out of 42 patients with necrosis of the distal tip of the proximal segment.[22] The surgical technique involved stripping the entire lateral and medial surfaces of the mandible up to the neck of the condyle. With more recent modifications of the VRO technique in which a soft tissue pedicle of medial pterygoid muscle attached to the posterior and medial aspect of the proximal segment is maintained, aseptic necrosis of the tip of the proximal segment is generally not a major concern.[23]

NONUNION OF THE MAXILLA

Nonunion, delayed union, or fibrous union of the maxilla may be due to both local and systemic factors. The blood supply to the maxilla may be compromised by poor surgical planning or may be questionable because of scarring from previous surgery, such as in the previously operated cleft palate patient. The presence of palatal scarring with large maxillary advancements may make it difficult to passively reposition the maxilla and may worsen hyponasal or hypernasal speech abnormalities as well as velopharyngeal incompetence (VPI) in cleft patients, and consideration may be needed for distraction osteogenesis, although the data regarding these issues indicate no significant differences between Le Fort surgery and maxillary distraction regarding speech

alterations and VPI. One of the major contributory factors to postoperative maxillary mobility is patients with parafunctional activity, excessive masticatory forces during function, or occlusal irregularities or interferences. In those patients in whom the maxilla has been moved in an anterior and inferior direction, there may be insufficient bony interfaces in the pillar regions (piriform rim and buttresses) to allow for complete osseous healing. Patients with systemic conditions that interfere with healing, such as diabetes mellitus, smoking, or an immunocompromised illness, require individualized case planning to minimize anticipated complications from poor bone healing. When an unstable maxillary movement (e.g., inferior repositioning) is anticipated, bone plates and screws may be combined with additional forms of stabilization, including skeletal suspension wires (circumzygomatic or piriform rim wires), as well as a period of intermaxillary fixation ranging from 1 to 6 weeks. With inferior repositioning of the maxilla, bone plates and bone grafts can be combined with adjustable pins for additional stability (Figure 61-4). When these methods are used, it may be acceptable to limit the use of intermaxillary fixation for a period of 1 week. In general, bone gaps of 5 to 6 mm or greater, resulting from large maxillary advancements, should receive allogeneic block bone grafting at the pillars of the maxilla, including the piriform rim and maxillary buttress regions. If the advanced maxilla has sufficient bone contacts in multiple regions, isolated defects may be filled with particulate allogeneic bone or alloplastic materials. In some cases, large bone gap defects in critical structural locations in the maxilla may require simultaneous autogenous block bone grafting with plate and screw fixation in order to provide for physical support.

After maxillary surgery, the primary physical finding that a nonunion exists is mobility of the maxilla during maximal intercuspation. The patient is generally able to move the maxilla superiorly by simply clenching the teeth together. Treatment of a mobile maxilla may be divided into early and late management. The use of short-term intermaxillary fixation is controversial and may not assist with bone healing across an established area of fibrous tissue interposition between bony segments and, in fact, may contribute to the nonunion by the constant pull of the mandible during attempted opening of the mouth. If maxillary mobility is noted in the early postoperative period, a short period of intermaxillary fixation may be beneficial to allow for neovascularization at the bone gap. Conversely, if the patient is already in intermaxillary fixation, removal may allow functional remodeling and bony consolidation. This is particularly important in the patient who has parafunctional occlusal activities, and selective occlusal adjustment after an occlusal analysis may be the most important means to allow bony union to occur. In addition, flat plane occlusal splints may be used to distribute occlusal forces more evenly and appropriately. Again, for minor occlusal discrepancies, selective occlusal equilibration

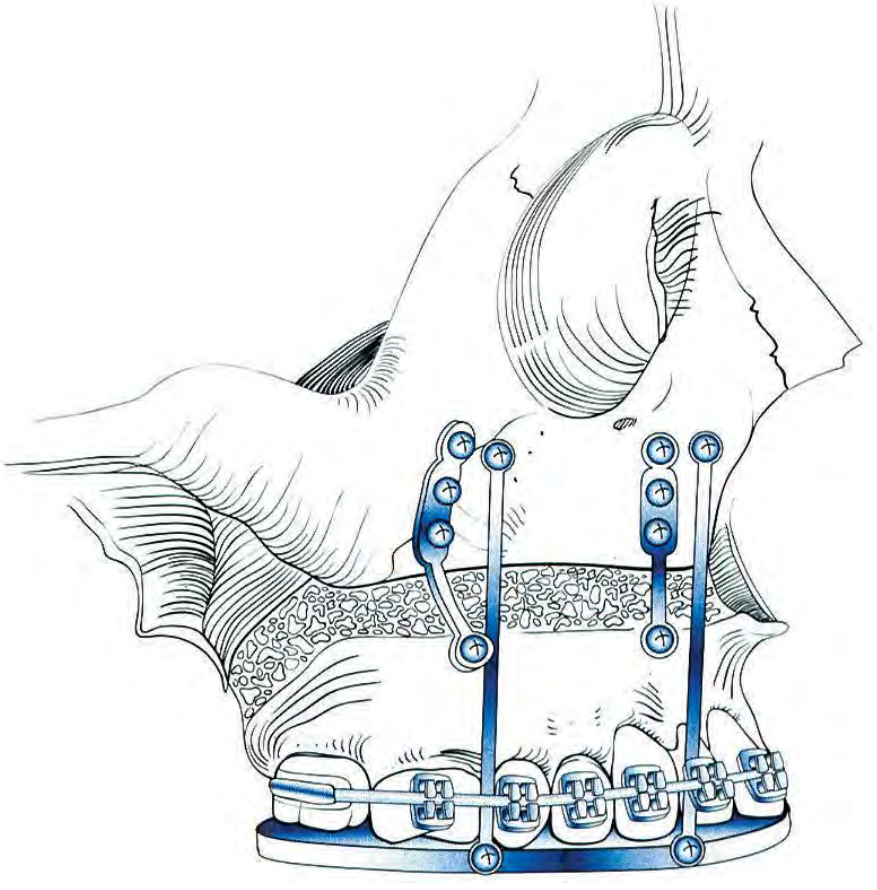


FIGURE 61-4. Inferior maxillary repositioning using bone grafts, plates, and adjustable pins.

may resolve premature contacts that may have contributed to mobility of the maxilla. Also, patients should be instructed on the use of a soft diet. Heavy intermaxillary elastics should be discontinued because, with function, they put intermittent strong forces on the maxilla by the attempts at opening of the mandible. For patients with posterior relapse, light short class III elastics can help prevent further movement and may allow osseous consolidation. However, all elastics must be used judiciously because they may aggravate the nonunion problem. Even if the final result is a minor class III malocclusion, this is usually easier to manage than a nonunion of the maxilla.

The late management of maxillary nonunion involves surgery for débridement of the fibrous tissue and either initial allogeneic bone grafting or autogenous bone block grafting and rigid stabilization of the maxilla at the buttress regions. This would include the use of bone plates and screws, particulate or corticocancellous allogeneic or autogenous bone grafts, auxiliary stabilization techniques, and possibly the use of alloplastic materials. The reoperation should be approached aggressively by completely mobilizing the maxilla and removing interpositional fibrous tissue to allow an osseous union.

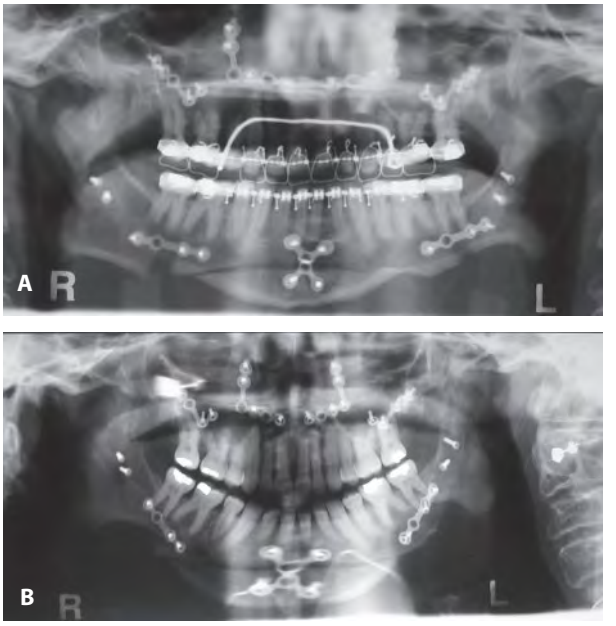


FIGURE 61-5. **A** and **B,** Rigid fixation failure 2 weeks after bimaxillary surgery.

NONUNION OF THE MANDIBLE

Nonunion or delayed union of the mandible may be due to avascular necrosis, insufficient bone contact, instability of the fixation devices, or instability of bone fragments. This complication may occur with SSOs and VROs. VROs warrant specific considerations, not only because a great deal of soft tissue and muscular attachements may be reflected off of the small proximal bone segment but also because the proximal segment may not lie in close apposition to the distal mandible and may, in fact, be unstable without the use of rigid fixation devices. Therefore, any significant parafunctional movement of the jaws or incidental postsurgical trauma in the early phases after surgery may contribute to a nonunion (Figure 61-5).

With regards to the SSO, larger advancements are of a greater concern than small advancements. For advancements greater than 7 mm, additional fixation may be required in order to maintain stability (Figure 61-6). Alternatively, skeletal suspension wires and a brief period of IMF have been shown to provide increased stability.[24]

Delayed union of a mandibular SSO may be treated with a short period of IMF. Alternatively, a second operation may be necessary with the application of additional plates and screws for stability. Nonunion of the mandible after a vertical subcondylar osteotomy may be more of a problem with edentulous patients or when a very short vertical cut is used (Figure 61-7). A second operation, to access the proximal segment from an extraoral approach to properly align and apply rigid fixation, may be necessary for correction of this complication.

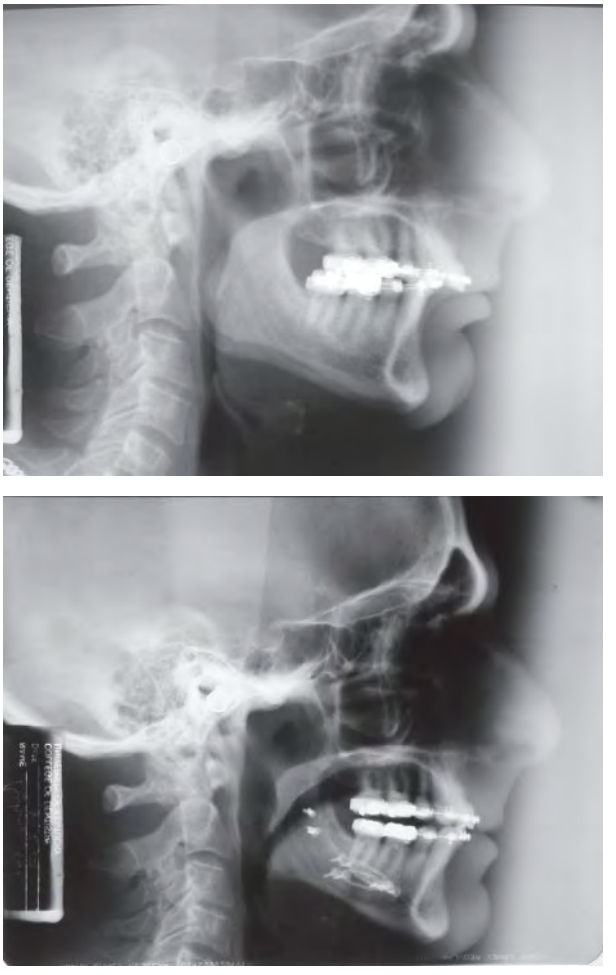


FIGURE 61-6. Large mandibular advancement using both monocortical plates and bicortical screws.

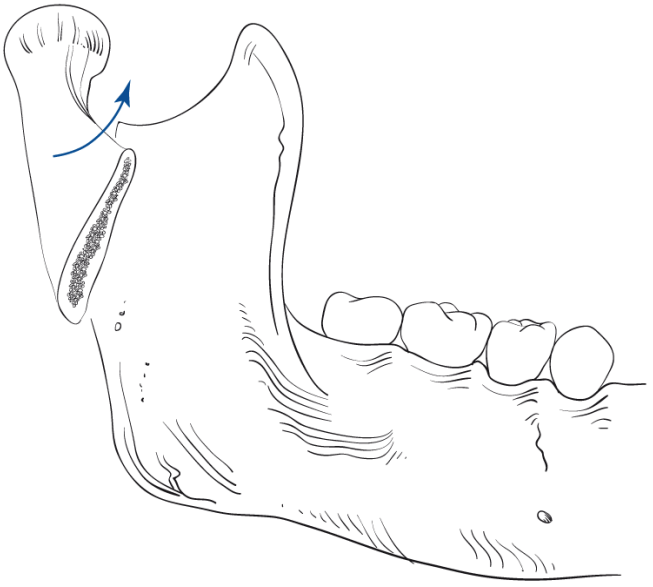


FIGURE 61-7. Severe condylar rotation is common with a short subcondylar VRO.

DENTAL AND PERIODONTAL INJURIES

Dental and periodontal injuries can be secondary to both vascular and nonvascular causes and are most frequently related to treatment planning errors or technical errors made at the time of surgery (Figure 61-8). Segmental procedures in the maxilla and mandible may cause a number of problems, including injury to tooth roots, loss of teeth, need for postoperative endodontic therapy, and the development of significant interdental periodontal defects.



FIGURE 61-8. Injured and exposed root surfaces adjacent to a segmental interdental osteotomy.

Regarding sufficient space between teeth for interdental osteotomies, preoperative orthodontic mechanics with coils can be used to maximize the space between root apices in the area of the planned interdental osteotomy. Periapical radiographs should be assessed for the direction of the root apices, which should be divergent at the planned osteotomy site. A minimum space of 3.0 mm is advocated between teeth for interdental osteotomies (1.0 mm on each root surface and 1.0 mm for the osteotome), and 5.0 mm is recommended for subapical osteotomies above the root apices to avoid injury to dental branches of the nerves.[25] Precise model surgery can greatly reduce the frequency of dentoalveolar injuries. The segmental surgery should be planned so that a minimal amount of bone is removed between osseous segments. Excessive manipulation and torquing of the bony segments should avoided but, when necessary, should be performed toward the root apices rather than at the gingival margin to minimize mucosal lacerations and subsequent periodontal defects. Segments should be tipped apart while maintaining interproximal tooth contacts whenever possible. Copious irrigation of fine fissure burs or reciprocating saw should be used when cutting through the outer cortex of the maxilla. This should be followed by gentle and progressive chisel placement in the oseteotomy site to permit gentle separation of the bone segments. When a bony palatal island is maintained during segmental maxillary surgery, the release of palatal soft tissues should occur under the bony island rather than toward the dentoalveolar segments. After the bony segments are gently separated, small amounts of bone may be judiciously removed toward the root surfaces. When larger wedges of bone are planned for removal, less bone should be removed initially as a wedge than the amount that was treatment planned, with careful additional bone removal as required to approximate the segments. When the bone segments do not fit together passively, the bony interferences may be gently sequentially reduced, because large bony defects after excessive removal of bone usually fill with scar tissue rather than with an osseous union.

With regards to mandibular segmental surgery, it is usually necessary to separate both the buccal and the lingual cortical plates, leaving only a small amount of lingual cortical plate near the cervical portion of the roots to be separated by a fine chisel. The use of a reciprocating, or micro-oscillating, saw is recommended to cut through the mandible, with careful palpation on the lingual surface in order to avoid iatrogenic soft tissue injury. Owing to the dense lingual cortical bone, the use of chisels is more hazardous in the mandible than in the maxilla, where they could fragment the lingual cortex or tear through the lingual pedicle region by applying excessive force once the lingual cortex has been separated. Therefore, chisels and osteotomes should be used with caution after the osteotomy has been completed with a saw, and the bony segments should be separated with minimal tapping of the osteotome through to the lingual surface.

FISTULA FORMATION

Postoperative fistulas in the oronasal and oroantral regions generally result from soft tissue injury at the time of surgery. Fistulas have been reported with isolated segmental osteotomies as well as Le Fort osteotomies.[26] This complication may occur as a result of using rotary instruments or saws or performing osteotomies that perforate the palatal mucosa at the time the segmental osteotomies are completed. Impingement of soft tissue in the segmental osteotomy site during segment repositioning and fixation may also result in tissue necrosis and subsequent fistula formation. Iatrogenic tearing of the palatal mucosa at the time of attempted tissue stretching may also result in nonhealing defects with the potential for tissue necrosis and communication between the nasal cavity or maxillary sinus and the oral cavity. This may occur most commonly when an osteotomy is created in the midline of the maxilla, where the palatal mucosa is extremely thin, with maxillary segmental expansion movements that stretch the midpalatal tissue, leaving a resultant tissue deficiency and nasal communication (Figure 61-9).

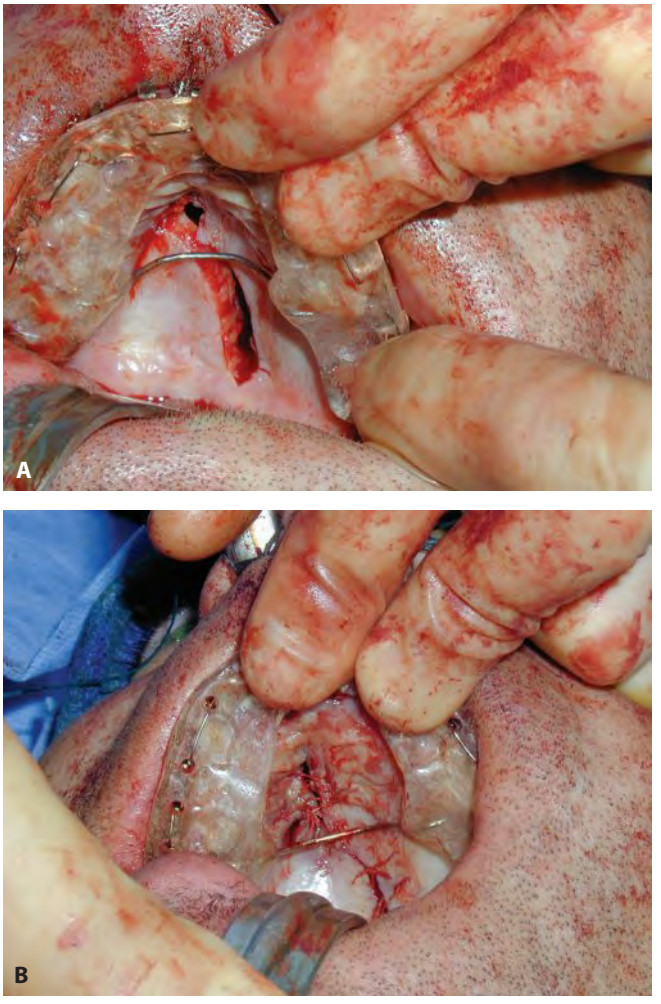


FIGURE 61-9. **A,** Oronasal communication during a two-piece Le Fort I osteotomy. **B,** Palatal mucosal release and sutures with good oral hygiene led to spontaneous closure.

Despite alterations in the method of osteotomy, careful soft tissue manipulation at the time of surgery in an attempt to prevent tissue perforation is the best method for prevention of postoperative fistula formation. When expansion is required, the palatal mucosa can be incised with two parallel incisions just medial to the greater palatine foramen; bony separation then occurs in the midpalatal region.[26] The tissue can stretch and expand in an area removed from the location of bony separation in the midline. An alternative technique involves making paramedian osteotomies in the nasal floor immediately adjacent to the lateral nasal walls. The overlying palatal mucosa is thicker soft tissue and somewhat more elastic than the thin midline palatal mucosa.[27] If a small tear of the palatal mucosa is noted after a bony cut, care should be taken to release the palatal tissue from above before expansion of segments to allow laxity and closer approximation of the mucosa to prevent a large soft tissue defect.

When a fistula is noted postoperatively, several measures may be used to allow the fistula to close spontaneously, and the prevention of sinus and nasal infections is essential. This may include systemic antibiotic therapy, nasal decongestants, and consideration for the provision of nasal drainage, if necessary. It may be advisable to construct an appliance that will obturate the fistula without placing pressure on the surrounding tissues that can contribute to spontaneous closure by providing a scaffold for tissue migration and also by reducing food and bacterial contamination at the site. Care must be taken to ensure that there is not excessive pressure on the palatal mucosa from a palatal obturator splint that can result in decreased vascularity, with further loss of soft tissue and underlying bone, creating a larger hard and soft tissue defect to address with surgical correction. If local measures, appropriate medical therapy, and fistula obturation have been unsuccessful, surgical closure of the fistula is generally required.

When considering closure of a fistula associated with segmental maxillary surgery, it is recommended that at least 6 months have elapsed to allow for complete revascularization of the individual maxillary segments, because a fistula may continue to close for 8 to 12 weeks, leaving a smaller soft tissue defect to correct surgically. Initially, a soft tissue flap should be raised from an area farthest from the bony segments with the least potential for loss of vascularity in the transfer. Buccal advancement flaps or local palatal island flaps may be used; however, if a large segment of the maxilla was involved with a significant loss of soft tissue, distant flaps should be considered, including the buccal fat pad, temporalis myofascial flap, or a tongue flap to transfer a large amount of well-vascularized soft tissue to the area of vascular compromise.

MANAGEMENT OF VASCULAR COMPROMISE

Objective measures to assess vascular compromise are limited to Doppler flowmetry of the maxillary gingiva that can be used to detect subtle decreases in the maxillary gingival

blood flow during and after a Le Fort I osteotomy[28]; however, its use is limited in the clinical setting and is usually reserved for research studies. The early clinical indications of vascular compromise after orthognathic surgery include blanching or a "dusky gray" color of the attached gingiva followed by a cyanotic appearance of the attached gingiva and adjacent unattached mucosa.[29] These overlying soft tissues can be an indicator of underlying bony involvement. Three typical vascular compromise scenarios are possible: (1) loss of vascularity to the soft tissues, with maintenance of bony perfusion; (2) loss of vascularity to the bone, with maintenance of soft tissue perfusion; and (3) loss of vascularity to both the bone and the soft tissues. If cyanosis is noted in the immediate postoperative period (hours to days after surgery), IMF, if present, should be released and the oral cavity should be inspected for stretching, kinking, or constriction of palatal soft tissues. Splints must be carefully evaluated to identify areas of excessive pressure on palatal soft tissues by the appliance and should be removed to evaluate the underlying tissues directly. If removal of IMF and recontouring or removal of palatal splints is not helpful or if the tissue is already necrotic, supportive care is necessary to attempt to minimize the amount of hard and soft tissue loss; however, eventual loss of the soft tissue and exposure of the underlying bone is an expected occurrence in these cases. In severe cases, any portion of a retained necrotic segment of palatal mucosa should be treated as an intraoral free graft or biologic dressing for the underlying bone, with meticulous daily irrigations with or without packing of the wound. As with all tissue grafts, this mucosa should be secured, because this stability may allow some degree of spontaneous revascularization. HBO therapy may be helpful to minimize bone loss while promoting neovascularization or at least limit the propagation of the area of necrosis. After sufficient time has elapsed to allow establishment of the specific zones of necrosis and dental injuries, débridement of the necrotic tissues and removal of nonvital teeth should be performed. In a delayed fashion, surgical reconstruction of the resultant hard and soft tissue defects varies based upon the size of the defects but may include autogenous bone grafting, free tissue transfer with a fibula flap and associated soft tissue, and prosthetic reconstruction with dental implants and implant-supported prostheses.

INFECTION

The incidence of postoperative infections after orthognathic surgery is low.[16,30–33] Chow and coworkers[30] revewed 1294 consecutively treated patients who underwent one- and two-jaw orthognathic procedures, and the complication rate was 9.7% overall, with a 7.4% incidence of postoperative infections. They noted that those patients who received only a single preoperative dose of antibiotic had a higher rate of infection than those with longer duration of perioperative antibiotic management, and as expected, two-jaw procedures had a higher incidence of infection than single-jaw procedures. The incidence of infection was almost equal between maxillary and mandibular osteotomies. In a series of 57 patients who underwent an SSO of the mandible, there was 1 case of infection, resulting in significant relapse of the orthognathic procedure.[31] Becelli and associates[32] had a 2.48% incidence of infection when examining 482 sides of patients who underwent a mandibular SSO. These occurred within the first month after surgery and were treated effectively with systemic oral antibiotics, although several of patients required screw removal and wound débridement. Bouwman and colleagues[33] reviewed 700 consecutive cases of a BSSO for advancement of the mandible, and in 15 cases, bicortical screws required removal owing to infection. Teltzrow and coworkers,[16] in a study of 1264 patients, noted a 2.8% incidence infection after a BSSO requiring extraoral incision and drainage, despite a protocol including a single dose of perioperative intravenous antibiotic.

As seen from this review, the incidence of infection with orthognathic surgery is low and varies from one study to another, with an overall incidence of perhaps 1% to 5% consistent with clean-contaminated wounds. In addition, most infections occur within the first few weeks after surgery because many of these are due to an infected hematoma, which are usually resolved with an intraoral incision and drainage procedure in the office. Infections due to loose hardware may occur early if there was insufficent stability at the time of surgery but also may be seen several months after the surgery following bony remodeling and resultant screw loosening.

Osteomyelitis after orthognathic surgery is especially rare. Teltzrow and coworkers[16] had one patient develop osteomyelitis after a BSSO, which resolved after decortication and long-term systemic antibiotic therapy.

NERVE INJURY

Maxillary Sensory Injuries

Sensory injuries in the maxilla after orthognathic surgery have not been studied as extensively as those seen with mandibular surgery. With a carefully placed low circumvestibular incision combined with gentle soft tissue retraction, nerve injury is generally inconsequential and limited to the terminal branches of the infraorbital nerve (V2). Spontanous recovery of sensation to the lip, cheeks, and nose usually occurs within 2 to 8 weeks after surgery. In contrast, paresthesias due to damage of the sensory nerve supply to the teeth, gingiva, and mucosa are more common. Decreased sensation to the mucosa is typically transient and normal sensation commonly returns within 6 to 12 months. On occasion, patients will experience permanent numbness intraorally on the palate and buccal gingiva, and therefore, in order to preserve sensation to the palate, some authors advocate preservation of the greater palatine neurovascular bundle whenever possible.[29] Also, failure of the teeth to respond to electric pulp stimulation may be temporary; however, permanent loss

of response to electrical, hot, or cold stimulation is not unusual and does not necessarily represent a tooth that requires endodontic therapy. The clinician must differentiate between a nonvital tooth and one that does not respond to stimulation but still has an intact blood supply. Many studies have shown decreased blood flow to the maxillary teeth during and after Le Fort osteotomy with pulpal blood flow studies, with return to normal within several months. A tooth that shows periapical radiolucency or a parulis with fistula formation upon examination may be a candidate for root canal therapy.

Mandibular Sensory Injuries

Sagittal Split Osteotomy

Although traction injuries to the inferior alveolar nerve may occur commonly during SSOs, frank transections of the inferior alveolar nerve occur less commonly.[34] The most likely time for this to occur is during the actual splitting process of the osteotomy. When the segments are being separated, care should be taken to visualize and protect the nerve, when possible. If the nerve is in the distal segment or encased in cortical bone, appropriate steps should be taken to release it. This may be as simple as releasing the nerve with a periosteal elevator from a medullary bone, or it may require additional bone cuts to release it from cortical bone. Laborious efforts to release the nerve should be avoided because this may cause worse iatrogenic injury to the nerve. One study suggested that low mandibular corpus body height and inferior position of the nerve in the class II retrognathic patient population may increase the risk for injury.[34] With a transection injury, repair of the nerve with one or more nonresorbable 6-0 to 8-0 monofilament nylon sutures placed in the epineurium has been recommended.[35] It may be necessary to perform a decortication laterally to release the nerve to the mental foramen for mobilization, especially if the plan is for a significant mandibular advancement that will further separate the proximal and distal nerve stumps and prevent direct repair without tension. It should be noted that one large series had a 3.5% incidence of transection of the inferior alveolar nerve, which occurred anterior to, or in the third molar region, in all instances.[15] Certainly, this is the location where the inferior alveolar nerve is most lateral and the buccal cortex of the mandible is thinnest, making the nerve most vulnerable to injury in this location. Nerve endings were approximated in nine patients by positioning the segments in close proximity, but without the need for epineurial sutures. The length of follow-up for these patients was 2 to 5 years, and all of the patients had some return of sensation to the normal inferior alveolar nerve distribution. Whether this represented spontaneous regeneration or new growth from the cervical plexus is unknown.

Conversely, if the transection occurs at the vertical bony cut, immediate repair may be difficult, and as mentioned, it may be necessary to expose more of the nerve in the distal segment. When excessive tension on the nerve repair site is present, the nerve may have to be exposed distally to the mental foramen to allow a tension-free repair. The need for such an extensive procedure needs to be weighed against the primary goals of the orthognathic surgical procedure.

Injury to the inferior alveolar nerve, in the absence of a transection, is frequently associated with the SSO of the mandibular ramus. Risk factors for neurosensory disturbance include advanced patient age, a simultaneous genioplasty procedure, and an increased magnitude of advancement.[36,37] Multiple techniques have been suggested to prevent these injuries, including varying the osteotomy design, cautious chisel placement, meticulous dissection technique, decompression of the lateral fragment if indicated, and perioperative steroid use. Vigorous medial retraction of the neurovascular bundle inferiorly during the horizontal osteotomy may cause the inferior alveolar nerve to be compressed against the lingula, and decreased intraoperative nerve conduction has been demonstrated.[34,38] Retraction on the medial aspect of the mandible should be done carefully to avoid compression nerve injuries. The most ideal location to create the lateral buccal (vertical) cut is in the first and second molar region where the cortex is the thickest, the mandible is the thickest, and the nerve is farthest from the lateral cortex.[15,39] Other recommendations to prevent nerve injuries are based on clinical experience, but no controlled studies have been done to determine whether one way is preferable over another.

Injury to the lingual nerve during an SSO can occur, but it is unusual.[40,41] The lingual nerve may be injured by overpenetration of bicortical screws used to rigidly fixate the osteotomy. The course of the lingual nerve near the medial surface of the mandible varies; therefore, any dissection on the lingual aspect of the mandible in the third molar region may temporarily or permanently injure this nerve.[42] As with inferior alveolar nerve injuries, lingual nerve injuries should be carefully followed and documented. If the nerve is visualized and has been transected, it should be repaired at the time of surgery. In general, symptoms of lingual nerve injuries after an SSO are rare and typically resolve with time.

VRO and Other Ramus Procedures

Although the vertical oblique osteotomy (VRO) of the mandibular ramus is an alternative to the SSO for horizontal mandibular excess and mandibular setback, this procedure may also result in permanent injury to the inferior alveolar nerve. The incidence of permanent paresthesia after an intraoral vertical subcondylar osteotomy has ranged from 9% to 11%.[43,44] Endoscopic approaches to the ramus may have a different incidence of nerve injury, but experience with these techniques is limited.[45] The incidence of nerve injury with the VRO procedure is mostly associated with the imprecise location of the inferior alveolar nerve. Traditionally, it has been suggested that a bulge on the lateral surface of the mandible (the antilingula) corresponds to the position of the lingula on the medial aspect of the mandible. Therefore, creation of an osteotomy posterior to the antilingula should theoretically avoid injury to the inferior alveolar nerve medi-

ally. Hogan and Ellis[46] showed that the bulge on the lateral surface of the ramus corresponds to the insertion of muscles and tendons and had no relationship to the medial position of the inferior alveolar nerve. Aziz and associates[47] showed in a cadavaric study that, in most instances, the position of the lingula was located posteroinferior to the position of the antilingula. In only 33% of the cadaver specimens, the lingula was found anterior to the antilingula. They noted that if the osteotomy was made 5 mm posterior to the antilingula, there was no risk of damage to the neurovascular bundle. Therefore, to avoid an injury to the inferior alveolar nerve, the saw blade should parallel the posterior border of the ramus and be placed 5 mm posterior to the bulge of the antilingula on the lateral aspect of the mandible. If, despite these precautions, postoperative paresthesia occurs, it should be carefully followed, although microneurosurgical repair is usually not warranted nor is it technically feasible.

Motor Nerve Injury

Injury to the branches of the facial nerve is much more common with extraoral approaches to the mandible than with intraoral orthognathic surgery. However, there have also been multiple reports of facial nerve injuries resulting from SSOs and VROs.[48–51] In one series that studied 1747 cases of SSO procedures, the incidence of facial nerve injury was 0.26%.[50] The degree of injury can vary from partial loss of motor function to total facial paralysis and is more often seen after a setback of the mandible than with mandibular advancement.[49] The possible causes of facial nerve injury during SSO setback include impingement on the nerve when the distal segment is moved posteriorly, fracture of the styloid process and subsequent displacement, and introduction of retractors behind the ascending ramus with impingement on the nerve. Most of the reported cases of facial nerve injury occurred with mandibular setbacks without use of the Hunsuck modification,[52] so the horizontal osteotomy extending to the posterior border of the mandibular ramus may have caused direct facial nerve injury. However, the most likely cause of facial nerve injury is pressure on the nerve trunk, either by the posteriorization of the distal segment or by the inadvertant placement of retractors behind the posterior border of the mandible (Figure 61-10). To prevent this complication, the horizontal osteotomy should extend just distal to the inferior alveolar neurovascular bundle and lingula into the retrolingular fossa when an SSO is used for mandibular setback. If a horizontal osteotomy extends to the posterior border, bony recontouring should be performed proximal to the lingula to prevent posterior overlap of the distal segment in the region of the posterior border of the ramus and the facial nerve. Unfortunately, the magnitude of the mandibular setback that causes this problem is unknown (possibly > 8 mm) and probably varies owing to variations in individual patient anatomy. Also, care should be taken when any retractors are placed behind the mandibular posterior border during all ramus osteotomy procedures.

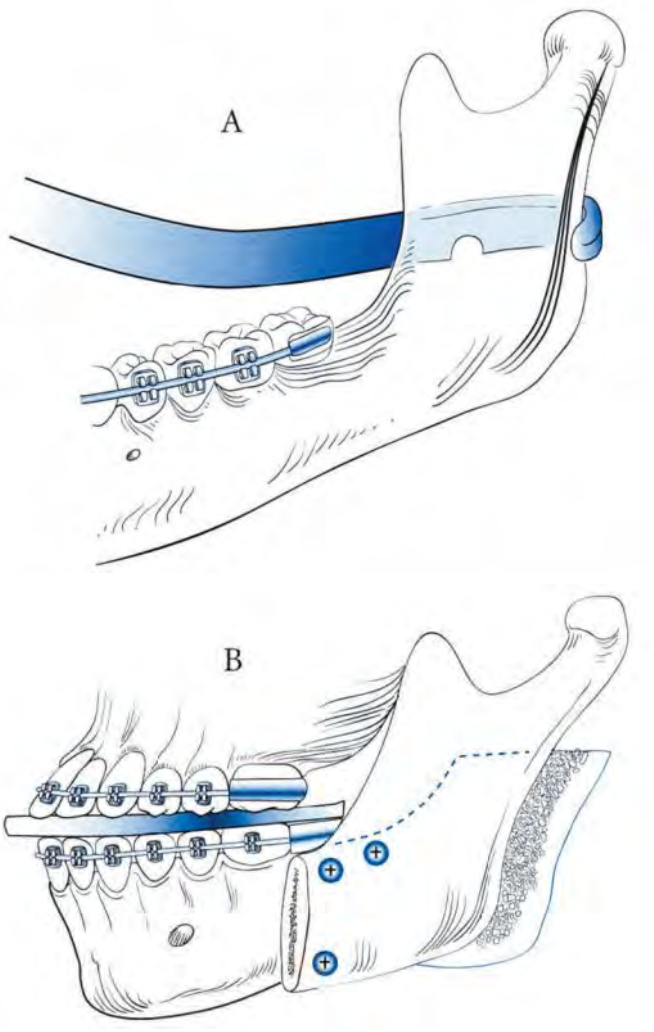


FIGURE 61-10. Causes of facial nerve injury with sagittal split osteotomy (SSO). **A,** Medial retraction behind ramus. **B,** Extension of the distal segment beyond the proximal segment posteriorly.

When a facial nerve paralysis occurs after surgery, a number of electrical tests can be used to determine the depth of injury and subsequent therapy. Electroneurography (ENOG), a study of peripheral nerve conduction, or electromyography (EMG), which is the detection and evaluation of electrical potentials from muscles, may be used diagnostically.[53,54] It is important to distinguish between an injury that causes segmental demyelination and one that causes distal wallerian degeneration. With axonal interruption, the ability to transmit a neuronal impulse is lost over a period of 5 to 7 days. When axonal degeneration occurs, the prognosis for complete spontaneous recovery is poor. When a high-grade nerve injury is noted, surgical exploration should be considered to rule out a partial or complete transection of the nerve. As long as the axon remains intact at the site of the conduction block, the nerve will continue to respond to electrical stimulation distal to the injury site even though paralysis may be present. Evoked electromyography (EEMG), a test in which the degree of muscle twitch elicited is recorded, has

been used as a prognostic test.[55] If the response to EEMG remains greater than 25% at 5 days, the injury is considered mild and the prognosis for spontanous recovery is good.[55]

Clinical management of the patient during the recovery phases of the paralysis can vary depending on the specific nerve branches, and the type of nerve injury, involved. When the patient has difficulty obtaining full eyelid closure, an eye patch and methylcellulose eye drops may be useful. Physical therapy such as heat, facial massage, and facial exercises performed twice a day have been suggested for neurosensory reeducation. Facial cream should be massaged into the skin around the eyes and mouth and over the midface, ideally using an electrical vibrating device. Biofeedback neurosensory exercises may consist of raising the eyebrows, blowing the cheeks, and grinning while visualizing the attempts in a mirror. Even though no facial movement may be noted, intact nerve fibers will be activated and the exercise will help to maintain muscle tone through electrical and mechanical stimulation. Systemic corticosteroids had been used orally, intramuscularly, and intravenously for facial nerve paralysis in an attempt to decrease perineurial edema from the injury.

NASAL AND SINUS CONSIDERATIONS

Alterations in Nasal Form: Septal Deviation

Le Fort surgery with maxillary repositioning requires manipulation of nasal components, associated facial musculature, and the maxillary sinus. As a result, alterations can occur with the internal nasal anatomy including position of the turbinates, nasal septum, and internal nasal valve. Adverse effects of maxillary osteotomies on the alar bases, nasal tip, supratip depression, and upper lip may result in an unaesthetic postoperative facial appearance.[56–58] The bony and/or cartilaginous nasal septum may be deviated before surgery, at the time of surgery, or during extubation. Therefore, the septum should be inspected before and after surgery. During a Le Fort I osteotomy, it is possible to align the septum at its inferior anterior caudal end of the nasal crest of the maxilla posterior to the anterior nasal spine region. At surgery, the septum is disarticulated from the entire nasal crest of the maxilla, and most commonly with maxillary impaction, the maxilla will encroach upon the presurgical vertical dimension of the nasal septum. Owing to this expected change during maxillary impaction, attention must be given to careful positioning of the septum at the time of surgery. Failure to do so may result in septal deviation and nasal airflow obstruction, or the end result may be an abnormal position of the columella and nasal tip deviation.[57] Several techniques are used to avoid septal deviation during superior maxillary repositioning, including resection of an appropriate portion of the inferior aspect of the bony (vomer) and cartilaginous nasal septum or creating a deep groove in the superior aspect of the maxilla in order to prevent septal deviation as well as stabilize the septum in the midline. In segmental maxillary osteotomies, creating a bony island with parasagittal palatal cuts may eliminate posterior superior pressure of the nasomaxillary crest on the nasal septum, although this will not eliminate pressure from the anterior portion of the maxilla on the nasal septum.

When septal deviation is recognized postoperatively, three choices for management should be considered (Figure 61-11), including immediate manipulation with manual repositioning, reoperation with reduction of the caudal septum, or septoplasty at a later surgical procedure, if indicated. If appropriate preventive measures for septal deviation were accomplished at the time of surgery, but the nasal septum appears to be asymmetrical, this may result from postoperative edema that may resolve spontaneously or may have occurred during extubation. In the early postoperative period, manipulation with a blunt instrument (e.g., Seldin elevator) placed within the nasal cavity on each side of the base of the nasal septum may allow for manual septal repositioning into a midline position (see Figure 61-11B and C). If rigid fixation has been used and the patient has no airway difficulties, short-term unilateral nasal packing on the side of the deviation may be considered. If septal deviation is due to lack of appropriate prevention during surgery, or if postoperative manipulation fails to result in correction, immediate reoperation with caudal septal resection, with or without the need to remove the rigid fixation for surgical access, may be indicated. If these approaches are not acceptable to either the patient or the surgeon, and the patient does not have significant airway difficulties or cosmetic concerns, the septal deviation may be reevaluated at a later date with consideration for a standard septoplasty procedure if necessary after complete resolution of edema and bony and soft tissue healing.

Alterations in Nasal Form: Internal Nasal Valve

An area of concern in maxillary surgery is alteration of the internal nasal anatomy, nasal airway resistance, and breathing patterns as a result of maxillary surgery. Expansion of the maxilla has shown little change in the nasal airway resistance, and many patients remain obligate mouth breathers even after maxillary expansion surgery.[59] Of greater concern is the possibility that superiorly repositioning the maxilla may decrease the normal nasal airflow owinge to an overall decreased dimension of the nasal cavity. Several studies have documented that the reverse is true[60–62] and that superior maxillary repositioning increases the nasal cross-sectional area, decreases nasal airway resistance, and increases nasal breathing. The explanation for this decrease in nasal airway resistance and improved airflow is related to an alteration in configuration of the angulation of nasal valve area.[60–62] The internal nasal valve is formed by the junction of the nasal septum and the upper lateral nasal cartilages and the normal angle formed in this region is 10 to 15 degrees. In contrast, the "external" nasal valve is essentially formed by the nares, which are composed of the floor of the nose and the soft tissues within the nasal sill. The increase in alar base width that



FIGURE 61-11. **A,** Deviated nasal septum postoperatively. **B,** Manual manipulation. **C,** Deviation correction.

results from elevating the soft tissues to expose the maxilla causes a slight widening of the internal nasal valve, increasing the angle of the internal nasal valve (>20 degrees), thereby reducing nasal airway resistance. Owing to the fact that this internal nasal valve is at the smallest cross-sectional area of the nose, slight alterations in this area have a significant effect on nasal breathing, whereas changes in larger intranasal areas have a much less effect on nasal airflow; this same phenomenon has been demonstrated in patients with cleft palates.[63] The effect of maxillary impaction on nasal airway resistance is determined preoperatively using the Cottle test, performed with lateral digital traction on the malar region to open the angle of the internal nasal valve while occluding the contralateral naris; an improvement in nasal airflow is typically expected with this maneuver.

Alterations in Nasal Form: Alar Base

In addition to the internal nasal changes, facial aesthetic changes in nasal morphology may result from maxillary surgery. Failure to properly manage the nasal septum, paranasal musculature, and labial mucosa may result in undesirable facial aesthetic results. Adverse changes in nasal and perioral configuration after maxillary surgery may include excessive alar base widening; increased prominence of the alar groove; upturning of the nasal tip (with an obtuse nasolabial angle); flattening, shortening, and thinning of the upper lip; and downturning of the labial commissures.[57] These complications may also be compounded by internal deviation of the nasal septum, or asymmetrical positioning of the columella and nasal tip due to septal deviation. These types of problems are difficult to manage and are best treated with prevention. The need to control alar base width and the necessity of reconstruction of paranasal and perioral musculature have been described previously.[56] It should be remembered that any maxillary movement results in alar base widening due to musculature reflection off of the anterior maxilla, and maxillary advancement contributes to even further alar base expansion. Therefore, all Le Fort procedures should incorporate a closure technique that includes an alar base cinch suture to control the degree of alar base widening and attempt to reestablish the presurgical alar base width. Undesirable lower lip changes can be prevented by the use of a low vestibular incision, and the use of a V-to-Y or double V-to-Y closure when indicated to preserve or increase upper lip vermilion display.

Postoperative Sinus Disease

Postoperative complications related to the maxillary sinuses are primarily a result of sinus infections, alteration or interruption of the sinus drainage system, and the persistance of an oroantral fistula. Although many patients experience sinus discharge or excessive nasal drainage and other sinus symptoms in the immediate postoperative period, true perioperative infections of the sinus region and the development of chronic sinusitis after Le Fort surgery are rare.[64,65] Between 2 and 6 months after surgery, there will be normalization of the bony and soft tissue structures in over 55% of the patients,[65] although, at 6 months, as many as 30% of Le Ffort osteotomy patients will show some latent sinus mucosal edema and lack of normal mucociliary function.[65] The pseudociliated stratified ciliated columnar epithelium of the sinus mucosa may also undergo a squamous metaplasia after surgery that may lead to alterations in normal sinus function.

Despite the rarity of postoperative sinus infections, there are several potential causes of infections after maxillary orthognathic surgery. The formation and retention of substantial hematomas in the sinus cavity is an obvious source of potential infection. Perioperative intravenous antibiotic prophylaxis with continued postoperative oral systemic antibiotics will help to prevent the blood clot from becoming

infected before resorption. Other potential causes of infection in the sinus include preexisting sinus disease, a history of smoking, dental infection due to trauma to the teeth during the surgery (segmental procedures), soft tissue ischemia and avascularity (again, with segmental surgery), and the presence of debris or retention of foreign bodies within the sinus. Foreign objects such as wires, bone plates, or screws are rarely the isolated cause of a sinus infection and do not appear to cause a significant increase in the incidence of infection after maxillary surgery.[64]

Preoperative assessment of patients presenting for maxillary surgery should include a history and clinical examination, with careful attention to symptoms of any preexisting maxillary sinus disease. Evaluation of preoperative radiographs may provide some information regarding the presence of occult sinus pathology. Postoperative management of sinus infections should include appropriate antibiotic therapy verified by culture and sensitivity, decongestants, intranasal vasoconstrictors, and irrigation of patent fistulae if present. Generally, sinus drainage can be managed to resolution within 10 to 14 days with these techniques. When a sinus infection is refractory to medical treatment, endoscopy, and possible surgical drainage of the sinus should be considered, and this sinus disease should be managed in a manner similar to treatment for patients with sinus disease who have not had Le Fort surgery with standard techniques.

UNANTICIPATED MANDIBULAR OSTEOTOMY FRACTURES

Management of Bad Splits

The intraoperative management of unusual osteotomy fragments (bad splits) is seen more frequently with mandibular procedures, especially the SSO, in which the osteotomy design has two right-angle turns to the ostetomy in which inadvertent splits in the bone may occur. The creation of additional osteotomy segments may occur on either the proximal or the distal bone fragments. The incidence of unfavorable fractures with a BSSO is 1.9% to 2.2%, with a slightly higher incidence when the third molars are present or have not been removed at least 9 months before surgery,[66,67] and depending upon the individual patient anatomy, these bad splits may occur in either the proximal or the distal segment. The standard method for intraoperative management of a bad split is to complete the separation of the proximal and distal segments and then assess the pattern of inappropriate fracture. Typically, bad splits can be addressed adequately via the same intraoral access used for the procedure, and transfacial access is rarely used to allow placement of additional fixation of bony segments. Management will vary depending on the size (small vs. large) as well as the specific location of the fragment, and some bad splits require no treatment. It must first be determined where the fracture deviated from the desired split, and of course, how to possibly prevent a similar occurrence on the other side or in the future. Often, it is necessary to remove the free segment to gain access to the remaining mandible. A saw or a bur may be used to create a groove on the intact mandible or individual fracture segments so that the SSO can occur along the original planned lines of osteotomy; chisels can be used to complete the desired split. The key to management is to visualize the damage and determine the precise orientation of the problem geometrically. Once the split is successfully completed, the distal segment can be advanced to its desired position. The position and size of the remaining fragments may make positioning of the condylar segment difficult. Segments are sequentially stabilized with plates and screws or wires to the remaining bony fragments. An extraoral approach may be an option, but is usually not necessary. The following examples will illustrate the management of specific unanticipated osteotomy fractures.

Proximal Segment Buccal Plate Fracture, Partial

The difficulty in managing free bone segments depends on the location and size of the fractured pieces of bone. A fragment may shear off the lateral aspect of the proximal segment, thereby leaving the distal mandible intact. Whenever a buccal fragment shears off, the usual cause is an inadequate bone cut at the inferior border of the lateral vertical osteotomy. The split must then be completed, and as mentioned, this can be done by making a deep groove on the inferior border and connecting it with the previous osteotomy as it extends down the external oblique ridge. With gentle manipulation and prying, and when necessary, sectioning bone, the segments can be separated as originally planned. Stabilization of the distal segment with application of IMF will allow easier stabilization of the free buccal plate fragment with screws and plates. Figure 61-12 shows a buccal plate fracture

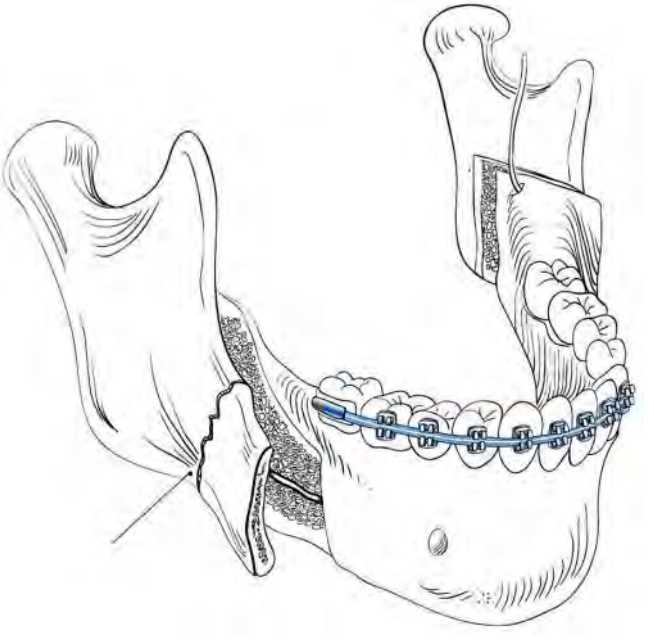


FIGURE 61-12. Proximal segment buccal plate fracture.

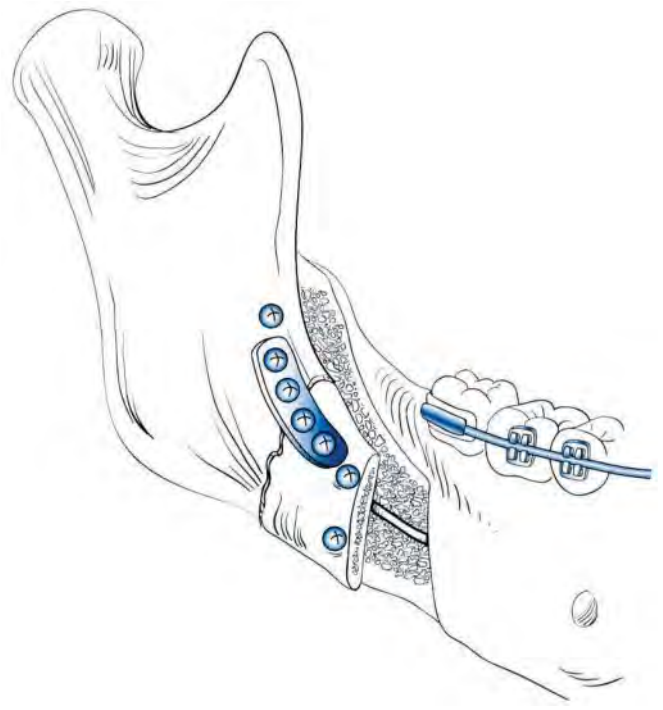


FIGURE 61-13. Rigid fixation used to stabilize the free fragment to the proximal segment; then bicortical fixation of the SSO.

and the main portion of the mandible is intact before completion of the split, so the free segment is stabilized with plate and screw osteosynthesis, as shown in Figure 61-13. Condylar position is not difficult to establish when a proximal segment is large enough to be positioned with adequate overlap and contact with the distal segment in its new position, and bicortical screws can be used, as usual, between the areas of contact of the two segments. With a small free buccal plate fragment, it is frequently easier to place a plate on the segment on the back table and then stabilize it to the proximal segment, although a very small buccal plate fracture segment may not need to be replaced, especially if it does not occur near the inferior border that might result in an unaesthetic postoperative result with excessive antegonial notching.

Proximal Segment Buccal Plate Fracture, Complete

When the buccal plate fracture occurs more superiorly, or there is a plan for a large mandibular advancement such that there will be no contact between the proximal and the distal segments when the occlusion is corrected (Figure 61-14), a different approach must be used. When the condyle and coronoid process are contained in the same proximal segment, owing to a horizontal osteotomy that is created too deeply on the medial ramus, and simulating a horizontal osteotomy of the mandible, control of condylar position is more difficult. The large proximal fragment should be stabilized with rigid fixation either in situ preferably or on the back table (but this

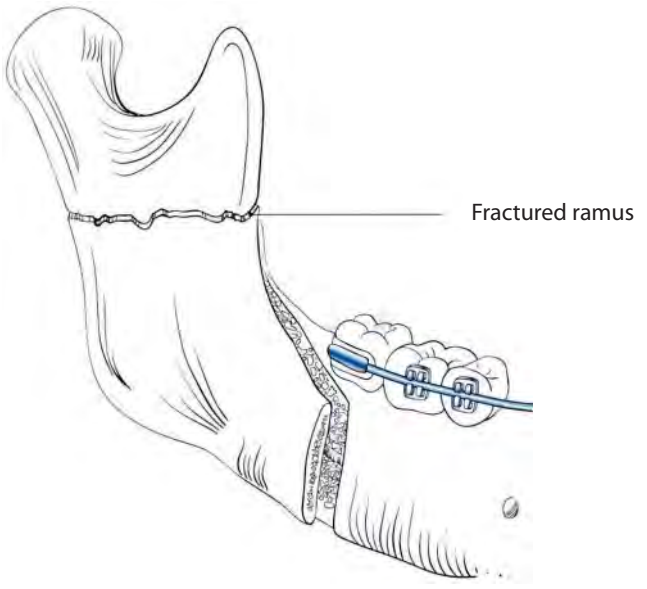


FIGURE 61-14. Horizontal fracture of the proximal segment.

would require disarticulation); the fragment should be then stabilized to the remainder of proximal segment (Figure 61-15). This may requires a percutaneous transbuccal approach for screw placement or the use of right-angled instrumentation. At this point, the proximal segment is easier to manipulate as a complete unit, and it can be fixated to the distal segment into its planned position. This can be accomplished with the use of several plates or bicortical screws. Control of condylar position may be established by posterior, superior, and vertical pressure on the proximal segment, followed by the use of a proximal-distal clamp (e.g., Jeter-van

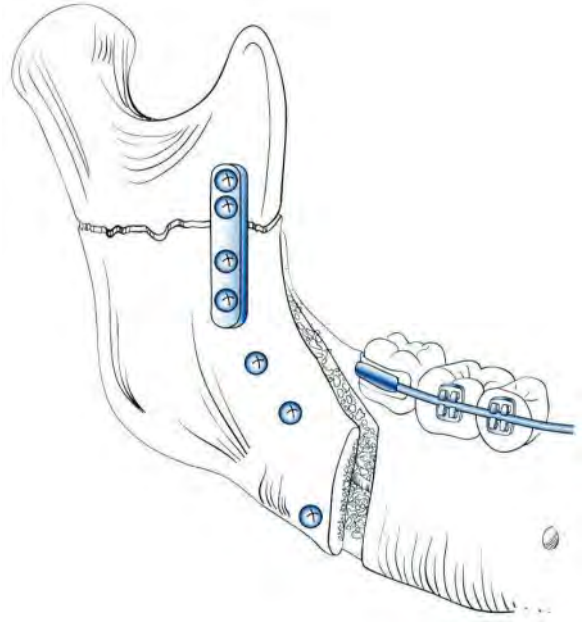


FIGURE 61-15. Fixation of the ascending ramus fracture with a plate and screws; then fixation of the SSO with bicortical screws.

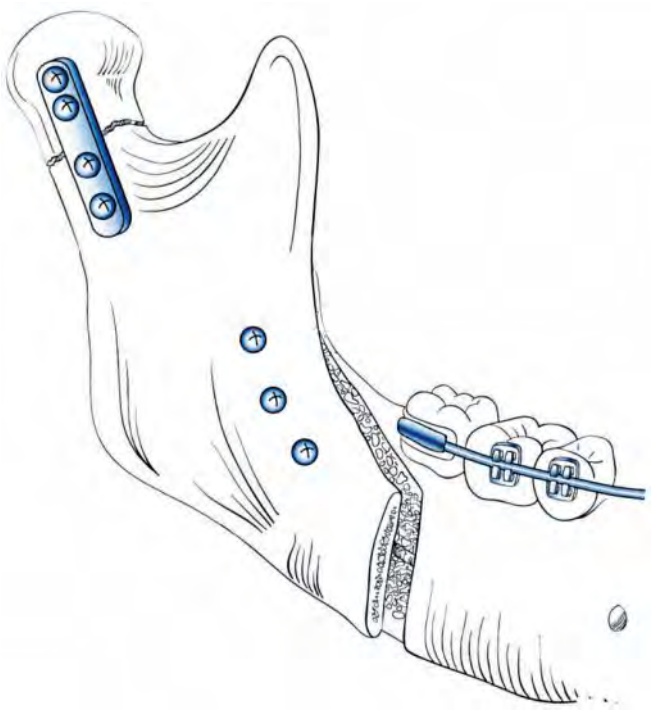


FIGURE 61-16. Fixation of the subcondylar osteotomy with a plate and screws; then bicortical SSO fixation.

Sickels) before placement of bicortical screws or by using the clamp on the coronoid process to stabilize the proximal fragment before screw placement.

Another bad split occurs with the creation of a subcondylar fracture with a condylar fragment separated from the proximal segment. In this case, the condyle must be rigidly fixated to the proximal segment to create a single proximal segment, and then the proximal and distal segments can be stabilized as usual for an SSO (Figure 61-16). Correct condylar positioning is extremely difficult to achieve in this setting, especially with a small condylar stump. The use of transbuccal percutaneous incisions may assist in the placement of a plate is placed on the condylar fragmant, and then the plate can be used as a handle to manipulate the condyle until holes can be drilled in the distal segment with screw placement and application of fixation. Alternatively, an endoscope-assisted technique can be used to improve visualization and application of fixation in this confined region.

Distal Segment Lingual Plate Fracture

Fortunately, fractures of the lingual plate of the distal segment occur less frequently than fractures of the buccal plate of the proximal segment. The underlying cause is frequently an impacted third molar, or it may be secondary to an incomplete ostetomy of the medial horizontal ramus or of the sagittal component of the SSO with the creation of a wedging effect on the medial aspect of the mandible (Figure 61-17). To prevent this type of inadvertant fracture, it is best to

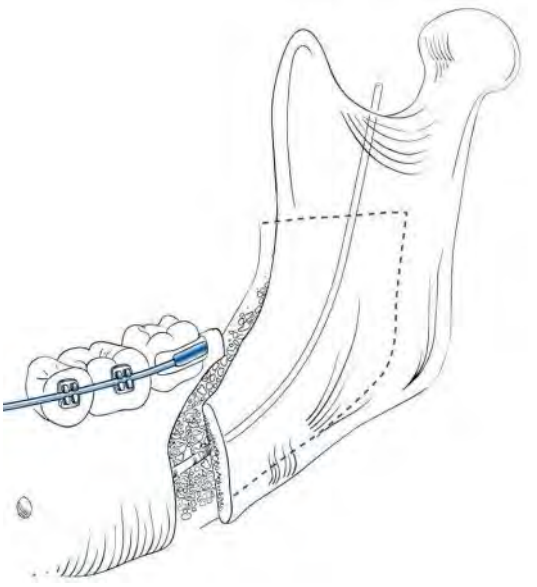


FIGURE 61-17. A lingual split of the distal segment in the third molar region.

remove mandibular third molars at least 9 months before surgery. When an unanticipated fracture occurs, the osteotomy must be completed along the original planned osteotomy lines. Because the resultant free lingual segment is not an obstruction when the osteotomy is completed, it generally maintains a substantial muscular and vascular pedicle attached to it from the mylohyoid muscle. The distal segment is then stabilized with the application of IMF, and the free lingual plate fragment is manipulated into an anterior position in contact with the distal segment and is fixated to the proximal segment with a lag screw technique using one or more bicortical screws. Then, one or more plates or a piece of titanium mesh may be used on the buccal aspect of the osteotomy site for fixation of the proximal and distal segments (Figure 61-18).

DISPLACEMENT OF THE PROXIMAL VRO SEGMENT

Excessive lateral displacement of the proximal segment can occur during a vertical subcondylar osteotomy. Depending on the geometry of the movement of the distal segment, the proximal, or condylar, segment may be displaced either laterally or medially. The desired position is to have the proximal segment in a lateral position to the distal segment of the mandible. Despite moderate flaring of the proximal segment, typically considerable remodeling occurs at the osteotomy site that allows accomodation of this position without any problems. Occasionally, however, the proximal segment will be flared excessively, especially with an asymmetrical movement of the mandible. This can be addressed intraoperatively by removing a second wedge at the sigmoid notch region to allow a more passive relationship of the proximal and distal segment without interferences that result in extreme lateral displacement of the proximal segment (Figure 61-19). Care

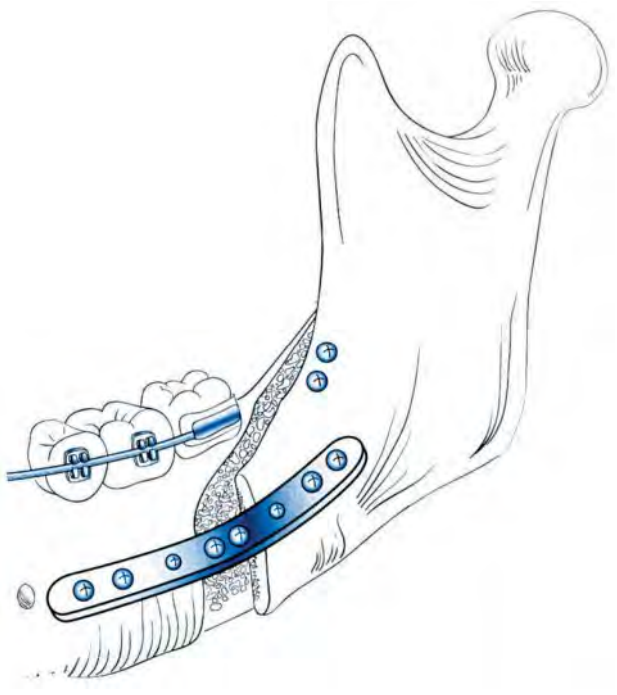


FIGURE 61-18. Two bicortical screws stabilize the free lingual segment to the proximal segment; then monocortical plate and screw fixation of the SSO.

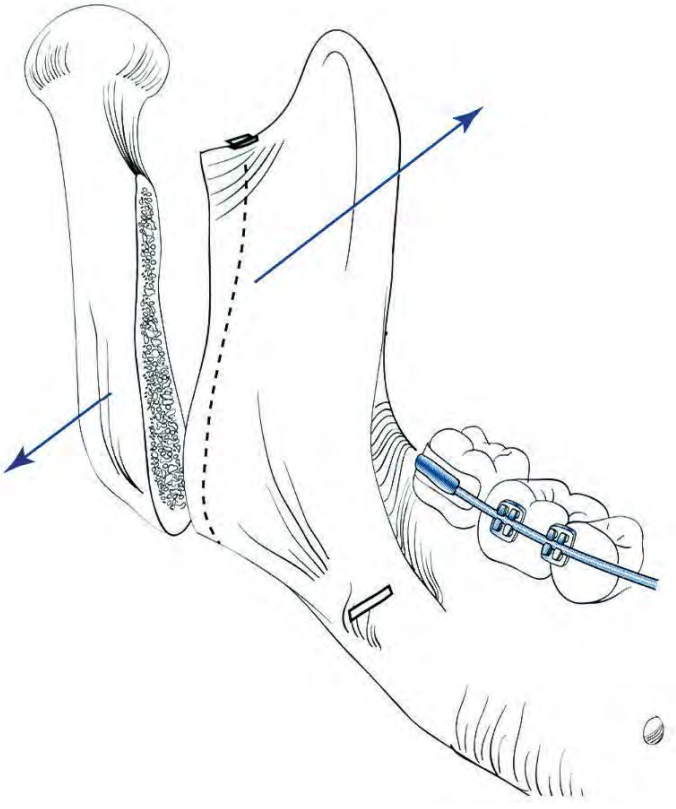


FIGURE 61-19. Excessive flaring of the proximal segment by premature contact at the sigmoid notch; correction by bone wedge removal from distal segment, wider at the sigmoid region than at the inferior border.

must be exercised when performin bony cuts in the sigmoid region because the masseteric branch, or the maxillary artery itself, can be injured iatrogenically. If excessive flaring is noted postoperatively either clinically or radiographically, the segment may be repositioned manually; but if this is not successful, reoperation with open repositioning may be necessary.

In some cases of asymmetry, the rotation of the distal mandible may cause the proximal condylar fragment to be displaced medial to the distal segment. There is a concern that this position may increase the incidence of nerve injury, but this has not been tested clinically, and in general, medial displacement of the proximal segment rarely causes a significant clinical problem. An uncommon patient complaint may be irritation of the posterior pharyngeal region due to contact from the proximal segment; if this occurs, the proximal segment should be repositioned or recontoured or a significant portion should be removed, likely in the inferior aspect of the proximal segment.

Finally, the relationship of the proximal and distal segments may form a butt joint with direct contact despite posterior repositioning of the distal segment. This would result in a clockwise rotation of the condyle that typically does not cause problems, but may contribute to short- and long-term relapse if there is significant tension on the posterior soft tissues with a tendency to displace the segments anteriorly.

PROXIMAL SEGMENT ROTATION

Lack of control of the proximal segment with an SSO can have several clinical effects with both aesthetic and functional consequences. The postoperative muscular influences are such that the proximal segment is pulled anteriorly and superiorly by the temporalis and masseter muscles, while the distal segment is pulled posteriorly and inferiorly by the mylohyoid, geniohyoid, genioglossus, and suprahyoid muscles (Figure 61-20). Therefore, this anterior and superior counterclockwise rotation of the proximal segment and clockwise rotation of the distal segment may result in an unpleasant cosmetic result with flattening of the gonial angle and notching the inferior border of the mandible in the antegonial notch region. This also may cause an unaesthetic protuberance in the cheek region secondary to the lateral positioning of the proximal segment. The type of fixation (wires vs. plates and screws) has been shown to affect the position of the proximal segment both during surgery as well as in the immediate postoperative period.[68]

The most ideal management of a rotated proximal segment is prevention of this complication. Several positioning appliances have been used to control the proximal segment during mandibular orthognathic surgery.[69–71] When rigid fixation is used, without positioning appliances, minimal insignificant rotation of the proximal segment may occur.[68] However, there is a tendency to rotate the proximal segment medially and superiorly as a result of large advancements.[72] This can lead to an unaesthetic result, especially if there was

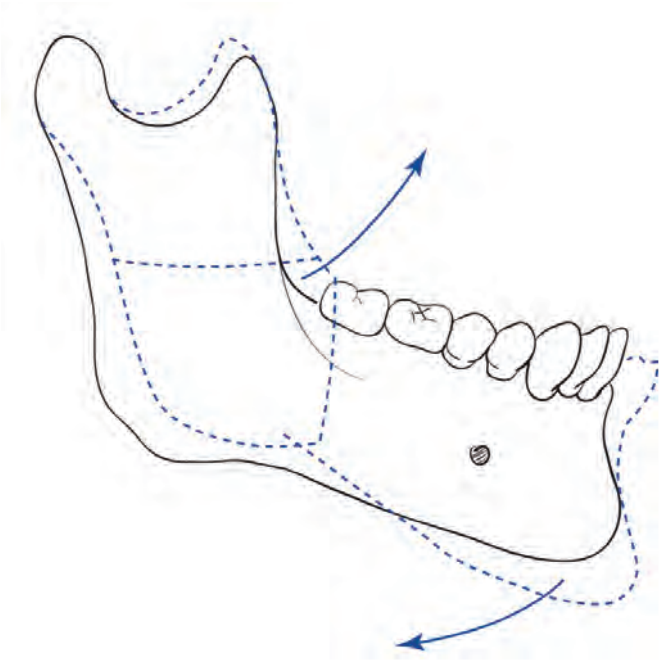


FIGURE 61-20. Diagram of relapse of mandibular orthognathic surgery, with counterclockwise rotation of the proximal segment and clockwise rotation of the distal segment.

any discrepancy in the height of the ramus before surgery (Figure 61-21).

The exact degree of rotation of the proximal segment that will cause clinically significant decreases in muscular bite forces or unaesthetic facial changes remains unknown; however, excessive rotation of the proximal segment should be assessed with regards to functional (decreased bite force generation or hypomobility) or aesthetic (loss of the gonial angle) problems, or both. An aesthetic problem in a patient with an acceptable occlusal result may be treated with the use of an alloplastic implant (e.g., high-molecular-weight porous polyethylene), customized for the specific defect based upon a stereolithography model. If the patient has an occlusal problem as well as aesthetic concerns, the SSO may need to be revised with surgery (Figure 61-22). When a malocclusion



FIGURE 61-21. Radiograph of short mandibular ramus.



FIGURE 61-22. Occlusal discrepancy with a postoperative anterior open bite deformity.

and unaesthetic result are combined with a decrease in bite force and/or hypomobility of the mandible, reoperation with application of rigid fixation must be combined with an aggressive postoperative physiotherapy regimen.

After any orthognathic surgical procedure, most patients demonstrate a limitation in the degree of maximum interincisal opening. The most dramatic decreases are seen after BSSO procedures.[69] TMJ hypomobility should be restored with the use of postoperative physiotherapy. Ellis[73] examined the range of mandibular motion after an SSO advancement osteotomy in monkeys, when either IMF or rigid osseous fixation was used. Animals that did not have IMF maintained after surgery demonstrated a greater range of motion in the early postsurgical period and attained the preoperative level of mobility by 12 weeks after surgery. Animals that underwent 6 weeks of IMF showed significant decreases in range of motion when compared with the rigid fixation group at each time period after surgery. Several clinical studies have shown that regardless of whether IMF or rigid fixation is used, with postoperative physiotherapy, a normal, or near-normal, range of motion will typically return by 2 years after surgery.[67,74]

There are several potential causes of hypomobility in patients undergoing orthognathic surgery. Scar tissue induced by the surgery may play a major role in limited mouth opening. However, postoperative immobilization can compound the effects of surgical dissection and have adverse effects on the muscles, joints, and surrounding connective tissues. Immobilization by itself induces muscle atrophy with a marked decrease in muscle fiber diameter. This problem may be compounded if the muscle is immobilized in a position where the overall length of the muscle has been shortened. In addition, the use of IMF causes deleterious effects on the TMJ, with a series of degenerative changes in articular cartilage and synovial membranes, with decreased function of the synovium, decreased production of joint fluid, and intraarticular stasis leading to fibrous changes in the TMJ with hypomobility.

Techniques used to eliminate or minimize the period of immobilization will likely decrease postsurgical hypomobility. Despite this, it is strongly suggested that all patients have routine presurgical evaluation of muscle and joint function and a systematic rehabilitation regimen as part of their postsurgical program. Mandibular ramal procedures are potentially the most harmful to the surrounding connective tissues of the jaws. Mandibular advancements, in particular, are susceptible to postoperative hypomobility. If rigid fixation is used, mild self-directed physiotherapy beginning 1 to 2 weeks after surgery may suffice, consisting of either active or passive jaw exercises. When a patient's progress is limited or when surgery has been associated with longer periods of IMF, more aggressive physical therapy is often required. If this is unsuccessful, intra-articular pathology may be responsible for the problem and additional steps may need to be taken to restore a normal range of motion, such as arthroscopic lysis and lavage, if indicated.[75]

TMJ DYSFUNCTION

Short-Term Disorders

TMJ dysfunction in patients undergoing orthognathic surgery deserves careful preoperative examination. A number of patients presenting for orthognathic surgery will have preexisting muscular temporomandibular dysfunction.[76–78] Although a small percentage of patients will develop symptoms as a result of surgery, the large majority will improve after surgery. It is generally accepted that achieving a more appropriate functional relationship of the jaws can improve TMJ symptoms, but orthognathic surgery should not be offered as a primary cure for TMJ problems. It has been generally accepted that class II patients with preexisting anterior disk displacement may benefit from SSO surgery because the consdyle is placed in a different relationship to the glenoid fossa, and this may alleviate the internal derangement by creating additional joint space to allow the disk to assume a more normal position between the cobdyle and the glenoid fossa. However, patients should be advised that orthognathic surgery may have a positive effect, negative effect, or no effect on internal derangement of the TMJ. After surgery, patients may have acute or gradual increases in a variety of TMJ symptoms, and acute exacerbations may be treated with anti-inflammatory medications and physical therapy. Gradual increases or chronic manifestations of TMJ problems are managed with standard protocols for patients with TMJ symptoms, using nonsteroidal anti-inflammatory drugs, soft diet, moist heat, and a bite splint and physical therapy, when indicated. Concern exists that with rigid fixation, there will be a higher incidence of TMJ dysfunction compared with the use of wire osteosynthesis, because the condyle is rigidly stabilized in a new position that may or may not be tolerable or adaptable for the condyle, disk, or glenoid fossa; however, clinical experience and research studies that have compared wire to rigid fixation show that the condyle is capable of adapting functionally to the new position even when rigidly fixated.[74,77]

Long-Term Disorders

Condylar resorption may occur with or without orthognathic surgery and may be responsible for delayed relapse in the young female, class II, high-mandibular-plane-angle patient who undergoes a significant advancement of the mandible. Other causes of delayed relapse after SSO advancement may include preexisting or iatrogenically induced internal derangements of the disk, and class II patients may be more prone to having disk displacement than class I or class III patients. The incidence of condylar resorption or progressive condylar remodeling ranges from 5% to 10% of the patients who undergo orthognathic surgery[79–84] As mentioned, patients who require a large magnitude advancement of the mandible, and who have preoperative TMJ symptoms, are more likely to have this problem than those who have smaller magnitude SSO advancements and no preexisting symptoms.[79,81] Condylar resorption has been noted to occur as far as 12 to 17 months after surgery.[83] Management of this complex problem includes bite splint therapy, with a possible role for anti-inflammatory medications,[83,84] and some surgeons advocate open joint surgery with synovectomy to aggressively address the synovial inflammation and halt the progression of condylar resportion. Secondary surgery to treat resultant anterior open bite deformities using costochondral bone grafting is unpredictable, and the rib graft is susceptible to the same resorption as the condyle in as many as 50% of cases.[83]

UNANTICIPATED MAXILLARY FRACTURES

Whereas the literature has focused on the management of bad splits with mandibular osteotomies, little attention has been focused on inadvertant osteotomies encountered during maxillary orthognathic surgery. With modified cuts of the maxilla (e.g., stepped osteotomy), the bone leading to the zygomatic buttress may be thin and more easily prone to fracture (Figure 61-23). Management may be accomplished by using a plate and screws on either side of the gap, or fractured segment of bone, and then reinserting the fragment behind the plate if necessary (Figure 61-24). In many cases, a small free segment of the thin anterior maxillary wall may be sacrified without any consequence, and this may be preferable to replacement as a free graft.

In addition, perhaps the most common unanticipated maxillary fracture with Le Fort surgery is the unpredictable fracture that occurs in the pterygoid plate region. Many studies using postoperative computed tomography scans have shown that the posterior osteotomy may occur in a variety of locations, including the maxillary alveolus in the area of the second molar, horizontally through the pterygoid plates, or even superiorly near the base of the skull. These inadvertent fractures may occur in these aberrant locations despite the appropriate use of the ptergoid plate osteotome in the

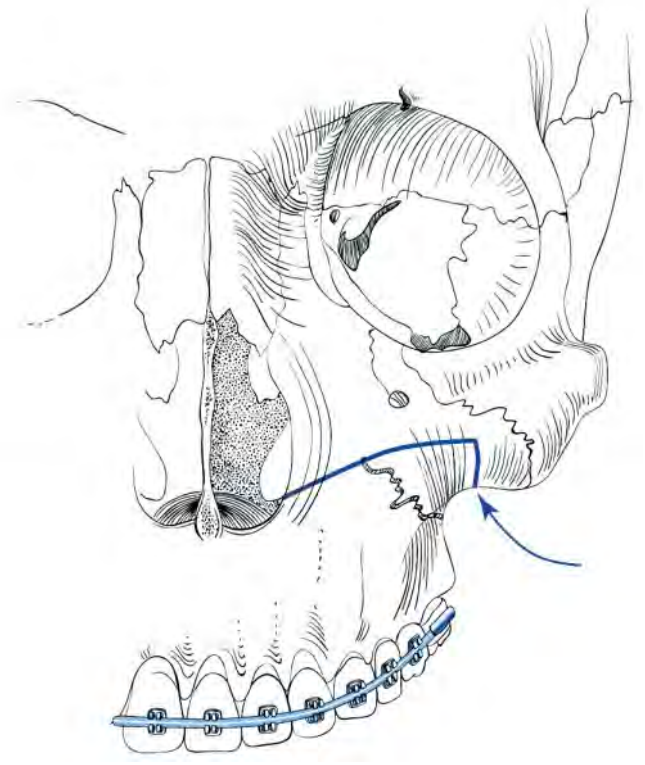


FIGURE 61-23. Fracture of the anterior wall of the maxillary sinus.

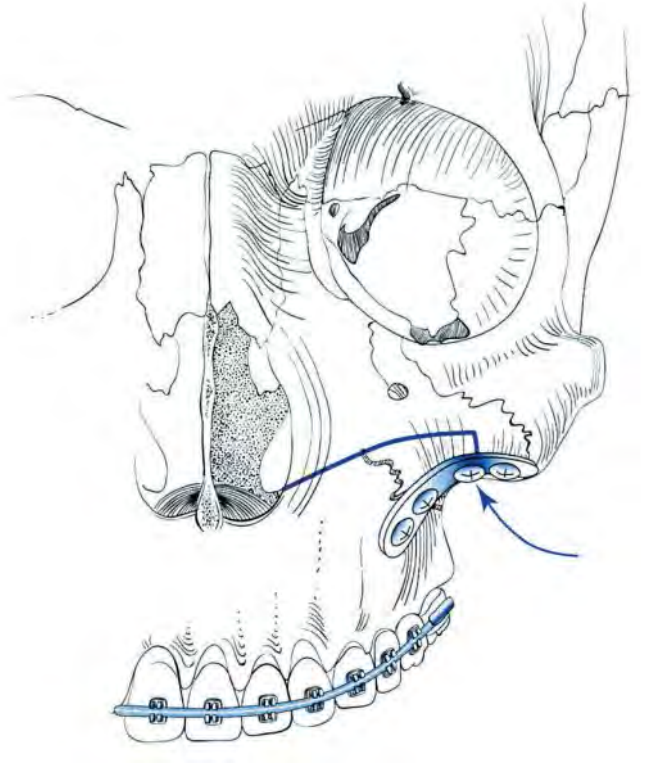


FIGURE 61-24. Rigid fixation to span defect; bone segment repositioned.

ptyerygomaxillary fissure, because access to the medial aspect of the pterygomaxillary junction is limited. Typically, these eccentric fractures have little clinical consequence in the majority of patients; however, the risks of neural and vascular injury, as well as the possibility of intracranial communication, do exist.

Despite careful preoperative treatment planning, either overimpaction or underimpaction of the maxilla may occur at the time of Le Fort surgery. This complication can be avoided with the use of internal, or preferably external, reference points. One option for external reference landmarks is to place a K-wire or Steinmann pin in the nasal bones at the nasion at the commencement of surgery and to take intraoperative measurements that will ensure that the maxilla is placed in the appropriate position at the end of surgery based upon the intended degree of impaction or downgrafting.[85] If the maxilla is found to be underimpacted afte surgery, very few options exist, although removal of plates and screws with placement of skeletal suspension wires in the outpatient setting has been advocated. Typically, a return to surgery to reposition and stabilize the maxilla into the correct position is warranted. When the maxilla is overimpacted, it is usually not possible to use multiple heavy vertical elastics in the early postoperative period to pull the maxilla inferiorly, and reoperation should be considered.

POSTOPERATIVE OCCLUSAL DISCREPANCIES

Occlusal abnormalities may be related to a number of factors in the preoperative, intraoperative, or postoperative phases of patient management. As a general rule, the majority of occlusal discrepancies between what was intended and what was obtained can be traced back to inaccuracies in preoperative patient records.[86]

Anterior Open Bite Malocclusions

Anterior open bites (apertognathia) after orthognathic surgery may be the result of technical difficulties seen with both the maxilla and the mandible at the time of surgery. With the maxilla, these include posterior interferences that are not recognized when IMF is applied during surgery; essentially, the IMF will attempt to close the "open bite" present due to posterior interferences and pull the condyles out of the glenoid fossa by doing so. If the maxilla is fixated with condyles not seated passively, with dislocation out of the glenoid fossa, when the IMF is released, there will be an immediate relapse with an open bite malocclusion. Occasionally, however, the open bite is not recognized until the next day owing to muscle relaxation and edema, and depending on the severity of the open bite, a return to the operating room for repositioning may be required.

Open bites that occur after orthodontic appliances have been removed may be due to relapse of surgically or orthodontically treated maxillary transverse discrepancies. Any molar interferences created after surgery may significantly

affect the development of an open bite anteriorly. Surgical and orthodontic correction of severe maxillary transverse discrepancies have been noted to be unstable,[87] and when relapse of the transverse correction occurs, it is usually manifested by an anterior open bite malocclusion. Management of late open bites will depend on their severity; they typically respond to posterior occlusal equilibration with enameloplasty of the molar interferences and rarely require reoperation. In addition, the development of an open bite in the long-term postoperative period may be due to condylar resorption with large mandibular advancements, as described previously.

In addition, anterior open bites have been noted to recur years after initial treatment with both orthodontics and surgery.[87,88] The stability of orthodontic therapy varies depending upon the orthodontic techniques used to manage the open bite relationship.[89] Rotation of incisors that are flared with closure of an open bite may be no more problematic than for other tooth movements. When extrusion of teeth has occurred with orthodontic mechanics, the results are less predictable, and the mechanism of this phenomenon is somewhat controversial. Tooth extrusion may have caused an increased sensitivity to external factors, such as the tongue and circumoral musculature, which may predispose the occlusion to apertognathia. Lack of stability or recurrence of the open bite is, therefore, thought to be due to the continued presence of etiologic factors (e.g., tongue thrust habit) and failure of biologic and functional adaptation. A variety of measures may be taken to correct these problems including orthodontic cribs or appliances to decrease the influence of external factors or surgical techniques such as a partial glossectomy, although these are not commonly performed.

Relapse of the Mandible

Relapse of the mandible after a BSSO procedure has been well documented in the literature, especially with larger magnitude advancements,[90–93] although the use of rigid fixation over wire fixation, or no fixation, has resulted in a significant reduction in expected relapse after orthognathic surgery. However, occlusal discrepancies may occur secondary to several reasons, including errors in the technical aspects of application of rigid fixation. Occlusal changes seen with rigid fixation may be secondary to unpredictable condylar torque, condylar sag, or incorrect placement of the proximal segment at the time of SSO surgery. These condylar positional discrepancies may result in anterior or posterior open bites or lateral shifts of the occlusion. Severe occlusal discrepancies may require revision of the orthognathic procedure. Minor discrepancies can be treated by early aggressive orthodontics with orthopedic movement of the bony segments and teeth. Posterior open bites of less than 3 mm c an be treated with vertical elastics or other orthodontic mechanics. Larger posterior open bites may require maxillary repositioning, possibly using only a posterior segmental maxillary osteotomy for the specific posterior open bite region. Anterior open bites represent failure to properly place the condyle in the glenoid fossa or instability at the osteotomy site with loss of fixation. The use of IMF with heavy anterior elastic traction may prevent reoperation when the cause is noted to be instability at the osteotomy site with loose or ineffective hardware, whereas condylar malpositioning may require revision surgery.

The ideal time to initiate correction of occlusal discrepancies is as soon after surgery as the problem is noted. Removal of the plates and/or screws in an outpatient setting, with the application of elastic traction therapy, may occasionally correct some postoperative malocclusions. Failure to place the condyle in the fossa, either unilaterally or bilaterally, requires evaluation as to whether orthodontic therapy can correct the problem, or more likely, whether a second revision surgery is necessary. A lateral shift of the occlusion in which the midline is deviated to one side is usually due to condylar torque at the time of surgery. In fact, this is the most common complication after BSSO surgery with application of rigid fixation, because the plates and/or screws place the condyles in an unnatural position that is different on each side, with a tendency for a slight shift of the occlusion in the early postoperative period. If using a proximal-distal segment clamp for application of rigid fixation, the proximal segment should lie passively against the distal segemnt, and shifts or torque of the proximal segment may need to be addressed with recontouring or bone shimming, or the clamp can be repositioned in a different location. After surgery, a midline shift due to torque of the proximal segment may be treated with orthodontics if 1 to 2 mm; larger midline discrepancies may rarely require reoperation.

Relapse of a skeletal class III mandibuar setback surgery upon release of IMF has also been noted, especially with larger degrees of posterior mandibular movement.[94–96] Theoretically, this may be caused by pushing the proximal fragment back during surgery, especially with VRO as described previously, and with the release of IMF, the mandible rotates forward. To prevent this problem, it has been suggested that the inferior border of both the proximal and the distal segments be aligned and that the medial sling (medial pterygoid muscle) be released to allow a passive fit of the segments.[95] Others disagree and believe that clockwise rotation of the proximal segment is not responsible for the relapse.[96] In addition, the use of a monocortical plate and screws on the proximal and distal segments may provide a more stable occlusal result than that seen with the use of bicortical screws (Figure 61-25). When this occlusal discrepancy is seen after surgery, the short-term use of class III elastics can help to correct the problem (1–2 mm discrepancy). If the discrepancy is greater than 3 to 4 mm, orthodontic mechanics or interproximal reduction (IPR) may be effective, although a second revision operation may be necessary.

As discussed previously, an anterior open bite may be seen after BSSO with relapse (Figure 61-26). This is usually due to a failure of the plates and/or screws placed at the time of fixation, or technical difficulties incurred at the time of



FIGURE 61-25. Monocortical plate and screws for SSO fixation.





FIGURE 61-26. Postoperative open bite after bilateral sagittal split osteotomy (BSSO). **A,** At 24 hours. **B,** After 1 week of occlusal elastic therapy.

osteotomy with resulting edema in the joints that typically resolves with time. However, an anterior open bite is much more commonly seen in patients after an intraoral VRO upon release of IMF.[97,98] Recommendations to prevent this problem include removal of the coronoid process and temporalis muscle influence, the use of skeletal suspension wires for additional fixation, using modified osteotomy cuts of the ramus to maximize bony contacts, and extending the postoperative period of IMF to at least 8 weeks.[98] Also, postoperative guiding elastics have been used for an extended period of 2 to 6 weeks when open bites have been noted in the immediate postoperative period.

MISCELLANEOUS COMPLICATIONS

Owing to the proximity of the nasal ETT to the maxillary, ETTs may be injured from drills, saws, or osteotomes during maxillary orthognathic surgery, and in some cases reintubation is required, while in others packing around the ETT is sufficient. In order to avoid this complication, a wire-reinforced anode ETT may be preferable, although the rarity of this occurrence does not warrant major changes to routine.

Alar rim injuries, with alar necrosis, are due to pressure on the alar rim from an nasotracheal tube. Care should be taken when wrapping the head so that there is no pressure on the tip of the nose or the forehead or ears from the ETT or its components.

Soft tissue emphysema in the cervical and facial regions has been noted after a variety of procedures that may be unrelated to orthognathic surgery. However, there are several reports of air in the soft tissues of the head, neck, and chest after Le Fort I osteotomies.[99] Subcutaneous emphysema of the cheeks is likely due to forceful blowing of the nose postoperatively, which allows air to escape into the surrounding tissues through the maxillary sinus ostium. Forceful coughing can allow air to pass into the retropharyngeal space and into the mediastinum. Alternatively, rupture of a perivascular bleb or traumatic introduction of air through the cervical fascia is also possible. Subcutaneous emphysema may be managed by observation, heat, and systemic antibiotics. Therapy for pneumomediastinum consists of close observation, cardiac monitoring, intravenous fluids, and antibiotics. Chest tubes or drainage of the mediastinum may be necessary, but supplemental oxygen, as well as pulmonary physiotherapy, should be used.

Epiphora

Epiphora may be seen after Le Fort osteotomy and is frequently due to edema of the nasal mucosa. Alternatively, the nasolacrimal ductal drainage system may be injured when a concomitant partial inferior turbinectomy is performed with during maxillary osteotomy. Damage to the nasolacrimal appartus may also occur if the bone cut for the Le Fort is placed too far superiorly along the medial wall of the sinus (i.e., lateral nasal wall). The incidence of nasolacrimal injury

is infrequent and usually transient and resolves spontaneously. Meticulous soft tissue dissection and carefully placed osteotomies around the medial aspect of the piriform aperture may decrease the incidence of epiphora. Persistent excessive eye tearing that does not decrease after 3 weeks may need to be addressed with a dacryocystorhinostomy procedure.

Auriculotemporal Syndrome

The auriculotemporal syndrome, syndrome of gustatory sweating, or Frey's syndrome, is an unusual complication tyically seen as a result of parotid gland surgery. After an injury to the auriculotemporal nerve, the symptoms are believed to be caused by a misdirected regeneration of parasympathetic fibers to denervated sweat glands, resulting in sweating of the cheek during mastication and salivation; this condition is diagnosed with an iodine-starch test. A number of authors have reported Frey's syndrome occurring after extraoral VRO and BSSO procedures,[100–103] with symptoms occurring between 3 months and 3 years after orthognathic surgery. Mild cases in which the patient may have symptoms only with spicy foods should be observed, because the symptoms may decrease with time. A variety of treatments have been suggested for more severe symptoms, including topical scopolamine patches and insertion of fascia lata or acellular human dermis matrix under the skin.[104] Because topical scopolamine has a number of undesirable side effects, there have been reports of the use of botulinum toxin as a successful treatment for this problem.[105,106]

Facial Scars

Although attempts are made to camouflage extraoral site incisions, unattractive facial scarring may occasionally occur. Egyedi and coworkers[107] noted 6 undesirable scars in a group of 100 patients with extraoral incisions for orthognathic surgery, although the criteria used to determine what was undesirable are unknown. Percutaneous incisions of 2 to 4 mm seldom leave significant scars, and the more common problem occurs when the epidermis adheres to the underlying fascia and muscle, with puckering or retraction of the skin. Scar revisions can improve the appearance of existing scars, although a scar will always be present. Other options for scar management include massage, injection of corticosteroids, and dermabrasion or laser resurfacing. With the use of intraoral techniques and endoscope-assisted surgery, most skin incisions can be avoided.

It should be noted that iatrogenic scarring of the cheek with skin retraction may occur during an SSO using bicortical screws placed with a transbuccal trocar approach. Whereas the majority of the skin puncture sites on the cheek heal without visible scars, there may be excessive scarring of the site, especially if the metal trocar becomes heated from the rotation of an eccentric drill bit during drilling the holes for the bicortical screws. The burn injury may require local wound care, intralesional steroid injections, or excision. Also, although the risk of salivary fistula exists through this trocar site, it would represent a rare occurrence.

Salivary Gland Injuries

Injuries to the parotid gland can occur with extraoral approaches for orthognathic surgical procedures. Painless swelling, parotid sialoceles, and fistulae have been reported in the first week after surgery.[108] The treatment of sialoceles or salivary fistulae may include the use of antisialogogue (anticholinergic) medications, pressure dressings, aspiration or drainage, and low-dose radiation to decrease salivary flow via induction of glandular atrophy. Sialography is not recommended in the acute phases of these injuries, because the contrast may extravasate into the soft tissues with inflammation or the procedure may create a larger fistula or increase the size of the current fistula. Resolution of a sialocele should be expected within 4 weeks with the appropriate use of nonsurgical therapies, and failure of these treatment options may require a more invasive surgical procedure.

Complications do occur during orthognathic surgery, and the clinician must be aware of the myriad of possible consequences in order to most appropriately manage or prevent these from occurring as well as to be able to adequately perform a detailed informed consent discussion with patients and families before orthognathic surgery. In most cases, complications from orthognathic surgery can be prevented with comprehensive diagnosis and treatment planning including a thorough history, clinical and radiographic examination, dental model and cephalometric analysis, and a meticulous attention to detail in the treatment planning phases for maxillary and mandibular surgery.

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62

CHAPTER

Cleft Orthognathic Surgery

Kevin S. Smith, DDS

In treatment of children with cleft lip and palate facial anomalies, the use of an interdisciplinary team approach continues to be of primary importance throughout adolescence. The coordination of care between restorative dentistry, surgery, orthodontics, and prosthodontics is critically important during this phase of reconstruction of the cleft lip and palate patient. As stated by the American Cleft Palate Association (ACPA), the minimum requirements for a cleft lip and palate team include a surgeon (oral and maxillofacial surgeon, otorhinolaryngologist, or plastic surgeon), a speech and language pathologist, and an orthodontist. These minimum requirements may be inadequate if the team does not provide a consultation for the other members who are truly important in the care of cleft lip and palate patients, specifically restorative dentistry and psychological counseling. Many children with cleft lip and palate have malformed teeth, dental crowding, missing teeth, supernumerary teeth, and ectopic eruption that frequently require careful evaluation and treatment by pediatric or general dentists during the phases of mixed dentition and into the early permanent dentition. Many children have underdeveloped, or missing, maxillary lateral incisors and may be candidates for dental implants and/or other prosthetic reconstruction. Consequently, these professionals are of utmost importance to the team. Sometimes overlooked in interdisciplinary care of these patients is psychological counseling. A significant number of these children have self-esteem issues associated with their facial deformities, especially during the formative preteen years, and families need to maintain an open mind and discuss these social issues associated with their cleft lip and palate child. A thorough team discussion about the consultations that can be obtained, and the services that can be provided, must take place to ensure that each team member has access to all aspects of care and to ensure that patient expectations are reasonable and appropriate. On occasion, families become very complacent with follow-up and recall to the interdisciplinary cleft palate clinic owing to the fact that they have had their immediate needs met once the cleft lip and cleft palate have been repaired surgically. If speech problems have been addressed, or do not exist, the family may be satisfied with the progress, and it is critical that the interdisciplinary team review the patient records to ensure that patients who have been noncompliant with regular team meetings attempt to reenter the regular team meetings, especially during late transitional dentition and early permanent dentition, to alleviate significant problems in treatment who may present late for treatment. When children are not followed routinely, and present late in the orthodontic and surgical sequencing for treatment of cleft lip and palate, the treatment becomes much more complex, social issues become much more significant, and the problems may increase exponentially (Figures 62-1 to 62-3).

Orthognathic care in the cleft lip and palate patient begins with development of the maxilla in the transitional dentition, and during this time, the orthodontist should take every opportunity to develop the transverse dimension of the maxilla. Expansion of the maxilla before alveolar cleft bone grafting will help to maintain the transverse dimensions of



FIGURE 62-1. Occlusal view of an 18-year-old who had inappropriate management of cleft dentofacial anomaly.



FIGURE 62-2. Anterior closed occlusal view of a patient with inappropriate cleft care. The patient was followed by a "cleft" surgeon, but without interaction from other cleft team members.

the maxilla and will alleviate potential transverse problems with orthognathic surgery once the child has reached adolescence. Before orthognathic surgery, orthodontists should be acutely aware of whether the child has a propensity for maxillary anteroposterior (AP) and/or vertical hypoplasia and should be careful not to treat these problems with extraction of teeth in the lower arch to try to maintain a class I occlusion. Early in the adolescent period, children who are treated in this manner have a greater problem later on once they have transitioned into a class III malocclusion and are missing significant lower arch dimension. The patient and family may initially think that the patient will not be a candidate for surgery, and this may have negative effects on the orthodontist-patient relationship if a surgery is recommended later. Statistically, the number of children who need to have orthognathic surgery and have had unilateral cleft lip and palate is approximately 25%.[1,2]

Other orthodontic considerations for the cleft patient requiring orthognathic surgery include preoperative orthodontic banding and bonding. It is critical to ensure that



FIGURE 62-3. Lateral cephalogram of a cleft patient presenting for correction of a severe dentofacial anomaly.

surgical rectangular arch wires and adequate surgical lugs are in place before surgery to allow the surgeon to manipulate the maxilla during the surgical procedure. The surgeon will have a difficult enough time manipulating the maxilla without having additional problems associated with poorly bonded orthodontic appliances with small arch wires and without adequate surgical lugs for intraoperative intermaxillary fixation. These small issues can provide an increasingly frustrating surgical procedure that may result in inadequate treatment in fixation.

Maxillary hypoplasia in three planes of space is a common problem encountered in children with cleft lip and palate, and there is controversy regarding what degree of this restricted growth is surgically induced and what degree of hypoplasia is intrinsic.[3] Decisions must be made by the interdisciplinary team, with parental and patient input, regarding whether the patient will have early surgery, end-of-growth surgery, or both. Many children entering junior high school, grades 6 to 8, may be experiencing psychosocial issues owing to the negative aesthetic effects of severe maxillary hypoplasia, and these children may be candidates for early orthognathic surgery with maxillary advancement surgery (Figures 62-4 to 62-6). Discussions about early orthognathic surgery must include an understanding by the family and patient that, in most situations, the patient will require secondary surgery after the cessation of facial growth.

Early in the history of orthognathic surgery, the application to children with cleft lip and palate dentofacial anomalies proved to be difficult. Many institutions, both before and after the introduction of rigid fixation, employed a philosophy of "splitting the AP discrepancy" between the maxilla and the mandible or avoided treatment of the maxilla completely.[4,5] Patients with severe maxillary hypoplasia may have had a Le Fort I osteotomy performed, repositioning the maxilla as anterior as possible allowed by the scar tissue restriction. There is typically adjunctive bone grafting and some form of additional fixation utilized in these cases of large maxillary advancements in the cleft patient to prevent relapse. Subsequently, the mandible was then repositioned posteriorly to achieve a class I canine and molar relationship with the maxilla. In many patient situations, the projection of the mandible may be in a relatively normal position, and essentially, the technique of treating to the deformity was employed. Treating to the deformity has significant ramifications in the form of compromised facial aesthetics and possible iatrogenic obstructive sleep apnea due to narrowing of the posterior airway (Figures 62-7 and 62-8).

In contemporary orthognathic surgery of children with cleft deformities, if cephalometrically and clinically, the mandible is in a normal AP position, it should not be repositioned posteriorly to split the difference with the maxilla; this will allow increased projection of the maxilla, elevation of the nasal tip, increased lip support, and the establishment of stable occlusion. Maxillary advancement does not correct the lip scarring or retraction or compensate for an unaesthetic lip repair, and it does not normalize anatomic landmarks.[6] The



FIGURE 62-4. Lateral cephalogram of a 12-year-old bilateral cleft lip and palate patient before orthognathic surgery.



FIGURE 62-5. Lateral cephalogram of the same 12-year-old with early intervention maxillary advancement. Secondary procedures were planned owing to continued growth.

skeletal surgery must occur before the final soft tissue revision, including any lip and nasal aesthetic procedures. The increased support provided by maxillary advancement for the nose and lip changes the cosmetic appearance without direct surgery on these structures. The judgment of the surgeon who performs the definitive lip and nasal surgery may be hindered if the skeletal deformity is not addressed before the final aesthetic soft tissue procedures.

Other presurgical orthognathic considerations for patients with bilateral cleft lip and palate include the use of palatal splints, especially in situations in which the bone graft is less than adequate in the alveolar cleft to help prevent fracture of the bone graft in this area and after the down-fracture and manipulation of the maxilla. A palatal splint will help stabilize the bone of the lateral segments as well as the premaxilla so that the intersegment bone does not fracture and will allow stabilization of the entire complex so that additional bone grafting can be accomplished during the orthognathic procedure, if indicated. Occlusal splints must be fabricated before surgery with consideration of whether overcorrection of the jaw movements will be planned. Posnick and Ewing[7] showed that 24 patients without pharyngoplasty with mean maxillary advancements of 6.7 mm relapsed immediately after surgery with a mean relapse of 2.0 mm over a 2-year follow-up period. Planned overcorrection in the model surgery and



FIGURE 62-6. Lateral cephalogram of the patient from Figures 62-4 and 62-5 at age 17. The patient asked the orthodontist for camouflage and did not want a second surgery. Note recurrent maxillary hypoplasia, not secondary to relapse, but continued mandibular growth.



FIGURE 62-7. Lateral cephalogram of a 17-year-old patient who had had early surgery for correction of maxillary hypoplasia.



FIGURE 62-8. The patient in Figure 62-7 had surgery and, due to severe maxillary scarring and inadequate maxillary advancement, also had mandibular setback. "Splitting the difference" causes negative facial profile changes and narrowing of the airway with risk of obstructive sleep apnea (OSA). Note changes in the chin-neck-throat angle and narrowed posterior airway space.

splint fabrication will usually decrease or eliminate relapse potential. Cleft patients with relatively small maxillary AP discrepancies will usually have excellent results with minimal relapse after maxillary advancement with overcorrection (Figures 62-9 to 62-12).

The procedure for performing maxillary osteotomies is described elsewhere in this text, and the important aspects of differences in cleft patients are discussed. Cleft patients undergoing orthognathic surgery many times may have had a prior pharyngeal flap surgery to correct hypernasality and velopharyngeal incompetence (VPI). The anesthesiologist should be made aware of the presence of the pharyngeal flap and that plans for alteration from the usual intubation protocol may be necessary. The endotracheal tube should not be forced through the nasopharynx because it can track submucosally along the posterior pharyngeal wall. Using a small catheter or an endoscope first through the endotracheal tube then gently through the lateral ports of the pharyngeal flap will facilitate the intubation (Figure 62-13). Another technique is to use a gloved finger and digitally palpate the right or left lateral pharyngeal port, depending on which side of the nose the endotracheal tube is placed, through the mouth and contact the finger with the endotracheal tube, ensuring that it follows the finger through the intended port.

A cleft maxilla differs from an intact maxilla because of the absence of soft and hard tissues and multiple prior surgical procedures that were required to repair and close defects. Perfusion of the mobilized maxilla is dependent on vessels coming from the overlying soft tissues, predominantly involving the palatal tissues. In cleft patients, this tissue is commonly scarred and fibrotic; therefore, care must be exercised when designing the incision to perform the osteotomy in order to maximize preservation of blood supply. With few exceptions, almost all patients may be treated with a Le Fort I osteotomy via a circumvestibular incision and maxillary down-fracture approach. For those with severe palatal scarring, who have previously undergone an island palatal repair, and those with bilateral clefts of the maxilla, an anterior buccal soft tissue pedicle should be left intact on the mobilized maxilla to maintain adequate vascular perfusion. Technically, this is a more challenging operation. Almost all unilateral



FIGURE 62-9. Unilateral cleft lip and palate patient before orthognathics. The patient is planned for only maxillary advancement with a 6-mm reverse overjet.



FIGURE 62-10. One year after maxillary advancement and rhinoplasty.

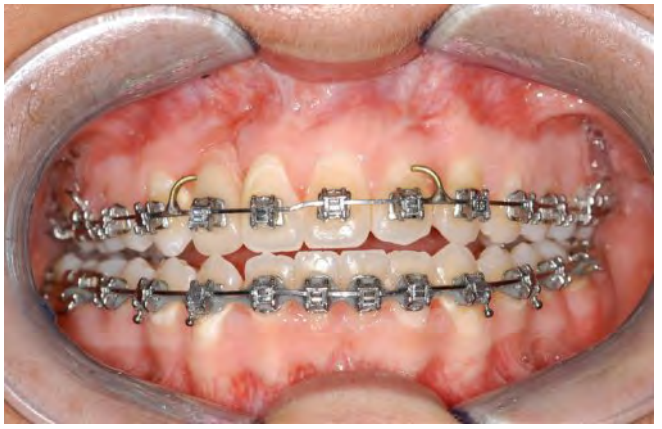


FIGURE 62-11. Preoperative occlusal view of a unilateral cleft lip and palate patient before maxillary advancement.



FIGURE 62-12. Postoperative dental occlusal view 1 year after maxillary advancement.

cleft patients can be treated with a Le Fort I osteotomy via a circumvestibular approach. The circumvestibular incision is made from the zygomaticomaxillary buttress to the opposite side, high in the mucobuccal fold (Figure 62-14).

Subperiosteal dissection exposes the entire lateral wall of the maxilla from the piriform rims to the pterygoid plates and from the alveolus, above the roots of the teeth, to the inferior orbital rim. The broad exposure permits excellent visualization of all planned osteotomies. At the conclusion of maxillary mobilization, this incision permits the maxilla to be down-fractured and pedicled entirely based upon the palatal soft tissues and the remaining buccal soft tissues below the incision. Good visualization and ease of mobilization are the major advantages of this approach. Hemorrhage control is performed with direct visualization. With this in mind, the incision design for bilateral cleft lip and palate patient is different even if there is a well-healed alveolar cleft bone graft in place. A tunneling technique should be accomplished with a small vertical incision in the midline and horizontal incisions on the lateral segments with a tunneling technique in between the cuspid and the lateral incisor areas (Figure 62-15). When an anterior buccal pedicle remains, the operation is technically more difficult. This preservation of labial soft tissues will prevent devascularization of the premaxillary bone segment and mucosa and can be accomplished in most cases without difficulty. For most patients with a cleft palate, the area of greatest resistance to mobilization of the posterior maxilla is the vertical portion of the palatine bone, located in the posteromedial aspect of the maxillary sinus. The bone is thick and access is limited, and this is the area of the descending palatine vessels that should be maintained, when possible. It is often desirable to segmentalize the maxilla of a cleft patient in order to improve occlusal relationships, but segmentation of the maxilla should be performed with caution, considering the compromised vascularity and scarring of the soft tissues.[8] The compromise of acceptance of a posterior cross-bite or other minor occlusal disharmonies may be preferred over the risk of avascular

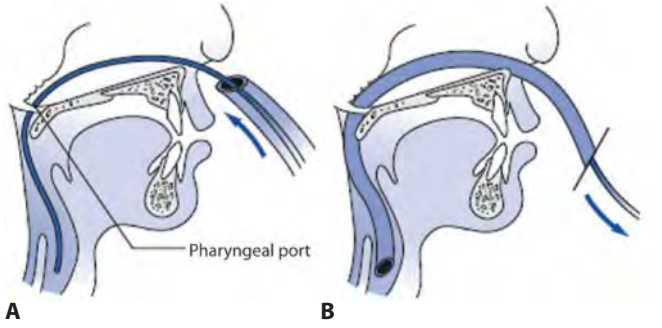


FIGURE 62-13. A catheter passed nasally through the one of the velopharyngeal ports serves to guide the nasoendotracheal tube past the pharyngeal flap.

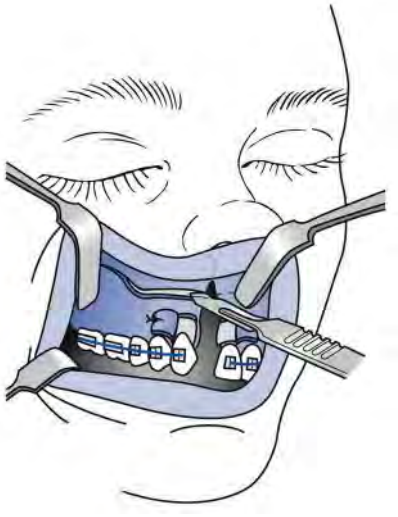
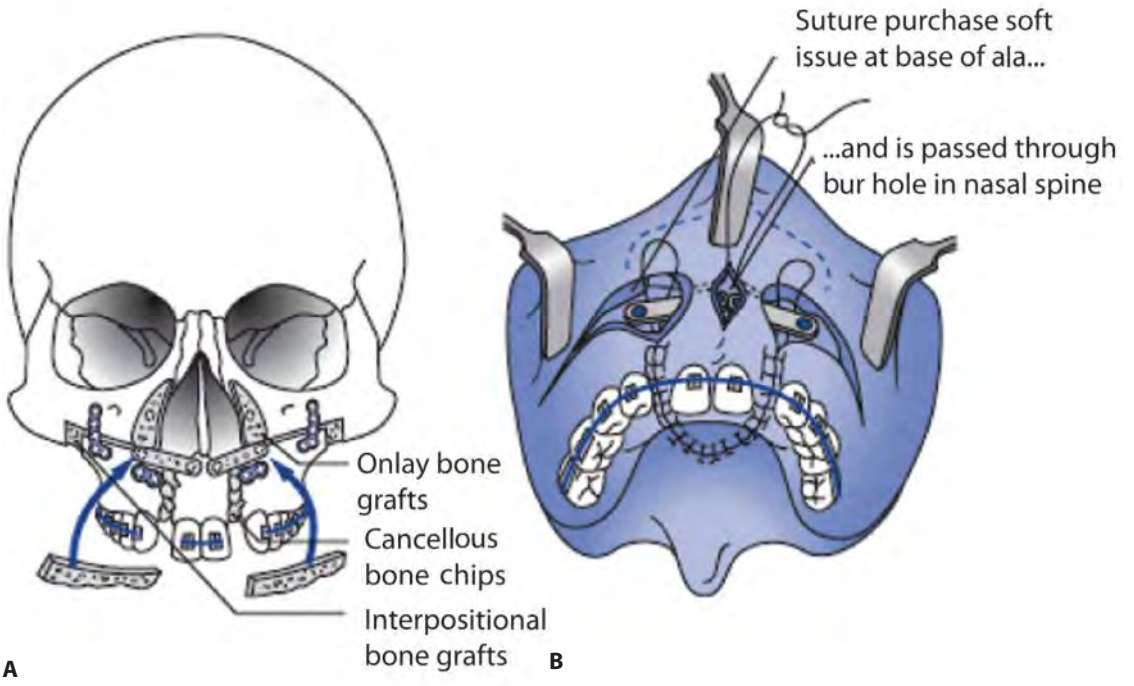


FIGURE 62-14. A high circumvestibular incision ensures adequate perfusion to the anterior maxilla. Note that the attached gingiva on both sides of the cleft is reflected and preserved. The remaining tissue lining the cleft walls is later incised and reflected palatally or buccally, depending on where it is needed for closure.



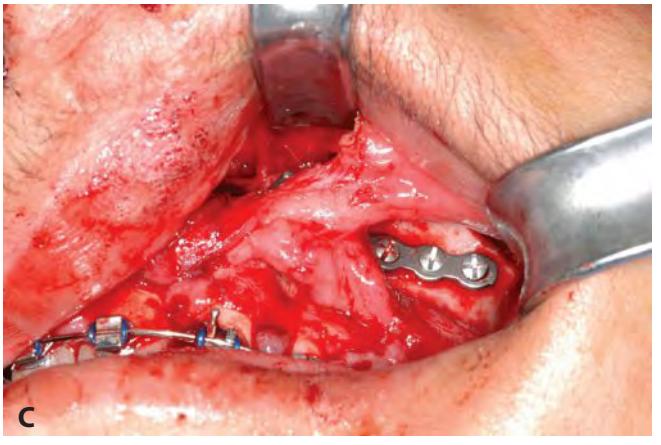


FIGURE 62-15. **A** and **B,** The premaxilla is secured with bone grafts, which are used to reconstruct the inferior piriform rim. These grafts are tunneled under the buccal flaps and secured to the anterior nasal spine anteriorly and to the lateral maxilla posteriorly. **C,** Intraoral view of the lateral maxillary incisions and medial incision with tunneling, providing an anterior pedicle to preserve blood supply to the premaxillary region. Note the rigid bone plates for osteotomy fixation.

necrosis of the soft and hard tissues after a segmental osteotomy. It is possible to close dental spaces with segmental osteotomies, and this is frequently done in residual alveolar clefts. During the osteotomy, closure of the nasal lining is of critical importance and, as stated earlier, requires bone grafting into the area to help stabilize the maxilla and to provide proper contours of the maxilla and alveolar bone.

General considerations in performing orthognathic surgery for cleft lip and palate patients include knowledge of differences in anatomy for children with and without cleft lip and palate deformities as compared with the typical skeletally mature adolescent orthognathic surgical patient. Patients with cleft maxillary deformities may have heavy buttressing of the maxilla, specifically at the piriform rim and pterygomaxillary buttress region of the posterior maxilla.[9] It is important to note this anatomic anomaly before orthognathic surgical procedures so that careful and thorough osteotomies can be completed in these regions to ensure that maxillary down-fracture can be completed without difficulty. Failure to weaken these buttress regions before mobilization may result in an unfavorable fracture, for example, in the pterygoid plate region extending to the skull base or orbit, and blindness has been reported after Le Fort I osteotomies in cleft patients.[9] Additional instrumentation that may help in down-fracturing and stretching of the soft tissue pedicle includes a modified Tessier rib-spreading forceps or a Smith sagittal split osteotomy–spreading forceps. These spreading forceps can easily fit posterior to the hard palate and allow slow controlled expansion of the scarred palatal soft tissue pedicle of the maxilla. This will increase the possible magnitude of down-grafting and advancement and improve the surgeon's ability to completely mobilize the maxilla.

Significant time must be taken to stretch the soft tissue pedicle in the vertical and AP dimensions to allow proper down-grafting and projection of the maxilla. Soft tissue handling is important after the down-fracture, and attempts at reconstruction of the nasal floor and closure of any residual fistulas will improve the final results of the surgery (Figure 62-16).

Residual bony defects can then be grafted to provide a better skeletal base for nasal aesthetic and alveolar and dental reconstruction in the future. The bone graft may be wedged into defects to help retain the proper maxillary position and promote osseous healing. Choices of bone augmentation range from iliac crest to the use of bone morphogenetic protein (BMP). Increasing clinical use of recombinant human bone morphogenetic protein-2 (rhBMP-2) has facilitated significant changes in maxillary bone grafting procedures within oral and maxillofacial surgery. Its use in cleft orthognathic surgery seems to be very appropriate. Consideration should be given to composite-type grafting scenarios where autogenous bone is used along with BMP. Bone grafts can be used for immediate stabilization with a membrane of BMP-impregnated collagen sponge over the site to help with enhanced bone formation in the region. Fixation of the cleft maxilla should be done with as heavy as fixation as possible for stabilization.[10,11] One consideration in the use of more rigid fixation is that the weak link in maintaining the position of the maxilla is not the plate or screws but the bone and bony contacts. Therefore, the surgeon should attempt to maintain as much bone-to-bone contact as possible and use bone grafts at the buttress regions, when necessary. The relapse potential of the osteotomized cleft maxilla is clinically significant in many situations, and a slow posterior migration of the entire maxillary complex may still occur, regardless of the size of the plates and screws or the amount of bony contacts.

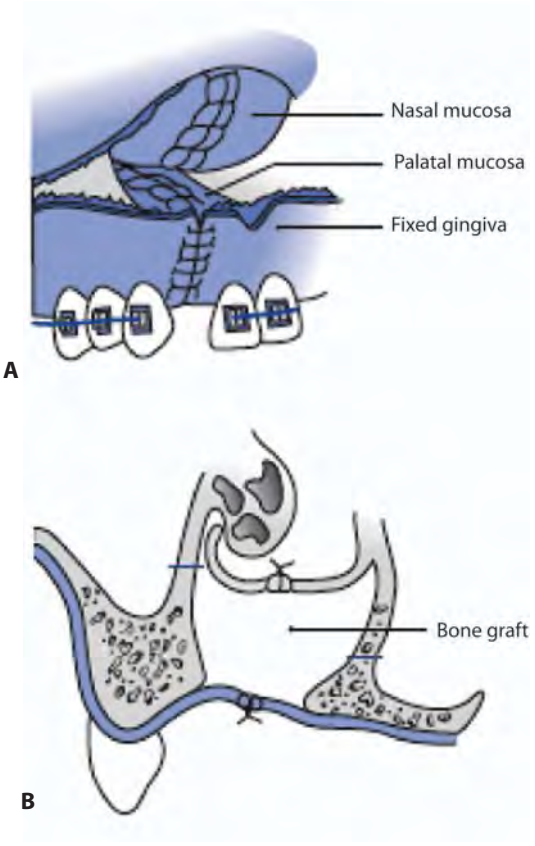


FIGURE 62-16. **A,** Buccal view shows the tissues lining the cleft elevated and sutured on the oral side. **B,** Deeper view into the cleft demonstrates closure of the oral and nasal tissues and the pocket for the bone graft.

CLEFT DISTRACTION OSTEOGENESIS

Any discussion of orthognathic surgery in the cleft lip and palate patient must include consideration for the use of distraction osteogenesis (DO). As discussed in Chapter 63, children with severe class III malocclusions remain one of the very few indications for DO in facial bone reconstruction. Chapter 63 reviews the basic biologic principles and concepts of DO; however, it should be noted that in the mid-1990s at the First International Conference on Craniofacial Distraction Osteogenesis, DO was introduced in the opening ceremony as the panacea for facial bone osteotomies in all aspects of orthognathic surgery and other facial deformity correction.[12] As time and surgical experience have progressed, there are very few indications for DO in orthognathic surgery and/or the correction of facial deformities. Adolescents with severe maxillary hypoplasia due to cleft deformities are appropriately treated with DO owing to the fact that there is slow expansion of the scar that has restricted the growth of the maxilla and the most common level of the osteotomy for DO is at the Le Fort I level.[13]

Various DO techniques and devices are available for maxillary distraction in the cleft patient, but a common device used is an external head frame (halo) distraction device, in which traditional maxillary osteotomies are performed for the procedure. Care should be taken to completely mobilize the maxilla, because the distraction device will not overcome inadequate mobilization of the maxilla and hardware failure will likely occur. Specially made bone plates are secured to the maxilla with screws, and these plates have a large diameter rectangular wire looping around the upper lip to the external surface of the skin in the paranasal regions. This wire is attached to an adjustable activation bar that can be manipulated in the vertical, horizontal, and transverse planes of space. The activation bar is then attached to a vertical bar positioned at the midline of the face and connected to the head frame distraction device (Figures 62-17 and 62-18). One of the major advantages of an external head frame distraction device is that the vectors of distraction can be controlled in three planes of space easily during the distraction process. Also, there is ready access to the activation arms of the distraction device, and most of the mechanisms of the device are readily visible with only the interface at the bone being in a buried position. Large magnitude linear distraction movements are possible, with average reported AP distraction movements of 8 to 15 mm and vertical inferior

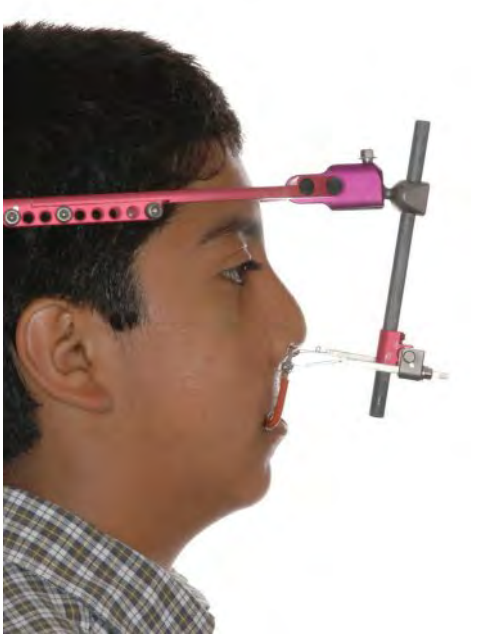


FIGURE 62-17. Lateral view of an external fixation distraction osteogenesis (DO) device. The cranial halo is fixated with multiple screws into the surface of the cranium. The vertical bar connects to the distraction arms, which are subsequently connected to the maxilla.

movements ranging from 1.6 to 13 mm.[13] Following the osteotomies and placement of the DO devices, the latency period is typically 5 days, and the distraction rate is usually 1 to 1.5 mm/day, depending upon the age of the cleft patient at the time of DO. Techniques that can aid in the proper final position of the maxilla with both internal and external DO appliances include occlusal splints and the use of intermaxillary elastics to guide the maxilla into the proper final location. During the DO activation period, alterations in the vector can be accomplished to allow the teeth to fit into the splint and the final resting position of the maxilla. The usual consolidation period, during which time the maxillary bone can heal, requires 2 to 3 months (8–12 wk). The removal of external frame distraction devices is simple and can be accomplished in the office with or without local anesthesia. The removal of the bone plates may be more complex and will require, at the minimum, local anesthesia and intravenous sedation. Patients may need to use a reverse-pull (protraction) headgear for night use for a period of 6 months after an external frame device has been removed to maintain the position of the maxilla and prevent relapse. Suzuki and colleagues[14] found a 22.3% horizontal relapse and a 53.7% vertical relapse during the first 6 months after DO surgery. Patients need to be prepared to wear the external head frame device for at least 3 to 4 months, and approximately 6 months of reverse-pull headgear in the postoperative period. Some have suggested that rigid fixation be accomplished after the distraction process, upon removal of the DO devices[15]; and these discussions should involve the orthodontist before surgery, but may serve to stabilize the maxilla further and prevent relapse. Disadvantages of external frame devices include the social appearance of the patient during treatment time, and other frequent complaints include difficulty finding a comfortable sleeping position and accidental trauma to the device from siblings, friends, and pets. The external frame device is large and bulky and may be easily caught on other objects or displaced by other people. Some patients decline the option of a head frame device owing to its appearance, especially if the child is in school. When using DO, the patient should be followed at regular intervals to ensure that the distraction process is proceeding as planned, and it should be recognized that the need for a reverse-pull headgear is also a disadvantage (Figures 62-19 to 62-21).

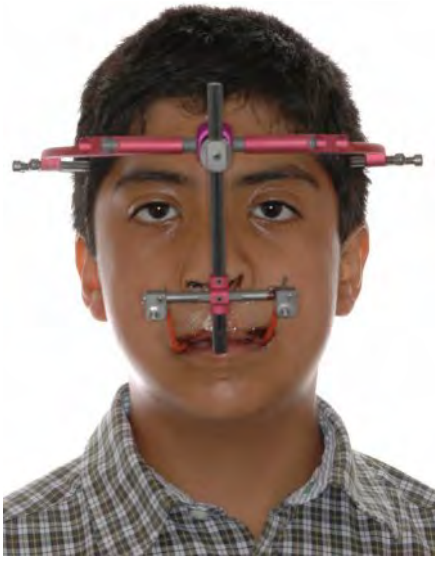


FIGURE 62-18. Frontal view of an external fixation DO device.

Internal distraction devices may also be used in the cleft patient and can work well and provide movement in the maxilla in the AP and yaw vector directions. Advantages of an internal device include increased patient acceptance, no visibility of the appliance externally, ability to remove the activation arms of the distractor once the distraction has been completed while leaving the distraction appliance in place for consolidation, and the ability to leave the extraction devices on for 6 to 9 months after the distraction process to allow for increased stability of the maxilla; this will eliminate the need for a post-DO reverse-pull headgear (Figures 62-22 to 62-26). Disadvantages of the internal devices are that the appliances are unidirectional in nature, they require a second surgery for removal, and they do not allow the surgeon to make significant changes once the appliances are in place and distraction is proceeding. In addition, placement of the devices in a parallel fashion bilaterally can be difficult for the surgeon. The surgical procedure includes typical osteotomies for the unilateral or bilateral cleft maxilla, downfracturing the maxilla to ensure adequate mobility of the maxilla, and then placement of the internal distraction devices and ensuring as close as possible paralleling bilaterally of the vector of distraction. Depending upon the need for



FIGURE 62-19. Lateral cephalogram of a patient with severe maxillary hypoplasia.



FIGURE 62-20. A patient after external frame DO with correction of the maxillary hypoplasia. The patient did not wear reverse-pull headgear in the postoperative phase.



FIGURE 62-21. Lateral cephalogram of the same patient from Figures 62-19 and 62-20, who had maxillary relapse secondary to lack of reverse-pull headgear and class III elastics, shows significant maxillary relapse.



FIGURE 62-22. Preoperative lateral view of a bilateral cleft patient before internal maxillary distraction for maxillary advancement.

increasing the vertical dimension of the face, the angle of the distraction device can be changed to allow for a "ramping down" of the maxilla in a downward and forward vector. Essentially, changes in the vertical position of the maxilla can be effected with the angulation of the device at placement but cannot be altered during the course of the distraction process. Most internal distraction devices are secured with screws into the buttress of the zygoma and onto the body of the maxilla, and the activation arms are placed, if not already part of the devices, and the wounds are closed. Again, the latency period is 5 days, and the rate of distraction is 1 to 1.5 mm/day divided into a rhythm of two to three times per day. The



FIGURE 62-23. Postoperative lateral view of a bilateral cleft patient 1 year after 8.5-mm internal maxillary distraction.



FIGURE 62-24. Preoperative lateral cephalogram shows the maxillary discrepancy in a bilateral cleft patient. The patient has severe palatal scarring, and internal maxillary advancement DO is planned.



FIGURE 62-25. Postoperative lateral cephalogram during post-distraction healing. Note that, although the distraction devices are not parallel, the advancement proceeded without difficulty. Overcorrection was not done because the appliances remained for 6 months.



FIGURE 62-26. Postoperative Panorex at 1.5 weeks shows good position of the internal DO devices.

internal distraction devices can correct for midline asymmetries during distraction by adjusting the yaw during distraction via activation of one device more than the other so that midline changes occur as well. As with the external frame distraction device, the use of occlusal splints with good interdigitation of teeth into the splint can help guide the treatment path of the distracting maxilla, and as mentioned, as distraction progresses, changing the quantity of distraction on the right or left will change the midline position of the maxilla. Depending upon magnitude of the maxillary midline discrepancy, intermaxillary elastics can be used to settle the maxilla into its final occlusal resting place in the occlusal splint. The maximum possible distraction distance of the maxilla with currently available internal DO devices is 30 mm. For some internal distraction devices, once the distraction process has been completed, the activation arms that are visible in the oral cavity can be removed, leaving the rigid devices in place submucosally. At this point, there is nothing visible to the patient intraorally during the consolidation phase, and while healing of the bone is occurring, the device is typically left in place for 4 to 6 months postoperatively, although the consolidation period may be shorter in the 3- to 4-month period, as with external devices. After consolidation, local anesthesia can be used to remove the bone plates and distraction devices intraorally, although in some cases, bone may have healed over the plates, requiring a more aggressive approach for removal in an operating room setting. In most situations, by allowing additional time for consolidation with the distraction device in place, the patient usually avoids the need for a reverse-pull headgear in the post-DO period. Again, standard plates and screws may be used for additional stabilization when the buried DO devices are removed. If mandibular surgery is required for either internal or external distraction, it can be accomplished at the time of the initial surgery so that the mandible can be placed in any required position. Once the rigid fixation is accomplished in the mandible, the final splint is placed and the maxilla is distracted into the newly repositioned mandible.

During cleft orthognathic surgery, whether it is by traditional Le Fort techniques or DO, closure of fistulae should be accomplished whether they are palatal in nature or in the area of the alveolar cleft. Incisions should be made to account for the closure of the fistula. Careful attention should be made for manipulation of the nasal mucosa and closure with placement of a resorbable membrane in the area, and/or bone grafting, and then closure of the oral mucosa as a three-layer closure technique. If there is a significant bony defect in the alveolar cleft region or the piriform region, this should be augmented with bone, using the options discussed previously.

POSTSURGICAL CONSIDERATIONS

If at any time there is an early sign of relapse of the maxilla in the postoperative period, reverse-pull headgear should be applied along with vertical and class 3 elastics to help

maintain the position of the maxilla in the healing phase and limit the amount of relapse. Patients and families must understand that there will be a secondary procedure to remove hardware, which may require general anesthesia in a hospital setting. The orthodontist provides a critical role in the postoperative management of these patients.

Velopharyngeal Considerations

It is possible for the cleft patient who has no VPI to develop VPI after maxillary advancement surgery. This is especially true in the cleft patient who has not had any form of pharyngeal flap for correction of speech problems.[16] Occasionally, it is necessary to section the pharyngeal flap to achieve the desired maxillary advancement; this may cause worsened hypernasality and should be discussed with patients and parents before maxillary surgical advancement. Although the literature is mixed, maxillary DO in the cleft patient has been reported to result in unchanged velopharyngeal function most commonly.. Many studies have shown no difference in worsening of hypernasality when comparing Le Fort to maxillary DO in the cleft patient. It should be remembered that worsening of hypernasality after maxillary surgery in the cleft patient, with or without DO, ranges from 6% to 17%, and in similar reports, 70% of children had an improvement in articulation after maxillary anterior repositioning.[13] Perhaps the most appropriate way to consider the effect of maxillary surgery on VPI is to classify the preoperative VPI status into mild, moderate, and severe. Maxillary advancement, with or without DO, may cause a worsening of one level, such that a mild VPI may progress to a moderate VPI that is well tolerated, whereas a severe VPI is certainly unlikely to change with maxillary surgery, and a pharyngeal flap may be required for correction. The concerning VPI status is the moderate VPI patient that may be tolerated presurgically but may become a severe VPI with significant symptoms postsurgically. This group should be the focus of future research in the area of VPI effects of Le Fort versus DO surgery in the cleft patient.

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CHAPTER

Distraction Osteogenesis of the Maxillofacial Skeleton

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Distraction osteogenesis (DO), osteodistraction, or callatosis, is a biologic process of new bone formation that occurs between bone segments that are separated by gradual incremental traction. This process commences when distraction forces are applied to the callous tissues that connect the divided bone segments and continues as long as these tissues are stretched by the applied forces. The traction generates tension that stimulates new bone formation in the gap parallel to the vector of distraction.[1] These factors are based upon the law of tension-stress that states that gradual traction on living tissues creates stresses that stimulate and maintain the regeneration and continued growth of both hard and soft tissues. DO is a surgical-orthopedic process, rather than an isolated surgical procedure, involving gradual separation of a fracture callous, which produces an unlimited quantity of new bone, with associated adaptive soft tissues changes, referred to as *distraction histogenesis.* Theoretically, the overlying soft tissue envelope, including the skin, subcutaneous tissues, muscles, fascia, and periosteum, respond favorably to the hard tissue movements with gradual elongation and accommodation and, therefore, a lower resistance and decreased overall bony relapse, although the clinical application of this theory has been questioned.[2] Although the principles of DO were developed many years ago, the application to the maxillofacial region began in the late 1980s and early 1990s.[3] Maxillofacial DO was applied to complex craniofacial deformities, including craniofacial microsomia, in an effort to grow bone and avoid grafting and to reduce bone resorption and relapse of surgical movements. In consideration of the evolutionary stages of surgery in general, these four stages include ablation, reconstruction, transplantation, and induction[4]; DO is classified within the most progressive stage of induction, or regeneration, in that bone is formed de novo as a type of endogenous tissue-engineered bone. In the 1990s, DO was welcomed with a major paradigm shift in the approach to the craniofacial asymmetry patient. Introduced as a new "disruptive technology" with wide applications in the maxillofacial region initially, DO was considered a panacea when, in fact, standard surgical techniques were perfectly adequate at the time. This overzealous enthusiasm has been followed by a period of intellectual reproach and a focus of the indications for this treatment scheme.[5] This reassessment phase has included a reappraisal of the myriad complications that may occur with DO as well as strategies for prevention and management.[6] In Ilizarov's opinion, ". . . there are no complications with the technique; there are only inexperienced surgeons causing problems for their patients,"[7] but perhaps the more important issue here is to define the indications for the use of maxillofacial DO. These indications include craniosynostosis and upper face deformities; maxillary and mandibular orthognathic discrepancies; mandibular condylar abnormalities including ankylosis; severe asymmetries of the maxillofacial region; maxillary alveolar cleft deformities; segmental defects of the jaws due to trauma or pathology; neonatal airway obstruction due to mandibular deficiency; and alveolar ridge deficiencies (Table 63-1). Despite the controversies surrounding the current indications for DO, the technique provides an additional arm in the treatment algorithm for congenital and acquired dentoskeletal deformities, with the advantages of no bone grafting or donor site morbidity and the ability to titrate the bony movements and grow an unlimited amount of new bone and with the disadvantages of a surgeon learning curve, vector control problems, and patient and parent cooperation during the entire DO process. With the application of maxillofacial DO to the appropriate clinical scenario, there are clear advan-

TABLE 63-1. **Indications for Maxillofacial Distraction Osteogenesis**

Severe maxillary deficiency
Severe mandibular deficiency
Transverse jaw deficiency
Temporomandibular joint ankylosis
Obstructive sleep apnea
Neonatal upper airway compromise
Hemifacial microsomia
Treacher Collins syndrome
Mandibular segmental defects
Craniosynostosis
Alveolar ridge deficiency
Alveolar cleft discontinuity
Alveolar cleft maxillary deficiency

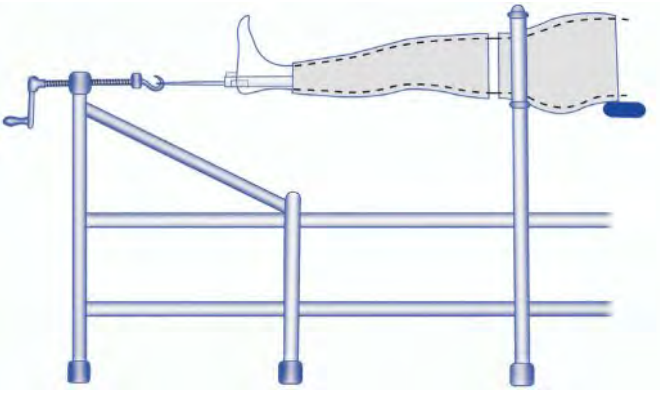


FIGURE 63-1. Early lower limb distraction osteogenesis (DO) traction device of Codivilla, 1905.

tages over conventional orthognathic and craniofacial surgical procedures, but it has become clear that DO will not replace standard surgical techniques, which can adequately accomplish the treatment goals in one surgical procedure, rather than through a process of gradual bone DO, which may take from weeks to months of treatment time.

HISTORY OF DO

DO was developed initially for orthopedic manipulation of long bones for a variety of problems. Although Ilizarov is generally credited with the concepts involved with distraction osteogenesis, it was Codivilla who first described the technique for femoral expansion in 1905[8] (Figure 63-1); and later, Abbott in 1927[9] used DO for tibial lengthening. In fact, Ilizarov is known as the "father of distraction osteogenesis," because he described the accepted principles of distraction osteogenesis in 1971.[10] 1951[C] that are still utilized clinically today. Ilizarov popularized the tension-stress effect by applying gradual traction to fractures and nonunions of endochondral long bones using large, external halo–type distraction devices (Figure 63-2) and recognizing that the increased cellular activity and metabolic response with neovascularization led to ossification of the site with bony union without the need for bone grafting. Ilizarov then realized that the mass or shape of these bones and joint articulations depends to a great extent on the available blood supply to the area as well as the functional burdens placed upon the bones (Wolff's law). Although orthodontists and oral and maxillofacial surgeons had been utilizing the principles of DO for years in the management of transverse maxillary deficiency, with both orthopedic sutural expansion in the growing patient and surgically assisted maxillary expansion in the adult, it was McCarthy who performed animal studies in the late 1980s that led to the clinical application of these principles to the maxillofacial region and the 1992 publication describing successful monofocal DO of the mandible in four children with craniofacial microsomia.[11] The average patient age was 6.5 years, with mandibular lengthening between 18 and 24 mm, with an 11- to 20-month follow-up period with minimal morbidity or complications. Before McCarthy, Snyder in 1973[12] used external DO devices in the canine mandible, and in 1977, Michieli and Miotti[13] used intraoral mandibular DO devices in the dog model. Throughout the 1990s, there was an explosion of new ideas related to DO device design, including external (extraoral and rigid external [halo] devices), internal (intraoral, subperiosteal, or buried), semiburied devices, toothborne or hybrid devices, and monofocal, bifocal, multifocal, and transport vectors, as well as the clinical indications for DO, including maxillary and mandibular deficiencies in the vertical, anteroposterior, and transverse planes, temporomandibular joint (TMJ) ankylosis, obstructive sleep apnea, neonatal airway compromise, mandibular continuity defects after tumor ablation or trauma, craniosynostosis, vertical and horizontal alveolar ridge deficiency, alveolar cleft maxillary deficiency, and alveolar cleft discontinuity. Since the explosion of interest in maxillofacial DO in the 1990s, surgeons who have experience with DO have focused the clinical indications for this technique and

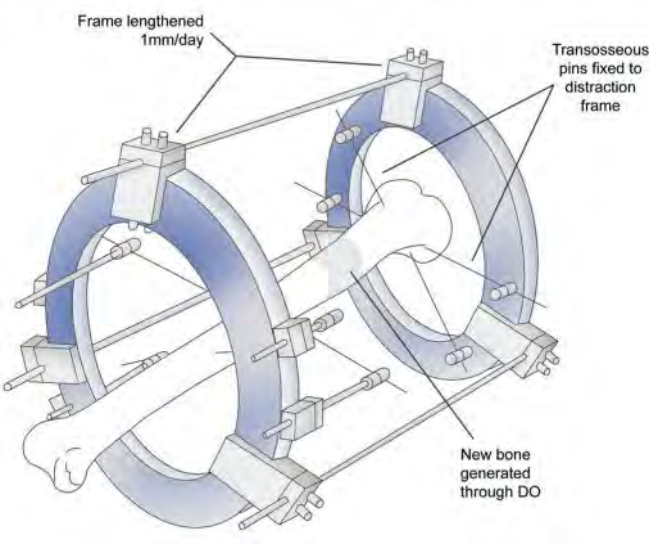


FIGURE 63-2. Lower limb lengthening halo DO apparatus of Ilizarov, 1951.

typically consider DO as an option only when conventional techniques for surgical correction are limited. In fact, only 4% of centers actively involved in performing DO on a regular basis have performed more than 100 cases of DO, making the designation of "experienced" a questionable misnomer. Even in the small number of known "DO centers," the reported incidence of complications with multivector DO is nearly 25%.[6] Therefore, in general, with other factors equal, the procedure with the lowest "cost" to the patient, in terms of finances, treatment time, and patient morbidity, should be used.

BIOLOGIC BASIS OF DO

Without a doubt, since the early 1990s, the unbridled enthusiasm about DO of the maxillofacial region has waned significantly since the resurgence of the technique and application to the maxillofacial region in the early 1990s.[5] Although the proposed benefits of DO that would allow it to replace traditional osteotomies for orthognathic surgery have not been borne out in clinical practice, DO maintains a place in the treatment of craniofacial malformations, specifically severe deformities in which there are poor conventional options for patient management. One of the major advantages of DO is the ability to lengthen bones to a greater degree than would be possible with orthognathic surgery, owing to the limitations based upon the envelope of discrepancy.[14] Unlike orthognathic surgery, the DO technique is not age-limited and could be applied not only to the skeletally immature child but also to the neonate with upper airway obstruction due to retrognathia in order to avoid a tracheostomy and permit normal speech and language development. DO is titratable, in that the clinician can continue to separate the bony segments until the desired results have been achieved (e.g., negative anterior overjet to account for some bony relapse or open airway at the tongue base in the Pierre Robin birth asphyxia patient). Also, the soft tissues are stretched slowly with DO so that they may gradually adapt to the bony movements and result in potentially less relapse than that achieved with conventional orthognathic surgery performed without rigid fixation techniques. In addition, regarding the TMJ, the mandibular condyle and disk position has additional time to adapt to any rotational changes that occur during distraction over a period of days or weeks, rather than acutely during orthognathic surgery in a period of minutes or hours. Also, with DO, there is no potential donor-site morbidity from the need for bone graft harvesting, which may be necessary, although uncommon, with orthognathic surgery. Both techniques are typically performed in the outpatient setting and, in fact, essentially in the same technical procedure, with application of distraction devices in lieu of plates and screws for orthognathic surgery. The cost of the DO procedure would typically be greater than orthognathic surgery because it usually requires two surgical procedures with general anesthesia in a hospital setting, and many more postoperative visits for the patient and family, as well as the additional cost of the actual DO devices, which is much greater than the cost of plates and screws. The disadvantages of DO include the additional time required for the entire treatment process, potential surgeon inexperience with a highly technique-sensitive procedure, the need for parent and patient cooperation, low incidence of major infection with maxillofacial DO, nerve paresthesia (trigeminal, facial nerves), TMJ dysfunction, facial scarring (pin tracts) from external devices, premature fusion of segments, malunion, device hardware failure with screw loosening and plate fracture, and relapse with the need for initial overcorrection. In addition, the issue of growth of the craniofacial skeleton after DO is unpredictable. In general, it is believed that in the syndromic patient, the deficient bone, or involved side of the jaw (e.g., hemifacial microsomia) has less inherent growth potential and may not grow normally after surgical correction, despite Wolff's law. Functional remodeling is expected to play a role in the growing process, yet overcorrection of 25% to 33% is advocated to allow for some relapse of the bony consolidate and expected lack of continued "normal" growth. Because more patients who had DO in the 1990s as children are growing into adolescence in the new millennium and at the present time, an assessment of these growth issues may allow further clarification in the future.

Another controversial advantage of DO in the adolescent cleft patient undergoing maxillary advancement is the theoretical lower incidence of velopharyngeal incompetence (VPI) development, or worsening, than with a standard Le Fort advancement. Based upon the present available literature, there is no consensus on VPI or speech alterations when comparing DO with Le Fort osteotomies.[15] In assessing the magnitude of maxillary advancement on VPI, other studies have shown no significant improvement in speech hypernasality, or VPI, with DO movements less than 15 mm.[16] Perhaps the most appropriate manner in which to consider the issue of VPI is to classify the preoperative VPI status into mild, moderate, and severe VPI, and then evaluate the possible outcomes of either Le Fort osteotomy or DO. In most cases, mild or moderate VPI cases may progress to moderate to severe VPI, whereas severe VPI is not likely to improve after maxillary advancement with either form of surgery, and other surgical options are usually required to address this problem independently (e.g., pharyngeal flap procedure).

The potential complications of the DO process are many and variable, and the problem is that not many surgeons or surgical teams have extensive experience in managing patients who have undergone DO. Complications of DO may occur during the preoperative phase, intraoperative phase, distraction phase, or postdistraction phase of the treatment process. In the preoperative phase, patient selection and vector planning are critical, and application of DO to the inappropriate patient or for the wrong indication may be problematic. Intraoperatively, an inadequate corticotomy may be performed, and the DO devices may be placed with an improper vector or with lack of sufficient device stability. During the distraction phase, the device may malfunction,

the vector may proceed in an improper fashion, and there may be dehiscence of the overlying soft tissues as well as facial scarring. In the postdistraction phase, there may be an inadequate total distraction distance, minor infections, a fibrous union, or malunion of the bone segments due to inadequate bony consolidation in the distraction gap resulting in potential bony relapse. Overall, many retrospective reviews[17] have shown that the most common complication with DO is nerve paresthesia (5% long-term), infection (<5%), condylar morphologic changes, incomplete osteotomy by the surgeon, device breakage, patient compliance problems, premature consolidation of bone, pin tract scarring, hypertrophic facial scarring, damage to teeth and roots, improper vector direction, TMJ ankylosis, hematoma at the distraction site, and periodontal problems. As expected, the more complex the DO device used (multivector vs. univector), the higher the incidence of complications, and patients of advanced age also have a higher incidence of complications, especially persistence of nerve paresthesia. In both animal[18] and clinical[19] studies, the incidence of prolonged paresthesia of the inferior alveolar nerve is variable, but generally remains low with mandibular DO corticotomies; and younger patients recover spontaneous neurosensory function more rapidly after nerve injury and to a greater degree or magnitude than older patients. With regards to the development of TMJ problems after DO, several reports have indicated the findings of hypomobility or fibrous ankylosis depend upon the specific direction and magnitude of mandibular movement, although more recent studies indicate that the TMJ accommodates to the DO process with no long-term functional TMJ abnormalities after mandibular DO.[20]

Regarding the overall process of new bone formation and transformation that occurs as a result of DO, this begins with the formation of a hematoma at the site of the corticotomy, or bone fracture, as expected, with organization of the clot as functional stresses are introduced with DO. As the process of DO progresses, the histologic appearance of the bone in the distraction gap becomes organized into four distinct zones of varying bone maturation (Figure 63-3), including a central zone of fibrovascular granulation tissue composed of collagen fibrils and new capillaries that are oriented parallel to the vector of distraction. The next zone is that of extending bone formation, followed by the zone of bone remodeling, and finally, the zone of mature bone, which gradually transitions into the normal adjacent native cancellous bone. Throughout the latency, activation, and consolidation phases of distraction, this bone gradually transforms from immature, woven bone with neovascularization to a more mature lamellar bone end product with minimal callus formation (Figure 63-4). As the consolidation continues, the periosteum becomes proliferative and will re-form a cortical outline around the newly formed cancellous marrow; this cortex is responsible for the strength of the bony consolidate that will prevent relapse.

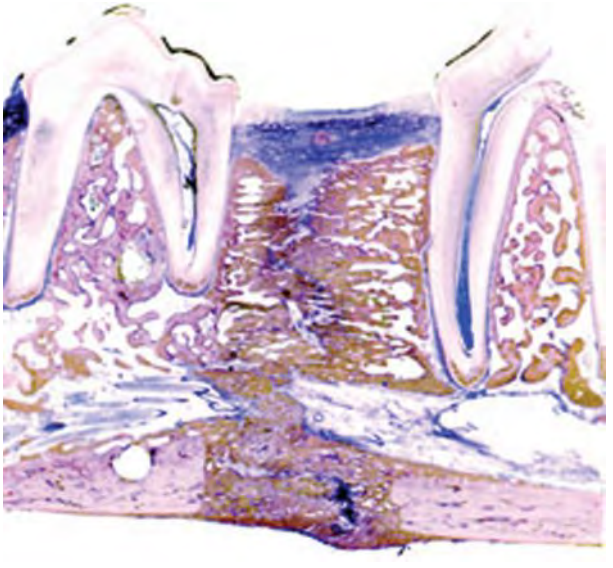


FIGURE 63-3. Healing of the distraction gap with four zones of bone formation (central, extending bone, bone remodeling, and mature bone zones).

PRINCIPLES OF DO

Although the original description of the technique of DO by Ilizarov has been modified and should be adjusted based upon the specific clinical scenario and location of distraction, the basic principles remain unchanged (Table 63-2). The first principle involves the *surgical technique* used to create the separation between the bones. For long bones and the mandible, a corticotomy is performed, rather than a complete osteotomy, in order to preserve the endosteal continuity and blood supply in the distraction gap before the initiation of distraction in order to support the internal milieu of the ensuing distraction gap and provide the most conducive environment for bone formation (Figure 63-5). This has been referred to as "rotational osteoclasis." In most cases, a reciprocating or micro-oscillating saw, or rotary handpiece with a thin fissure bur, is used to perform the majority of the corticotomy, and a bone chisel and osteotome are used to complete the bony fracture. In the mandible, the location of the corticotomy is typically in the body, angle, or ramus region depending upon the intended distraction vector, horizontal (body), oblique (angle), or vertical (ramus), and a corticotomy will not section across the inferior alveolar canal or damage developing tooth buds that would occur if a complete osteotomy were performed. For the maxilla, a true corticotomy is not possible owing to the nature of the thin bone so, a standard Le Fort osteotomy is generally performed. At the same time as the corticotomy, the DO device is applied to the mandible, and the specific device and planned vector dictate the angulation of the corticotomy itself; so, in many cases, the device is applied before performing the corticotomy in order to visualize the area for the planned corticotomy and for placing a score mark in the midpoint between the stabilization plates and the screws of the DO device. Depending upon the specific DO device used, it may be secured to the mandible from an extraoral

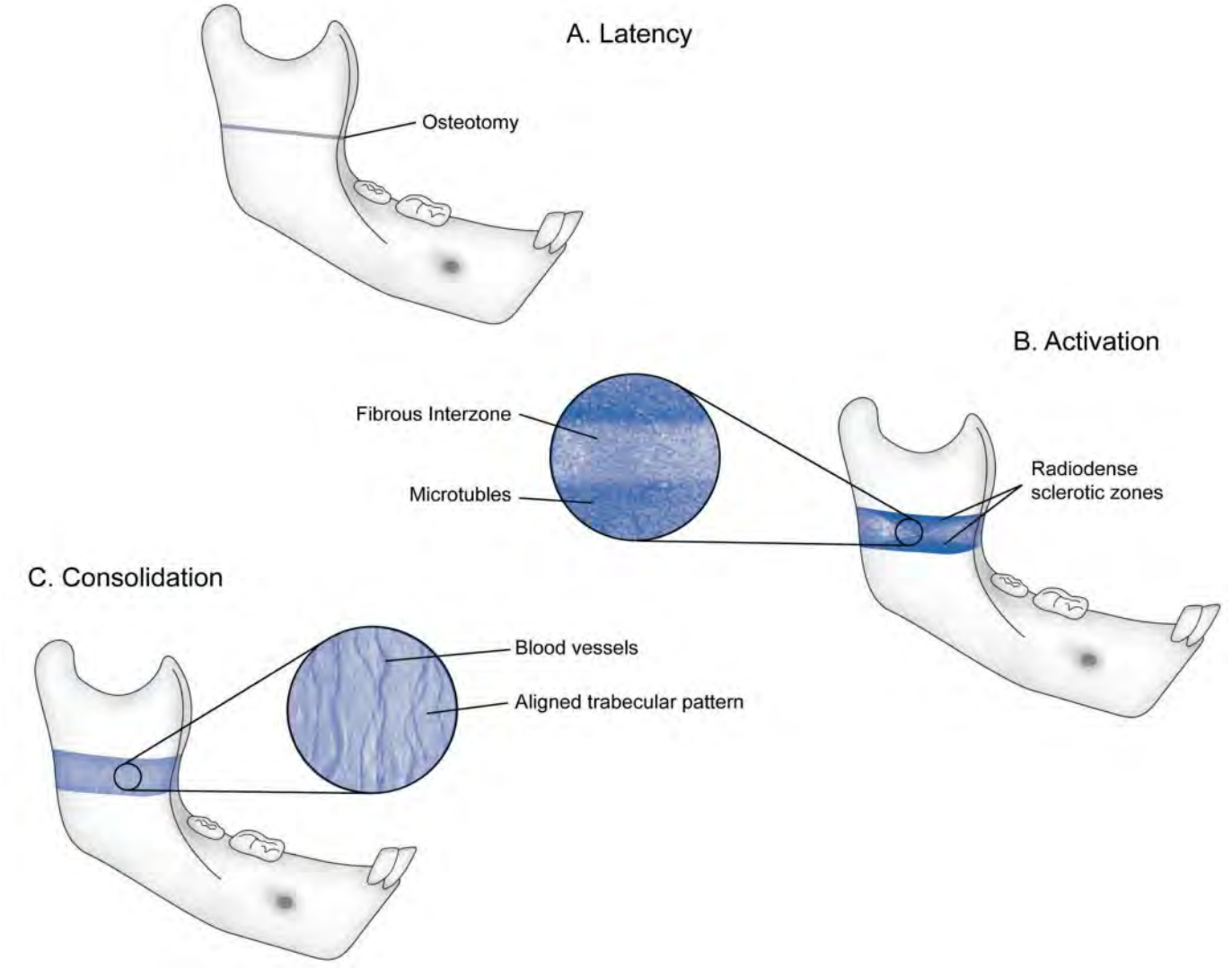


FIGURE 63-4. Gap bone healing during latency, activation, and consolidation.

transfacial approach using transcutaneous pins that are then secured to the devices, or transoral DO devices may be secured to the mandible with standard bone screws. The extraoral pins should be placed parallel to each other and perpendicular to the distraction vector, and in order to minimize pin tract scarring, it is advisable to pinch the skin and subcutaneous tissues together between the pins during transcutaneous pin placement. Before wound closure during surgery, the distraction devices must be activated to confirm that movement of the bone segments will proceed in an uninterrupted fashion, because failure to achieve segment distraction at the time of surgery must be corrected at this point in time during surgery. Many times, this maneuver will stretch the cancellous bone, but may also minimally convert the corticotomy to an osteotomy through physical separation of the bony segments. After activation of the devices at surgery, deactivation should occur in order to bring the bone edges in close proximity without compressive forces.

TABLE 63-2. **Principles of Distraction Osteogenesis (Ilizarov)**[7]

1. Surgical procedure
   a. Corticotomy
   b. Device application
2. Latency period (3–7 days)
3. Activation period
   a. Rate (1.0 mm/day)
   b. Rhythm (daily to four times/day)
4. Consolidation period (8–12 wk)
5. DO device removal

DO = distraction osteogenesis.

The second principle involves the *latency period*, defined as the interval between the initial surgical procedure (corticotomy and device application) and the device activation, typically ranging from 5 to 7 days (Table 63-3). The primary reason for the delay is to permit the establishment of a fibrovascular bridge across the endosteum of the bony segments that will act to support future bone formation during

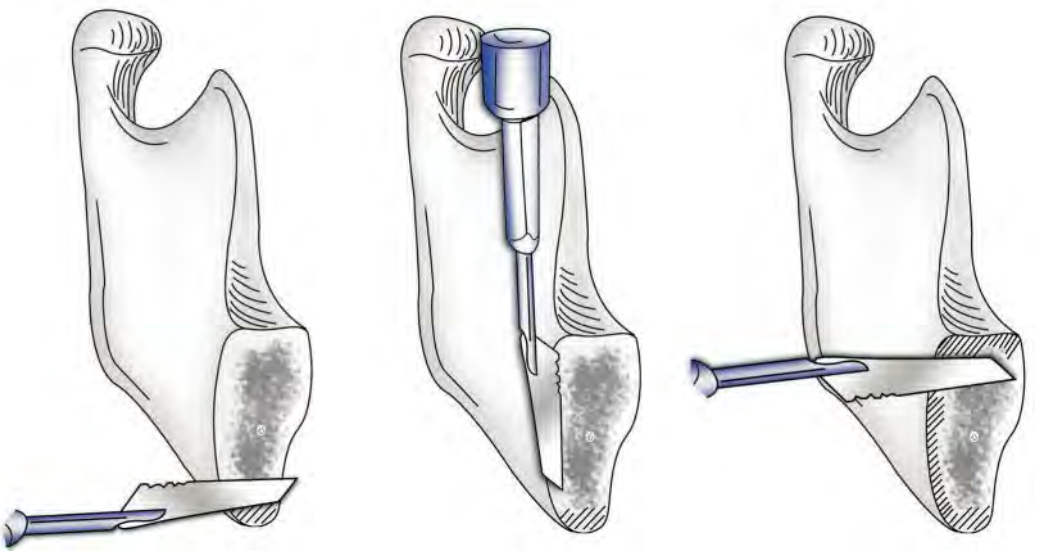


FIGURE 63-5. Corticotomy in posterior mandible with a reciprocating saw.

the distraction period. This time delay is prudent considering that even if a corticotomy is performed, there may be transient interruption of endosteal continuity and vascularity owing to the initial surgical insult as well as postsurgical edema. Within the first 24 hours, there is vascular ingrowth and mesenchymal cell proliferation, and within 24 to 72 hours, the early stages of granulation tissue (fibroblasts and capillaries) formation begin. Therefore, the supportive fibrovascular connection across the corticotomy site should be established in a 5- to 7-day period in the skeletally mature patient. The specific length of time chosen for this latency period is critical because waiting too long may result in premature fusion of the segments with inability to perform distraction and not waiting long enough may result in the lack of a stable vasculature in the bony gap leading to a nonunion, or fibrous tissue interposed between the bony segments at the completion of distraction. The latency period is also age-adjusted, such that a shorter period is used for children (2- to 3-day latency) and neonates (zero-day latency), whereas a longer period is employed for elderly patients (7- to 10-day latency).

The third DO principle involves the *activation period*, which includes both the rate and the rhythm, or frequency, of distraction. For the typical adult patient, the usual rate of distraction is 1.0 mm/day in divided increments, generally 0.5 mm twice daily. Again, age plays a significant role in the decisions regarding rate, such that in children and adolescents, a rate of 2.0 mm/day may be chosen, whereas for neonatal distraction, the rate can vary from 3.0 to 5.0 mm/day. If the rate is too slow, especially in the very young patient, there may be premature fusion of the proximal and distal segments, whereas too rapid a distraction rate may result in a fibrous nonunion because the microvasculature may be compromised. The specific rhythm used is a subject of debate because, initially, it was believed that continuous distraction throughout the day would be ideal, although it appears as if incremental, intermittent traction of the bony segments produces advantageous periosteal stresses that serve to enhance bone regeneration in the distraction gap. Therefore, twice-daily (bid) rhythms are employed in most patients for ease and patient compliance, so that distractions may occur at set times each day (e.g., 8:00 AM and 8:00 PM). If there is discomfort during the distraction activation, especially toward the end of the process when bone healing is advanced and consolidation is beginning, the rhythm may be adjusted to three times daily (tid) or four times daily (qid) schedules (e.g., 0.25 mm qid), and predistraction analgesics (nonsteroidal anti-inflammatory drugs [NSAIDs]) may be helpful for patient comfort. Typically, each specific distraction device is supplied with a screwdriver to activate the distraction, with an "arrow" inscribed on the screwdriver, as

TABLE 63-3. **Distraction Protocols**

Location	Latency Period (days)	Rate (Total mm/day)	Rhythm	Consolidation Period (or 2 × Activation Period)
Mandible or maxilla: adult	5–7	1.0	bid	8 wk
Alveolar ridge	5–7	0.5–1.0	bid-tid	4 wk
Transport segment	5–7	1.0	bid-tid	4 wk
Mandible: neonatal	0	2.0–4.0	bid-qid	2 wk
Mandible: child	2–3	2.0	bid	3–4 wk
Mandible: elderly	7–10	0.5–1.0	qd-bid	10–12 wk

well as the distance of distraction achieved with one complete 360-degree turn of the screwdriver to ensure that patients or parents perform the turns correctly. Multivector devices may add confusion because there may be more than one movement performed during each activation of the device (x, y, and z axes). It should be remembered that the bony regenerate is "moldable" during this period, and it could be influenced by the use of orthodontic elastics, which can also affect the occlusal relationships of the dentition. This plasticity of the distraction bone can be used to bend the bone in cases in which vector correction is required (e.g., use of vertical anterior elastics for closure of an anterior open bite malocclusion).

The fourth principle of distraction involves the *consolidation period*, or period of neutral fixation, that has the greatest degree of variability based upon surgeon preference. This is the period of time for bony consolidation that occurs between the completion of distraction and the removal of the distraction device. In the adult patient, this is usually 8 to 12 weeks, whereas in the young patient, there may be sufficient strength in the bony regenerate immediately after completion of distraction to allow for removal of the DO devices immediately after distraction. It has been suggested that the consolidation period should equal twice the activation (distraction) period, so that the time is based upon total distraction distance; for example, if the activation period was 14 days, then the consolidation period should be approximately 28 days in length. During the consolidation period, a large, bulky extraoral multivector device may be replaced with a lightweight carbon fiber rod supplied by the companies for patient comfort. There is extreme variability in the length of the consolidation period because it is unclear exactly when device removal is appropriate and will not result in relapse owing to premature removal. An objective means of evaluating whether bony stability would exist if the DO devices were removed is limited to radiographic documentation that a bony cortex exists or that there is sufficient bone fill in the distraction gap. Because this period of the DO process is the least understood and adds the most time to the overall DO process, there has been a great deal of research interest in acceleration of the consolidation period by treating the distraction gap region with either chemical factors, such as growth factors, cytokines, insulin-like growth factor-I (IGF-I), fibroblast growth factor (FGF), transforming growth factor-beta-1 (TGF-β-1), and interleukin-6 (IL-6), as well as physical factors, including ultrasound stimulation, low-level laser stimulation[21] and electromagnetic therapy.

The fifth, and final, principle of distraction involves the *removal of the DO device*. Depending upon whether the devices are intraoral, extraoral, buried, or pin-retained, removal may be a simple procedure performed under local anesthesia or intravenous sedation in an outpatient clinic or it may require more extensive surgery performed under general anesthesia in an operating room setting. There are technical developments ongoing that may allow the foot plates of buried devices to remain attached to the bone, possibly with resorbable materials (e.g., oly-G-lactic acid [PGLA], poly-L-lactic acid [PLLA] co-polymers), which would make the removal of buried devices simpler without the need for extensive reexposure of the surgical site. Further refinements and technologic advances in materials may allow fabrication of completely resorbable DO devices in the future. It should be noted that, in some cases, surgeons may choose to place titanium bone plates and screws in a conventional orthognathic surgical manner at the time of DO device removal ("hybrid" DO-orthognathic procedure) to prevent relapse in specific circumstances in which relapse is a major concern (e.g., Le Fort advancement in the cleft patient).

PATIENT EVALUATION AND VECTOR PLANNING

A comprehensive history and clinical examination should be performed in a manner similar to that done for the orthognathic surgical patient with an asymmetry. Particular attention should be directed toward examining the patient in a natural head position (NHP), despite asymmetries in orbitozygomatic position, ear deformities, and occlusal cants. Frontal, profile, bird's-eye, and worm's-eye views are helpful to visualize both hard and soft tissue volume and landmarks. A functional TMJ examination should be performed, especially with mandibular asymmetries. The standard radiographic evaluation should be used, including periapical radiographs to plan for interdental osteotomies, if necessary, a panoramic radiograph to assess the dentition and condylar/ramus morphology, and a posteroanterior cephalometric radiograph to assess orbitozygomatic discrepancies and occlusal cants. Care must be taken to maintain the patient's head in the NHP during the cephalometric films, such that in cases of microtia, or melotia (low-set ears), the ear rod on the affected side is allowed to contact the scalp passively in the temporal region, and it is not placed within the external auditory canal as on the unaffected side. The use of three-dimensional computed tomography (CT) scanning and computer planning is becoming invaluable in the assessment and treatment planning of craniofacial asymmetries and also in DO device design and vector planning.

The choice of distraction device depends upon several variables including the specific anatomic abnormality, the desired distraction vector, the overall magnitude of bony movement, the planned surgical access as well as access for device activation, patient and parent preferences, and surgeon experience. The DO devices are classified according to several factors. In relation to the skin surface, DO devices may be external or internal, and these internal devices may be buried or semiburied and placed transorally or transfacially. Devices may also be classified according to the type of tissue that anchors the devices, including boneborne, toothborne, or hybrid (a combination of bone- and toothborne) devices. Lastly, devices are classified according to the number of potential vectors of movement (Figure 63-6) as monovector (or univector or unidirectional, one vector),

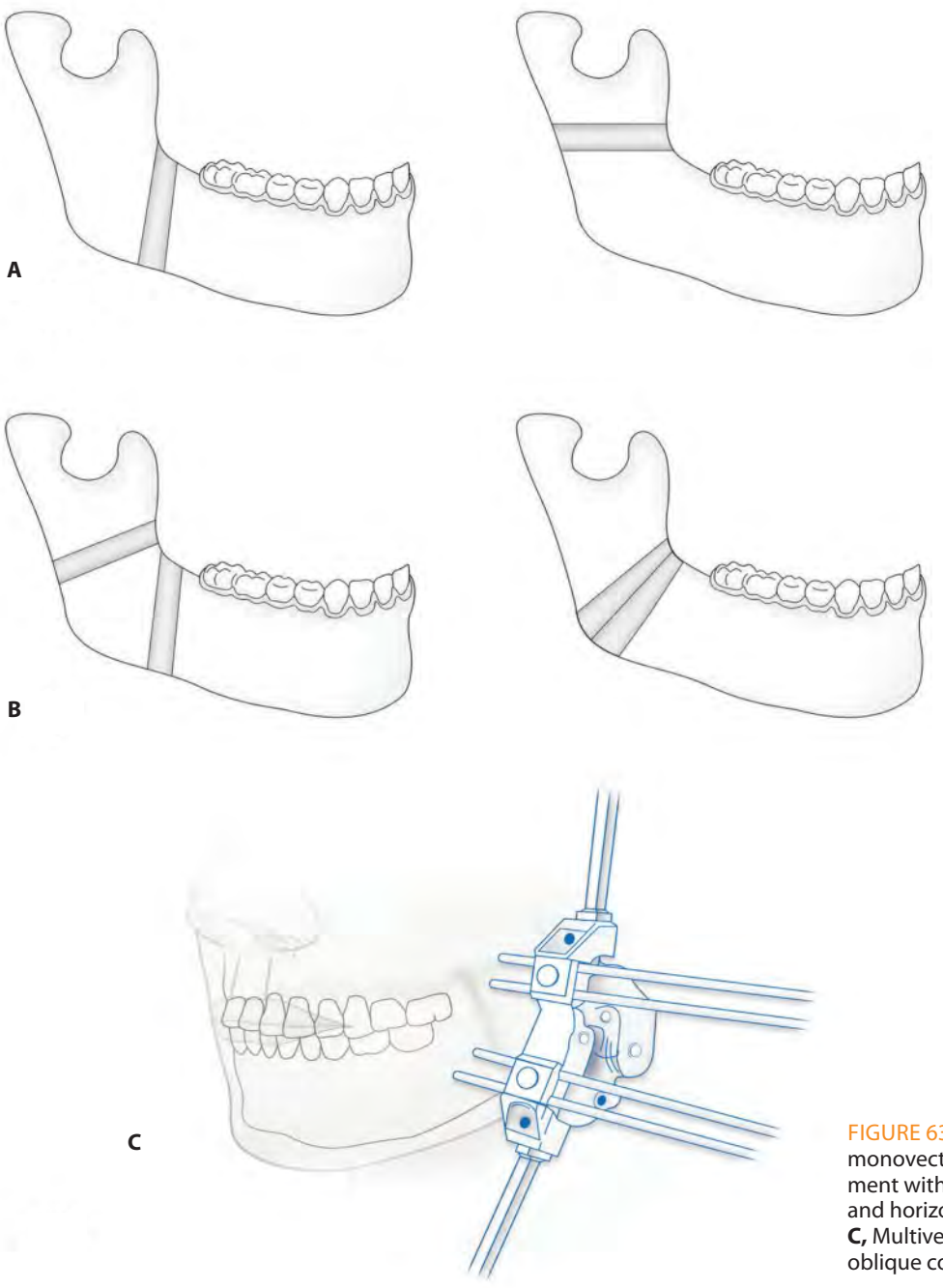


FIGURE 63-6. **A,** Vertical and horizontal monovector corticotomies. **B,** Bivector movement with either two corticotomies (vertical and horizontal) or one oblique osteotomy, **C,** Multivector movement achieved with one oblique corticotomy and a multivector device.

biplanar or bidirectional (two vectors), and multivector (or multiplanar, more than three vectors) devices. In general, the need to use a multivector device requires external placement with potential facial and pin tract scarring; but this problem should be balanced with the advantages of ease of access to the activation devices and the full control of multiplanar vectors in three planes of space (x, y, and z axes). Monovector devices usually require only one corticotomy and use a "straight screw" type device to achieve simple linear elongation of bone in a vertical, horizontal, or oblique vector. A bidirectional device may require two corticotomies performed perpendicular to each vector and the use of a specific device to achieve both vector movements, or a specialized device may be used with only one corticotomy to achieve biplanar movement (Figure 63-7). For maxillary distraction, either internal or external devices may be used (Figure 63-8) and the RED (rigid external device), which uses a maxillary occlusal splint as well as an external multivector control arm, is used for maxillary and midface distraction in cleft and craniosynostosis cases.

The issue of distraction vector, or trajectory of movement of the bone segments, is important to consider for both the maxilla and the upper face as well as the mandible. However, most efforts have focused on distraction vector planning in the mandible (Figure 63-9). The three mandibular vectors are based upon the relationship of the direction of the corticot-



FIGURE 63-7. Oblique osteotomy with vertical and horizontal vector Wood distraction device.

omy to the occlusal plane. A vertical vector, perpendicular to the occlusal plane, is achieved with a horizontal osteotomy of the ramus, whereas a horizontal vector, parallel to the occlusal plane, results from a corticotomy performed vertically in the body or angle region of the mandible. An oblique vector, resulting in both vertical and horizontal elongation of the mandible, will result when the corticotomy is performed at an angle between zero degrees (the vertical vector) and 90 degrees (the horizontal vector) to the occlusal plane, generally in the angle region of the mandible. With regards to vector planning, it must be recognized that there are both biologic and mechanical forces that will significantly affect the direction of distraction (Figure 63-10). The biologic vectors result from the neuromuscular soft tissue envelope including the muscles of mastication and suprahyoid musculature. These forces may influence the distraction vector unpredictably, and the surgeon has little control over these factors. The mechanical vectors, conversely, are under full control of the surgeon and orthodontist involved in the care of the distraction patient. These mechanical forces include the distraction device itself, the intercuspation of the teeth, bone segment contact, the TMJ apparatus, and of course, the angulation of the corticotomy and DO device placement that determines the actual direction of vector movement. As an example of the result of the combined biologic and mechanical vector forces, a vertical distraction device in the ramus will result in counterclockwise rotation of the mandible with a posterior open bite occlusion, whereas a

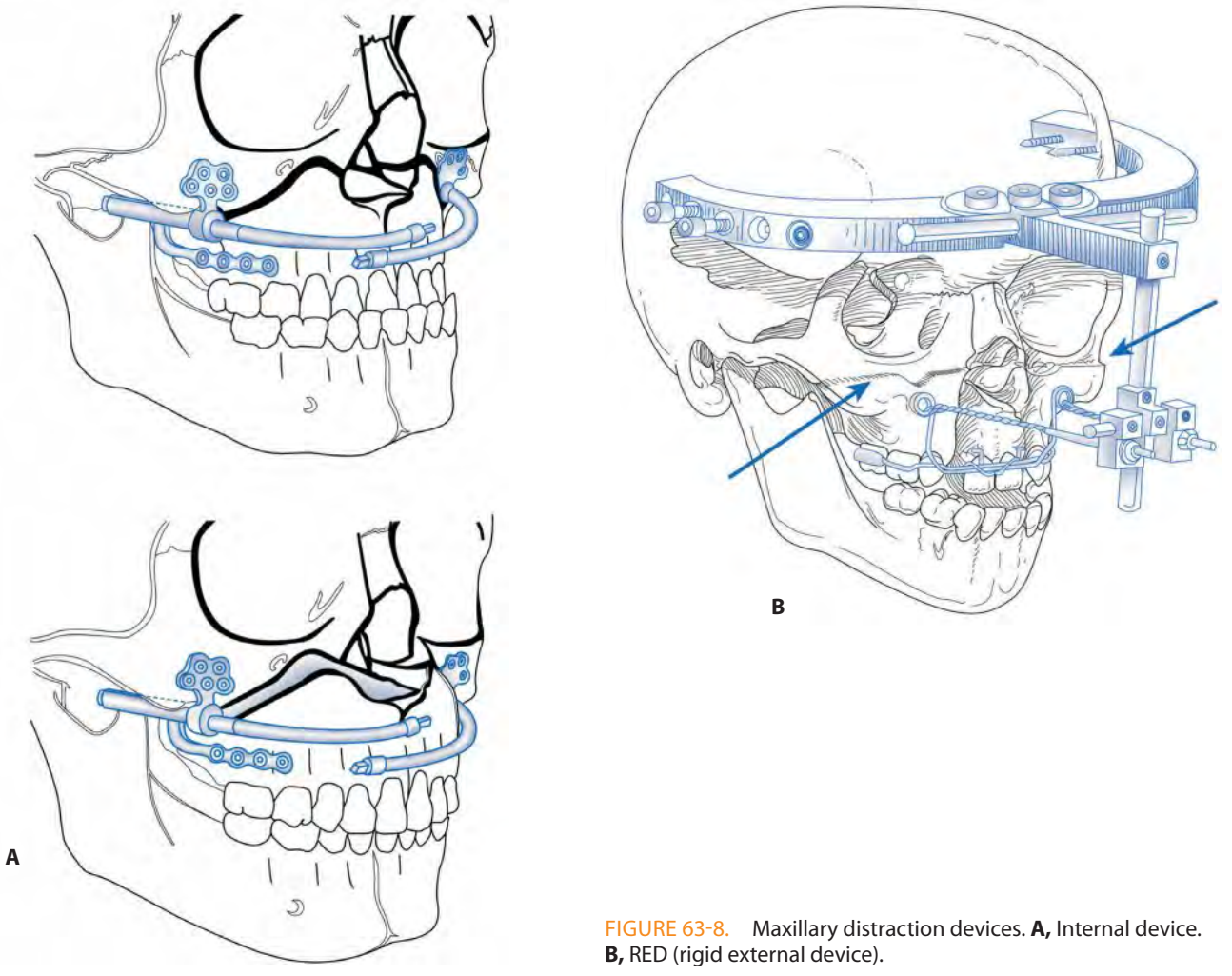


FIGURE 63-8. Maxillary distraction devices. **A,** Internal device. **B,** RED (rigid external device).

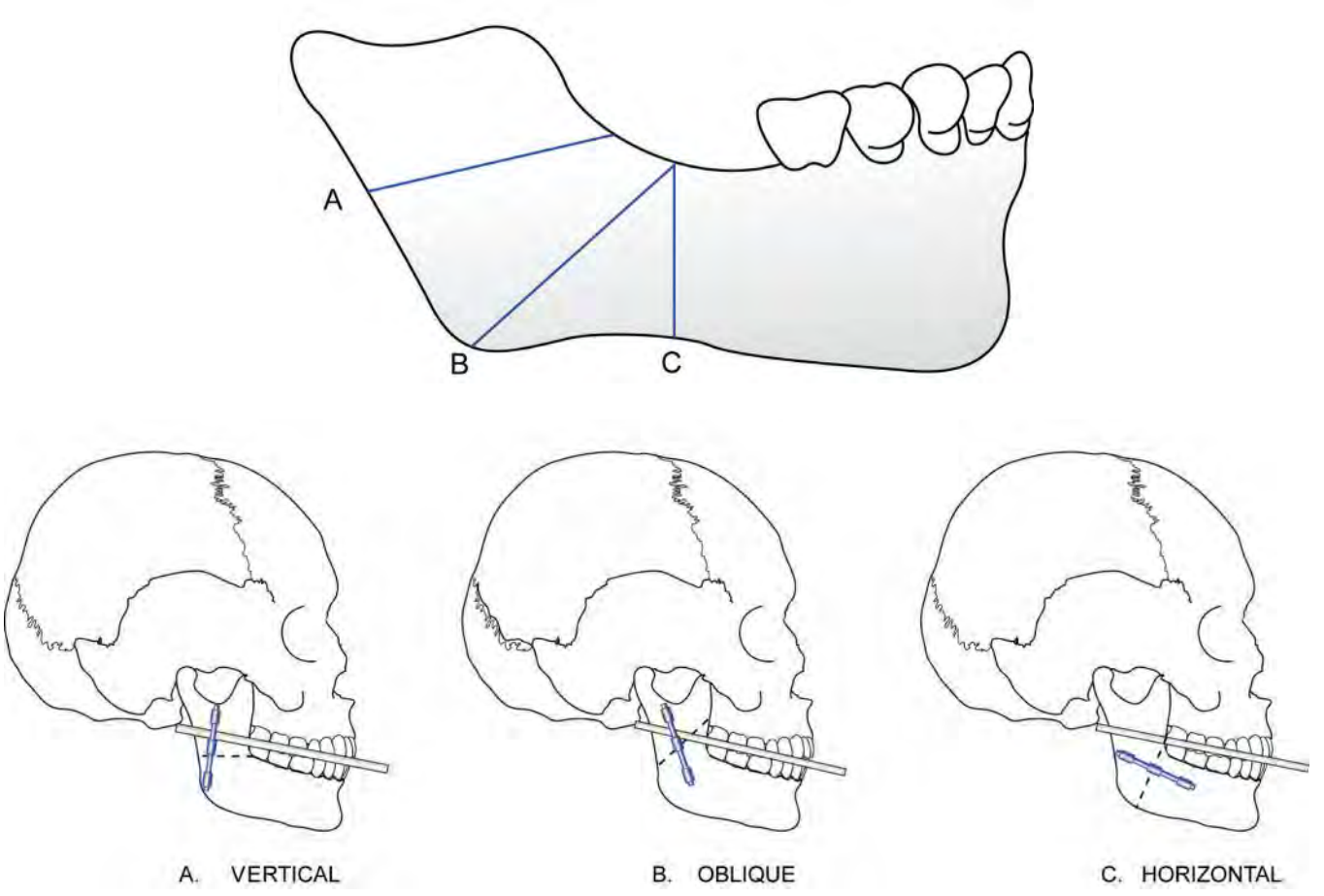


FIGURE 63-9. **A,** Osteotomy design for mandibular distraction. **a,** Horizontal osteotomy results in vertical vector. **b,** Oblique osteotomy results in oblique vector. **c,** Vertical osteotomy results in horizontal vector. **B,** Mandibular distraction vectors. **a,** Horizontal osteotomy *(dotted lines)* (parallel to occlusal plane) results in vertical vector. **b,** Vertical osteotomy (perpendicular to occlusal plane) results in horizontal vector. **c,** Oblique osteotomy results in oblique vector.

horizontal mandibular distraction vector has the tendency for clockwise rotation of the mandible with the development of an anterior open bite occlusion, due to the biologic vectors, including the suprahyoid musculature (Figure 63-11). Additional considerations in vector planning involve unilateral cases in which rotational forces also influence movement of the mandible (e.g., hemifacial microsomia). In an attempt to determine the precise placement of a distraction device to achieve the desired vector, a geometric formula may be used to allow proper positioning of the pins, or screws, of the distraction device. The pin (or screw) placement angle is determined by measuring the vertical and horizontal deficiencies in the mandible as well as the gonial angle of the mandible (condylion-gonion-menton), because this will dictate the intended vertical and horizontal movement of the mandible. The pin placement angle is determined (The formula is shown in Figure 63-11.): 180 subtracted by the gonial angle and multiplied by the product of the vertical deficiency (condylion-gonion distance) divided by the total deficiency (vertical plus horizontal [gonion-menton distance] deficiency). In the example shown with a 10-mm horizontal and a 10-mm vertical deficiency, with a gonial angle of 140 degrees, the pin placement angle is 20 degrees to the mandibular plane (gonion-menton). This formula can assist the surgeon if three-dimensional imaging and planning is not available. Remember that in the maxilla, the vector is mostly dependent upon the specific DO devices chosen (usually bilateral devices are necessary), including buried internal devices and REDs. There is generally a horizontal, or anteroposterior, vector in most maxillary distraction cases; however, vertical change may be achieved with intermaxillary vertical elastics or by variable adjustment of the external device and RED.

Regarding the technical aspects of distraction, the bone segments may be separated in a monofocal, bifocal, or trifocal fashion (Figure 63-12). The monofocal mode will separate two bone surfaces in a standard DO fashion, whereas a bifocal distraction will utilize a transport disk of bone that is moved through the soft tissues toward another segment of bone. The trifocal mode will move two segments of bone as transport disks toward each other, and specialized devices are available to assist with transport distraction. The bifocal and trifocal modes of transport distraction are typically used for reconstruction of segmental defects of the mandible

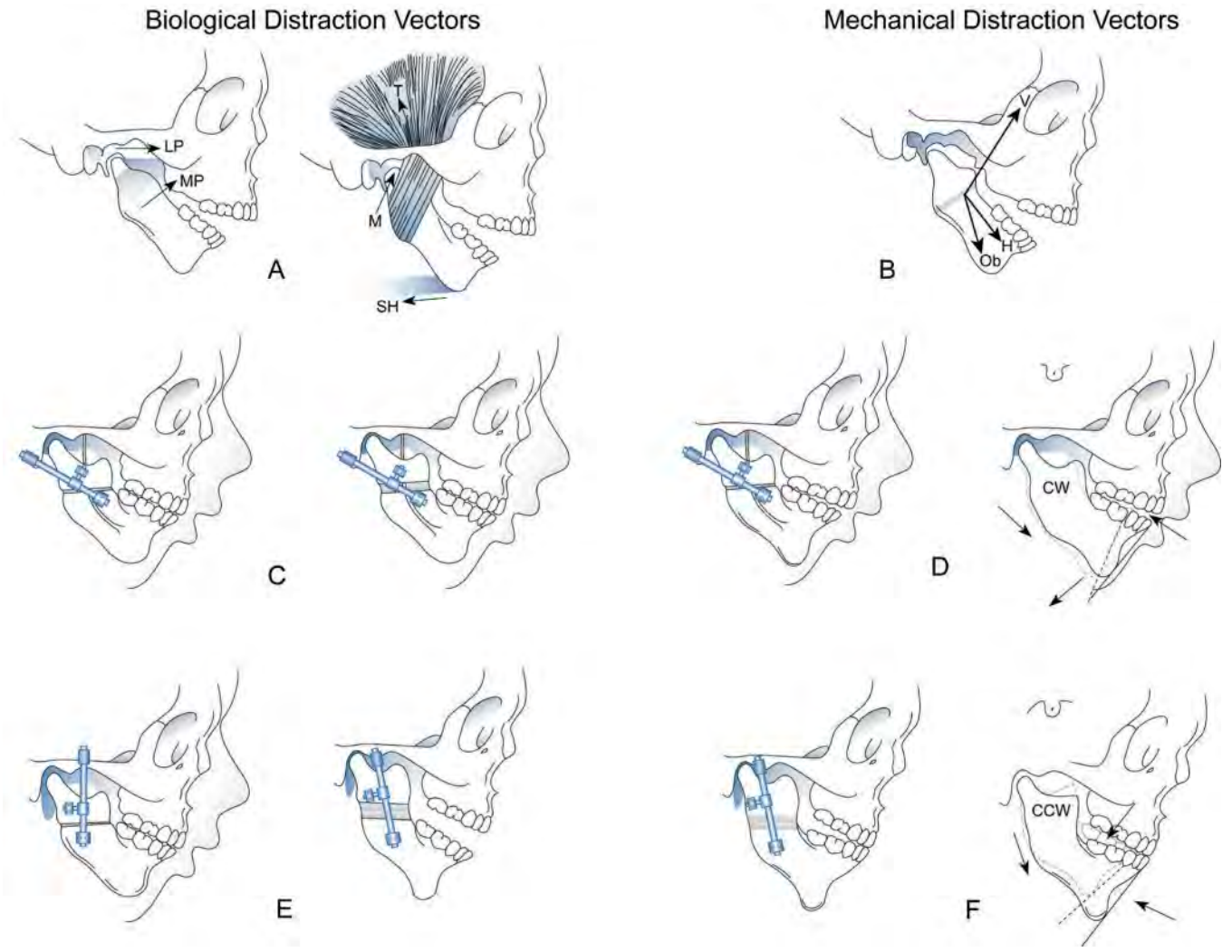


FIGURE 63-10. **A,** Biologic distraction vectors. LP = lateral pterygoid; M = masseter; MP = medial pterygoid; SH = suprahyoids; T = temporalis. **B,** Mechanical distraction vectors determined by corticotomy design and device placement. H = horizontal; O = oblique; V = vertical. Horizontal (H) vector **(C)** results in clockwise rotation (CW) **(D)** of the mandible with an anterior open bite occlusion. Vertical (V) vector **(E)** results in counterclockwise rotation (CCW) **(F)** of the mandible with a posterior open bite occlusion.

(Figure 63-13). With transport distraction, the transport disk of bone will develop a fibrocartilaginous cap on the advancing front of the bone segment as it moves through the subcutaneous soft tissues. When used for mandibular segmental defect reconstruction, this cartilage cap must be resected at the time of device removal and may require additional bone grafting at the junction site as well as the use of rigid fixation devices for stabilization. The formation of this cartilage cap can be used as an advantage in cases of TMJ reconstruction with DO (Figure 63-14). For example, in cases of TMJ ankylosis, after gap arthroplasty, a vertical ramus osteotomy (VRO; or variation) is performed with application of a distraction device with a vertical vector to distract the proximal bone segment into the newly created glenoid fossa. This particular transport disk will become the new condyle with a desirable cartilaginous cap to prevent future ankylosis, and it will function as a normal condyle as it assumes a more normal anatomic size, shape, and position.[22]

ORTHODONTICS FOR MAXILLOFACIAL DO

The orthodontist may play a critical role in the complex management of the patient undergoing DO as an integral member of the team for growth assessment, occlusal, and aesthetic evaluation and treatment planning.[23] The role of the orthodontist is in vector planning and modification, if necessary, during distraction, preparation of the dentition for DO, management of the dentition during active DO, and finalizing the occlusion at the completion of DO. The three phases of orthodontics for DO include predistraction, active distraction, and postdistraction treatment. Pre-DO orthodontics is similar to pre-orthognathic surgery preparation using fixed orthodontic appliances to eliminate dental compensations, coordinate arches, correct transverse deficiencies, correct occlusal disharmonies, eliminate crowding and potential dental interferences with distraction (e.g., retroclined maxillary incisors inhibiting forward mandibular movement), and

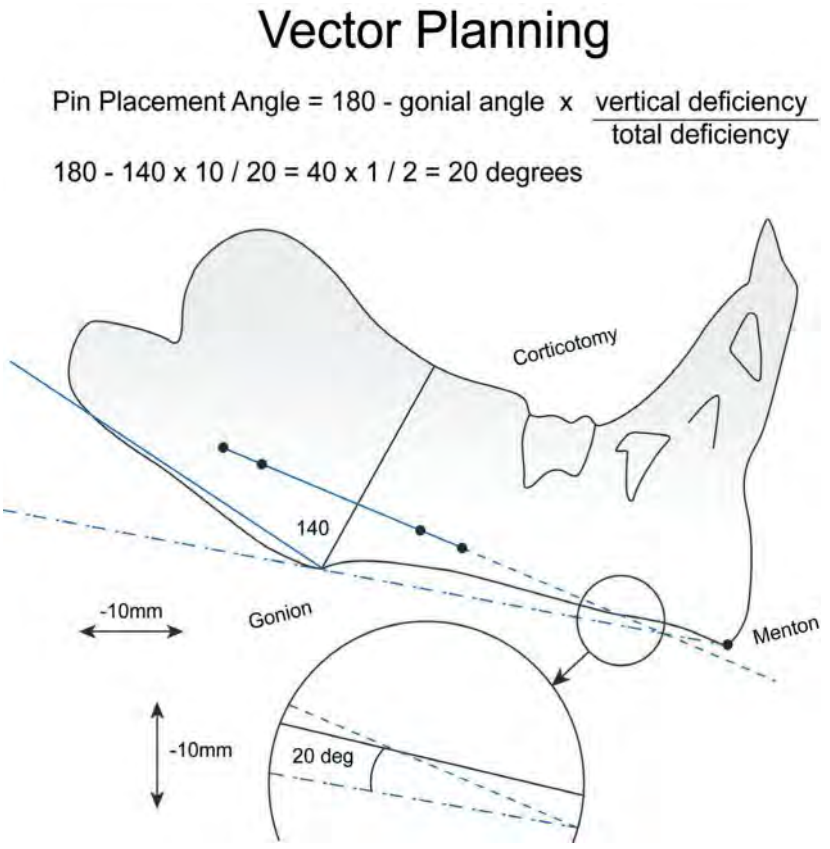


FIGURE 63-11. Vector planning formula for mandibular distraction. In the example, there is a 10-mm vertical and a 10-mm horizontal discrepancy that results in a pin placement angle of 20 degrees in reference to the mandibular plane (menton-gonion).

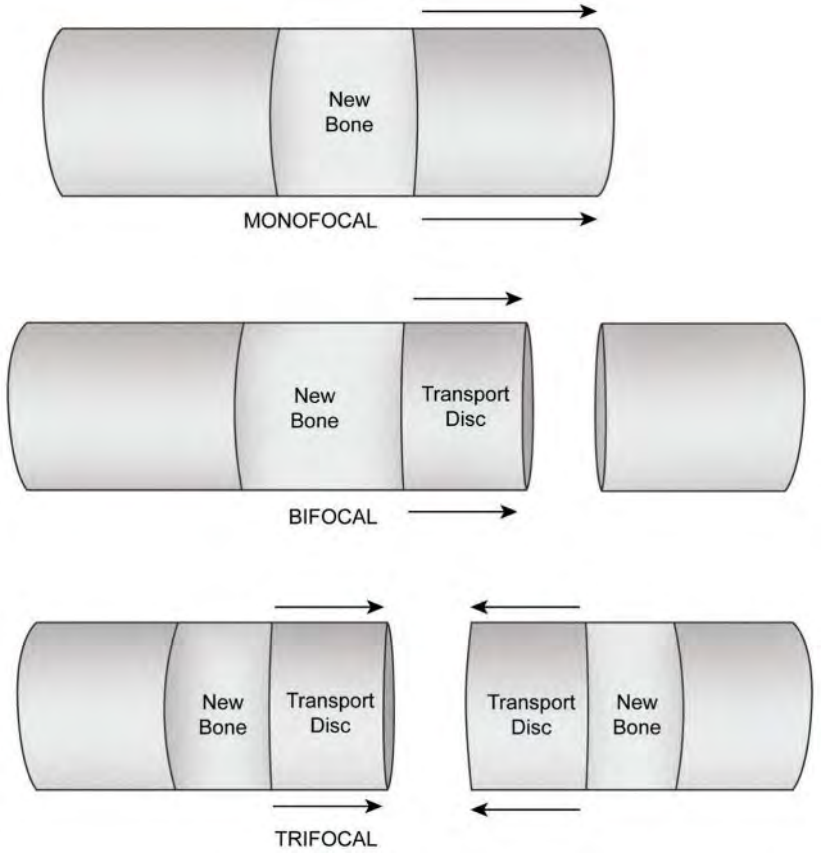


FIGURE 63-12. Monofocal, bifocal, and trifocal distraction schemes, based upon movement of the transport segment of bone.

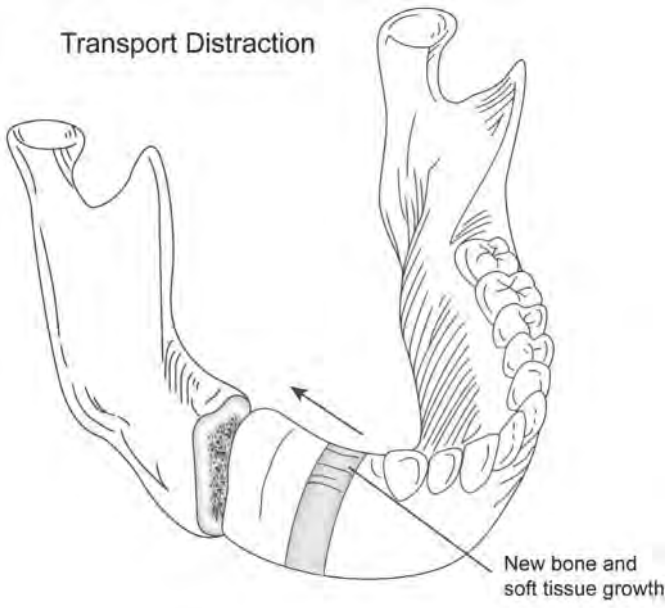


FIGURE 63-13. Transport distraction for segmental defects of the mandible. As the transport segment advances through soft tissues, a fibrocartilaginous cap forms that requires resection after DO.

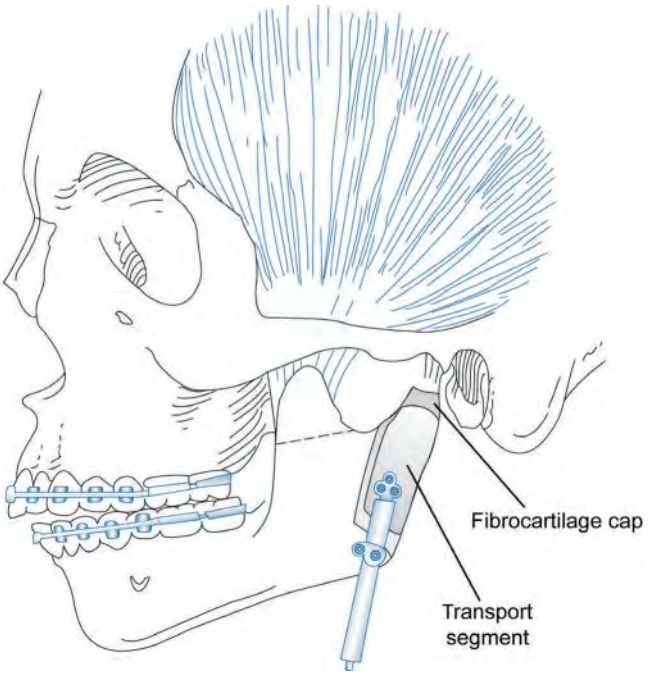
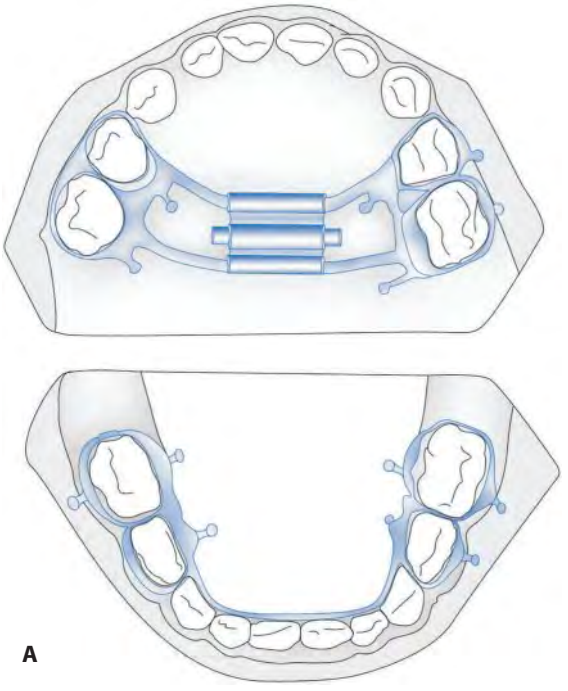


FIGURE 63-14. Condylar segment transport distraction. After condylectomy, a modified vertical ramus osteotomy with application of a DO device with vertical vector (and coronoidectomy). The transport segment moves toward the glenoid fossa with formation of a neocondyle with fibrocartilaginous cap.

creating root divergence at a tooth-bearing corticotomy site. As mentioned, orthodontic traction and occlusion is considered to be one of the mechanical vectors that is under the control of the clinician during active distraction. Dynamic changes are going on in the young patient with regards to the dentition and occlusion with transitioning from a primary to permanent dentition, vertical growth of the alveolar process and jaws, and possibly premature occlusal contacts that develop during distraction resulting in functional shifts that may affect the vector movement. Therefore, active DO orthodontic intervention may include occlusal equilibration, the use of neutral occlusal bit plates, and fabrication of specific orthodontic stabilization devices to control mediolateral interarch relationships during distraction, especially with unilateral distraction vectors (Figure 63-15). For unilateral mandibular distraction, two concerns are control of developing laterognathism and contralateral cross-bite occlusion.



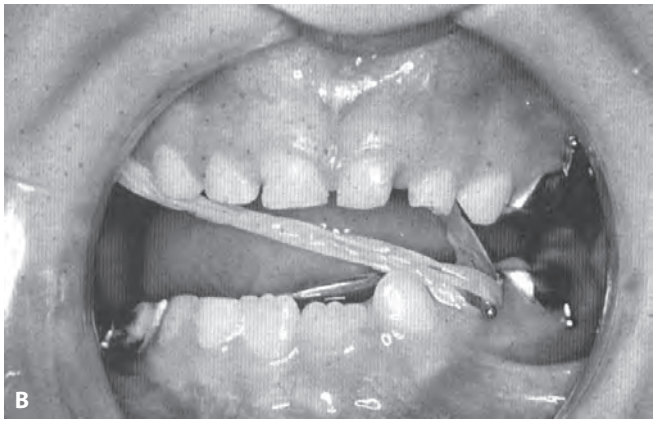


FIGURE 63-15. **A,** Orthodontic stabilization devices used for DO. **B,** Cross-arch elastics to control laterognathism and unilateral cross-bite elastics.

Laterognathism is the development of an asymmetrical habitual occlusion during DO that results in functional shifts toward the unaffected side, or in the direction of the distraction vector, that may limit the magnitude of distraction. Although functional appliances may be used, typically cross-arch elastics are used to control laterognathism, but these forces directly oppose the forces of DO, and therefore, overcorrection may be indicated. For contralateral cross-bite control, transpalatal and lingual arches with both intermaxillary cross-arch elastics and unilateral cross-bite elastics are used. Orthodontics with vertical elastics can also be used to decrease or eliminate developing open bites during distraction, by a combination of molding of the bony regenerate as well as tooth extrusion. Also, for vertical vectors in the ramus region to lengthen the ramus (e.g., hemifacial microsomia), it may be necessary to maintain the ipsilateral posterior open bite after mandibular plane leveling with a bite plate and sequential plate reduction to allow unimpeded maxillary growth. If active orthodontic intervention is used during distraction, then minimal postdistraction orthodontics may be required. Post-DO orthodontics must consider whether orthodontic finishing can be completed in the nongrowing patient or whether a planned malocclusion was performed with the expectation of future growth (e.g., a class III occlusion with anterior occlusal cross-bite expecting future maxillary growth). Again, functional appliances (e.g., bionator therapy) may be used for up to 6 months after DO, depending on patient age, to mold the regenerate during ossification. Finally, after bony consolidation, teeth may be moved orthodontically into the area of the distraction gap.

MANDIBULAR DISTRACTION

Mandibular anteroposterior hypoplasia constitutes the majority of dentoskeletal deformities that are treated with orthodontics and orthognathic surgery, and DO should not be considered in cases in which standard orthognathic surgical techniques can be employed (sagittal split osteotomy [SSO], VRO). When considering the indications for DO, the magnitude of the skeletal discrepancy must be severe enough to warrant distraction, and typically, these types of deformities are due to congenital craniofacial abnormalities that may be a component of a syndromic diagnosis, such as hemifacial (or craniofacial) microsomia, Pierre Robin sequence, Treacher Collins, Goldenhar's, or Nager's syndrome, or may also be associated with the myriad signs and symptoms of obstructive sleep apnea. In addition to congenital deformities, acquired mandibular retrognathis may result from condylar trauma or pathology that leads to TMJ ankylosis. In addition, DO can be applied to the mandible to correct transverse mandibular deficiency with mandibular widening via distraction as well as segmental mandibular defects managed with transport distraction osteogenesis. Typically, DO is considered for these severe deformities in which conventional orthognathic options are limited owing to the young age of the patient or the degree of bony movement, which may not allow application of rigid fixation, or in whom plates and screws may interfere with future growth, and the magnitude of soft tissue (skin, muscle, nerve) stretch that may not respond well to acute lengthening, and before the introduction of rigid fixation devices, may have contributed to relapse of the bony segments. As mentioned previously, a wide variety of distraction devices are available for mandibular DO procedures, and these include external, internal, semiburied, toothborne hybrid devices, with monofocal, bifocal, multifocal, and transport distraction vectors. In cases of severe horizontal and vertical ramus deficiency, either a multifocal multivector device or a combined distraction device may be used to accomplish both vertical and horizontal distraction vectors simultaneously (Figure 63-16). In cases of unilateral vertical ramus hypoplasia, for example with hemifacial microsomia, a stable univector distraction device can be used to level the mandibular occlusal plane and allow unimpeded future growth of the maxilla using a maxillary splint that is sequentially reduced during vertical maxillary growth (Figure 63-17). An alternative option for a patient with a unilateral vertical ramus deficiency (e.g., hemifacial microsomia) is to consider performing bimaxillary osteotomies, application of intermaxillary fixation (IMF), and the use of only a mandibular distraction device that will result in distraction of both the mandible and the maxilla owing to the use of IMF (Figure 63-18). Segmental defects of the mandible can be treated with the use of transport DO as mentioned previously, and ankylosis of the TMJ can also managed with the use of a transport segment of bone that is distracted toward the glenoid fossa in order to create a new condylar head with fibrocartilage. In a manner similar to surgically assisted maxillary expansion, mandibular expansion with distraction may be accomplished with a midline mandibular osteotomy and the use of an external or internal device (Figure 63-19).

A major indication for mandibular distraction has become popular as an alternative to tracheostomy or tongue-lip adhesion[24] in the neonate born with severe mandibular anteroposterior deficiency and airway obstruction with birth asphyxia due to isolated obstruction at the base of the tongue. Most often, this technique is considered for the Pierre-Robin (1923)[25] sequence patient with the triad of mandibular anteroposterior hypoplasia, a U-shaped cleft palate, glossoptosis due to an in utero problem of mandibular hypoplasia with lack of tongue descent, and failure of closure of the palatal shelves in the midline. The airway obstruction is present at birth in 70% of infants and results in a mortality rate of 2% to 26% even in experienced centers. A variety of methods have been used to address the airway, including the EXIT (ex utero intrapartum treatment) procedure in which immediate intubation is performed upon a staged delivery of the infant with the head delivered first for intubation. In general, for mild airway obstruction in the neonatal period, positional head therapy (prone, lateral, head extension positions) is usually effective to open the posterior pharynx, relieve the tongue obstruction, and maintain arterial oxygen saturation. For moderate airway obstruction, nasal stents can be placed

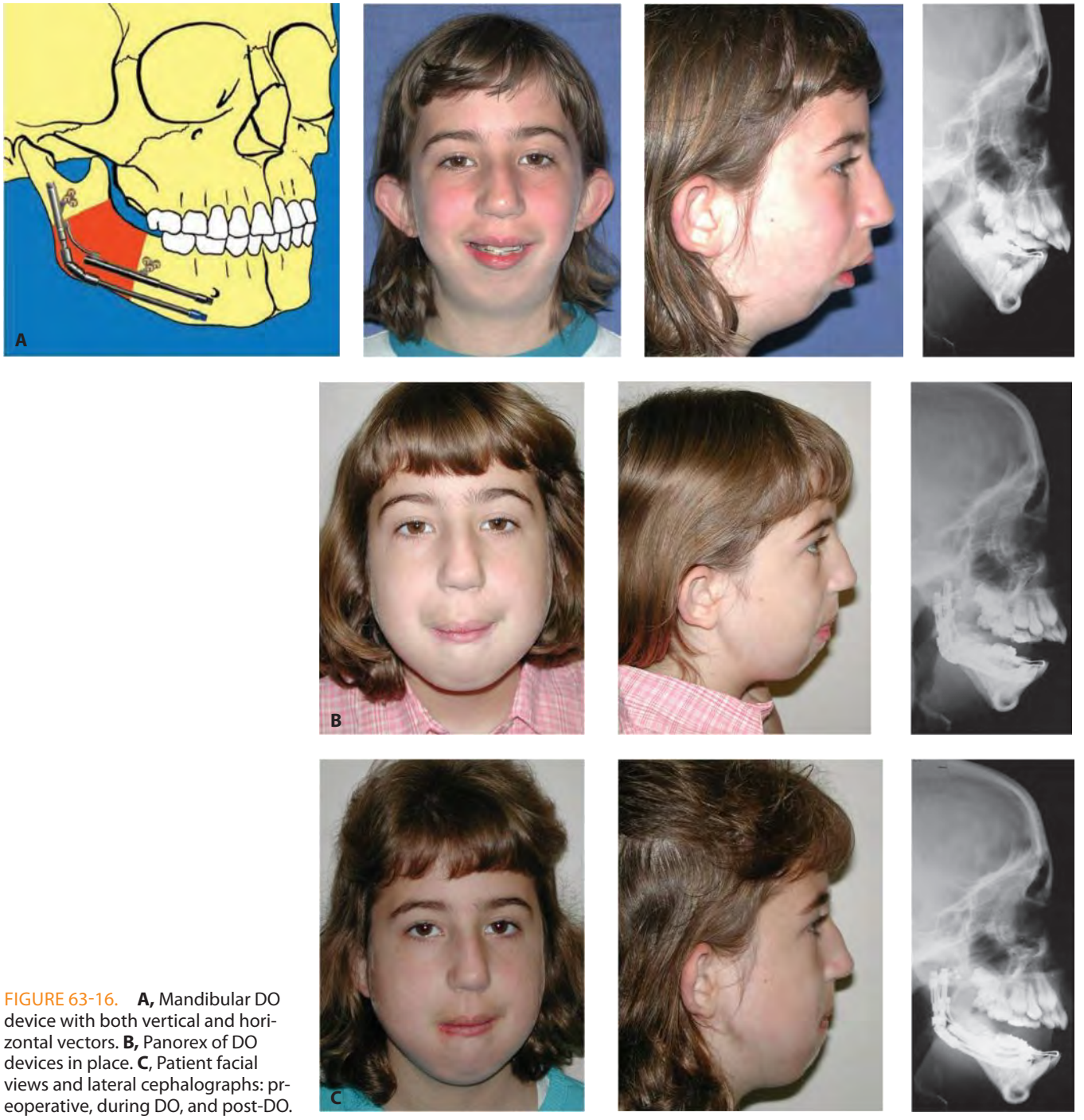


FIGURE 63-16. **A,** Mandibular DO device with both vertical and horizontal vectors. **B,** Panorex of DO devices in place. **C**, Patient facial views and lateral cephalographs: preoperative, during DO, and post-DO.

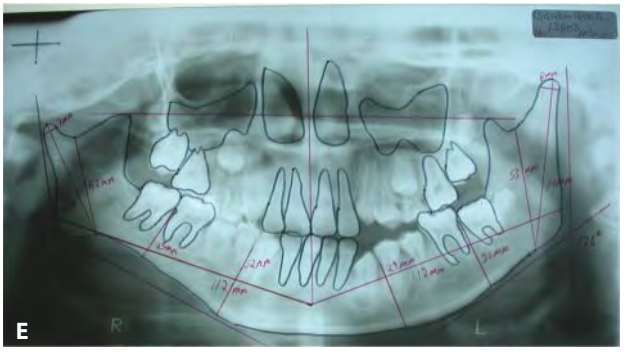
using a 3.0 endotracheal tube or other nasal airway device, such as a nasal trumpet. For severe obstruction, with difficulty maintaining inspired oxygen concentrations above 90% most of the time, either an anterior glossopexy (tongue-lip adhesion) procedure can be used with a reported success rate less than 50%; a tracheostomy, which does not address the cause of the problem, can be performed with significant long-term morbidity including tracheostomy care, psychosocial issues, speech developmental delay, skin scarring, tracheal stenosis, and an average time of 18 to 36 months until removal; or mandibular DO can be performed in order to correct the etiology of the problem and allow future mandibular growth, without significant morbidity. Among the few problems reported with neonatal mandibular distraction, alteration in the morphology of the condyle with reported cases of TMJ ankylosis has occurred,[26] although other reports show no ankylosis and an adaptation of the condyle to the distraction process.[27] The DO procedure will serve to advance the mandible, elevate the hyoid bone, and relieve the obstruction at the tongue base; one of the major benefits of



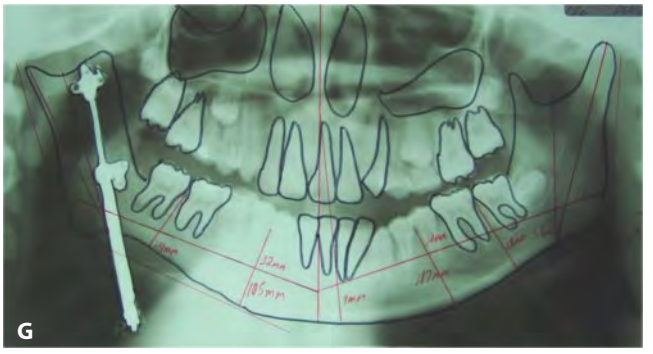


















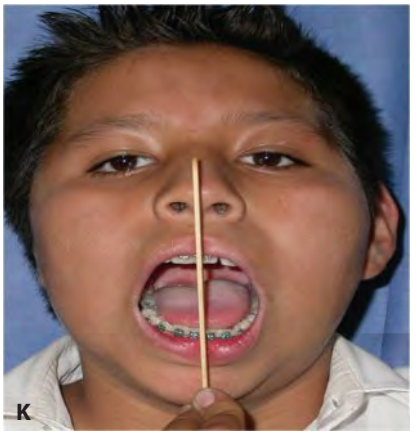


FIGURE 63-17. Mandibular DO for right hemifacial microsomia using a vertical vector device with creation of a posterior open bite on the affected side. **A–E,** Preoperative. **F,** Device activation during surgery. **G** and **H,** Post-DO films. **I** and **J,** Post-DO clinical with leveled mandibular occlusal plane, bite splint to allow maxillary growth, and **K,** excellent midline mouth opening.

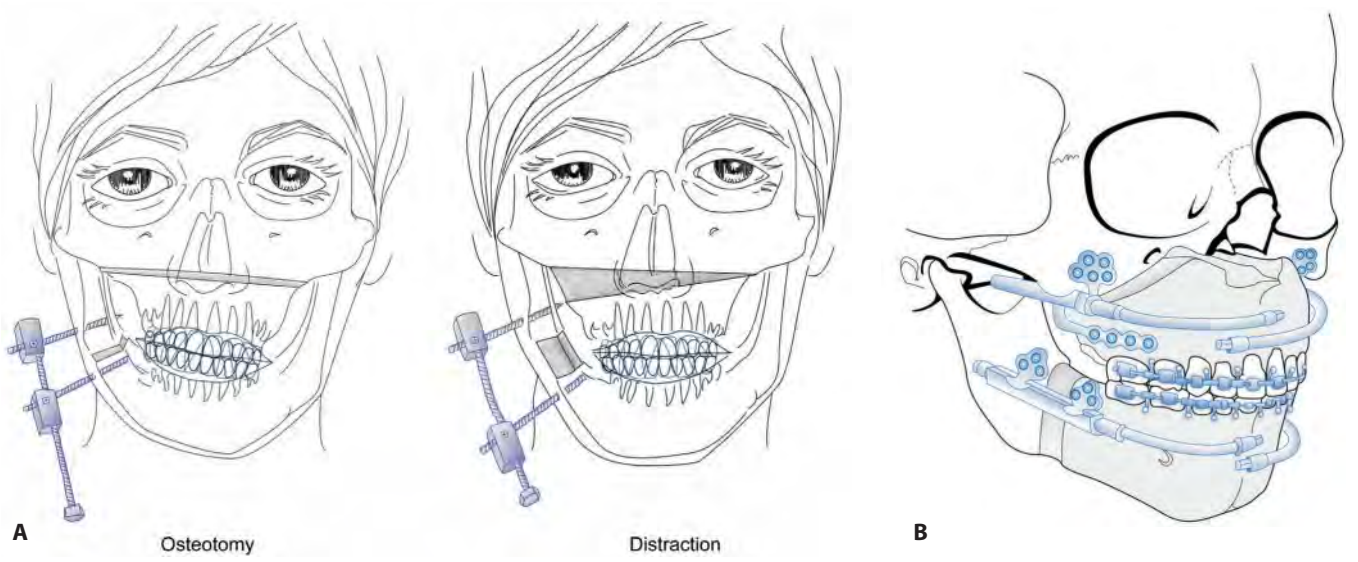


FIGURE 63-18. **A,** Bimaxillary DO for hemifacial microsomia accomplished with unilateral mandibular corticotomy, Le Fort osteotomy, and placement of a mandibular DO device and use of intermaxillary fixation (IMF) to distract the mandible and maxilla simultaneously. **B,** Alternative technique for bimaxillary DO, requiring coordinated synchronous activation of each device.

DO is the ability to titrate the degree of mandibular forward movement and continue until the airway obstruction has been relieved and confirmed by laryngoscopy. Owing to expected relapse in the young patient, overcorrection is advocated, and creation of a reverse overjet class III jaw relationship is preferable at the time of DO completion. The presence of other abnormalities of the upper airway must be ruled out with direct laryngoscopy, because laryngomalacia or tracheomalacia would be contraindications to DO, and tracheostomy is warranted for severe airway obstruction. It is important to consider the distraction device options carefully for the very small mandible, and consideration should be given toward computer planning with SLA (stereolithography) and customized semiburied device fabrication when possible, in order to achieve the precise final position of the distal segment of the mandible (Figure 63-20). Conventional options for neonatal distraction include either internal plate and screw devices or external pin devices (Figure 63-21). Typically, the distraction rates in the neonate are accelerated over the typical adult rate of 1.0 mm/day to a 4.0-mm/day distraction rate in divided increments, and, in this manner, 2.0 cm of distraction may be accomplished within a period of 7 to 10 days while the patient is in the intensive care unit of the hospital. After a consolidation period of approximately twice the distraction period (14–30 days), the devices can be removed; resorbable devices are available and clinical research continues to determine efficacy.

MAXILLARY DISTRACTION

Maxillary DO has been performed for years by oral and maxillofacial surgeons and orthodontists using surgically assisted maxillary expansion techniques to address transverse maxillary deficiency. Similar considerations are made for maxillary DO as with mandibular DO in that DO should not be used in cases in which conventional orthognathic surgical procedures can be used. Maxillary DO at the Le Fort I level is typically reserved for cleft lip and palate patients, who have had numerous previous surgical procedures with severe maxillary anteroposterior hypoplasia, palatal scarring

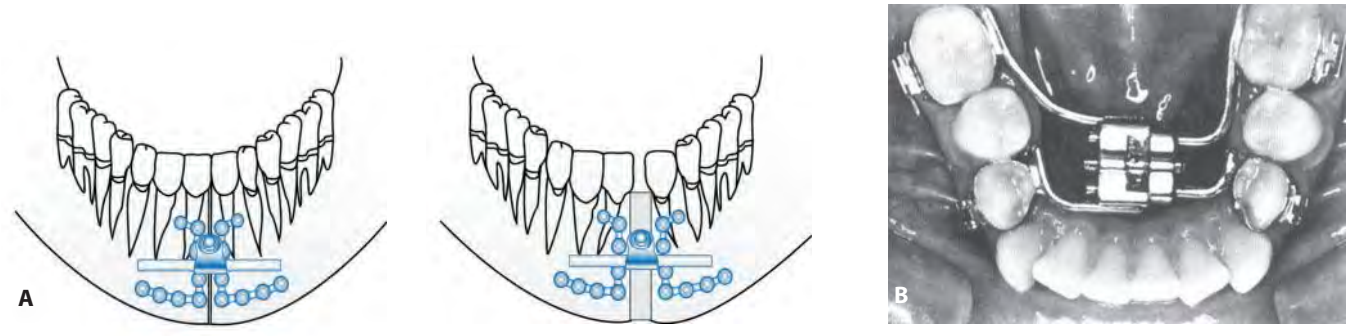


FIGURE 63-19. **A**, Mandibular widening by midline corticotomy and DO device placement. **B,** Midline osteotomy with a mandibular modified hyrax appliance.

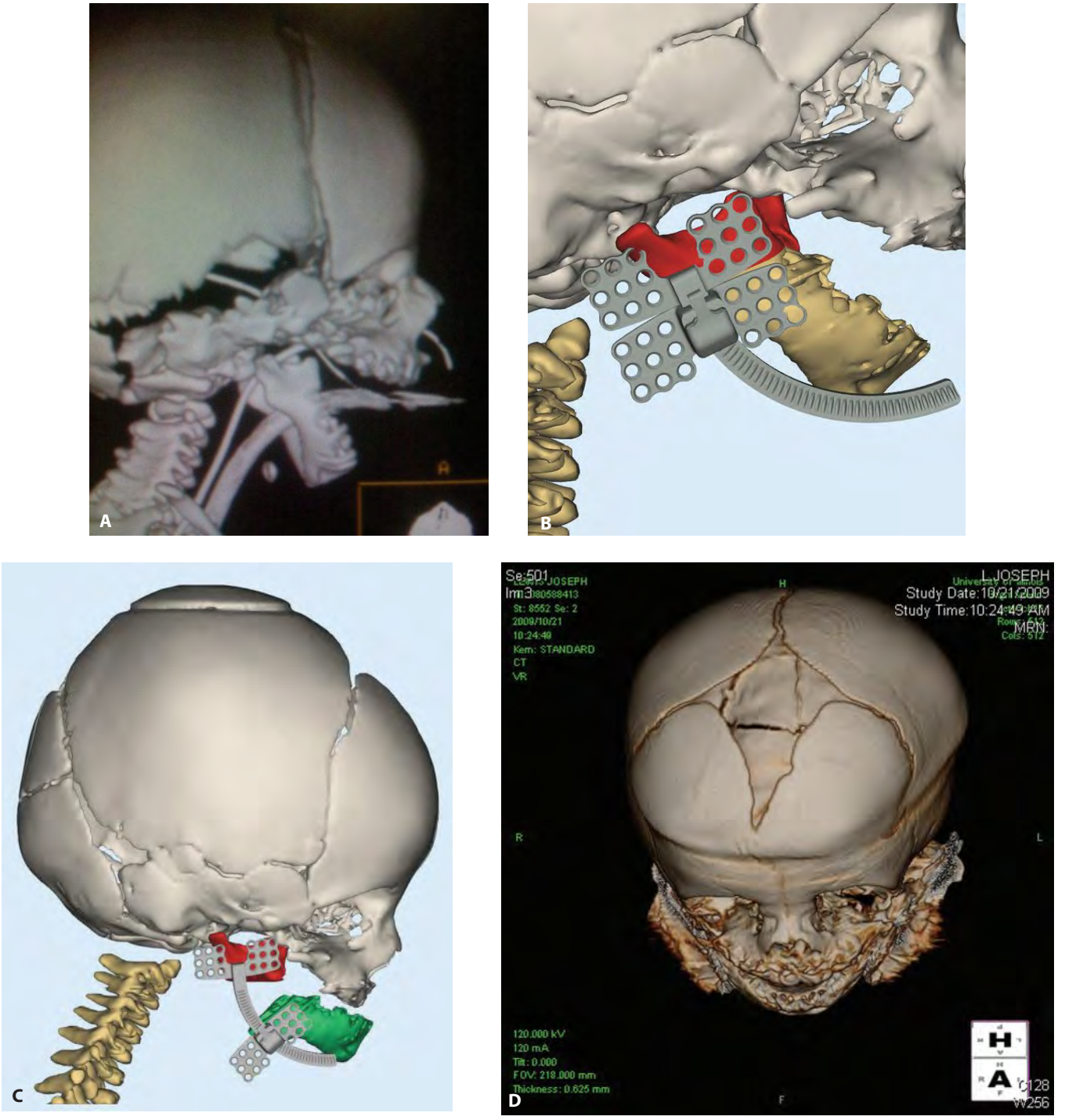
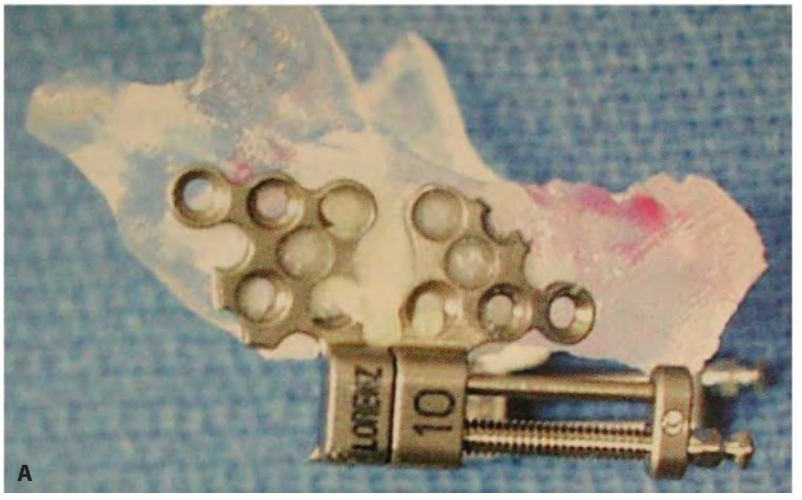


FIGURE 63-20. Owing to unique mandibular deformity in this case of Pierre-Robin sequence **(A),** a customized curvilinear DO device **(B)** was manufactured to achieve the precise advancement and rotation of the distal mandible with DO **(C),** and DO continued until the airway was open and a reverse overjet was obtained **(D).**

(possibly with a pharyngeal flap), and compromised blood supply to the maxillary hard and soft tissues. These factors may result in difficulties with standard Le Fort surgery with problems with mobilization of the maxilla into the final forward position, an increased relapse potential compared with conventional orthognathic surgery even with rigid fixation, and worsening of speech and swallowing abnormalities or VPI, especially in the presence of a pharyngeal flap. Application of maxillary DO at the Le Fort II level may be used for unusual midfacial deformities of the nasomaxillary complex such as Binder's syndrome; use at the Le Fort III level is generally for cases of syndromic or nonsyndromic craniosynostosis, such as Apert's, Crouzon's, and Pfeiffer's syndromes. In most cases of maxillary DO performed at the Le Fort I











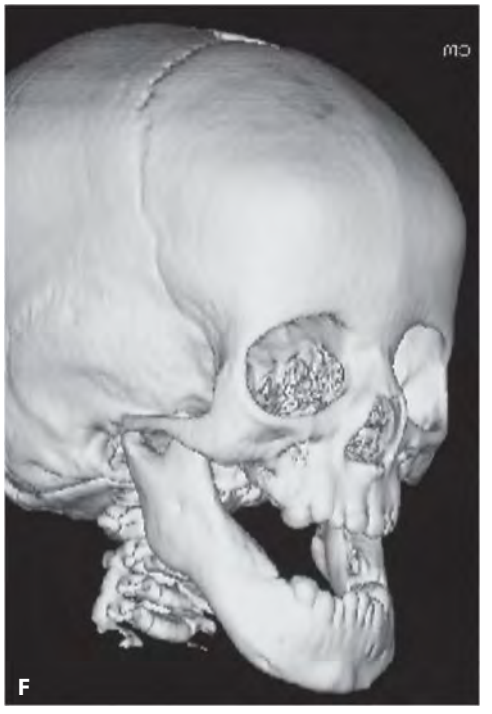


FIGURE 63-21. Neonatal DO for airway management may include buried DO devices **(A),** external devices stabilized with cortical pins **(B),** or transfacial pins with external devices **(C** and **D). E–F,** Pre- and post-DO computed tomography (CT) scans with mandibular advancement and airway improvement. *(Continued)*

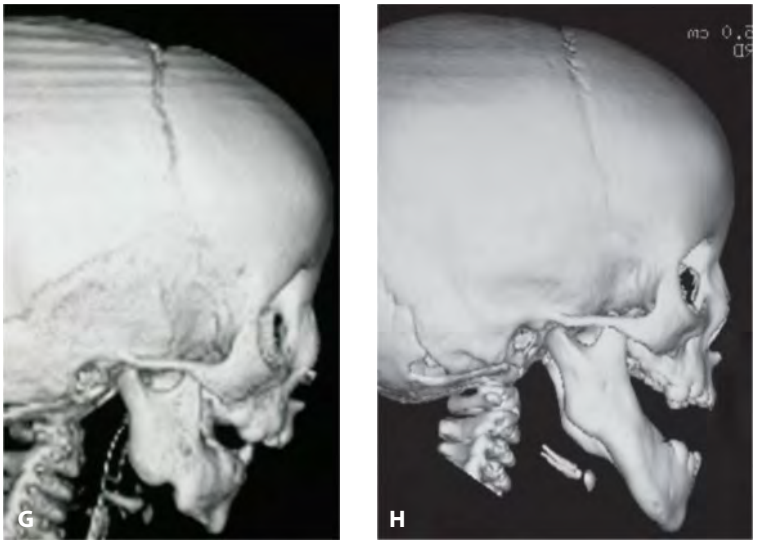


FIGURE 63-21. *(Continued)* **G–H,** Pre- and post-DO computed tomography (CT) scans with mandibular advancement and airway improvement.

level, semiburied intraoral devices are adequate (Figure 63-22), but for cases of more severe deformities in cleft patients in whom additional vector control is required, a RED may be used.

In a fashion similar to that used for mandibular transport DO, maxillary segmental distraction may be used, especially in the area of an alveolar cleft defect for closure without the need for bone graft procedures, using specialized distraction devices (Figure 63-23).

Maxillary distraction may also be used at the Le Fort III level for craniosynostosis cases, although the usual unidirectional vector of distraction fails to address the complex three-dimensional nature of the skull and cranial base abnormalities (Figure 63-24), and perhaps these deformities benefit from a one-stage surgical approach with cranial recontouring and application of small or resorbable fixation, without the need for DO device removal at a second surgical procedure, despite the potential DO advantages of maintenance of blood

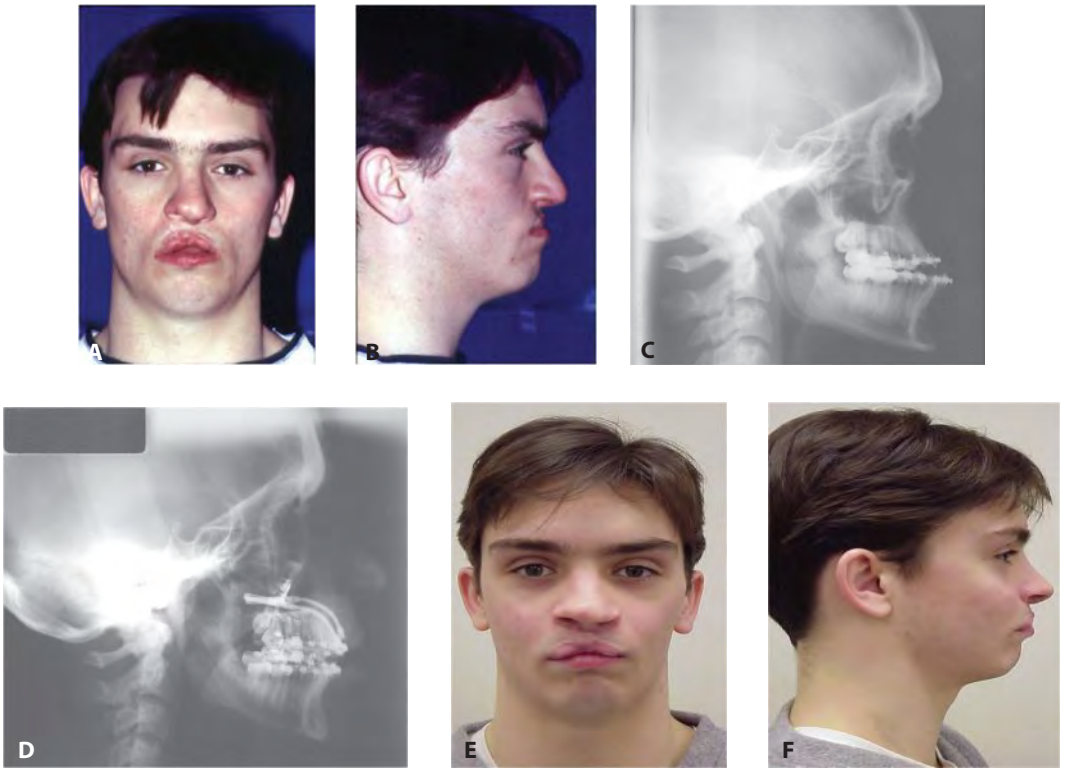


FIGURE 63-22. **A** and **B,** Maxillary DO in a cleft lip and palate patient using buried transmucosal DO devices. **C** and **D**, Pre- and postoperative lateral cephalographs. **E** and **F,** Post-DO facial and profile views.

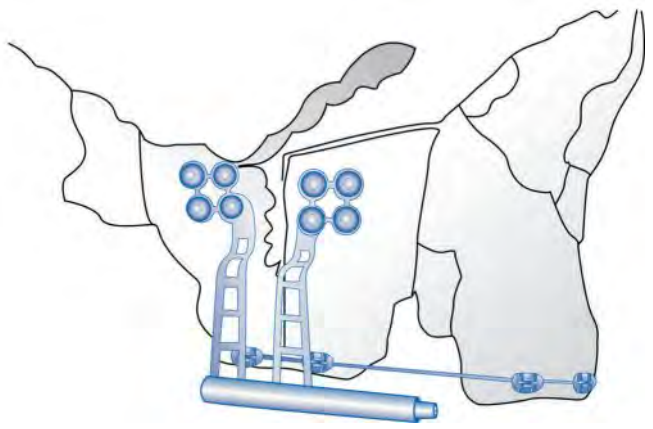


FIGURE 63-23. DO device used for alveolar segment DO in a cleft alveolus to avoid bone grafting.

supply, less intraoperative blood loss, and lack of soft tissue limitation to cranial remodeling. Each patient must be evaluated independently to determine the most appropriate surgical procedure to achieve the desired result with the least patient morbidity.

ALVEOLAR RIDGE DISTRACTION

The subject of alveolar ridge distraction is covered in Chapters 11 and 12, but it deserves mention in this section on DO, because the indications and expected advantages of the technique are consistent with the other applications of DO to the craniomaxillofacial skeleton. The technique also represents an alternative form of transport DO that has been discussed previously, with the transport segment pedicled on the lingual periosteum and soft tissues for maintenance of blood supply, and a vertical vector of the planned bony distraction.

In areas of alveolar ridge deficiency, alveolar ridge DO offers an alternative option to autogenous or allogeneic or alloplastic bone grafting or guided bone regeneration, with the principal benefit being the soft tissue expansion of the attached and unattached gingiva during the process of distraction, in order to avoid a soft tissue dehiscence that frequently occurs with vertical alveolar ridge augmentation (Figure 63-25). Alevolar ridge DO is by no means a panacea for reconstruction of the alveolar ridge, because there may be several complications including poor vector control with an inadvertent lingual orientation or deviation of the transport segment due to the soft tissue pedicle, as well as unpredictable osseous healing in the distraction gap[28] (Figure 63-26), that may require bone grafting at the time of endosseous implant placement, thereby negating the expected benefit of avoidance of bone grafting.

THE FUTURE OF MAXILLOFACIAL DO

In the maxillofacial region, the application of DO maintains specific indications including severe skeletal deformities of the maxilla and mandible that are not amenable to correction by conventional orthodontic and orthognathic surgical treatment methods. Because one of the major limitations or disadvantages of the DO process is the length of the consolidation period, basic and clinical research will continue to focus on methods to shorten the consolidation period by accelerating the process of osseous healing in the distraction gap using both locally applied stem cells, growth factors, and cytokines, as well as the application of external sources of bone stimulation. Development of customized devices specifically designed with an exact vector for the individual patient is an option using three-dimensional computer treatment

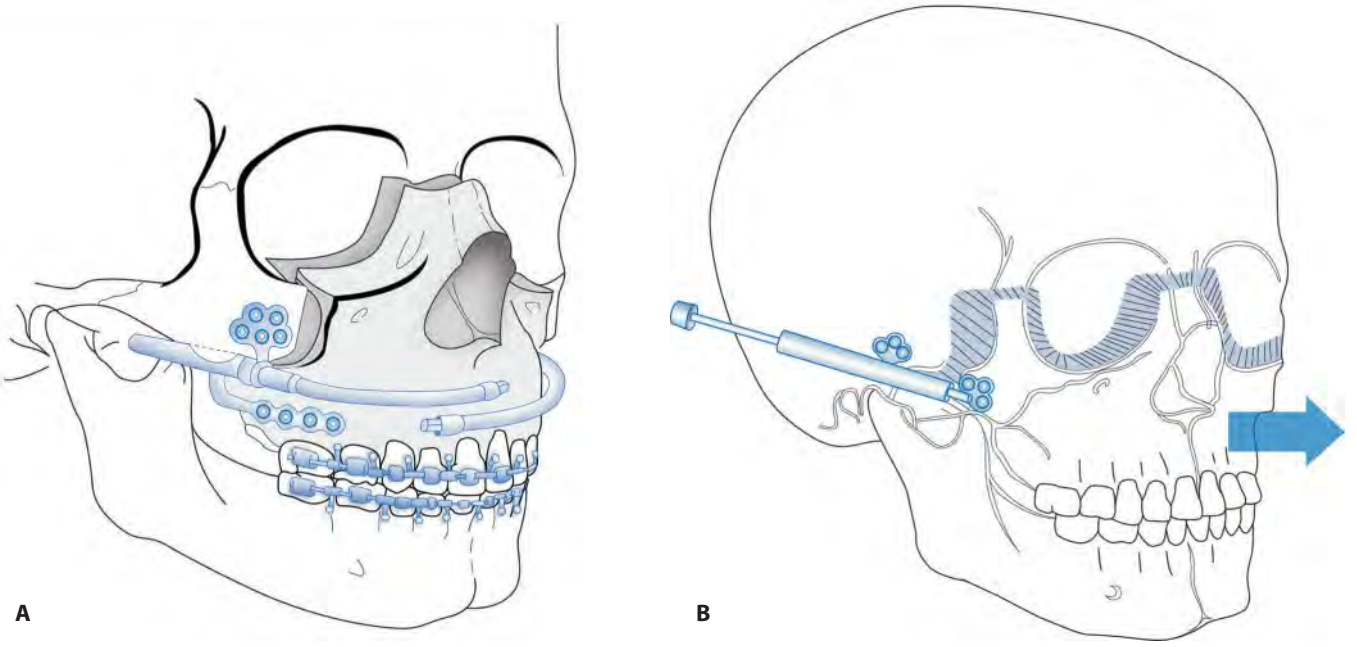


FIGURE 63-24. DO at the Le Fort II **(A)** and Le Fort II **(B)** levels with semiburied devices.

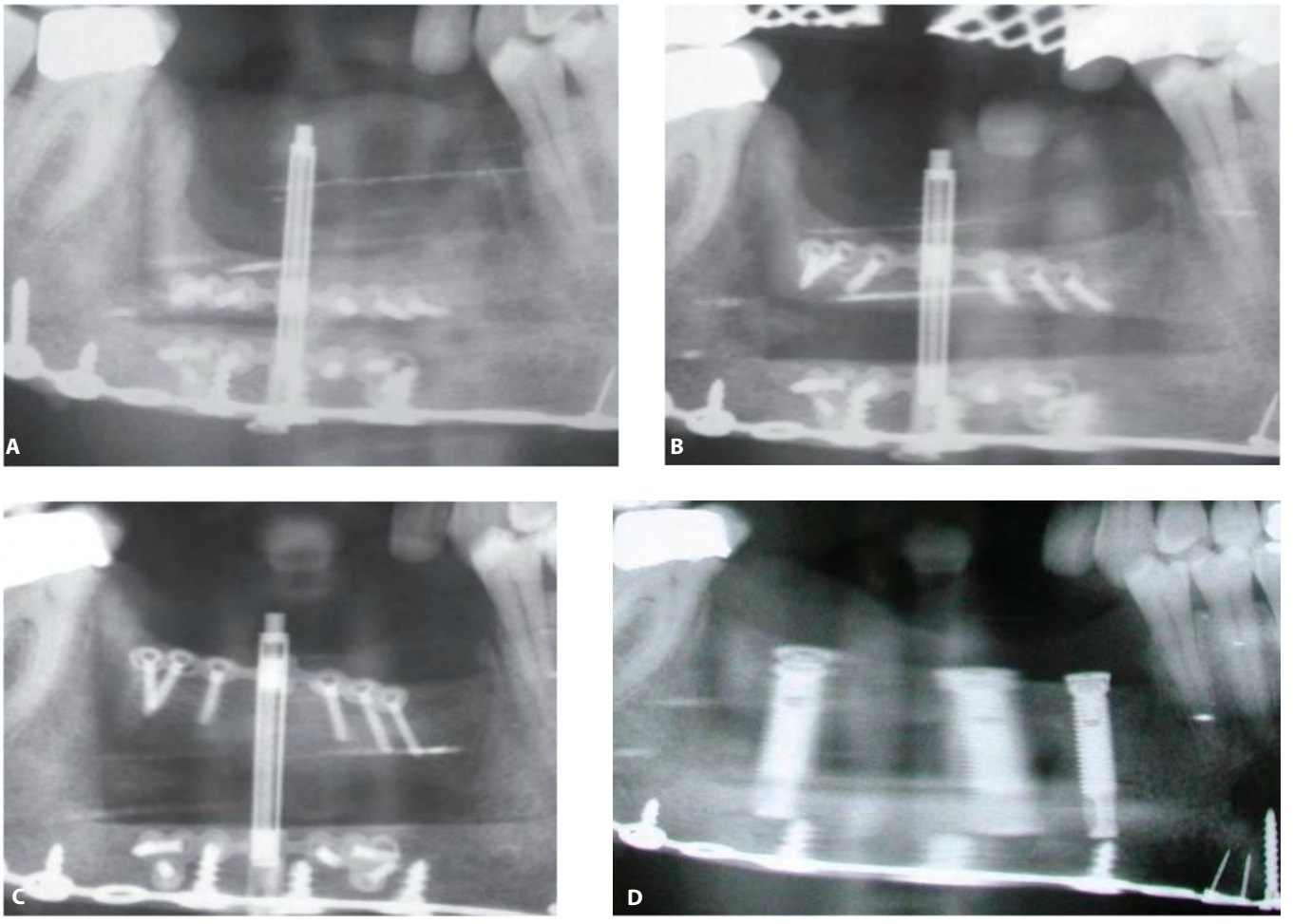


FIGURE 63-25. **A–D.** Alveolar DO with endosseous implant placement.

planning, and further developments may allow cost containment in the future. The development of automatic devices with internal preprogrammed daily incremental device activation will continue, as well as the refinement of resorbable materials that can be used for distraction, especially in the pediatric age group, that will provide sufficient rigidity to allow bony movement despite significant soft tissue restriction and then undergo spontaneous resorption at the appropriate time, in order to avoid the morbidity of an additional surgical procedure for device removal.

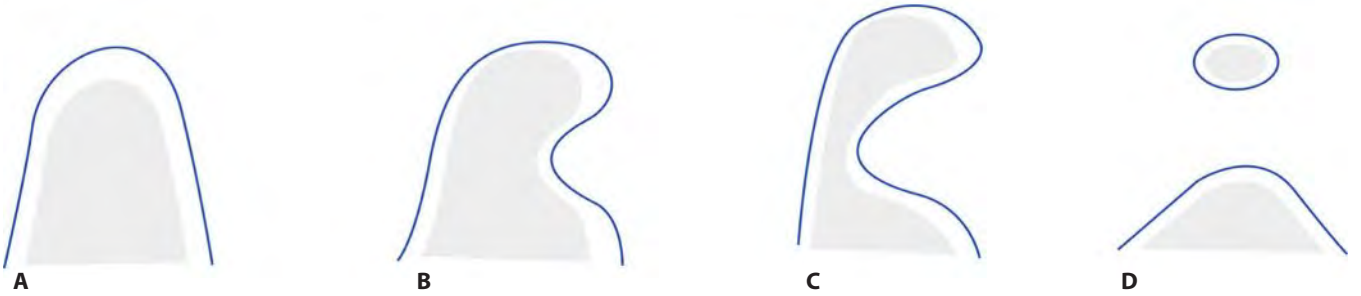


FIGURE 63-26. Possible final outcomes of alveolar DO. **A** shows a nearly normal alveolar ridge morphology. **B** shows a minor buccal concavity defect. **C** shows a major buccal defect, but with lingual/palatal bony continuity. **D** shows a complete separation of the transport segment from the native bone. B and C are most common.

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CHAPTER

Surgical and Nonsurgical Management of Obstructive Sleep Apnea

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Sleep and dreaming have been sources of mystery and fascination since biblical times. Sleep consists of inevitably recurring episodes of readily reversible relative disengagement from sensory and motor interaction with the environment.[1] The function of sleep remains a mystery, and only in recent years has there been research into specific symptom complexes and causes of sleep disorders. In 1979, the Association of Sleep Disorders Center and the Association for the Psychophysiological Study of Sleep published the first classification of sleep and arousal disorders.[2]

Modern sleep research became possible in 1924 when Hans Berger,[3] a German psychiatrist, described the recording of human electroencephalography. Loomis and colleagues in 1935[4,5] published a quantitative description of the four levels of sleep based on electroencephalogram (EEG) characteristics. The historic discovery of a cyclic phase of sleep characterized by rapid conjugate eye movements was made by Aserinsky and Kleitman in 1953.[6] Subsequent studies confirmed this to be a very active phase of sleep that correlated closely with dreaming.[7]

NORMAL SLEEP STAGES

Normal sleep architecture includes both quiet sleep (non–rapid eye movement [non-REM] sleep) and active sleep (rapid eye movement [REM] sleep). Non-REM sleep consists of four stages that are based largely on the original criteria of Loomis and colleagues.[4,5] Stage 2 predominates and accounts for 45% to 50% of total sleep time. The four stages of non-REM sleep represent progressively deeper sleep marked by the increasing appearance of high-amplitude slow waves in stages 3 and 4, which are collectively known as *delta sleep*. Non-REM sleep is characterized by a general slowing of all levels of activity. Progression through all four stages of non-REM sleep usually occurs rapidly after sleep onset. REM sleep occurs after non-REM sleep has been established, and the first REM period normally occurs after 70 to 90 minutes of non-REM sleep. The average duration of a period of REM sleep is approximately 20 minutes. The initial REM period of the night is usually very brief, but subsequent REM periods become longer. During an average night of REM/non-REM cycle progression, four to six REM periods normally occur at intervals of 60 to 90 minutes. REM sleep occupies about 20% to 25% of total sleep time in a healthy young adult. REM sleep EEG patterns look very similar to those seen during the wakeful state. Generalized skeletal muscle atonia (except for the ocular muscles) and absence of reflexive activity are other features unique to REM sleep. Marked physiologic changes also occur during REM sleep. Temperature, blood flow, and oxygen use in the brain are increased. Heart rate, blood pressure, and respiration show dramatic fluctuations and increase in average rate.

During sleep, the control of respiration is influenced by two systems: the metabolic control system and the behavioral control system.[8] The influences of hypoxia and hypercarbia on ventilation are the predominant components of the metabolic control system of respiration. This system predominantly controls respiration during non-REM sleep. The behavioral control system governs respiration during voluntary activities, such as swallowing or speaking, and may

suppress the ventilatory response to metabolic stimuli. During REM sleep, the effects of hypoxia and hypercarbia on ventilation are much less than during non-REM sleep, and the behavioral control system may predominate. With a blunted response to hypoxia and hypercarbia, irregular respirations, and decreased skeletal muscle tone of the upper airway muscles during REM sleep, an episode of partial or complete airway obstruction with apnea or hypopnea may occur.

SLEEP APNEA SYNDROME

The sleep apnea syndrome is a disorder characterized by abnormal breathing in sleep and sleep fragmentation. At least 30 episodes of apnea occur during 7 hours of nocturnal sleep in these patients. *Apnea* is defined as the cessation of airflow from the nostrils and mouth for at least 10 seconds. These apneic episodes can result in hypoxemia, hypercarbia, systemic and pulmonary hypertension, polycythemia, cor pulmonale, bradycardia, and cardiac dysrhythmias. Sudden death has occurred in patients with sleep apnea. Throughout the night, the alternating episodes of apnea and arousal from sleep may occur as frequently as 400 to 600 times, with each typical apnea episode lasting 15 to 60 seconds. These episodes can amount to as much as 50% of a night's sleep. The frequent disruption results in symptoms similar to those of sleep deprivation. These include excessive daytime sleepiness, fatigue, depression, personality changes, and impotence. These dysfunctional symptoms are common primary complaints and are often the reason people seek treatment.

Epidemiologic data suggest that sleep apnea syndrome may be quite common, particularly in its milder forms. In fact, obstructive sleep apnea (OSA) is the second most common sleep disorder, insomnia being the most common. A 1993 Sleep Commission Report estimated that 20 million Americans have sleep apnea, with the majority being undiagnosed and untreated.[9] The exact prevalence is unknown, but sleep apnea syndrome may affect up to 2% to 3% of adult males.[8] In certain populations, the prevalence may be as high at 10%. Most patients are diagnosed after age 40, but sleep apnea can occur at any age. There is a strong male predilection, with men outnumbering women by up to 8:1 until menopause. This implies a hormonal influence. The cost for diagnosis and treatment of this sleep disorder accounts for over $50 million (U.S.) in hospital bills each year. Overall, sleep disorders and sleepiness cost the U.S. economy a minimum of $15.9 billion in direct costs each year.[10]

Classification

Central sleep apnea, OSA, and mixed sleep apnea are the variations of apnea that occur in the syndrome. In central sleep apnea, respiratory muscle activity ceases simultaneously with airflow at the mouth and nostrils.[11] This disorder is found in patients with central nervous system (CNS) insufficiency that affects the outflow of neural output from the respiratory center to the diaphragm and other muscles of respiration. CNS disorders associated with central sleep apnea include brainstem neoplasms, brainstem infarctions, bulbar encephalitis, bulbar poliomyelitis, spinal surgery, cervical cordotomy, and primary idiopathic hypoventilation.

Patients with central sleep apnea have been treated with some success by using respiratory-stimulating drugs such as theophylline, progesterone, and acetazolamide. In severe central apnea, modalities of treatment have included phrenic nerve pacemaker implantation to ensure regular respiration during sleep and nocturnal mechanical ventilation with a negative-pressure ventilator for more severe cases. There are no simple and convenient methods of treatment for mild central apnea.

The most common type of sleep apnea by far is obstructive. This is characterized by sleep-induced obstruction of the upper airway that results in cessation of airflow with preservation of respiratory effort, respiratory center drive, and diaphragmatic contraction.[11]

Mixed sleep apnea is a combination of central and obstructive apnea. This pattern begins with an episode of central apnea with no airflow detectable at the mouth and nostrils and no respiratory muscle activity. The pattern ends with an episode of obstructive apnea with cessation of airflow only at the mouth and nostrils.[11]

Differential Diagnosis

Profound hypersomnolence is a characteristic feature of both sleep apnea and narcolepsy; hence, they are often confused. However, unlike sleep apnea, narcolepsy affects both sexes equally, with most patients experiencing the onset of symptoms around or shortly before puberty.[12] The first symptom to appear with narcolepsy is usually excessive daytime somnolence (EDS). The sleep attacks can range from mild to severe and are characterized by the sudden onset of overwhelming sleepiness that lasts 30 seconds to 20 minutes. After brief naps, the narcoleptic usually feels refreshed and relatively free from disturbing symptoms for up to 2 hours. Serious accidents, marital discord, and the inability to hold jobs frequently result from these sleep attacks. Another feature of narcolepsy is the abrupt loss of muscle control (cataplexy). Attacks can be particularly disabling because they are characteristically precipitated by emotional experiences such as laughter, anger, or excitement. Additional associated symptoms of narcolepsy include sleep paralysis and hypnagogic hallucinations. Sleep paralysis is the skeletal muscle atonia of REM sleep persisting into the awake state. Hypnagogic hallucinations are REM sleep imagery occurring while falling asleep. Patients are sometimes misdiagnosed as schizophrenic if hypnagogic hallucinations are prominent.

Diagnosis of narcolepsy is made by documenting sleep-onset REM periods during a nocturnal polysomnography.[12] In normal sleep, REM sleep is usually not seen until about 70 to 90 minutes into sleep. The clinical features of narcolepsy probably represent abnormal manifestations of REM sleep.

Treatment modalities for narcolepsy include behavioral therapies, CNS stimulants, tricyclic antidepressants, or monoamine oxidase inhibitors (only in resistant cases) and L-tryptophan.[13]

Other disorders that may be confused with sleep apnea syndrome include sleep-related abnormal swallowing syndrome, gastroesophageal reflux, depression, alcohol or drug dependence, and sleep-related nocturnal myoclonus.

History of OSA Syndrome

OSA has a remarkably short history, considering the incidence and disabling symptoms of the syndrome. Burwell and coworkers published the first description of the syndrome in 1956.[14] Their report compared an obese, somnolent, polycythemic patient with the sleepy red-faced boy, Joe, in the Charles Dickens novel *The Posthumous Papers of the Pickwick Club* (1837). However, Burwell and coworkers did not link their patient's excessive daytime sleepiness to nocturnal sleep fragmentation. In 1966, Gastaut and associates[15] were the first investigators to demonstrate repeated apneas in pickwickian patients during sleep. They correctly attributed the excessive daytime somnolence in these patients to nocturnal sleep fragmentation caused by repeated apneas.

The misdiagnosis of narcolepsy in patients with sleep apnea and the general skepticism of EDS as a valid clinical sign are the two main reasons sleep apnea syndrome was overlooked for so long.

Clinical Manifestations

Sleep apnea patients present with a variety of symptoms and clinical manifestations. Patients with OSA most often complain of EDS. The patients may experience serious social, economic, and emotional problems from the EDS associated with this disorder. The uncontrollable desire to sleep may predispose the patients to occupational or automobile accidents.

Almost all patients or their bed partners give a history of heavy, loud snoring that has usually been present for several years before the EDS was noted. The snoring is produced from the passage of air through the oropharynx causing vibrations of the soft palate. Typically, the snoring is interrupted periodically by apneic episodes that last 30 to 90 seconds. Bed partners usually describe an episode in which the snoring stops and the patient seems to stop breathing for a period of time. A loud snort followed by a hyperventilation usually signals an end to the apneic episode.

Other common presenting complaints are morning headaches and nausea that result from the hypercarbia that develops with the hypoventilatory episodes. Depression, personality changes, and intellectual deterioration may also develop.

The systemic hypertension that is a common finding in OSA may be related to the catecholamine release triggered by the systemic hypoxemia. In more advanced severe cases, pulmonary hypertension, polycythemia, and cor pulmonale may develop and become life-threatening. However, most patients do not manifest these disturbances because their ventilation during wakeful periods is sufficient to prevent these complications of chronic hypoxia.

A prominent sinus dysrhythmia is commonly associated with the apneic episodes. The extent of bradycardia is directly proportional to the severity of the oxygen desaturation. The greatest degree of cardiac slowing occurs in obstructive apneas in which a Müller maneuver is performed. Increased vagal efferent tone mediates the bradycardia.

The development of severe and life-threatening medical complications from the apneic events clearly depends on the frequency, duration, and degree of hypoxemia and associated hypertensive response.

Physical Findings

A major feature of OSA is obesity. The increased body weight correlates with increased frequency of apnea and the severity of hypoxemia. However, the morbidly obese, somnolent, hyperventilating patient with cor pulmonale represents only a small number of sleep apnea patients. Lower body mass index (BMI) patients with OSA often have more abnormal cephalometrics than obese people.[16,17]

Obstruction can occur at a number of points in the airway. Physical examination of these patients may reveal hypertrophy of the tonsils or adenoids, retrognathia, micrognathia, macroglossia, deviation of the nasal septum, a thick short neck, or tumors in the nasopharynx or hypopharynx. Both primary and secondary medical conditions are associated with OSA, owing to their effects on the upper airway anatomy. These may include temporomandibular joint disorders, myxedema, goiter, acromegaly, and lymphoma.

Most patients with classic OSA have no identifiable craniofacial anomaly. However, there does appear to be a significant subpopulation of sleep apnea patients with craniofacial anomalies.[18,19] Lowe and colleagues[20] found several alterations in craniofacial form in subjects with OSA that may reduce the dimensions of the upper airway and subsequently impair stability of the upper airway. A sample of 25 adult male patients with moderate to severe OSA showed a posteriorly positioned maxilla and mandible, a steep occlusal plane, overerupted maxillary and mandibular teeth, proclined incisors, a steep mandibular plane, a large gonial angle, increased upper and lower facial heights, a posteriorly placed pharyngeal wall, and an anterior open bite in association with a long tongue.[20] Bacon and coworkers[18] evaluated 32 patients with sleep apnea by cephalometry and demonstrated an anteroposterior shortening of the cranial base, a posterior facial compression with narrowing of the pharyngeal airway, and an increased lower facial height. Rivlin and associates[21] reported on nine OSA patients with posterior displacement of the mandible. The number of apneas correlated with the total posterior displacement.[21]

DIAGNOSIS

Physical Examination

A diagnostic evaluation includes a thorough history and physical examination, fiberoptic endoscopy, radiologic evaluation, and polysomnography. Little additional information can be gained from routine laboratory tests. Except in severe cases, pulmonary function tests, electrocardiogram (ECG), arterial blood gases, and chest radiographs are often normal during wakefulness in sleep apnea patients.

Other diagnostic tests that may aid in evaluating sleep apnea patients include a complete blood count (CBC), serum electrolytes, and thyroid function tests. Secondary polycythemia may be revealed by a CBC, and nocturnal carbon dioxide retention may be reflected by increased bicarbonate levels. Hypothyroidism, a contributing cause of sleep apnea, may be identified from thyroid function studies.

After a complete history is obtained from the patient and his or her bed partner, a complete clinical examination of the mouth, nasal, pharyngeal, and laryngeal areas is performed. The emphasis of the clinical examination should be the identification of anatomic abnormalities that may contribute to or produce obstruction during sleep. The nose is examined for a deviated nasal septum and enlargement of the turbinates. Micrognathia, retrognathia, and macroglossia may be noted in examination of the oral cavity. Occasionally, masses or tumors in the nasopharynx or hypopharynx may be noted. In the pharynx, adenotonsillary hypertrophy, a long soft palate, a large base of the tongue, and excess pharyngeal mucosa are potential causes of obstruction. The larynx is examined for vocal cord webs and paralysis of the vocal cords. OSA patients may present with any combination of these anatomic abnormalities.

After topically anesthetizing the nasal cavity and pharynx, a fiberoptic endoscope is introduced through the nose. In sequential fashion, the nasopharynx, oropharynx, hypopharynx, and larynx are examined. The appearance and position of the soft palate, base of tongue, and lateral pharyngeal walls are evaluated. Changes in the position of the base of the tongue such as forward movement with protrusion of the mandible are noted. The appearance of the pharyngeal airway and degree of pharyngeal wall collapse is noted while the patient performs a modified Müller maneuver. To accomplish this maneuver, the patient attempts to inspire with the mouth and nose closed. Increased negative pressure in the pharynx will demonstrate the point of collapse.

Cephalometric Examination

A lateral cephalogram is routinely obtained in the radiologic evaluation of sleep apnea patients (Figure 64-1). Cephalometric analysis is performed to identify any skeletal and soft tissue abnormalities that may exist. The advantages of cephalometry are its easy access, low cost, and minimal radiation exposure. However, it should be recognized that there are obvious limitations of evaluating a three-dimensional area with a two-dimensional lateral cephalometry.

Mandibular or maxillary position can be evaluated by a number of methods including the SNA (sella-nasion–A point) and SNB (sella-nasion–B point) angles. Patients with skeletal deficiencies are more likely to have obstruction at the base of the tongue or at the level of the soft palate. Riley and colleagues[22,23] determined that OSA patients had an inferiorly positioned hyoid bone, a longer-than-normal soft palate, and a narrowing at the base of the tongue. The position of the hyoid bone is determined by drawing a perpendicular line from the mandibular plane (MP) through the hyoid bone (H). The mean MP-H distance for normal subjects is 15.4 ± 3 mm (see Figure 64-1). The position of the hyoid bone is important because it serves as a central anchor for the muscles of the tongue and thereby partly determines tongue position. Soft palate length is measured from a line drawn from posterior nasal spine (PNS) to the tip of the soft palate shadow (P). The mean PNS-P distance in normal subjects is 37 ± 3 mm. Posterior airway space (PAS) is determined by a line drawn from point B through the gonion (Go) intersecting the base of the tongue and the posterior pharyngeal wall. Figure 64-2 demonstrates change in PASs after maxillomandibular advancement (MMA). Mean PAS in normal subjects was determined to be 11 ± 1 mm. Lower face height is measured from the anterior nasal spine (ANS) to the menton (Me).

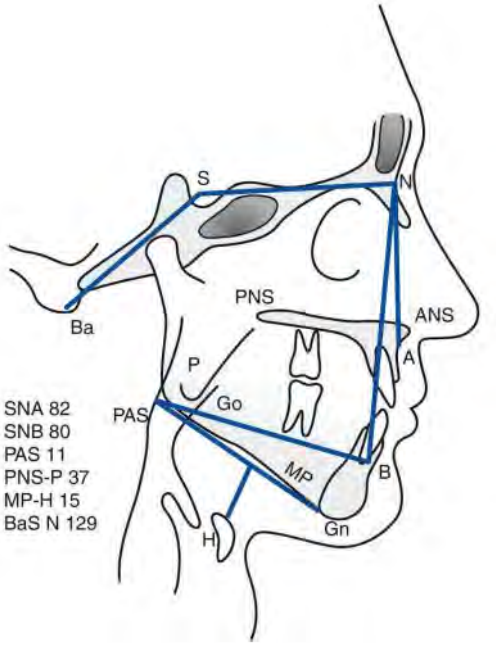


FIGURE 64-1. Cephalometric screening used for initial evaluation of patients with obstructive sleep apnea syndrome. A = A point; ANS = anterior nasal spine; B = B point; Ba = basion; Gn = gnathion; Go = gonion; H = hyoid; MP = mandibular plane; N = nasion; P = palate; PAS = posterior airway space; PNS = posterior nasal spine; S = sella; SNA = sella-nasion–A point; SNB = sella-nasion–B point. (Adapted from Tiner BD, Waite PD. Surgical and nonsurgical management of obstructive sleep apnea. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1535.)

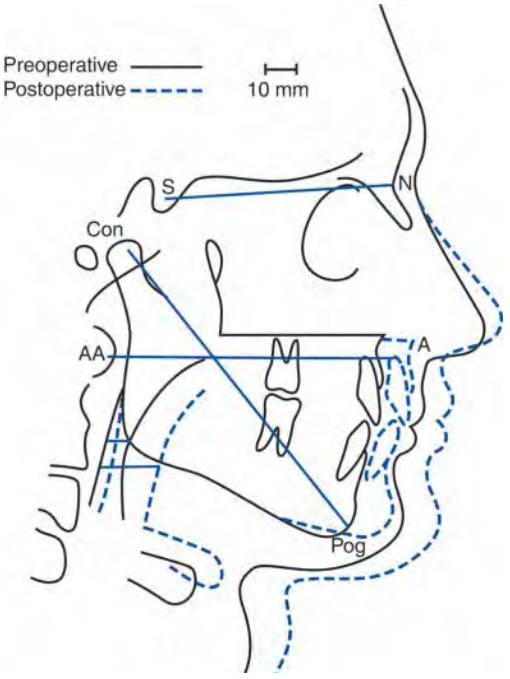


FIGURE 64-2. Change in posterior airway space after maxillary and mandibular advancement. A = A point; AA = anterior edge of atlas; Con = condylion; N = nasion; Pog = Pogonion; S = sella. (Adapted from Tiner BD, Waite PD. Surgical and nonsurgical management of obstructive sleep apnea. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1536.)

There is no absolute value for this measurement in OSA patients. However, some studies have shown an increased lower face height and a shortened cranial base with OSA patients.[18]

Computed Tomography

Computed tomography (CT) is an alternative to cephalometry and has been used to provide a quantitative assessment of the upper airway at various levels. With three-dimensional CT reconstructions, Lowe and coworkers[24] found OSA patients with larger tongue surface areas and smaller airway surface areas. Haponik and associates[25] found significantly decreased cross-sectional areas of the nasopharynx, oropharynx, and hypopharynx in OSA patients when compared with control subjects by using CT scanning. Some authorities believe that the airway can be assessed only by a CT scan. However, Riley and colleagues[23,26] compared patients who had three-dimensional CT scans and found a statistically significant correlation between the PAS measured on the lateral cephalogram and the volume of the pharyngeal airway measured on CT scans. Waite and Villos[27] demonstrated by helical CT analysis that MMA increases both anteroposterior and lateral dimension of the airway at all levels from nasopharynx to hyoid. Many studies are currently being done to determine the effects of patient position and changes in airway. A cephalogram and a CT scan are static evaluations at a fixed time of a dynamic system and they should be viewed as only part of the overall evaluation of the patient.

Polysomnography

Nocturnal polysomnography remains the gold standard for establishing the diagnosis of sleep apnea, quantitating its severity, and determining the success of treatment modalities. The study is performed in a sleep laboratory and the patient's sleep is monitored overnight. At least 4 hours of total sleep time must be recorded for a diagnostic study. The components of the polysomnogram include the EEG, electrooculogram (EOG), electromyogram (EMG), and ECG (lead V2). Sleep staging and architecture are determined by the EEG, EOG, and EMG tracings. Potentially lethal cardiac dysrhythmias are detected by the ECG. Oxygen saturation is measured by ear oximetry. A 5% or greater decrease in arterial oxygen saturation from baseline is significant during episodes of apnea or hypopnea. Respiratory effort and breathing pattern are measured using respiratory inductive plethysmography or by measuring intrathoracic pressure changes with an esophageal balloon catheter. The distinction between an episode of central apnea and obstructive apnea is made by correlating airflow at the nose and mouth with movement of the abdominal and thoracic respiratory muscles. Central apnea occurs if both airflow and respiratory muscle movement stop simultaneously. An episode of obstructive apnea occurs when airflow at the mouth and nose ceases but respiratory muscles in the abdomen and thorax continue to move dysfunctionally.

Of particular interest are the number of respiratory events (apneas and hypopneas), the number of oxygen desaturations below 90%, and the lowest oxygen desaturation. The respiratory disturbance index (RDI) can be calculated from these data:

$$\text{RDI} = \frac{\text{Apneas + Hypopneas}}{\text{Total sleep Time}} \times 60$$

An RDI greater than 5 is considered abnormal and an RDI greater than 20 is considered clinically significant, because EDS usually does not occur below this level. OSA also becomes clinically significant when oxygen desaturation events fall below 85%.

Site of Obstruction

After a complete presurgical evaluation, each patient is grouped according to the site of obstruction: type I, oropharynx; type II, oropharynx and hypopharynx; and type III, hypopharynx. In a review of 40 OSA patients, Riley and coworkers[28] found the majority of patients had a type II obstruction (soft palate and base of tongue).

The mandible, base of tongue, hyoid, and pharyngeal wall are intimately related by their muscular and ligamentous attachments. The mandible is related to the base of the tongue by the genioglossus muscle. The tongue, through

multiple muscular and connective tissue attachments, is related to the hyoid bone and to the mandible in such a way that retraction of the mandible results in a narrowing of the airway and posterior movement of the tongue. Compensatory mechanisms exist in non–sleep-apneic patients to prevent occlusion of the airway. However, in sleep-apneic patients, these mechanisms do not exist or are unable to compensate adequately.

Obstruction of the upper airway is primarily prevented by the action of the pharyngeal dilating muscles contracting in phase with respiration. Reduced muscle tone is normal and prominent during REM sleep. However, OSA patients may have a significant reduction in muscle activity during non-REM sleep so that the pharynx becomes narrower and airway resistance increases. In patients with abnormal skeletal development, the reduction in size of the resting airway may predispose them to upper airway obstruction during sleep.

The patency of the upper airway is determined by a balance between the pharyngeal musculature and the negative oropharyngeal pressures that are generated from resistance to airflow in the nasopharynx. Because the airway of OSA patients is unstable even at rest, any structural narrowing of the airway will eventually hinder the muscular component of the balance. Collapse of the airway in OSA is primarily a result of high intraluminal negative pressures associated with hypotonic pharyngeal wall musculature and disproportionate anatomy in either the oropharynx or the hypopharynx or both. Disproportionate anatomy includes any combination of large base of tongue, long soft palate, narrow mandibular arch, shallow palatal arch, or retrognathic mandible.

MEDICAL TREATMENT

Once the diagnosis has been confirmed, the treatment approach for sleep apnea is determined by the severity of the physiologic derangements and the predominant type of apnea. Regardless of the predominant type of apnea present, all patients should be cautioned that certain drugs may precipitate or exacerbate OSA. Alcohol and other CNS depressants have been shown to aggravate sleep apnea and even to precipitate apnea and oxygen desaturations in normal persons.[29]

Weight loss and nasal continuous positive airway pressure (CPAP) are the initial modes of therapy that should be initiated in obese patients with moderate OSA. A study of 16 patients who lost an average of 20 kg showed fewer apneas, reduced oxygen desaturations, and less daytime sleepiness than a control group of patients who did not lose weight.[30] Many patients can relate weight gain in preceding years to an increase in severity of their OSA symptoms. Unfortunately, weight loss by dietary measures is seldom sustained, and OSA symptoms recur with weight gain. Riley and associates[31] found that 47 of 50 OSA patients who were between 20% and 100% overweight at the time of diagnosis had regained all the weight they had initially lost 5 to 7 years later.

The role of oxygen therapy in the treatment of sleep apnea is controversial. In a study by Motta and Guilleminault,[32] the administration of oxygen increased the duration of apneic episodes and led to worsening of acidosis and hypercarbia during both REM and non-REM sleep. It is unknown how many of their patients had chronic obstructive lung disease. Other studies have shown that supplemental oxygen therapy consistently reduced the severity of oxygen desaturation and decreased the frequency of apnea.[33,34]

The combined experience of these reports suggests that oxygen therapy limited to a flow rate of 2 L/min can be used safely in most OSA patients and will produce beneficial effects on respiration. The dangers of profound hypoxemia are greater than the concerns of prolonged apnea, acidosis, and hypercarbia. The effects of oxygen therapy on a patient with severe airway obstruction or chronic respiratory acidosis should be monitored with oximetry or polysomnography.

Several drugs have been used in the treatment of OSA syndrome with variable results. The carbonic anhydrase inhibitor acetazolamide stimulates respiration by producing a metabolic acidosis. This drug reduced the number of apneas and decreased the severity of oxygen desaturations in a group of patients with central sleep apnea.[35] However, in several cases, acetazolamide given to patients with mild OSA produced more frequent OSAs of longer duration.[36] Therefore, acetazolamide is probably not indicated in the management of OSA syndrome.

Some patients with OSA benefit from the respiratory stimulant effect of progesterone, especially those with the obesity-hypoventilation syndrome.[37–40] Progesterone increases alveolar ventilation and improves oxygenation, but its effect on frequency of apnea is limited. Major side effects that limit its long-term use include decreased libido, alopecia, and impotence.

The tricyclic antidepressant protriptyline is the most effective and best studied drug for the treatment of OSA.[41] In a study of 12 patients, Smith and colleagues[42] showed a reduction in apnea frequency and oxygen desaturation during non-REM sleep, in addition to a decrease in REM sleep. Protriptyline produces its beneficial effect by a preferential stimulation of upper airway muscle tone and by decreasing the percentage of time spent in REM sleep, thereby reducing the more severe REM-related apneas. Anticholinergic side effects such as dry mouth, constipation, urinary retention, and impotence are frequent and limit its use.

Oral Appliances

The use of a variety of prosthetic devices is another approach to treatment. The nasopharynx and the posterior tongue are the two anatomic areas of concern. Insertion of a nasopharyngeal airway has been used to prevent upper airway occlusion at the level of the soft palate.[43] The American Sleep Disorders Association recommends that oral appliances may be used in patients with primary snoring, mild OSA, or in those with moderate to severe OSA who refuse or are intolerant of nasal CPAP. The U.S. Food and Drug Administration has granted market clearance for 32 oral appliances for snoring but only 14 of these have received market clearance for

TABLE 64-1. **Food and Drug Administration Approved Oral Appliances for the Treatment of Obstructive Sleep Apnea**

Appliances	Manufacturer
Adjustable PM Positioner	Jonathan A. Parker, DDS
Elastic Mandibular Advancement, Triation (EMA-T)	Frantz Design, Inc.
Elastic Mandibular Advancement	Frantz Design, Inc.
Elastomeric Sleep Appliance	Village Park Orthodontics
Equalizer Airway Device	Sleep Renewal Inc.
Herbst	Orthodontics, SUNY at Buffalo
Klearway	Great Lakes Orthodontics, Ltd.
NAPA	Great Lakes Orthodontics, Ltd.
OSAP	Snorefree, Inc.
PM Positioner	Jonathan A. Parker, DDS
Silencer	Silent Knights Ventures, Inc.
Sleep-In Bone Screw System	Influence Inc.
SNOAR Open Airway Appliance	Kent J. Toone, DDS
Thornton Oral Appliance	W. Keith Thornton, DDS

treatment of snoring and OSA (Table 64-1).[44] Common side effects of oral appliance therapy include excessive salivation, xerostomia, soft tissue irritations, transient discomfort of the teeth and temporomandibular joint (TMJ), and temporary minor occlusal changes. Uncommon, more serious complications include permanent occlusal changes and significant TMJ discomfort.

Removable anterior repositioning splints have been used somewhat successfully to temporarily advance the mandible while passively bringing the tongue forward with it.[44–46] The optimal amount of forward movement is between 50% and 75% of the patient's maximum protrusive distance. An important design feature of these devices is that the appliance must maintain the mandible in the forward position while the patient is asleep. Bear and Priest[47] used a mandibular anterior repositioning splint to determine whether surgical advancement of the mandible would have any lasting and positive effect on a patient's OSA.

A tongue-retaining device (TRD) that pulls the tongue forward without moving the mandible forward has also been used successfully in some patients with mild to moderate OSA.[48,49] The TRD functions by placing the tongue into a cup or bubble positioned between the anterior teeth. Surface adhesion holds the tongue in place and the appliance requires that the patient's jaw remains partially open. One disadvantage of the TRD is that the tongue is not always held forward because surface tension of the tongue in the bubble is lost after a time. The TRD and mandibular anterior repositioning splints both force nasal breathing, which can be difficult for patients with inadequate nasal airways.

Arguably, the most researched oral appliance is the Klearway titratable appliance developed by Alan Lowe, DMD, PhD, at the University of British Columbia, Canada. It features a maxillary and mandibular component connected with an adjustable screw mechanism (Figure 64-3). The components are made of a thermoactive acrylic resin that is slightly soft at body temperatures and very compliant at high temperatures. This property decreases major tooth discomfort and considerably increases retention in those patients who have lost a significant number of teeth. A unique feature of the Klearway appliance is that it permits both lateral (1–3 mm) and vertical (1–5 mm) jaw movement during sleep, which reduces the risk of TMJ and jaw muscle discomfort. This movement also facilitates oral breathing in patients with nasal airway obstruction. The screw mechanism of the appliance allows for an 11-mm anterior movement of the mandible with a total of 44 incremental steps of 0.25 mm. In a study of 38 patients with moderate to severe OSA by Lowe and coworkers,[50] the Klearway appliance reduced the RDI to less than 15/hr in 80% of the moderate group and in 61% of the severe group. The Klearway appliance is marketed worldwide by Great Lakes Orthodontic Ltd., Tonawanda, NY).

Another commonly used and effective oral appliance is the Herbst appliance, which is an anterior mandibular positioning device. It consists of two full-coverage clear acrylic components snapped onto the maxillary and mandibular teeth connected with two rod and tube attachments that allow vertical opening, protrusion, limited lateral movement, and no retrusive movement. It is used only at night and advances the mandible 5 to 7 mm or at least 75% of the patient's maximum protrusive distance. A study by Clark and associates[51] on 24 patients with mild to severe OSA using the Herbst appliance showed a significant improvement in the RDI after 4 months of appliance use in 58% of the subjects on the postappliance polysomnogram.

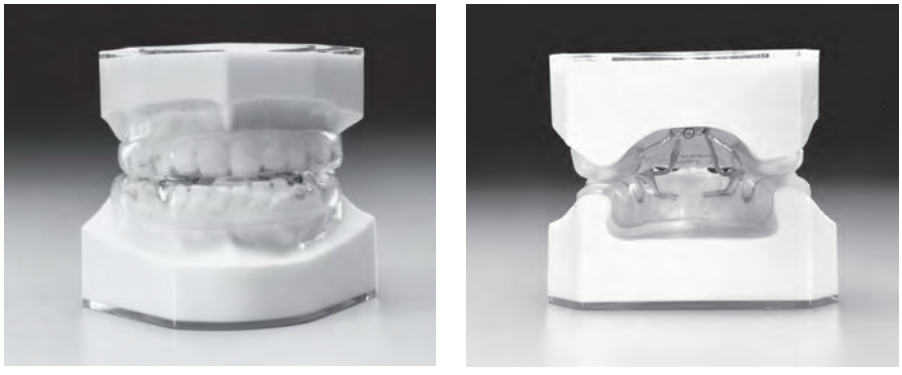


FIGURE 64-3. Klearway oral appliance.

Another disadvantage of oral appliances is the need to wear them nightly. As with any device, compliance has been shown to be a problem with oral appliances. If appliance therapy is successful, further treatment options may include mandibular advancement surgery to achieve the same forward tongue position on a permanent basis.

Continuous Positive Airway Pressure

CPAP through the nose has been shown to be quite successful in treating a broad range of OSA patients and is at present the most successful nonsurgical treatment.[52–54] The nasal CPAP is administered while the patient is asleep by means of a tight-fitting mask that is connected to a compressor. A CPAP of 7 to 15 cmH_2O acts as a pneumatic splint of the upper airway and prevents passive collapse of soft tissues during respiration. Stimulation of mechanoreceptors of the genioglossus muscle leading to increased airway tone has also been suggested as a mechanism of action. Sullivan and colleagues were the first to report the successful treatment of sleep apnea with nasal CPAP in 1981.[55] In most cases, this therapy is effective in eliminating apneas and hypopneas, improving arterial oxygen saturations, reducing or eliminating excessive daytime sleepiness, and eliminating sleep disruption and fragmentation. CPAP may be combined with surgery and weight loss, or it may be used as a sole form of therapy. Although initially recommended for short-term relief of sleep apnea, the use of nasal CPAP for long-term care of patients has increased over the past few years. In recent years, bilevel positive airway pressure (BiPAP) systems that allow independent regulation of inspiratory and expiratory pressures and the newest modification in CPAP systems, auto-CPAP, have been used to more effectively treat OSA and increase tolerance and compliance.[56,57] Auto-CPAP units adjust the CPAP throughout the night rather than delivering one fixed pressure. Optimal CPAP is delivered to the patient adjusting for positional changes, alcohol or sedative effects, sleep-state–dependent changes (REM vs. non-REM), and the effects of upper airway infections or congestion. BiPAP ($2,500) and auto-CPAP ($1,600) systems are more expensive than traditional CPAP ($600–$800) systems.

Despite the uniform success of this therapy, patient compliance remains a problem. Compliance rates at 12 months have been reported as low as 54%.[58] The average nightly use of CPAP is 4.8 hours and the rate of use is usually determined in the first week of use. Overall, approximately one third of patients love CPAP, one third struggle with CPAP but eventually tolerate it, and one third hate CPAP and never use it. Patient dissatisfaction results from nasal dryness and congestion, sore throat, dryness of the skin and eyes, claustrophobia, and the inability to tolerate the noise, discomfort, or mask. Careful patient selection and follow-up are essential if nasal CPAP is selected as a treatment modality.

SURGICAL TREATMENT

Surgery has been the primary form of therapy for OSA. To be successful, the surgical procedure must either bypass the obstructive area or prevent collapse of the soft tissues in the upper airway at the obstruction. Many patients and surgeons tend to view surgical treatment of OSA as a quick and permanent cure. However, a clear definition of what constitutes a cure is lacking in the literature. This problem often makes a determination of the efficacy of individual surgical procedures difficult. Only objective data obtained from a postoperative polysomnogram can be accepted as proof of efficacy for surgical procedures. Currently, the procedures used in the surgical treatment of OSA include tracheostomy, nasal surgery, uvulopalatopharyngoplasty (UPPP), and several orthognathic surgical procedures. Selection of the individual procedure is determined by the severity of the sleep apnea, the presence of a maxillofacial skeletal deficiency, the site of the obstructive segment, and the presence of morbid obesity.

Tracheostomy

Tracheostomy was the first efficacious surgical procedure for treating OSA, performed by Kuhlo and coworkers in 1969.[59] It is almost 100% curative in relieving the signs and symptoms of OSA because it bypasses all the potential obstructive sites in the upper airway. After tracheostomy, there is a rapid and striking reduction in daytime somnolence and a marked improvement in sleep architecture owing to a major reduction in the frequency of arousals. Sinus dysrhythmias, bradycardia, pulmonary hypertension, hypoxemia, and apnea all improve dramatically with the procedure. Tracheostomy clearly is an effective surgical treatment for patients with OSA.

The disadvantages of a permanent tracheostomy can have a devastating effect on sleep apnea patients. Almost all patients experience psychological depression from the social and medical problems associated with a lifelong tracheostomy. The tracheostomy leaves the patient aesthetically disfigured and exposes the patient to common local complications such as bleeding, infection, pain, and granulation tissue formation. Patients are also at increased risk for the more serious complications of tracheal stenosis or erosion into an adjacent blood vessel. Because of these disadvantages and complications, a permanent tracheostomy should be reserved for severe cases of OSA with significant cardiovascular symptoms. Simmons and coworkers[60] have suggested that tracheostomy should be the primary therapy for all patients who spend substantial time in severe oxygen desaturations below 50% and for those who have life-threatening cardiac dysrhythmias during sleep apnea. Tracheostomy may also be used as an interim treatment until adjunctive procedures to reconstruct the upper airway are completed.

Nasal Surgery

Significant obstruction in the nasal cavity has been shown to cause excessive daytime sleepiness, sleep fragmentation, hypopneas, and periodic breathing during sleep.[61] In most patients with moderate to severe OSA, nasal obstruction is not the major contributing factor. The obstruction may be due to a deviated nasal septum, nasal polyps, or enlargement of

the turbinates. In these patients, septoplasty, nasal polypectomy, or turbinectomies are usually helpful only as adjunctive surgical procedures in the treatment of OSA. Unless the obstruction in the nasal cavity is severe, surgical correction usually will not yield any significant improvement on a repeat polysomnography.

Uvulopalatopharyngoplasty

The oropharynx and soft palate can cause significant airway obstruction during sleep. At least 10% of persons older than 40 years snore regularly and significantly. Loud and intermittent snoring is found in almost all patients with OSA. In many cases, habitual snoring is present for many years before sleep apnea is diagnosed. Ikematsu[62] followed a large number of habitual snorers over several years and found that 91% of these patients had decreased oropharyngeal dimensions and longer soft palates and uvulas than normal subjects. He was able to eliminate their snoring by surgically excising the excessive soft tissue in the palatal folds and partially excising the uvula.

With minor modifications, Simmons and coworkers[60] and Fujita and associates[63] popularized the UPPP for the treatment of OSA. The procedure was designed to eliminate oropharyngeal obstruction by performing a tonsillectomy and adenoidectomy, excising the uvula, removing redundant lateral pharyngeal wall mucosa, and resecting 8 to 15 mm along the posterior margin of the soft palate.

The surgical technique of UPPP varies to some degree by patient and surgeon, but the basic goal is to shorten the palate and widen the PAS (Figure 64-4). A mucosal incision is created with electocautery on the anterior surface of the soft palate. The dissection is frequently carried laterally to include the palatine tonsil. The tonsillar bed is coagulated and hemostasis achieved. Palatal muscle is excised and mucosa from the nasopharynx is pulled forward for primary closure. Multiple interrupted resorbable sutures are placed. If the tonsil is removed, the mucosa of the anterior fauces pillar is closed to the posterior fauces pillar. This attempt to remove redundant pharyngeal tissue and stretch or tighten the posterior pharyngeal wall results in constriction. In addition, frequently by shortening the soft palate, the width of the soft palate is thickened, as demonstrated cephalometrically. Lymphoid tissue from the tonsillar fossa can be removed separately or in conjunction with the uvula (Figure 64-5). The amount of velum to be excised is determined by the location of palatal competence and closure of the nasopharynx. These can be estimated or identified during nasopharyngoscopy. Palatal incompetence can occur but usually is of minimal concern if the patient swallows carefully. Pain with swallowing usually lasts for several weeks.

UPPP results in symptomatic improvement in the patient and eliminates habitual snoring in almost all cases. However, reports show that significant objective improvement on the postoperative polysomnogram ranges only from 41% to 66%.[58,60,64,65] This procedure only eliminates the obstruction at the level of the soft palate and does not address

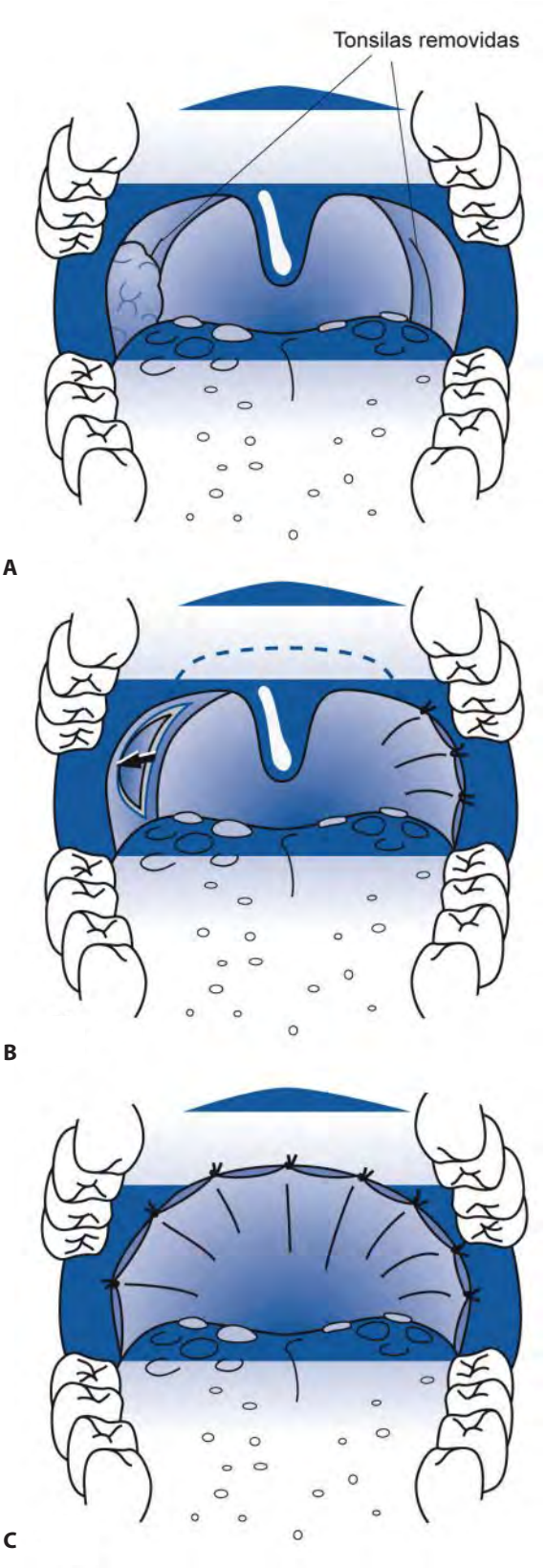


FIGURE 64-4. **A–C,** Uvulopalatopharyngoplasty. Tonsils and uvula are removed and the anterior pillar is closed to the posterior pillar. (Adapted from Tiner BD, Waite PD. Surgical and nonsurgical management of obstructive sleep apnea. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1540.)

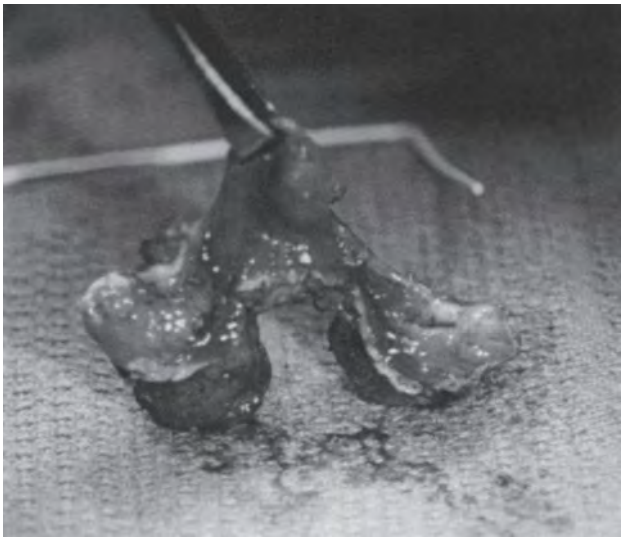


FIGURE 64-5. Surgical specimen of tonsils and uvula. (Reproduced with permission from Tiner BD, Waite PD. Surgical and nonsurgical management of obstructive sleep apnea. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1541.)

obstructions occurring in the hypopharyngeal and base of tongue areas. Many patients have more than one site of obstruction. If UPPP is performed when the presurgical evaluation demonstrates obstruction localized to the soft palate–tonsil area, the success rate of the surgical procedure approaches 90% treating OSA.[23,26]

Complications from UPPP are related to changes in palatal function. Permanent velopharyngeal incompetence occurs in approximately 5% of patients and is more common during the first 2 months postoperatively. Patients experience occasional reflux of liquids into the nose and mild nasal air escape during speech. However, hypernasal speech and changes in the quality of the patient's speech are usually not seen. Simmons and colleagues[64] reported a 5% to 10% rate of minor wound infections that resolved with antibiotics. Palatal stenosis is definitely a risk with this operation and occurs in approximately 1% of patients. It occurs more frequently with excessive resections of the posterior tonsillar pillars and injudicious use of electrocautery. Postoperative pain after UPPP is significant, and narcotic analgesia should be used with caution to prevent sedation-induced exacerbation of OSA. Postsurgical deaths have resulted from the combination of pharyngeal edema and narcotic use.

Laser-Assisted Uvulopalatoplasty

In the late 1980s, Dr. Yves-Victor Kamami (Paris, France) designed a procedure to reshape and recontour the soft palate under local anesthesia with a CO_2 laser to treat snoring and selected patients with OSA syndrome.[66] He originally named the procedure "laser resection of the palatopharynx" (LRPP). Initially, the procedure was accomplished in four or five sessions spaced at monthly intervals. Over time, the procedure evolved into a one-stage technique for most patients. It consisted of two paramedian vertical incisions placed lateral to the uvula extending up toward the junction of the hard and soft palates for 2 to 3 cm. A second horizontal incision was placed just under the roof of the uvula, leaving a small uvula to prevent centripetal scar formation. Over a 5-year period, Kamami[66] treated 63 OSA patients with this technique. The RDI was reduced by more than 50% in 55 patients that were classified as successful responders. The RDI improved from 41.5 to 16.9 for the average responder, and for the entire group, the average RDI improved from 41.3 to 20.3.

In the early 1990s in the United States, Dr. Yosef Krespi modified the procedure and renamed it "laser-assisted uvulopalatoplasty" (LAUP). He initially used the procedure to treat loud habitual snoring. In a study of 280 patients treated in the office under local anesthesia, 84% were cured with an average of 2.7 sessions.[67] Overall results for OSA patients treated with LAUP are far less encouraging, with an average successful surgical response of 52.2%.[68] Based on these findings, the current main indications for LAUP include loud habitual snoring, upper airway resistance syndrome, and mild OSA (apnea index < 20). All snoring patients who elect to undergo LAUP should be evaluated for OSA preoperatively and again postoperatively if OSA was previously diagnosed. If not, then the patient and surgeon may be lulled into a false sense of security by eliminating the snoring without eliminating the undiagnosed OSA, potentially increasing patient morbidity and mortality.[69]

The most common complication after LAUP is a moderate to severe sore throat. Patients experience pain 8 to 10 days after surgery and reach their peak pain intensity on the fourth or fifth postoperative day. Pain control is achieved with oral analgesics and anesthetic gels. The risk for velopharyngeal insufficiency is low because the procedure is frequently done in stages and the surgeon has the opportunity to evaluate speech and soft palate function after each session. Patients are also at low risk for bleeding and infection. The great majority of patients can eat, drink, and speak almost immediately and can resume full activities the following day.

Orthognathic Surgery Procedures

Various orthognathic surgical procedures have been described for the treatment of OSA. The majority of patients have airway obstruction at the level of the soft palate and at the base of the tongue (type II obstruction). Orthognathic surgical procedures can change the size of the airway in several regions. Mandibular advancement and genial advancement probably work by changing the position of the mandible and hyoid bone with subsequent effects on the genioglossus and hyoglossus muscles. OSA patients with identifiable craniofacial anomalies can clearly benefit from a variety of these procedures.

Mandibular Advancement

Total mandibular advancement was the first orthognathic surgical procedure used in the treatment of OSA. Kuo and

colleagues in 1979[70] and Bear and Priest in 1980[47] reported complete reversal of sleep apnea symptoms in patients with horizontal mandibular deficiency treated by mandibular advancement. More recently, Alvarez and coworkers[71] reported the successful treatment of an edentulous patient with sleep apnea by mandibular and genial advancement.

A bilateral sagittal ramus osteotomy is usually the procedure of choice for total mandibular advancement. The amount of advancement is determined preoperatively from the orthognathic surgery database. Adjunctive orthodontic treatment is frequently necessary to obtain the desired occlusion and to eliminate dental compensations that would otherwise limit the amount of advancement. After advancement with the standard surgical technique, the fragments are rigidly fixed with screws or bone plates. For large advancements of 7 mm or more, long-term stability is enhanced with a 5- to 7-day course of maxillomandibular fixation and skeletal suspension wires. In advancements of 6 mm or less, maxillomandibular fixation is usually not necessary.

The exact reason for how mandibular advancement improves OSA is not clearly known, but the suspected effect is the pulling of the tongue forward off the pharyngeal wall. This effect is created by anteriorly moving the insertion of the genioglossus and geniohyoid muscles. If this were the only factor, anterior chin procedures would be equally effective as total mandibular procedures. Variations of geniotomies have been designed to maximally pull the tongue muscles forward.

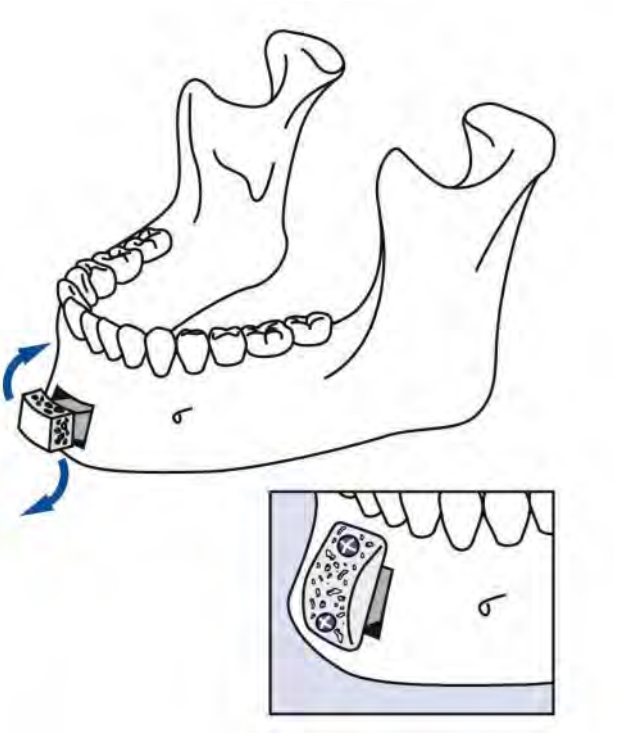


FIGURE 64-6. Genial tubercle advancement. The outer table of the symphysis is removed and the inner table is secured with 2-mm screws. (Adapted from Tiner BD, Waite PD. Surgical and nonsurgical management of obstructive sleep apnea. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1542.)

Genial Advancement

A rectangular osteotomy apical to the teeth but maintaining the inferior border of the mandible allows the genial tubercles with their muscular attachments to be maximally advanced with minimal cosmetic change (Figure 64-6). A modified vestibular mucosal incision is made in the anterior mandible. Periosteum is reflected down to the inferior border. An oscillating saw is used to make parallel horizontal cuts that include the genial tubercle. The osteotomy is designed in a shape similar to a drawer so that it can be pulled outward with the genial muscles. The bone must be broad enough cuspid tocuspid to be rotated 90 degrees and set on top of the buccal cortex. The outer cortical and cancellous bone of the rectangle can then be removed and the inner cortex rigidly fixed with bone screws. Any hemorrhage from the cancellous bone should be controlled.

This procedure does not change the aesthetic chin or advance the anterior belly of the digastric muscle, which may be helpful in suspending the hyoid. In contrast to this procedure, a horizontal sliding geniotomy does advance the genial tubercles and the anterior belly of the digastric muscle.

Genial Advancement with Hyoid Myotomy and Suspension

In 1984, Riley and associates[72] described an alternative technique in which an inferior mandibular osteotomy and an associated hyoid myotomy and suspension were used in the treatment of OSA (Figure 64-7). This technique is similar to a horizontal mandibular osteotomy, which is commonly used for advancement genioplasty. The osteotomy is designed to include the genial tubercle on the inner cortex of the anterior mandible where the genioglossus muscle attaches. Repositioning the anteroinferior segment of the mandible forward with the attached genioglossus muscle theoretically pulls the tongue forward and improves the hypopharyngeal airway. In conjunction with the osteotomy, the body and greater cornu of the hyoid are isolated through a submental incision. The infrahyoid muscles are transected, taking care to remain on the hyoid bone at all times to avoid injury to the superior laryngeal nerves (see Figure 64-7A). This allows the hyoid bone to be pulled anteriorly and superiorly. Strips of fascia or nonresorbable suture are passed around the body of the hyoid and attached to the intact portion of the anterior mandible to complete the hyoid suspension.

In 1989, Riley and colleagues[73] published a review of 55 patients with OSA who were treated with inferior mandibular osteotomy and hyoid suspension. Forty-two patients had obstruction at both the oropharynx and the hypopharynx and received concomitant UPPP and inferior mandibular osteotomy with hyoid myotomy and suspension. The remaining 6 patients were determined to have obstruction localized to the base of the tongue and underwent the osteotomy and hyoid suspension only. All patients were reevaluated by polysom-

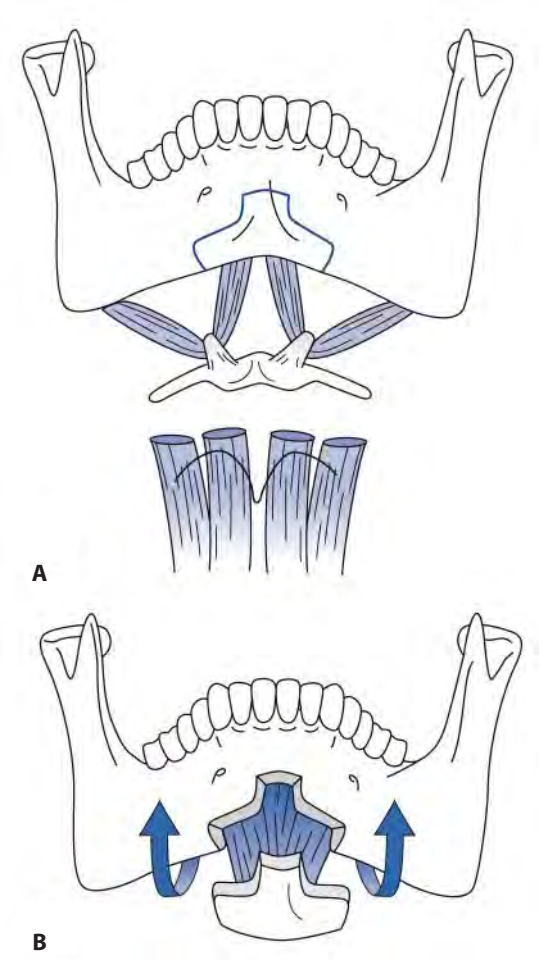


FIGURE 64-7. Inferior mandibular osteotomy and hyoid myotomy. **A,** Omohyoid, sternohyoid, and thyrohyoid muscles released (see Figure 64-8 for more detail on muscular relationships). **B,** Inferior segment is advanced anteriorly and locked on the anterior mandible. (**A** and **B,** Adapted from Tiner BD, Waite PD. Surgical and nonsurgical management of obstructive sleep apnea. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1543.)

nography 6 months after surgery. Thirty-seven patients (67%) were considered to be responders to surgery based on the polysomnogram results. Genioglossus advancement ranged from 8 to 18 mm with a mean of 13 mm. All responders to surgery showed significant improvement in their RDI and oxygen desaturation events. Eighteen patients (33%) were considered nonresponders and failed to show significant improvement by polysomnography. The presence of preexisting chronic obstructive pulmonary disease (COPD) was found to be a determining factor in increasing the risk of failure.

In 1994, Riley and colleagues[74] reported on a new modified technique for hyoid suspension that fixed the hyoid to the thyroid cartilage instead of the anterior margin of the mandible. When this modified technique was performed with inferior mandibular osteotomy, in lieu of the original hyoid suspension technique, the surgical response rate (with or without UPPP) was raised to 79.2%. The 5 nonresponders in this study of 24 patients achieved postoperative RDI values close to levels at which they would have been considered surgical responders.

Long-term follow-up of these patients has shown that the indication for this procedure is limited. Patients with normal pulmonary function, normal skeletal mandibular development, the absence of obesity, and moderate OSA are candidates for treatment with inferior mandibular osteotomy with hyoid myotomy and suspension.

The most serious reported complication from a hyoid suspension has been severe aspiration in one patient, in which the thyrohyoid membrane was totally sectioned.[28] Other complications have included wound infections, transient sensory disturbances of the mental nerve, and mandibular fracture. An advantage to hyoid suspension is that it circumvents the need for maxillomandibular fixation and does not affect the occlusion.

Maxillomandibular Advancement

Combined advancement of the maxilla and mandible with or without hyoid suspension is the most recent and efficacious surgical procedure for the treatment of OSA. The surgical technique includes a standard Le Fort I osteotomy in combination with a mandibular sagittal split osteotomy for advancement of the maxilla and mandible. A concomitant inferior mandibular osteotomy with or without hyoid myotomy and suspension, as previously described, is also performed. This surgery may result in a significant facial change, which is most often favorable (Figures 64-8 and 64-9). Several authors have described the use of MMA in treating large series of OSA patients.[75–80] In a series of 23 patients, Waite and coworkers[75] performed a high sliding horizontal genioplasty without the hyoid myotomy and suspension. All patients were reevaluated by polysomnography at 6 weeks postoperatively. The surgical success with MMA was 65% based on a postsurgical RDI of less than 10. Riley and associates[76] reported the largest series of OSA patients treated with MMA in which 98% (89 of 91) were successfully treated based on a postoperative RDI of less than 20 with at least a 50% reduction in the RDI compared with the preoperative study. It should be noted that 67 of the 91 patients (74%) did not receive phase 1 therapy based on their two-phase protocol for reconstruction of the upper airway. Despite this, the MMA was labeled a phase 2 procedure. In 1997, Hochban and colleagues[77] reported a 98% success rate on 38 OSA patients consecutively treated with a 10-mm MMA as the primary surgery, without any adjunctive procedures. Their criteria for success were based on the more rigid postoperative RDI of less than 10. Patient selection for MMA was based on subjective symptoms of excessive daytime sleepiness, an RDI of greater than 20, and specific craniofacial characteristics determined cephalometrically Only 2 patients who were morbidly obese were treated surgically. Based on their excellent results, the authors concluded that a stepwise algorithm of staged surgical procedures was not justified. In

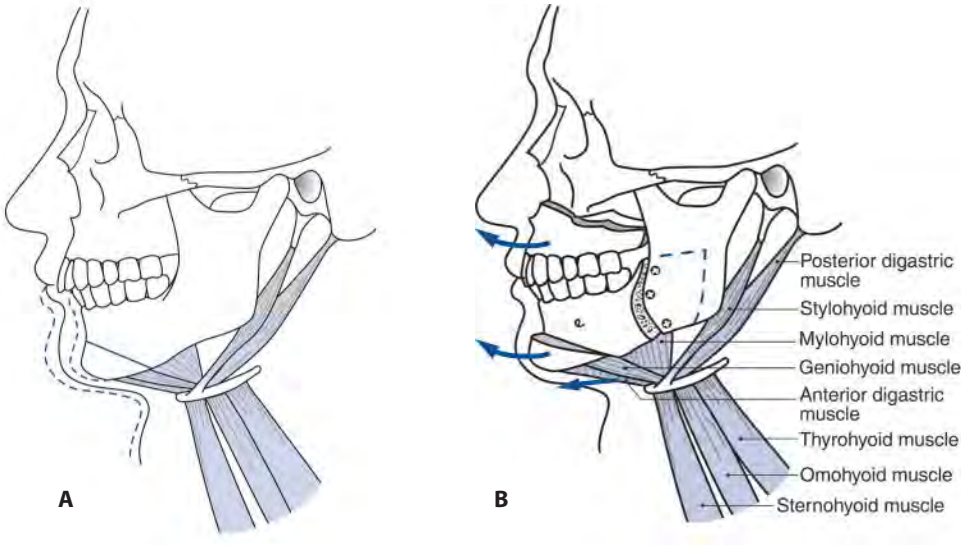


FIGURE 64-8. Schematic drawings of preoperative **(A)** and postoperative **(B)** two-jaw advancement, genial advancement, and hyoid suspension. (**A** and **B,** Adapted from Tiner BD, Waite PD. Surgical and nonsurgical management of obstructive sleep apnea. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1544.)

a series of 50 OSA patients consecutively treated with MMA, Prinsell[78] reported a 100% success rate based on a postoperative RDI of less than 15, an apnea index (AI) of less than 5, or a reduction in the RDI and AI of greater than 60%. In this series, occasional concomitant nonpharyngeal procedures and an anterior interior mandibular osteotomy were accomplished with the MMA as a single-stage operation. In 1999, Lee and coworkers[79] proposed a three-stage protocol for the surgical treatment of OSA patients. All 35 patients in their series had type II obstruction with collapse at the oropharyngeal and hypopharyngeal areas. Stage 1 reconstruction consisted of a UPPP and inferior sagittal osteotomy with genioglossus muscle advancement, or an anterior mandibular osteotomy. If stage 1 was unsuccessful, patients advanced to stage 2, which consisted of MMA with rigid fixation. A hyoid myotomy and suspension was the sole component of stage 3 reconstruction. Based on postoperative polysomnography, 69% (24 of 35) were considered surgical respondents based on an RDI of less than 20. Of the 11 stage 1 failures, 3 elected to proceed to stage 2 reconstruction with MMA. All patients who underwent MMA had a postoperative RDI of less than 10, indicating a 100% response rate. No patient required stage 3 reconstruction. Bettega and associates[80] treated 51 consecutive OSA patients according to the Stanford two-step surgical procedure. Forty-four patients had phase 1 surgery with a success rate of 22.7% (10 of 44). Twenty patients underwent MMA as part of phase 2 in the protocol. Of these, 75% (15 of 20) were considered to be surgical responders based on a postoperative RDI of less than 15 and at least a 50% reduction in the RDI. Of the 5 failures, 3 had postoperative RDIs of less than 20.

The PAS consistently increases with MMA. However, there is no direct relationship between the gain in PAS and the remission of sleep apnea. MMA is effective for patients who have obstruction at the base of the tongue. This surgical treatment is the most efficacious procedure for expanding the pharyngeal airway and improving or eliminating OSA. It remains the best current alternative to tracheostomy.[81] Indications for this procedure include severe mandibular deficiency (SNB < 74 degrees), morbid obesity, severe OSA (RDI > 50, oxygen desaturations < 70%), hypopharyngeal narrowing, and failure of other forms of treatment.[82] The success rate of MMA appears to increase when adjunctive procedures such as UPPP, partial glossectomy, septoplasty, or turbinectomies are



FIGURE 64-9. Preoperative **(A)** and postoperative **(B)** photographs of a patient with obstructive sleep apnea syndrome who underwent two-jaw surgery with a genial advancement. (**A** and **B,** Reproduced with permission from Tiner BD, Waite PD. Surgical and nonsurgical management of obstructive sleep apnea. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1545.)

included in the treatment plan. This lends support to the theory that most OSA patients have multiple levels of obstruction.

Adjunctive orthodontic therapy is usually indicated in patients selected for MMA. Presurgical orthodontics improves the postoperative occlusion and eliminates preexisting dental compensations that would otherwise limit the amount of advancement. Maximum advancement of the facial skeleton and maintenance of a functional occlusion and acceptable aesthetics are the goals of surgical-orthodontic correction.

The osteotomies are rigidly fixed with miniplates and bicortical screws (Figure 64-10). With large advancements (>7 mm), skeleton suspension wires and a short course of maxillomandibular fixation (1 wk) can be used to reduce surgical relapse. Potential complications of MMA include surgical relapse, nonunion, bleeding, malocclusion, infection, unfavorable changes in facial appearance, and permanent or temporary sensory disturbances of the inferior alveolar and infraorbital nerves.

The long-term skeletal stability of MMA has been shown to be quite good. Louis and colleagues[83] showed a mean relapse of 0.9 ±1.8 mm among 20 maxillary advancement patients who underwent MMA for OSA. The mean follow-up period was 18.5 months (range 6–29 mo). When the patients were divided into three groups reflecting small (≤6 mm), medium (7–9 mm), and large (≥10 mm) advancements, there was no statistical difference in the measured relapse among the groups. Rigid fixation was achieved with four miniplates and no bone grafts were used in any of the maxillary advancements. Nimkarn and coworkers[84] reported on 19 OSA patients who underwent MMA with simultaneous genioplasty and found relatively stable long-term (>12 mo) surgical stability of the maxilla and mandible. Maxillary and mandibular advancement was stable over the long term in both the vertical and the horizontal planes. With the exception of gonion in the vertical plane, there was no statistically significant correlation between the amount of surgical advancement and the amount of postsurgical instability.

Mandibular Setbacks

In a small number of patients, a mandibular setback procedure can be the initiating factor in the development of OSA. Riley and associates[85] reported on two women who developed OSA syndrome after mandibular osteotomies for correction of class III malocclusion and skeletal prognathism. Neither patient had any symptoms of sleep apnea before surgery. Postoperatively, both patients began to snore loudly. Evaluation by polysomnography confirmed the presence of OSA syndrome. A comparative examination of the preoperative and postoperative lateral cephalograms of each patient showed a more inferiorly positioned hyoid bone and a narrowing of the pharyngeal airway as a result of the mandibular setback procedure.

In an attempt to identify those patients potentially at risk for OSA, all patients who are planned for mandibular setback procedures should be questioned preoperatively and postoperatively about the presence or absence of snoring, excessive daytime sleepiness, or observed apneas during sleep. Although the vast majority of patients who undergo mandibular setbacks are able to adapt to the changes in the skeletal and muscular apparatus, a subset of patients may be at risk for developing overt signs of OSA after mandibular setbacks.

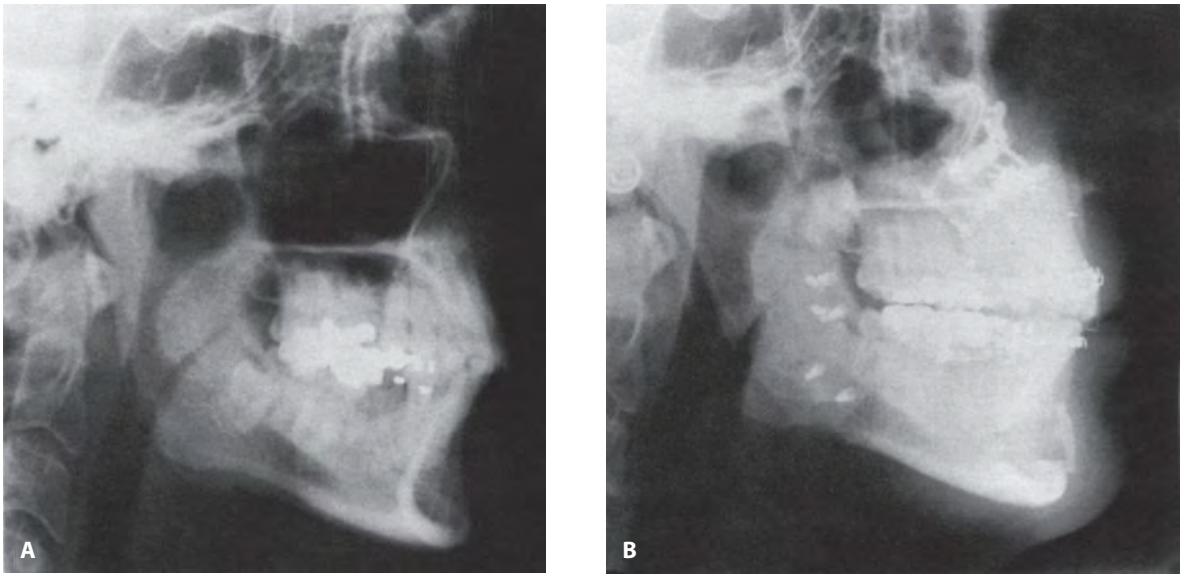


FIGURE 64-10. Preoperative **(A)** and postoperative **(B)** radiographs of a two-jaw surgery and genial tubercle advancement for sleep apnea. (**A** and **B,** Reproduced with permission from Tiner BD, Waite PD. Surgical and nonsurgical management of obstructive sleep apnea. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; p. 1546.)

COMPLICATIONS

Surgical complications in treatment of OSA may be severe and different from those related to routine orthognathic surgery. Sleep apnea patients are often older and more obese and, by the nature of their disease, have difficult airways. Obesity is a known co-morbidity associated with multiple medical issues such as hypertension, diabetes, heart disease, pulmonary dysfunction, poor intravenous access, and difficult airway management.

Respiratory failure is the major complication of airway surgery. Respiratory obstruction is a common cause of death in sleep apnea, especially after UPPP. Anesthesia protocols exist for management of the sleep apnea patient owing to physical characteristics such as obesity, short neck, small jaw, large tongue, difficult laryngoscopy, and poor mask ventilation. Jet ventilation and fiberoptic intubation are valuable techniques.[86]

Obesity, hypertension, COPD, myocardial infarction, and stroke are more common in the patient with OSA.[87] The surgeon should anticipate these medical problems and be prepared to manage appropriately. Standard intravenous access and central lines are much more difficult to obtain in the obese patient. Large obese surgical patients are more difficult owing to intraoral access, and obesity is often associated with increased infection and poor healing. Hypotensive anesthesia may be contraindicated and postoperative blood pressure management more complicated in the presence of pain control. Renal perfusion is often reduced in this group and antihypertensive medication will complicate fluid management. Older patients with hypertension and poor cardiac function need closer management of fluid status than the routine healthy orthognathic patient. People with COPD and low baseline oxygen saturations may desaturate quickly if oversedated postoperatively, and poor oxygen saturation may contribute to poor healing, dehiscence, and bone nonunion. Coronary artery disease especially stents, arrhythmias and strokes is often treated with anticoagulation or antiplatelet therapy. Extensive upper airway surgery such as MMA or tongue base or palate surgery is at a great risk for hemorrhage.

Patient positioning on the operating room table for large obese patient is clearly a challenge. Many sleep apnea patients just do not fit the average operating room table and careful positioning with padded support must be provided. Aggressive arm slings that wrap the shoulder upward often produce chronic pain and postoperative musculoskeletal pain.

Deep venous thrombosis has a higher association with extended surgery time, abdominal girth, and obesity. Sequential compression devices should be used on all OSA patients and maintained until adequate ambulation.[88,89] Antiplatelet therapy or low-dose heparin is not recommended owing to the continued bleeding in the maxilla, sinus, and nasal area. Soft tissue ecchymosis may create airway obstruction.

Malocclusion and inferior alveolar nerve paresthesia are the two most common postoperative complaints. MMA surgery is much different than orthognathic surgery because the intention is to maintain the same occlusion but move both the upper and the lower dentition forward, thereby increasing the upper airway space. Large skeletal changes are often necessary to open the airway but also pose greater challenges in maintaining the intricate relationship of intercuspation. Even a small percentage of relapse in a large advancement will result in a nonfunctional malocclusion. Patients with presurgical abnormal occlusion but good dentition should be considered for presurgical orthodontics. Patients must know in advance the risk of malocclusion and realize orthodontics is not considered medically necessary. Because the magnitude of skeletal change is greater than with orthognathic surgery, the skeletal fixation techniques must be modified.

Paresthesia may be more problematic in this patient subset owing to the greater skeletal change and the age distribution. Older age groups typically do not adjust to nerve injury as well as younger patients. Most nerve injuries improve slowly with time, but low-dose medical therapy with clonazapam, amitriptyline, or gabapentin can be very beneficial. Microneurosurgery in this patient group is unknown. Even complaints of paresthesia in the maxilla are reported that may be due to larger Le Fort advancements causing delayed infraorbital re-innervation and/or larger defects in the lateral wall of the sinus associated with chronic sinusitis. Most patients will tolerate mild paresthesia for the benefit of better breathing.

Typical patients that present for MMA are middle aged and, therefore, often have unique dental considerations, such as periodontal disease, missing teeth, impacted teeth, implants, and special prosthodontic factors. These factors may prevent presurgical orthodontics but also complicate treatment. Dental implants and extensive bridges do not behave like independent teeth with physiologic movement (Figure 64-11). Sometimes, malocclusion can be corrected by equilibration, new restorations, or even simple



FIGURE 64-11. Postoperative malocclusion of the posterior dentition due to a deep curve of Spee and an extensive prosthetic bridge.

extractions. Periodontal disease, bone loss, and calculus can often be the cause of surgical wound infections. The geniotomy incision is the most likely to dehisce and become infected, perhaps owing to age-related periodontal factors.

Skeletal relapse is a common problem in all surgery, but in orthognathic surgery, this is often corrected by the orthodontist. Up to 20% relapse is reported in orthognathic surgery, which would be only 1 mm if the skeletal change was 5 mm. But in MMA surgery, the skeletal change is often 10 mm and a 20% relapse would be 2 mm, which may be beyond the envelope of treatment in orthodontics or equilibration. Larger skeletal changes imply larger bone gaps and less bone contact. Larger bone gaps also imply longer bone plates with greater flexibility. Therefore, the surgeon must use techniques that reduce relapse such as bone grafting and reinforced plating materials.

Hardware failure and plate fracture are not uncommon in skeletal surgery (Figure 64-12). Routine fixation systems are not designed for MMA surgery, and thin flexible plates used in larger bone defects will bend and flex, ultimately leading to stress fractures. This can be worsened by the surgeon bending and work-hardening the plate to fit an osteotomy gap. Large maxillary advancements anatomically do not have good bone apposition, and chronic movement with mastication will cause delayed healing, nonunion, or stress fracture of the plating system. Prebent OSA plates are stronger in the gap area and control the amount of advancement. Prebent OSA plates do not require the surgeon to bend the plate to fit the osteotomy step and are, therefore, stronger. Bone grafting the lateral wall of the maxilla is advisable owing to the limited bone contact and prevention of soft tissue ingrowth. This will improve stability, increase healing, and reduce relapse.[90]

Mandibular fixation of the sagittal split osteotomy can be done in several different ways. Bicortical position screws are probably the strongest, but when they fail, the entire construct is lost. Bicortical screws may compress the inferior alveolar nerve, and if rotation of the segments occurs, there will be poor adaptation of the proximal and distal segments. Monocortical plates are perhaps kinder to the TMJ and nerve but more flexible. Stronger, stiffer alloys are available that improve the flexibility of the plate. Mandibular fixation in OSA patients usually requires a modification because the advancement is greater, there is less bone apposition, and the patients are stronger and larger. Double monocortical plate per side or superior bicortical position screws with a monocortical plate will increase the engineering support (Figures 64-13 and 64-14). Fixation of the osteotomy is ultimately the surgeon's choice based on multiple factors.

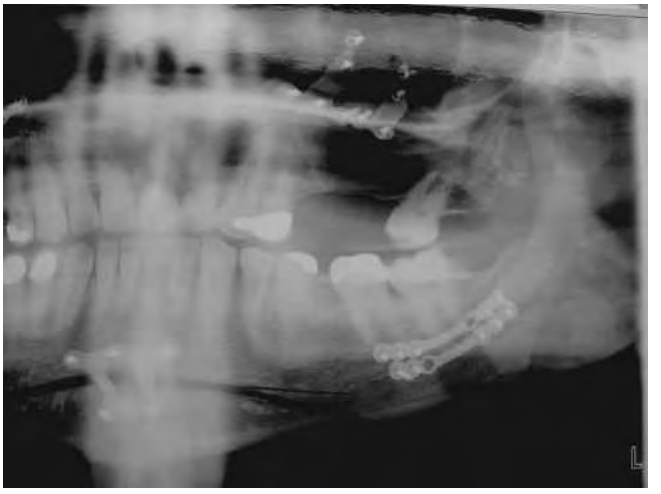


FIGURE 64-13. Double monocortical fixation increases strength in large advancements.

SUMMARY

Because the OSA syndrome is a complex disorder, the type of treatment selected should be tailored to the individual patient based on the relative risks and benefits of the therapy and the

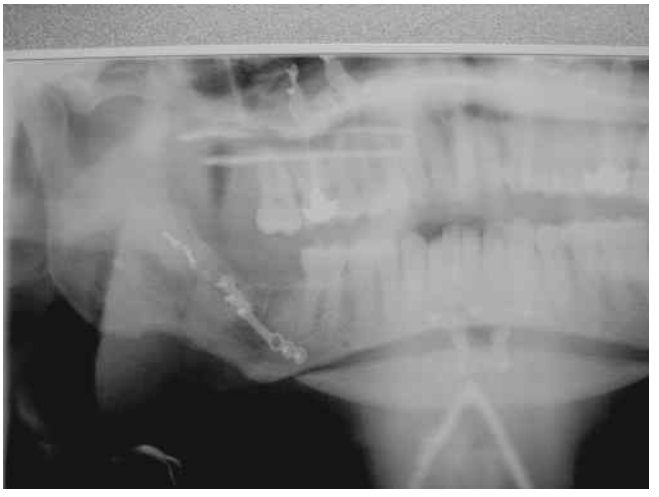


FIGURE 64-12. Plate fracture in the early postoperative period.



FIGURE 64-14. Combination of both monocortical and bicortical fixation screws.

TABLE 64-2. **Results of Maxillomandibular Advancement for Obstructive Sleep Apnea***

Results	RDI	Desaturation†	Patients (*N*)	Percent of Total
Excellent	≤10	0	20	28.2
Good	≤10	≤20	26	36.6
Satisfactory	≤10	>20	15	21.1
Poor	>20	>20	10	14.1

RDI = respiratory disturbance index.
*Maxillomandibular advancement surgery results for 71 obstructive sleep apnea syndrome patients classified by polysomnography.
†Number of oxygen desaturations < 90%.

severity of the disease. Although a subset of the patients who present with OSA has an identifiable craniofacial anomaly, care must be used in choosing a simple mechanistic therapy. The success of the chosen therapy should be evaluated both subjectively and objectively. There is no clear agreement on what constitutes a cure of sleep apnea. Most authors use the RDI in assessing severity of disease and success of treatment. However, all agree that the potentially significant physiologic consequences that can be life-threatening result from hypoxemia. In some cases, patients after treatment have no oxygen desaturations below 90%, but in terms of RDI, they are considered not cured and are deemed treatment failures.

A more reasonable approach would be to define the concept of success in terms of "excellent," "good" "fair," and "poor" and to avoid using the term "cured" in assessing treatment outcomes. These terms could be quantitatively approached assigning lowest oxygen desaturation and RDI parameters to each one. In Table 64-2, the results of 71 patients treated by MMA are assessed by these criteria. In managing patients with severe sleep apnea, a "cure" is seldom achieved with a single surgical or medical treatment (tracheostomies excluded). However, MMA may significantly improve a patient to the point that nonsurgical therapies are more efficacious, if needed at all.

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SECTION 8

Facial Aesthetic Surgery

65

CHAPTER

Blepharoplasty

Tirbod Fattahi, DDS, MD, FACS

INTRODUCTION

Blepharoplasty, commonly referred to "eyelid lift," is a widely accepted and utilized aesthetic surgical procedure. According to a recent survey, blepharoplasty of upper and lower lids was the third most commonly performed cosmetic procedure in United States in 2008, trailing body liposuction and breast enhancement.[1] Contributing to its popularity is its relative ease of performance, ability to be done in an office setting with minimal anesthesia, and lack of a prolonged recovery period

The history of blepharoplasty to correct "lid disorders" dates all the way back to the first century A.D. in Rome when Aulus Cornelius Celsus described excision of eyelid skin.[2] Since then, multiple refinements and modifications have been proposed.

Eyes are the "windows to the soul"; attractive eyes are one of the main features of an aesthetic and balanced face (Figure 65-1).[3] In order to achieve an excellent result, blepharoplasty of upper and lower lids demands a clear understanding of the appropriate preoperative diagnosis, regional anatomy, surgical execution, and management of any postoperative complications. Perhaps more important than the aforementioned concepts, it is paramount to remember that any cosmetic surgical procedure requires an appreciation of what the patient wants. Discrepancies between what the practitioner wants to treat and what the patient wants to have treated can lead to suboptimal results. Often times, patients will present to the clinician's office with a specific complaint regarding the appearance of the periorbital region. Based on the vast amount of information available on the internet, most patients can be quite focused about their concerns. Considering the complex anatomy of the periorbital region, the clinician must formulate a clear diagnosis and treatment plan that corresponds to the patient's concerns. For example, a patient may not be satisfied with the appearance of the upper eyelids and complain that she has difficulty applying makeup to the upper lids simply because they are so "sagging." This patient may present for what she perceives as an eyelid issue. Often times, however, these patients suffer from brow ptosis and actually need brow elevation in order to address the "sagging" eyelids (Figure 65-2). Therefore, proper preoperative communication and treatment planning is absolutely necessary in order to obtain satisfactory results and a "happy" patient. The purpose of this chapter is to familiarize the reader with commonly used nomenclature, surgical anatomy, patient selection, surgical execution, and potential complications associated with upper and lower lid blepharoplasty.



FIGURE 65-1. Open, attractive, youthful eyes. (From McCurry S. National Geographic, June 1985; 167: cover photo.)

NOMENCLATURE

As is common with many aesthetic procedures, multiple "phrases" and "terms" are used to describe the appearance of the upper and lower lids. This can sometimes lead to confusion for the novice surgeon. A list of commonly used diagnoses follows:



FIGURE 65-2. Patient complaining of "heavy eyelids." Examination revealed brow ptosis in addition to dermatochalasia of eye lid skin.



FIGURE 65-4. Patient with congenital ptosis of the upper lids and down-slanting of the lateral canthal angle.

- **Dermatochalasia:** This is a nonspecific term implying excess skin; this is applicable to any part of the facial skin; typically due to a weak bony foundation (hypoplastic infraorbital rim in case of the lower lids), aging process, exposure to sunlight, genetics, and environmental causes (Figure 65-3).
- **Blepharochalasia:** This is a more specific term implying an inflammatory component to redundant skin of the lids. This is often related to angioedema and episodic swelling and edema of the periorbital region.
- **Blepharoptosis:** This acquired or congenital condition relates to drooping of upper lids. This is usually secondary to disinsertion of the levator palpebral superioris aponeurosis (LPS) from the upper tarsal plate (Figure 65-4). This condition is different from brow ptosis, which often causes excess upper lid skin. Blepharoptosis can occur simultaneously with dermatochalasis and blepharochalasis of lids and requires repair of LPS dysfunction.
- **Pseudoptosis:** This term should not be confused with blepharoptosis; this "ptosis," albeit not a true ptosis, is due to the appearance of "drooping" upper eyelid skin without any levator dysfunction. This is simply excessive, heavy lid skin causing a "drooping appearance." There is no indication for ptosis surgery in these patients.
- **Prolapsed fat pads:** Prominent lower lid fat pads or the medial fat pad of the upper lid can present as a "bulge" secondary to weakening of the orbital septum. The term "prolapsed" is more accurate than "herniated" because the septum is still intact, albeit weak, as opposed to a herniation in which there is a distinct opening through which fat bulges (Figure 65-5).
- **Hypertrophic orbicularis oculi:** This is a distinct and hypertrophic portion of the pretarsal component of the orbicularis oculi in the lower lid. Although this can be considered aesthetic in many individuals, in some patients, this condition may exacerbate the "bulge" in the lower lid.
- **Prolapsed lacrimal gland:** This infrequent occurrence is due to the weakness of the septum of the upper lid, causing a unilateral or bilateral fullness in the lateral aspect of the upper lids due to the descent of the lacrimal gland.
- **Lower lid malposition:** This general term applies to any degree of abnormality associated with the lower lid. This could encompass frank ectropion, entropion, lower lid rounding or shortness, and lateral canthal dystopia (Figure 65-6).

ANATOMY

Surgical anatomy of the eye lids is rather complex.[4–10] There are numerous structures in both upper and lower lids with similar functions, but different names (Figure 65-7). Also, it



FIGURE 65-5. Patient with prolapsed fat pads of the lower lids and dermatochalasia of all four lids.



FIGURE 65-3. Patient with dermatochalasia of the eyelids and normal brow position.

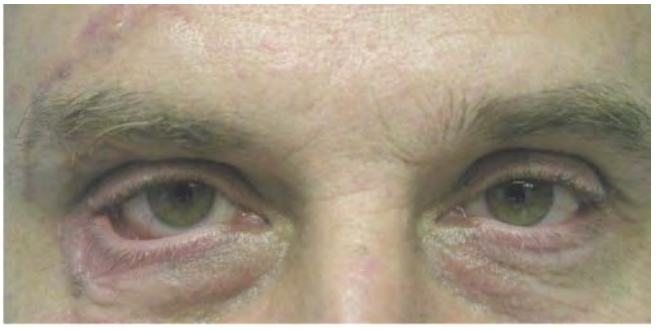


FIGURE 65-6. Right side lower eyelid rounding and frank ectropion.

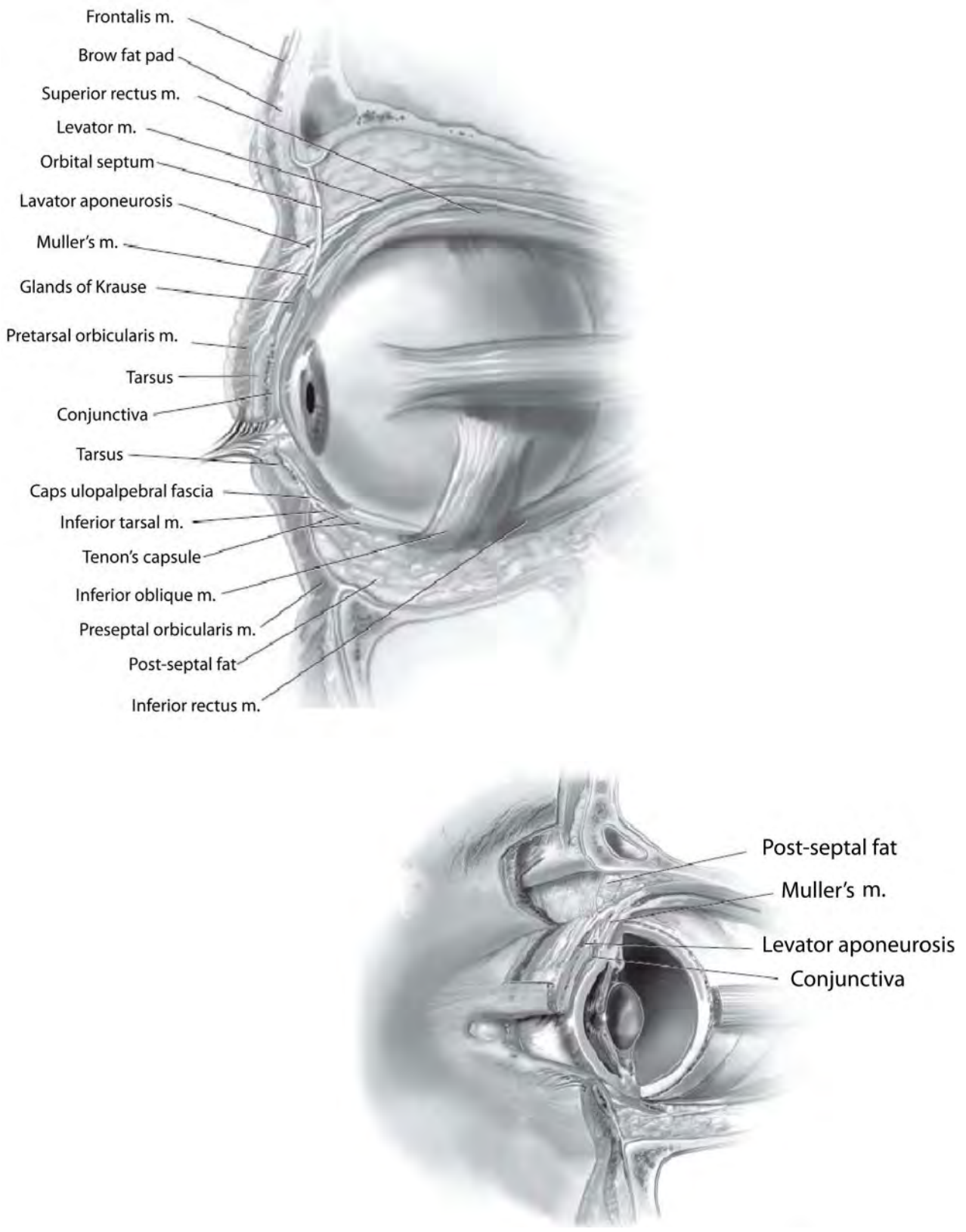


FIGURE 65-7. Anatomic details for upper and lower eye lids. (From Fattahi T. Blepharoplasty. In Fonseca RJ, ed. Oral and Maxillofacial Surgery, 2nd ed. St. Louis, Mo.: Elsevier; 2009: 579–594.)

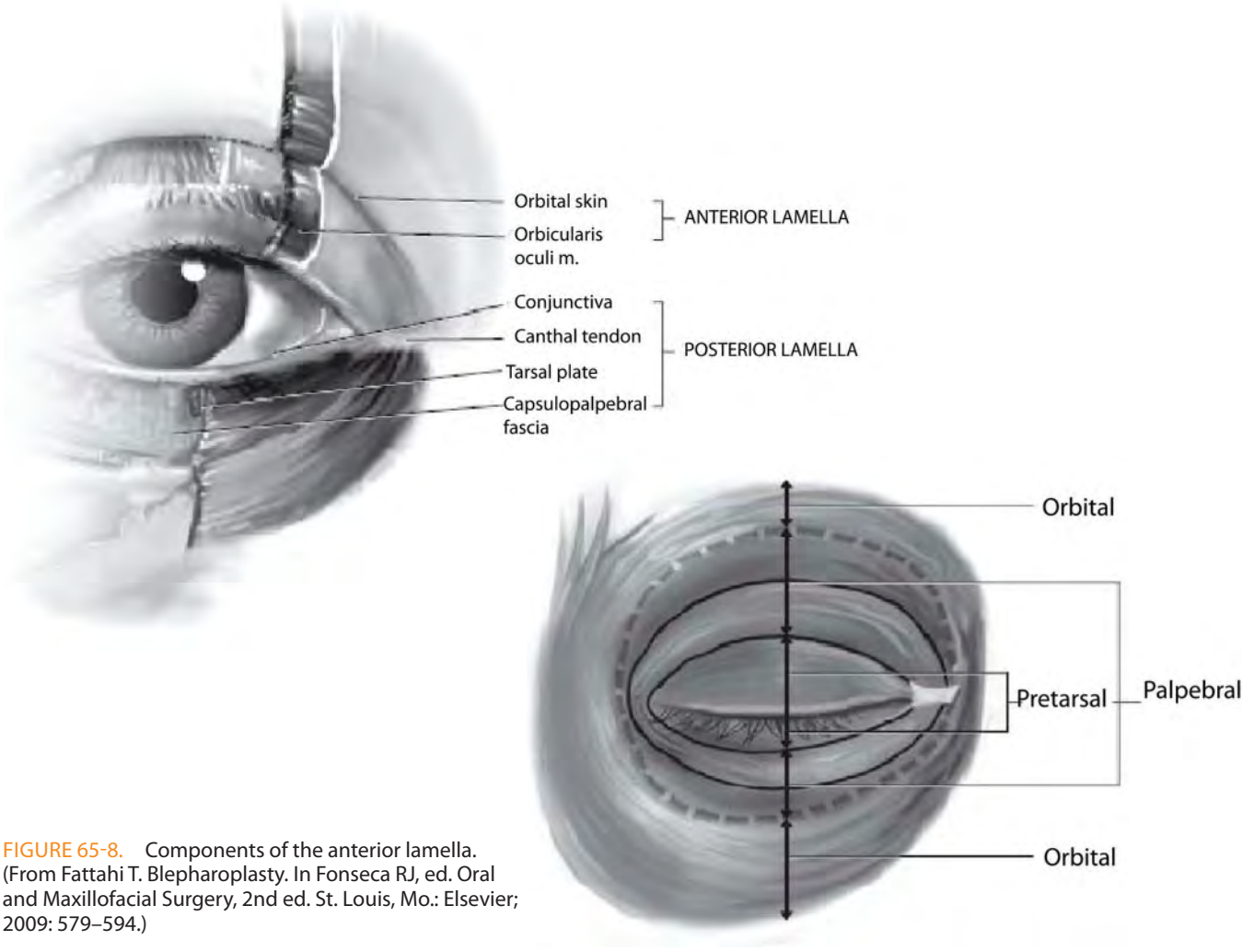


FIGURE 65-8. Components of the anterior lamella. (From Fattahi T. Blepharoplasty. In Fonseca RJ, ed. Oral and Maxillofacial Surgery, 2nd ed. St. Louis, Mo.: Elsevier; 2009: 579–594.)

is imperative to remember that, at times, surgical alterations of the lid anatomy may involve alteration of the brow or midface structures as well. Therefore, an understanding of the surgical anatomy of those particular areas is also helpful.

The most consistent method toward understanding the surgical anatomy of the upper and lower lids is to divide each lid into three lamellae: anterior, middle, and posterior. Each lamella has several components, which are discussed individually.

- Anterior lamella: skin, orbicularis oculi
- Middle lamella: orbital septum, orbital fat pads
- Posterior lamella: lid retractors, suspensory system, tarsus, conjunctiva

Anterior Lamella

The anterior lamella includes the eyelid skin and the orbicularis oculi muscle (Figure 65-8). Upper and lower lid skins are the thinnest skin on the body; the average thickness of the epidermis complex in the adult lid is about 130 μm.[11] This thinness can be considered both an attribute and a disadvantage to the surgeon; although the lid skin is amenable to massaging after surgery if necessary, it is also quite susceptible to scarring if handled improperly during surgery.

A clinically significant region of the skin of the upper lid is the supratarsal crease. This crease (two distinct creases in many patients) is the point of attachment of the upper septum to the aponeurosis of the levator palpebral superioris muscle. This crease is usually found approximately 8 to 10 mm cephalad to the upper lid margin. There are no well-defined creases in the lower lid in most patients; however, with age and exposure to the environment, lower lid creases/rhytids begin to develop just inferior to the lashes. These creases run in a medial to lateral direction and can be utilized for placement of incision for a transcutaneous lower lid blepharoplasty.

The orbicularis oculi is a circular skeletal muscle encompassing the lids and adjacent tissues. The muscle is innervated by the facial nerve (seventh cranial nerve) and has two major components: palpebral and orbital. The palpebral portion is further subdivided into pretarsal and preseptal. The orbital portion originates from the superomedial and inferomedial orbital rim, the maxillary process of the frontal bone, the frontal process of maxilla, and the medial canthal tendon. The fibers sweep across the eyelids, onto the forehead and the cheek regions, respectively. The orbital portion covers the corrugator

supercilii muscle. This portion of the orbicularis oculi is rarely encountered during blepharoplasty. Only involuntary movements are associated with the orbital portion of the muscle.

The preseptal and pretarsal components of the palpebral portion of the muscle have voluntary and involuntary movements. The pretarsal portion is firmly attached to the tarsal plates; the preseptal component is anchored medially to the medial canthal tendon and laterally attach to the lateral canthal region. The palpebral components of the orbicularis oculi are responsible for the blink reflex and moving the marginal tear film toward the lacrimal puncta.

Middle Lamella

The middle lamella comprises the orbital septum and the orbital fat pads (Figure 65-9). The septum is essentially a fascial membrane separating the eyelids from the deeper orbital contents. The septum maintains the orbital fat pads on its posterior surface. In both lids, the septum originates from the arcus marginalis, the confluence of the periorbital periosteum and the periosteum of the facial bones. In the upper lid, the septum attaches to the levator aponeurosis, approximately 2 to 5 mm above the superior edge of the tarsal plate; this fusion represents the clinically significant supratarsal crease. This crease may not be present in approximately 50% of the Asian population. In this group of patients, the septum attaches directly to the superior aspect of the upper tarsus (Figure 65-10). In the lower lid, the septum attaches directly on the inferior aspect of the tarsal plate. The orbital septa of the lower and upper lids actually meet each other medially just deep to the orbicularis oculi muscle around the posterior lacrimal crest. Laterally, the septum fuses with the lateral canthal tendon region.

Just deep to the septum, one finds the preaponeurotic orbital fat pads and the lacrimal gland (Figure 65-11). In the upper lids, there are two fat pads: nasal and middle. The lacrimal gland occupies the space laterally. These two fat pads separate the septum from the underlying levator aponeurosis. The tendon of the superior oblique muscle separates the central fat from the nasal fat compartment. In the lower lids, there are three fat pads: medial, central and temporal. The medial fat pad is the most vascular; this is of clinical significance during fat removal in blepharoplasty in order to prevent postoperative retrobulbar bleed. The inferior oblique muscle separates the medial from the central fat pad. The temporal fat pad may have more than one component.

Posterior Lamella

Lid retractors, suspensory system, tarsus, and the conjunctiva make up the posterior lamella of the lids (Figure 65-12). Lid retractors oppose the action of the orbicularis oculi. In the upper lid, the retractors include the levator aponeurosis (fascial condensation of the levator palpebral aponeurosis [LPS]), and Müller's muscle. LPS is a parasympathetically innervated muscle (cranial nerve III); from the lesser wing of the sphenoid bone, it travels superior to the superior rectus muscle and becomes aponeurotic as it attaches to the lower two thirds of the anterior surface of the upper tarsal plate. Müller's muscle is a sympathetically innervated (superior cervical chain) muscle originating from the inner surface of the LPS and inserting on the superior edge of the upper tarsal plate. Both muscles are elevators of the upper lid. In the lower lid, the lid retractors include the capsulopalpebral fascia and a lesser defined muscle known as the

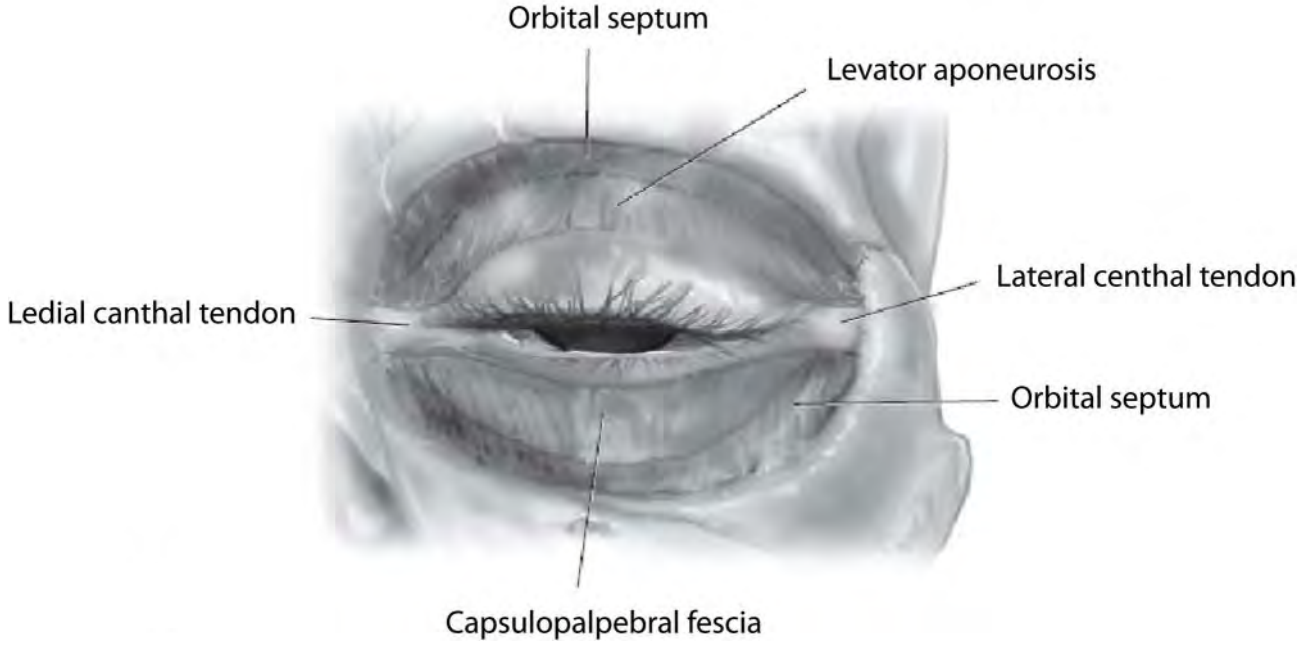


FIGURE 65-9. Components of the middle lamella. (From Fattahi T. Blepharoplasty. In Fonseca RJ, ed. Oral and Maxillofacial Surgery, 2nd ed. St. Louis, Mo.: Elsevier; 2009: 579–594.)

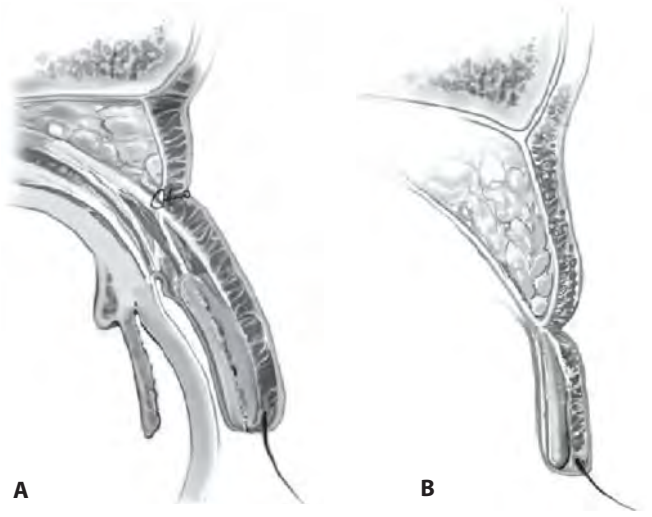


FIGURE 65-10. Differences in the attachment of levator aponeurosis in Asians **(A)** and non-Asians **(B).** (**A** and **B,** From Fattahi T. Blepharoplasty. In Fonseca RJ, ed. Oral and Maxillofacial Surgery, 2nd ed. St. Louis, Mo.: Elsevier; 2009: 579–594.)

inferior tarsal or *palpebral muscle,* also known as *Horner's muscle.* The capsulopalpebral fascia is an extension of the inferior rectus muscle attaching to the inferior edge of the lower tarsal plate. Horner's muscle is a portion of the deep head of the orbicularis oculi muscle. These two lower lid retractors are analogous to the levator aponeurosis of the LPS and Müller's muscle of the upper lid, respectively.

The two hammock-like suspensory systems of the upper and lower lids include Whitnall's ligament and Lockwood's ligament, respectively. They are also known as the *superior* and *inferior transverse suspensory ligaments.* They prevent the globe from craniocaudal movement. Whitnall's ligament is a fascial condensation originating from the trochlea and attaching to the orbital lobe of the lacrimal gland. This ligament is found just superior to the LPS, but deep to the septum. Lockwood's suspensory ligament is a fascial condensation of Tenon's capsule (bulbar fascia) running between the medial orbital walls and attaching to the lateral retinaculum. Lockwood's suspensory system is just anterior to the capsulopalpebral fascia.

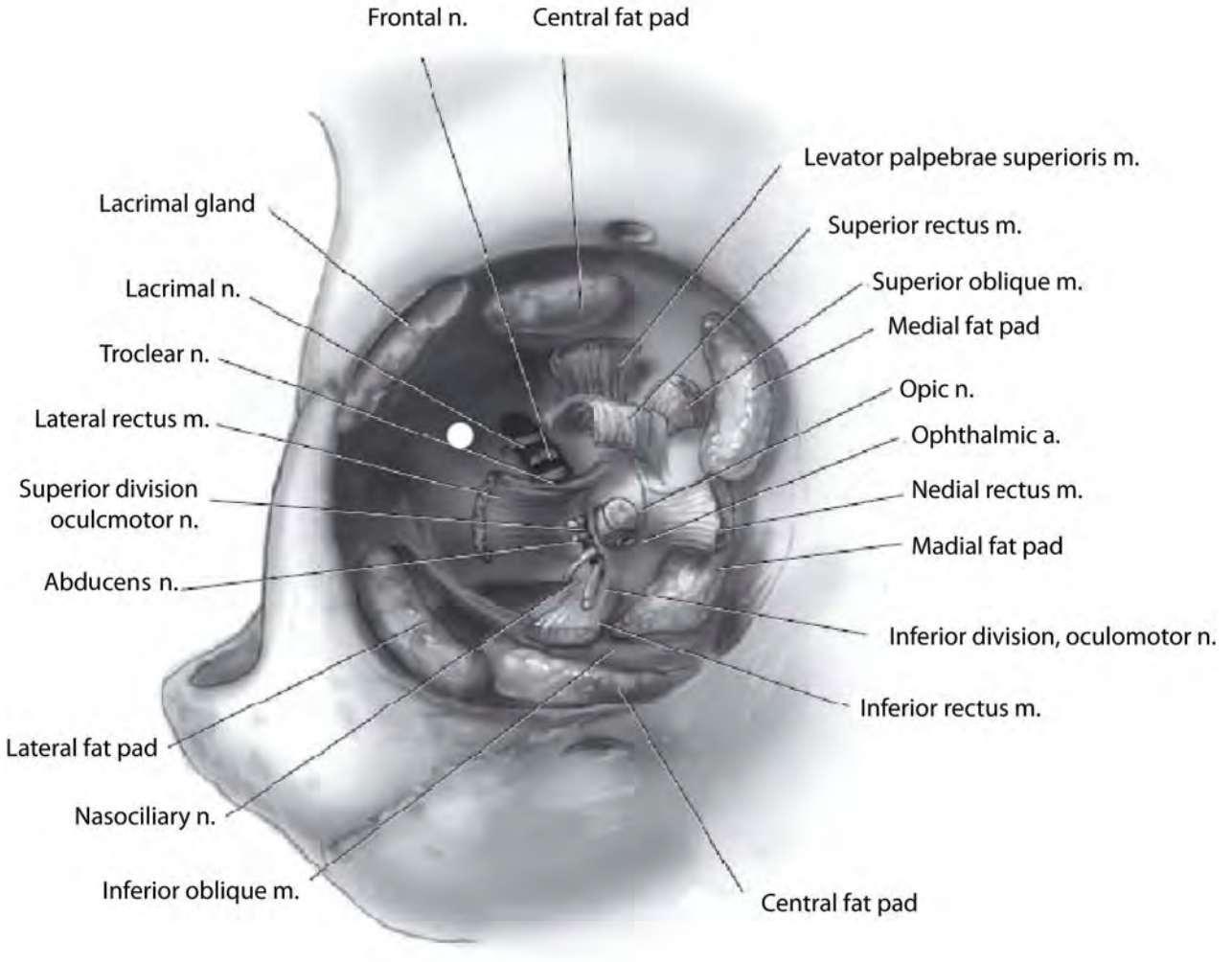


FIGURE 65-11. Anatomy of the right eye with septum removed. (From Fattahi T. Blepharoplasty. In Fonseca RJ, ed. Oral and Maxillofacial Surgery, 2nd ed. St. Louis, Mo.: Elsevier; 2009: 579–594.)

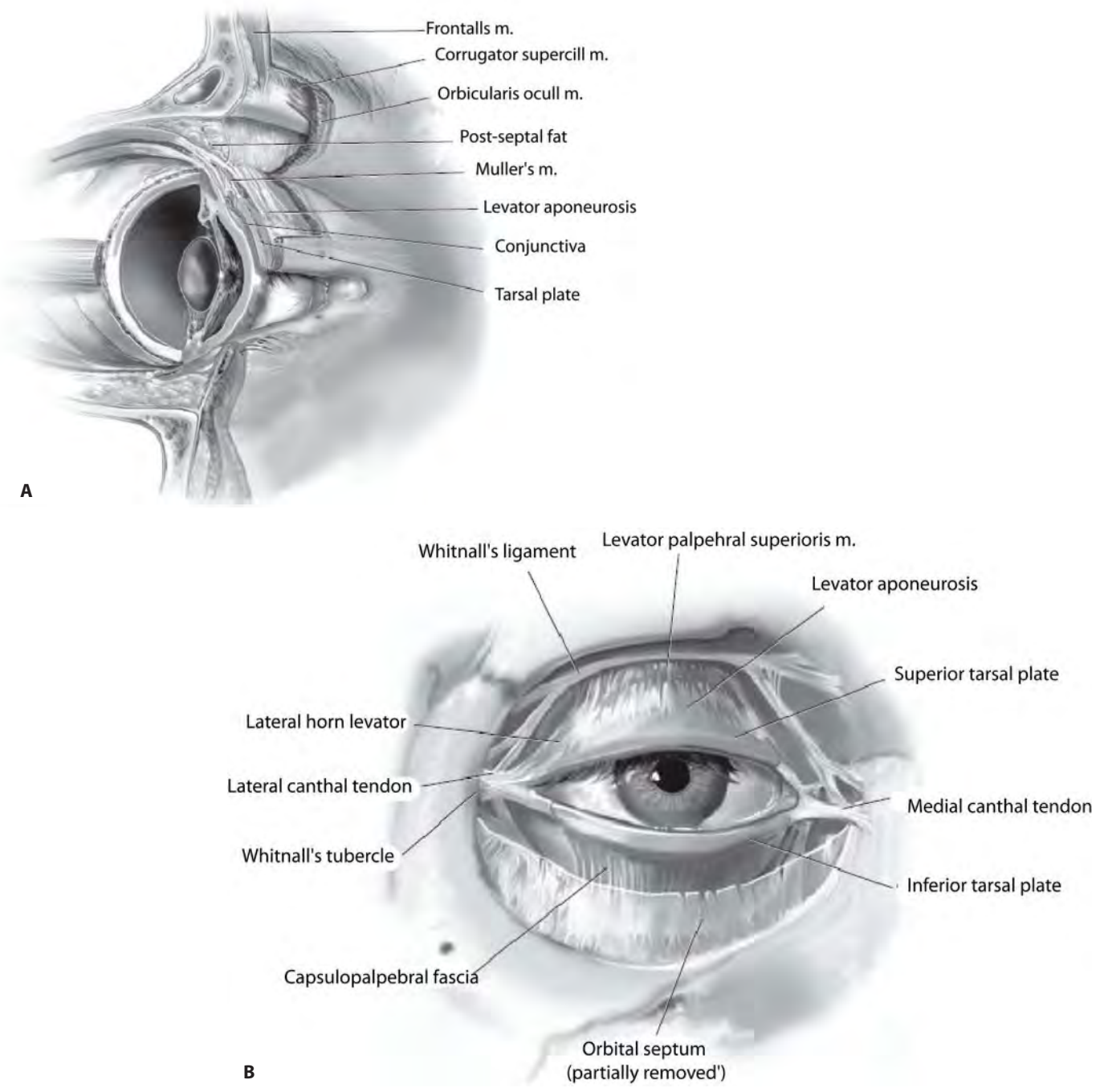


FIGURE 65-12. **A** and **B,** Components of the posterior lamella (**A** and **B,** From Fattahi T. Blepharoplasty. In Fonseca RJ, ed. Oral and Maxillofacial Surgery, 2nd ed. St. Louis, Mo.: Elsevier; 2009: 579–594.)

Tarsal plates provide the main skeletal support to the upper and lower lids. They are made of dense fibroelastic tissue lined with meibomian glands. In the lower lid, the tarsal plate is only 4 to 6 mm in height; in the upper lid, the plate is 8 to 10 mm in height.

Conjunctiva makes up the last component of the posterior lamella. It lines the most inner aspect of the lids (palpebral conjunctiva), forms the fornix, and then turns onto the globe and covers the anteriormost aspect of the cornea (bulbar conjunctiva).

Although not specifically components of the posterior lamella, the lateral and medial canthi play a significant role in the transverse and spatial relationship of the lower and upper lids. Lateral and medial canthi are lateral and medial fibrous extensions of the tarsal plates. Both canthi have two heads: superficial and deep (anterior and posterior). The anterior limb of the medial canthus and the posterior limb of the lateral canthus are thicker than their counterparts. The two limbs of the medial canthal tendon cover the lacrimal sac; the two limbs of the lateral canthal tendon cover Eisler's fat pad.

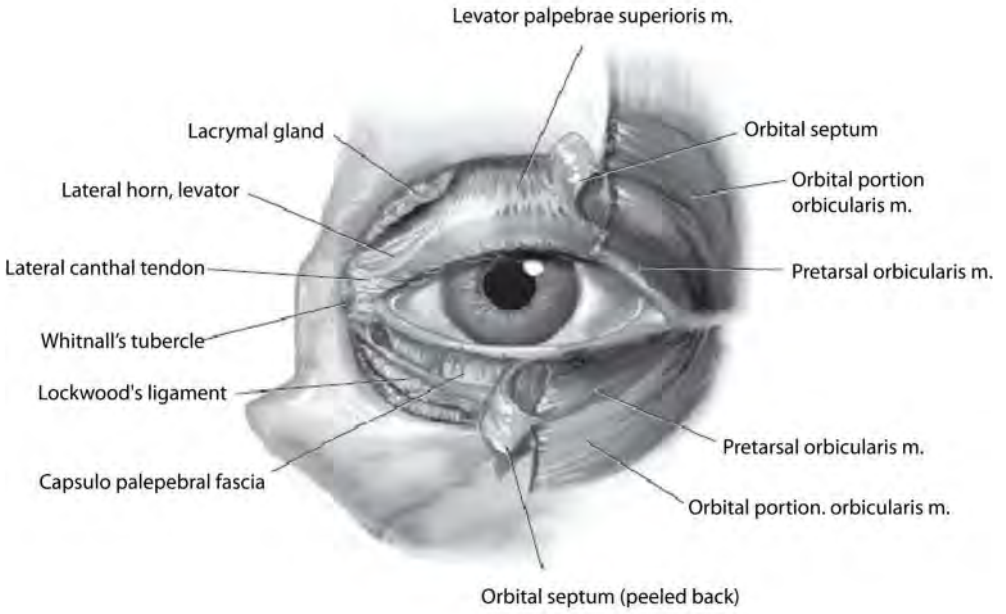


FIGURE 65-13. Components of the lateral retinaculum. (From Fattahi T. Blepharoplasty. In Fonseca RJ, ed. Oral and Maxillofacial Surgery, 2nd ed. St. Louis, Mo.: Elsevier; 2009: 579–594.)

The lateral retinaculum (LR) is a clinically significant structure in the lateral orbital rim (Figure 65-13).[12–14] LR is essentially a labyrinth of connective tissue anchored within the lateral orbital rim that maintains the position, integrity, and function of the globe. It is attached to Whitnall's tubercle, a bony protuberance 10 mm inferior to the zygomaticofrontal suture and 3 mm posterior (deep) to lateral orbital wall rim within the zygomatic bone. The following structures attach to the LR:

- Lateral canthal tendon (posterior limb).
- Lateral horn of LPS aponeurosis.
- Lockwood's ligament of the lower lid.
- Check ligament of lateral rectus muscle.

Innervation and Blood Supply

Sensory innervation of the eye lids is provided by the fifth cranial nerve (trigeminal); specifically, the first and second divisions (ophthalmic and maxillary). The lacrimal nerve travels in a superior and lateral direction providing sensation to the lacrimal gland, upper lid, and brow region. The frontal nerve divides into the supraorbital (centrally) and supratrochlear (medially) to provide sensation to the upper lid, midbrow, forehead, scalp, and nasal bridge. The nasociliary nerve gives off branches to provide sensation to the globe. The infraorbital nerve emerges from its foramen to provide sensation to the lower lid, midcheek, and upper lip and anterior maxillary teeth. The zygomatic nerve divides into the zygomaticotemporal and zygomaticofacial nerve to provide sensation to the temple and lateral brow and lateral aspect of lower lid and cheek respectively.

The blood supply to the eyelids is provided by the internal and external carotid systems. The ophthalmic artery, facial artery, and infraorbital artery are the main arteries in this region. The ophthalmic artery is a branch of the internal carotid system and has multiple branches (supraorbital, supratrochlear, ethmoidal, marginal, and external nasal). The facial artery becomes the angular artery in the medial canthal region and anastomoses with the branches of the ophthalmic artery medial to the lacrimal sac. The infraorbital artery emerges from the maxillary artery and supplies the lower lid region. The marginal and peripheral cascades (anastomoses between the internal and the external carotid systems) within the lid proper are responsible for the blood supply to the lashes and tarsus (marginal) and the conjunctiva and lid muscles (peripheral).

The venous system mirrors that of the arterial system, although deep anastomoses with the pterygoid plexus of veins and the superior ophthalmic vein exist that may lead to a retrograde infection.

Lacrimal System

The lacrimal system begins with the lacrimal gland sitting in the lacrimal fossa. Tears are produced in the gland and then travel in a lateral to medial direction toward the lacrimal puncta by the blink reflex. Accessory lacrimal glands within the upper and lower fornices are known as *glands of Kraus* and *Wolfring*. Tears enter the upper and lower canaliculi, approximately 8 to 10 mm away from the lacrimal sac. Both canaliculi have a 2-mm vertical component before making a 90-degree turn to form the common canaliculus. The common canaliculis runs deep to the pretarsal orbicularis oculi to empty into the nasolacrimal duct, which then travels into the inferior meatus approximately 15 mm from the nasal floor.

PATIENT EVALUATION

Perhaps the most significant component of any aesthetic surgery is a thorough understanding of the patient's needs and desires. The surgeon must recognize the patient's complaint and then determine whether the patient's expectations are realistic. Patients seeking blepharoplasty usually complain of "bags under the eyes," "excess skin in the upper lids," "always looking tired," "deceased vision laterally," and "inability to apply makeup to the upper lids."[15–17] It is important to establish the exact reason a patient is seeking to have surgery. This is facilitated by giving the patient a hand mirror and asking him or her to point to the areas of concern.

Once the chief complaint has been elicited, a complete medical history must be obtained. Medical conditions such as thyroid disease, heart disease, liver dysfunction, bleeding disorders, and seasonal allergy and edema must be ruled out. Many of these conditions may interfere with the proper diagnosis and surgical execution of blepharoplasty. It is particularly important to determine whether a patient has had LASIK (laser-assisted in situ keratomileusis) surgery in the past 6 months because the conjunctiva can become anesthetic after this procedure and mask symptoms associated with dry eyes.

Once the history has been completed, a step-by-step approach must be undertaken to evaluate the lids. With all aesthetic procedures, one must first gauge an overall view of the *entire* face before focusing on the specific area of concern. This overall image often provides a tremendous amount of information about the aging process that can then be relayed to the patient during the evaluation of the face.

After this overall assessment, the clinician must evaluate the position of the brows and forehead. If a patient appears to have dermatochalasia of the upper lids, one must ascertain that the dermatochalasia is not due to brow ptosis. Performing an upper blepharoplasty in the presence of brow ptosis will only cause further descent of the brow and lead to an unaesthetic result. If the brow appears ptotic, the patient may need to have a brow elevation (e.g., endoscopic, open, browpexy) *before* upper blepharoplasty.[18] There are specific measurements for an ideal brow for males and females. Generally, there are three parts to an eyebrow: medial, apex, and tail. In a male, all three regions should be aligned with the supraorbital rim. A masculine brow is one with little elevation in its apex or tail. Conversely, a female brow should have a slight elevation medially (0–2 mm compared with the underlying supraorbital rim), elevate to 10 mm at the apex (compared with the underlying supraorbital rim), and then slightly descend toward the tail aspect. A complete evaluation of the brow and forehead is described in Chapter 68.

Next, attention should be directed toward the upper lids. Determination of the presence or absence of a supratarsal crease must be made. Typically, the supratarsal crease is approximately 9 to 11 mm above the lid margin. This can be measured with a ruler while elevating the brow with one hand and asking the patient to look downward. If the supratarsal crease is significantly greater than 9 to 11 mm, one must consider the possibility of levator disinsertion from the septum. With the brow kept in its normal position, excess upper lid skin must be pinched between two fingers to determine the amount of dermatochalasis. This can be subjectively graded from 0 to 3, with 3 being significant hooding in which excess lid skin is sitting on top of the lashes to 0 being no redundant tissue. Assessment of the upper lid fat pads must also be done. Usually, one finds prolapsed medial fat pad in the upper lid, especially in an older patient. Another important consideration is the presence of lid ptosis (blepharoptosis).[7,19] This can be determined in two ways. The first method is to measure the palpebral fissure. This is the distance between the central aspect of the upper lid and the central aspect of the lower lid with the patient gazing in a primary position. This width should be almost 10 mm bilaterally. A significant decrease in palpebral fissure width should raise the suspicion for upper lid ptosis. The other method is the margin reflex distance-1 (MRD-1). This is the distance between the light reflex in the patient's cornea and the central upper lid margin with the patient's eyes in the primary position of gaze. The upper lid usually covers 2 mm of the superior limbus of the iris (Figure 65-14). The normal MRD-1 is approximately 4 to 5 mm; a smaller number may indicate upper lid ptosis. The MRD-1 is more accurate than the

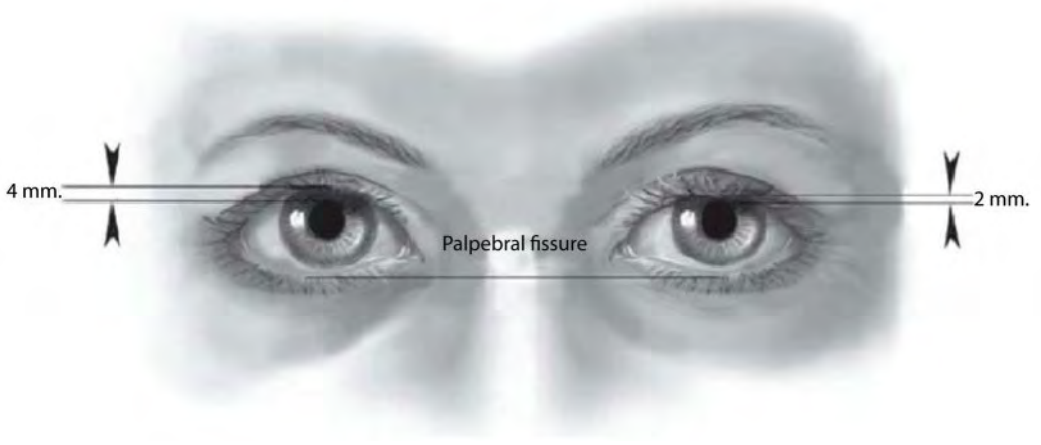


FIGURE 65-14. Normal margin reflex distance-1 (MRD-1) of the right eye and abnormal MRD-1 of the left eye denote left upper eyelid ptosis.



FIGURE 65-15. Asking the patient to look up will accentuate the borders of the lower eyelid fat prolapse.

palpebral fissure width because the latter can be altered by lower lid malposition (rounding, ectropion).

After the upper lid, focus should be directed at the lower lids. The presence of the fine rhytids just inferior to the lashes should be noted. Any excess skin in the lower lid subunit should be assessed. One can usually find prolapsed fat pads along the lower lids. The presence of prolapsed lower lid fat pads can be assessed by asking the patient to look upward or by gently pushing on the upper lids (while the eye is closed) to determine any lower lid "bulges" (Figure 65-15). A hypertrophic orbicularis oculi muscle can be differentiated from a lower lid prolapsed fat pad by asking the patient to smile; animation of the muscles of facial expression emphasizes hypertrophy of the orbicularis oculi muscle. Lower lid retraction can be assessed by measuring the distance from the inferior limbus to the central portion of the lower lid. Usually, the lower eye lid is at the level of the inferior limbus. Another method is the MRD-2, the distance from the light reflex of the cornea to the central aspect of the lower eyelid with the patient's eyes in primary gaze. This distance should be approximately 5 to 5.5 mm.[20] This distance is increased in lower lid retraction.

Lower lid laxity should also be determined by the snap and distraction test (Figure 65-16). The snap test is performed by pulling the lower lid downward and then releasing it to determine how quickly the lid "snaps" back into place. This should occur in less than 1 second or with one or two blinks. The lid distraction test is performed by pulling the lid away from the globe in an anterior and inferior direction. If the lid is distracted more than 7 mm, the lid is considered abnormally loose. If the snap or distraction tests are positive, the surgeon should consider a lid-tightening procedure at the time of blepharoplasty in order to prevent postoperative lid malposition.

Consideration should also be given to the midface region. If there is evidence of midface ptosis (e.g., prominent nasolabial fold, nasojugal fold), the patient should be advised about other indicated procedures such as midface lift or fat repositioning.

Other components of the physical examination should include a visual acuity examination. If there is diminished vision due to excessive hooding of the upper lids or to other intrinsic globe issues, a formal visual field test by an ophthalmologist or optometrist should be done. Many times, insurance companies will require such a test before authorizing upper blepharoplasty. Extraocular movements should be recorded preoperatively to ensure full and unrestricted movement of each periorbital muscle. Bell's phenomenon should also be assessed to prevent corneal exposure and desiccation in case of a residual lagophthalmos after surgery. The patient is asked to close her or his eyes tightly; the clinician then opens the lid to assess position of the cornea. A normal Bell's phenomenon will roll the globe and cornea in an upward position upon forceful closure of the lids (Figure 65-17).

If the patient has reported symptoms of dry eyes, a Schirmer test should be done to evaluate tear secretions. After drying the inferior fornix of the lower lid, a Schirmer strip is bent and inserted in the lateral aspect of the lower fornix. Room lights may need to be dimmed to prevent reflex





FIGURE 65-16. Snap and distraction test. **A,** In the snap test, the lower eyelid is pulled inferiorly to assess how quickly it resumes its normal position next to the globe. **B,** In the distraction test, the lid is pulled in an anterior direction to assess its laxity.



FIGURE 65-17. Normal Bell's phenomenon. Note the upward rotation of the iris as the lids are forcefully opened.



FIGURE 65-18. Fine forceps grasps the excess upper lid skin until the lashes evert.

blinking. A normal Schirmer test is 15 mm of wetness on the strip after 5 minutes. Five to 10 mm of wetness suggests hyposecretion, whereas less than 5 mm is highly indicative of dry eyes. If the Schirmer test is abnormal, further investigation is warranted. These patients will definitely require a very "conservative" blepharoplasty and may need frequent eye lubricants postoperatively.

A complete set of photographs is an absolute necessity in any aesthetic surgery. For blepharoplasty, frontal repose, frontal animated (smile), three quarters, profile, and a photograph with the patient looking in an upward gaze are sufficient.

SURGICAL PROCEDURES

Upper lid blepharoplasty is begun by marking the patient in the preoperative area.[21–24] The patient should be in a sitting position facing the surgeon. Most patients have a supratarsal crease. This crease can be used as the inferior aspect of the incision if the crease is in a normal position. The brow is elevated with the nondominant hand. A fine-tipped marker is used to outline the crease. The incision is marked just above the superior punctum and extends laterally and fades into one of the crow's feet. It is important to maintain the incision within the eyelid subunit and not extend too far laterally. The upper portion of the incision is determined by pinching the excess upper lid skin by a fine blunt forceps until the eyelashes began to evert (Figure 65-18). This position is marked at its highest position. This will serve at the superior incision once it is connected medially and laterally to the inferior incision. The basic shape of the incision is nearly an ellipse or oval (Figure 65-19). Although a skin/muscle flap can be elevated simultaneously, the easiest method of flap elevation is a skin-only flap. This is done quite easily after administration of local anesthetic with a vasoconstrictor for hemostasis purposes. The skin flap is incised and undermined by iris scissors. Hemostasis is obtained by electrocautery. At this point, hypertrophic or ptotic orbicularis oculi can be excised cautiously. This will further expose the orbital septum, which will need to be incised if preaponeurotic fat pads (usually the nasal fat pad) need to be removed. A transverse incision through the orbital septum will expose the underlying medial and central upper lid fat pads (Figure 65-20). The prolapsed portion of the fat pads are then grasped with hemostats, and cut with scissors; electrocautery is applied to the cut edges of fat pads to ensure complete hemostasis before releasing the clamp and allowing the fat pads to retract posteriorly. Closure is not done until both sides are performed to ensure equal amount of skin, muscle, and fat resection. A slight lagophthlmos (<1–2 mm) is desirable at this point (Figure 65-21). This resolves within a few days. Skin closure is with 6-0 sutures. If a supratarsal crease does not exist, a skin-muscle flap can be elevated simultaneously. The inferior edge of the incision in this situation would be approximately 9 to 10 mm above the lashes. A skin-muscle flap will allow creation of a new supratarsal crease by attaching the levator aponeurosis directly to the inferior edge of the incision.

For lower lid blepharoplasty, the first decision to be made is whether a transconjunctival or transcutaneous blepharo-



FIGURE 65-19. Typical markings for upper eyelid blepharoplasty.



FIGURE 65-20. Exposure of the nasal fat pad after incising the septum.

plasty is to be performed. If the patient has prolapsed lower fat without excess skin, a transconjunctival lower lid blepharoplasty is indicated. Of note, it is also possible to "reposition" the fat in the nasojugal area via a transconjunctival approach if needed. If however, excess skin is present in the lower lid, the surgeon has three choices: (1) a transcutaneous lower lid blepharoplasty with removal of fat and skin, (2) a transconjunctival lower lid blepharoplasty to remove the fat with a skin-tightening procedure at the same time or later time (i.e., chemical peel, CO_2 laser resurfacing). and (3) a lower eyelid "pinch" blepharoplasty.[25–33] This decision must be made preoperatively and discussed with the patient at length. If a transcutaneous lower lid blepharoplasty is considered, a lid-tightening procedure must be strongly considered at the same time to prevent postoperative lid malposition, especially if the patient has preoperative lid laxity (i.e., positive snap or distraction tests, previous lower lid blepharoplasty).

For transconjunctival lower lid blepharoplasty, the patient is marked in the preoperative area by gently pushing on the globe or asking the patient to look up and determining the areas of prolapsed fat. These regions are marked with a marking pen. A transconjunctival lower lid blepharoplasty is performed by gentle retraction of the globe with a globe retractor, followed by incising the palpebral conjunctiva and



FIGURE 65-21. Slight lagophthalmos (metallic eye shields in place) after upper eyelid blepharoplasty

capsulopalpebral fascia with electrocautery (Figure 65-22). The incision is approximately 5 to 6 mm inferior to the lid margin. This will allow easy access into the three lower lid fat pads without disrupting the orbital septum. With gentle pressure on the globe, fat is easily protruded into the surgical field.[34] Each prolapsed fat pad is clamped with a hemostat, excess fat is trimmed, and electrocautery is used to cauterize the clamp before release to ensure hemostasis. It is prudent to ensure that no muscle is attached to the fat before resection. Both sides are then evaluated simultaneously to ensure equal removal of fat. The incision is closed with resorbable , and if a skin-tightening procedure is to be done at the same time, the surgeon proceeds as appropriate.

A transcutaneous lower lid blepharoplasty is initiated with skin markings for the ideal incision. Preoperative marking for the location of prolapsed fat pads is done as described previously. The ideal location of a transcutaneous lower lid blepharoplasty incision is within 3 to 4 mm of the lower lashes (Figure 65-23). The incision is started medially just below the punctum and extends laterally. At the lateral canthal region, the incision should rise, following the upward curve of the lower lid, and then gently descend and blend in with one of the crow's feet. A skin-only flap is first elevated. Once the skin flap is retracted inferiorly, a muscle flap is blunted dissected and elevated in a steplike fashion. This step should decrease the incidence of lower lid retraction because the skin incision and muscle incision will not be at the same location. Once the muscle has been elevated, the orbital septum can be clearly seen. A transverse incision is made through the orbital septum, exposing the underlying fat pads. Each fat pad is approached individually. Excess fat is clamped and removed and hemostasis obtained as previously described. Once the fat has been removed, attention is directed toward the lateral aspect of the incision where the lateralmost portion of the preseptal orbicularis muscle flap (off the inferior aspect of the incision) is suspended to the lateral retinaculum with a 4-0 suture. This pexing of the muscle to the thick fascial condensation of the lateral orbital wall will significantly reduce the risk of lower lid malposition postoperatively.[35] Once this has been done, the inferior skin flap is grasped at its lateral edge and pulled cephalad; excess skin is then removed with scissors. Next, the muscle flap is closed, followed by closure of the skin incision using 6-0 sutures.

A "pinch" lower eyelid blepharoplasty is indicated in patients with no fat prolapse but with excess eye lid skin. This procedure begins with "pinching" the excess skin with fine forceps in a cephalad direction toward the lashes; then the "rolled" lower eyelid skin is simply excised with the incision being placed 3 to 4 mm below the lashes. There is no muscle or fat excision in this maneuver (Figure 65-24). Closure of the skin incision is performed using 6-0 sutures.

POSTOPERATIVE CARE

Because upper and lower lid blepharoplasty is considered a "clean" surgical wound, perioperative antibiotic coverage for

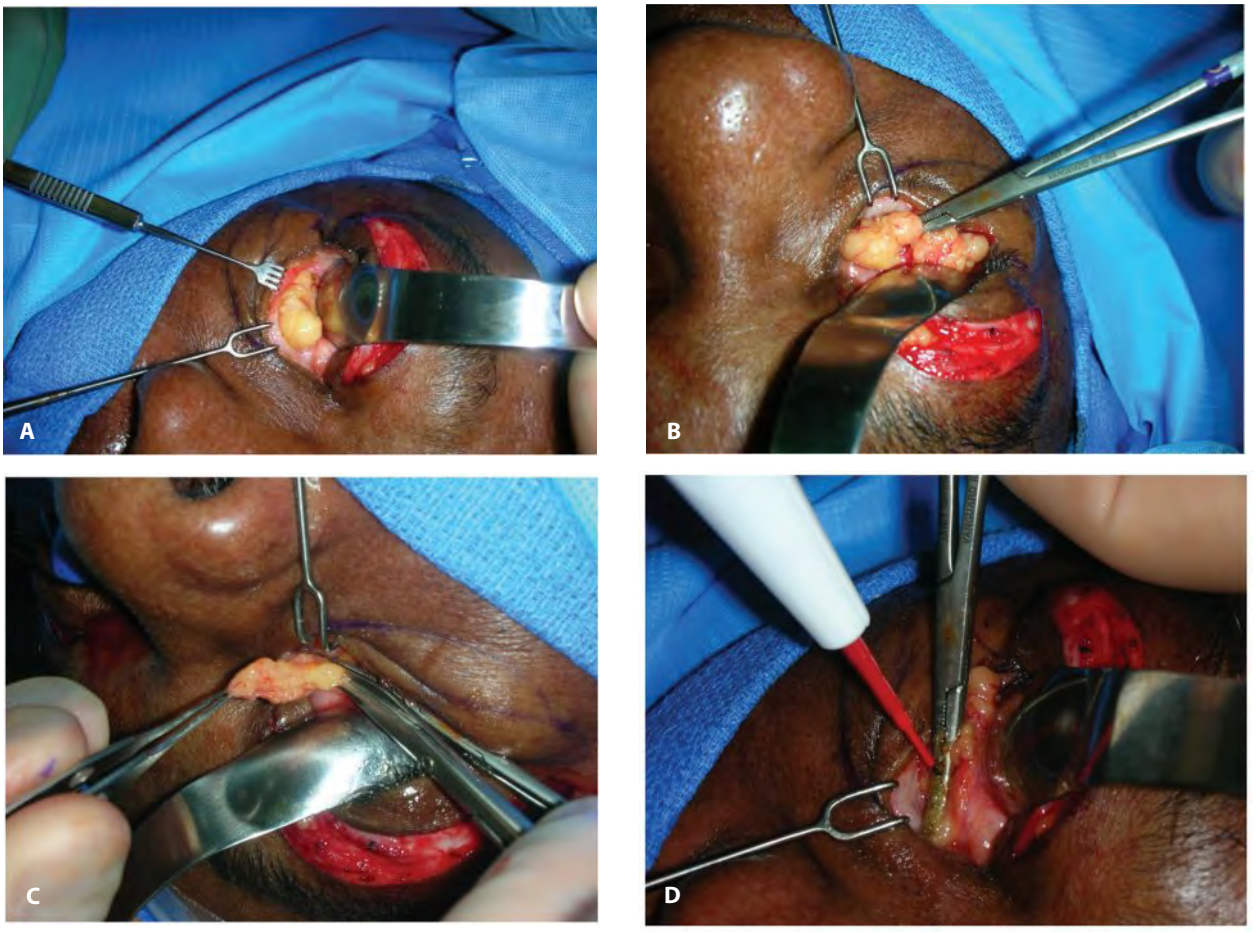


FIGURE 65-22. Lower eye lid transconjunctival blepharoplasty. **A,** Exposure of the fat pads. **B,** Clamp is applied at the base of each fat pad. **C,** Scissors are used to cut the excess fat. **D,** Electrocautery is utilized on the clamped fat pad for hemostasis before releasing the fat pad.

greater than 24 hours is not recommended. All patients should have ice cold compresses applied to the surgical site in the recovery room and for the first 48 hours postoperatively. No contact lenses should be worn for the first few days after surgery. Patients are instructed to avoid exercise and heavy lifting for the first 2 weeks postoperatively to decrease the chance of capillary tears and bruising.



FIGURE 65-23. Placement of lower eyelid transcutaneous blepharoplasty. The incision is approximately 4 mm inferior to the lower lashes.

Ophthalmic steroid eyedrops may be used to decrease chemosis. All nonresorbable sutures are removed in 5 days. Steri-Strips can be used to support the incision if necessary (Figure 65-25).

COMPLICATIONS AND MANAGEMENT

There are minor and major complications associated with blepharoplasty.[2,7,36–39] Fortunately, the major problems are rare. Minor issues include chemosis, bruising, subconjunctival hemorrhage, and blurred vision. All of these issues are transient and will resolve within the first 7 to 14 days after surgery.

More major complications include those associated with aesthetics, lid position, and function of the eyes. Aesthetic complications involve persistence of orbital fat pads, excess or redundant skin, and asymmetry between the two sides. This can be prevented by precise preoperative assessment. The lateral fat pad of the lower lid is usually the most difficult to access. Most underresections are associated with this particular pad. Persistent excess skin postoperatively can be prevented by measuring the patient while the patient is sitting. When the patient lies supine, the brows raise;

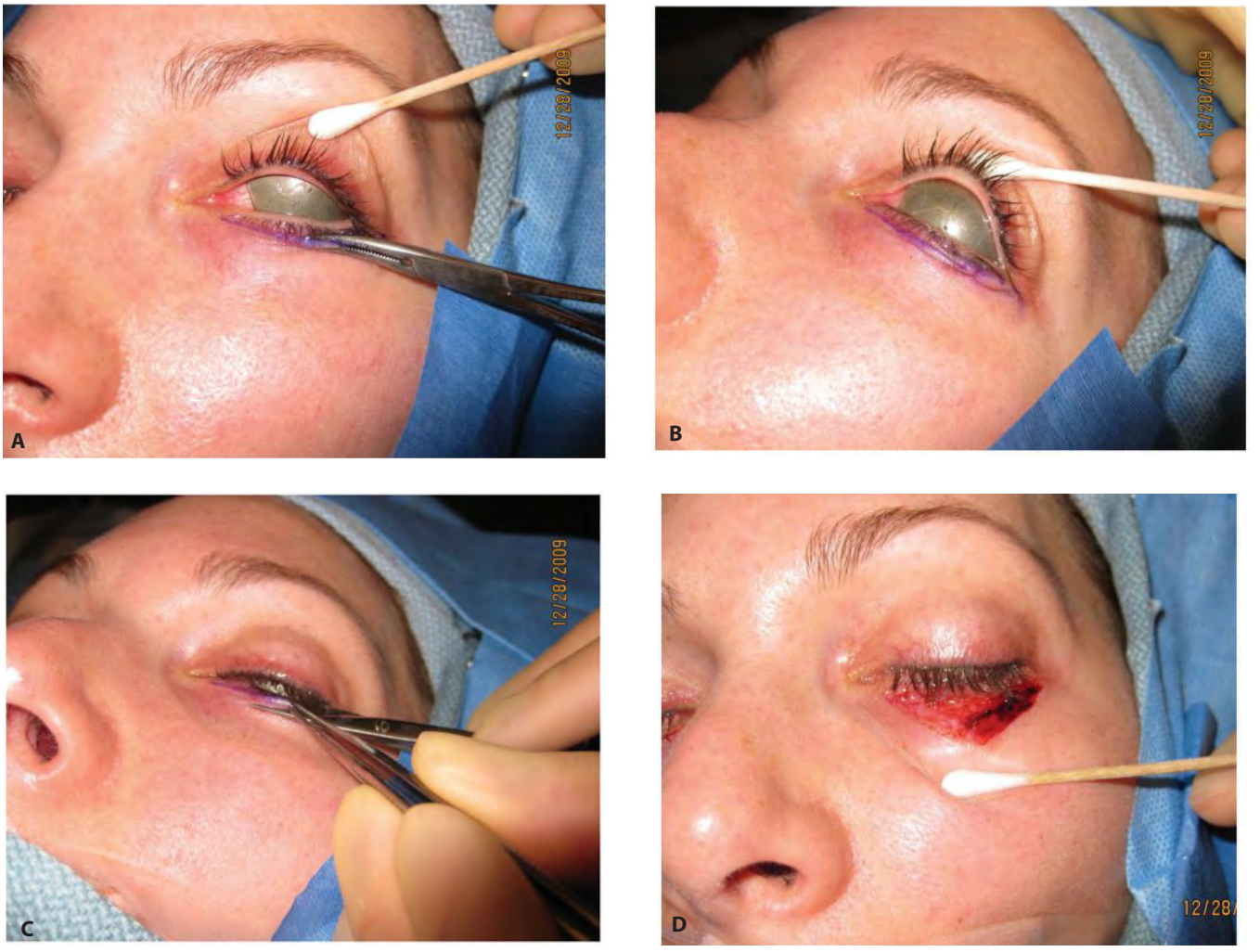


FIGURE 65-24. Pinch blepharoplasty. **A,** Excess skin is pinched with forceps. **B,** Appearance of pinched skin before excision. **C,** Excision of skin only. **D,** Underlying orbicularis oculi muscle. Note amount of skin resection

this can remove some of the dermatochalasia and lead to underremoval of excess skin. Asymmetries usually occur owing to unequal removal of fat and skin from both sides. All fat and excised skin should be kept and labeled throughout the procedure to ensure equal amount of resection (assuming there were no asymmetries preoperatively). Asymmetries can also be due to the position of the supratarsal crease or incision. It is imperative to measure the distance between the lashes and the crease (incision) if any doubt exists regarding potential asymmetries between two sides.

Lid malposition can occur if excess skin was removed or if there was lid laxity preoperatively, or both. After a conservative transcutaneous lower lid blepharoplasty, a minor rounding (<1 mm) of the lower lid can occur without any adverse sequela. This is usually amenable to massage. However, if the rounding is greater, or if there is frank ectro-



FIGURE 65-25. Typical appearance after blepharoplasty.

pion, this will need surgical repair in most cases. Although massage and taping of the lids can help, most patients will need a lid-tightening procedure, mucosal graft for posterior lamella lengthening, tarsorraphy, or skin graft for anterior lamella lengthening. This is the main reason the author advocates pexing of the preseptal orbicularis muscle to the lateral retinaculum in every transcutaneous lower lid blepharoplasty.

Perhaps the biggest complications after blepharoplasty are related to functional issues. Dry eyes after blepharoplasty are usually due to a preexisting condition or excessive skin resection leading to a persistent lagophthalmos or both. This requires application of artificial tears, taping of the lids, and massage. Referral to an ophthalmologist or oculoplastic surgeon may be warranted if the condition persists. Diplopia after blepharoplasty can occur if the superior oblique or the inferior oblique muscles are injured during surgery. Care must be taken during fat removal to ensure that all muscle and fascia have been removed from the fat pads before excision. Electrocautery near these muscles can also cause disturbances of vision. Persistent diplopia can be a serious problem and must be referred to an ophthalmologist. Retrobulbar bleeds and blindness are the gravest complications of upper and lower blepharoplasty. Meticulous hemostasis is mandatory during surgery to decrease the chance of postoperative bleeding. Intense, unilateral pain, and progressive proptosis and chemosis are hallmarks of a retrobulbar bleed; this requires emergent attention by an inferior canthotomy and cantholysis to decrease the intraocular pressure and evacuate any clots. If this pressure is allowed to increase, optic nerve ischemia can occur that will cause irreversible visual disturbance. The canthotomy and cantholysis should be kept open for 24 to 48 hours. Once the situation has been resolved, the lateral canthal tendon can be repaired.

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66

CHAPTER

Basic Principles of Rhinoplasty

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For many cosmetic surgeons, rhinoplasty is one of the most challenging surgical procedures. A clear understanding of nasal anatomy is critical in order to provide an aesthetic result that does not compromise nasal function. Developing a pattern of analysis of the nose is vital for proper diagnosis and for determining the most appropriate treatment plan. Numerous rhinoplastic techniques have been described. Some surgeons favor an endonasal approach whereas others believe that an external approach is more desirable. Each surgeon must become familiar with all technique options in order to address the wide variety of challenges of rhinoplasty surgery.

The goal of this chapter is to give a broad overview of the diagnosis and treatment of nasal deformities. It is by no means exhaustive because multiple textbook volumes have been written on this subject. The reader should gain an understanding of nasal anatomy and determine how to systematically analyze the nose. Both endonasal and external rhinoplasty are described.

NASAL ANATOMY

A clear understanding of nasal anatomy is important to successfully perform nasal procedures and decrease the incidence of complications.

Surface Anatomy

The terms used to describe the surface anatomy of the nose are important in nasal form analysis and for treatment plan formulation (Table 66-1). For descriptive purposes, the spatial relationships are described as cephalic, caudal, dorsal, basal, anterior, posterior, superior, and inferior (Figure 66-1).

Skin and Soft Tissue

The soft tissue that overlies the bone and cartilage may influence the final result of rhinoplasty. The thickness of the skin will determine how it will redrape after performing a rhinoplasty. The skin thickness varies along the dorsum of the nose. The skin is fairly thick and mobile in the region of the nasion. It quickly thins over the nasal dorsum and is generally thinnest and most mobile in the middorsal region (rhinion). In the distal third of the nose, the skin tends to be more thick and adherent and has an increased sebaceous content.

A patient with thin skin will show dramatic changes with alteration of the underlying bone and cartilage, and this limits room for error because little is camouflaged by the thickness of the skin. Conversely, for thick-skinned individuals, more aggressive sculpturing of the nasal skeleton must be performed in order to effect significant changes. Although thick skin may mask imperfections, it does not redrape as well and can result in underlying fibrosis and formation of a polybeak deformity (supratip scarring). Better results are possible with thin-skinned patients; however, the margin for

TABLE 66-1. **Surface Anatomy of the Nose**

Glabella: the most forward projecting point of the forehead in the midline at the level of the supraorbital ridges.
Radix: the junction between the frontal bone and the dorsum of the nose.
Rhinion: the anterior tip at the end of the suture of the nasal bones.
Dorsum: the anterior surface of the nose formed by the nasal bones and the upper lateral cartilages.
Supratip break: the slight depression in the nasal profile at the point where the nasal dorsum joins the lobule of the nasal tip.
Infratip lobule: the portion of the tip lobule that is found between the tip-defining points and the columellar-lobular angle.
Tip-defining points: there are four tip defining points: the supratip break, the columellar-lobular angle, and the most projected area on each side of the nasal tip formed by the lower lateral cartilages.
Alar sidewall: the rounded eminence forming the lateral nostril wall.
Alar-facial junction: the depressed groove formed on the face where the ala joins the face.
Columella: the skin that separates the nostrils at the base of the nose.

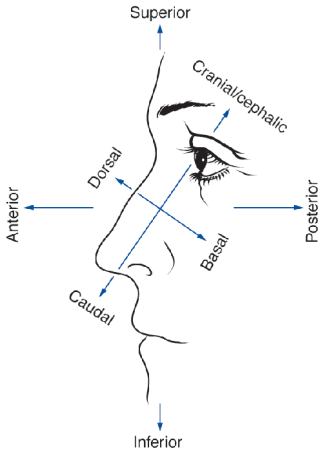


FIGURE 66-1. Spatial descriptors. In describing the relationship of one anatomic unit to another, many terms are used. The standard relationships are anterior, posterior, superior, and inferior. The nose is also described in terms of dorsal, basal, caudal, and cranial (or cephalic) positions. (Adapted from Austermann K. Rhinoplasty: planning techniques and complications. In Booth PW, Hausamen JE, editors. Maxillofacial Surgery. New York: Churchill Livingstone; 1999; p. 1378.)

error is smaller. The surgeon must sometimes modify the technique depending on the type of skin of the patient.

Superficial Musculoaponeurotic System and Nasal Musculature

The muscles of the nose are encased in the nasal superficial musculoaponeurotic system (SMAS). This is a fibromuscular layer that separates the skin and subcutaneous tissue from the nasal cartilage and bone. The SMAS of the nose is in continuity with the SMAS of the face. During rhinoplastic surgery, the dissection is performed beneath the SMAS. Violating the SMAS will often result in increased bleeding, scarring, and postoperative edema.

The muscles of the nose can be divided into four categories: the elevators, the depressors, the compressors, and the dilators (Figure 66-2). The muscles of significance are the paired depressor septi nasi. These muscles can result in drooping of the nasal tip during smiling. This added tension on the nasal tip must be recognized preoperatively and addressed by resection in order to achieve a cosmetic result.[1]

Blood Supply

There is a rich blood supply to the subdermal vascular plexus of the nose that arises from branches of both the internal and the external carotid arteries. The blood supply from the internal carotid artery that supplies the external nose includes the dorsal nasal artery and the external nasal artery. The dorsal nasal artery is a branch of the ophthalmic artery. The external nasal artery is a branch of the anterior ethmoid artery.

The external nose is also supplied by branches of the facial artery and the internal maxillary artery, which originate from the external carotid artery. The facial artery branches include the angular artery, lateral nasal artery, alar artery, septal artery, and superior labial artery (Figure 66-3).

The internal nose is supplied by the internal and external carotid branches. The ophthalmic artery, a branch of the internal carotid, branches into the anterior and posterior

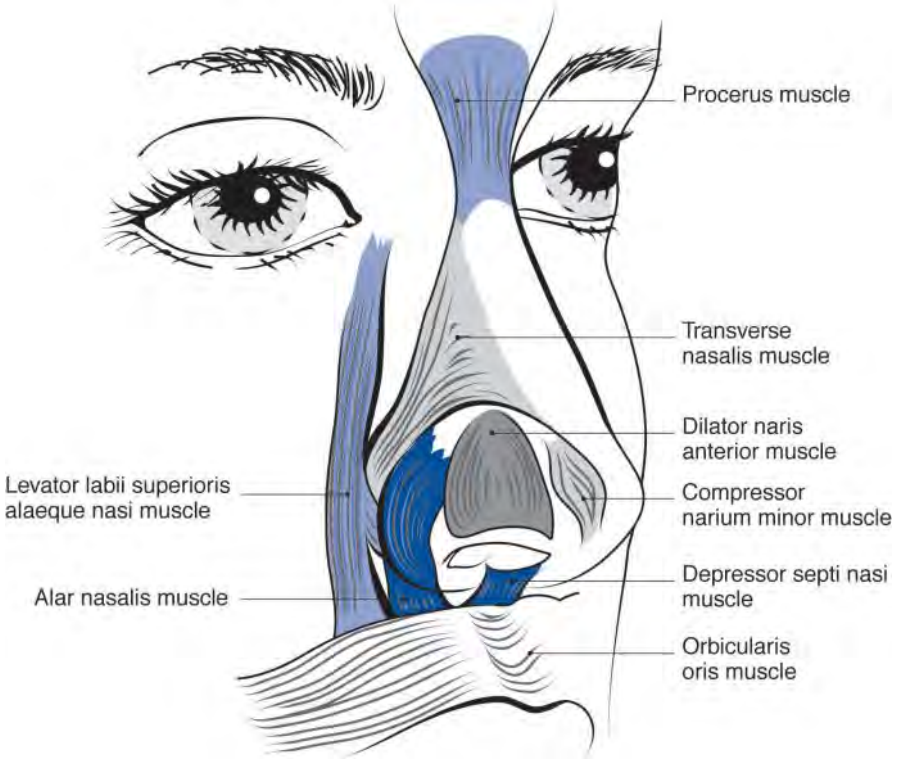


FIGURE 66-2. Nasal musculature. The muscles of the nose are grouped into the elevators *(light blue),* the depressors *(dark blue),* the compressors *(light gray),* and the dilators *(dark gray).* (Adapted from Jewett B. Anatomic considerations. In Baker SR, editor. Principles of Nasal Reconstruction. St. Louis: Mosby; 2002; p. 17.)

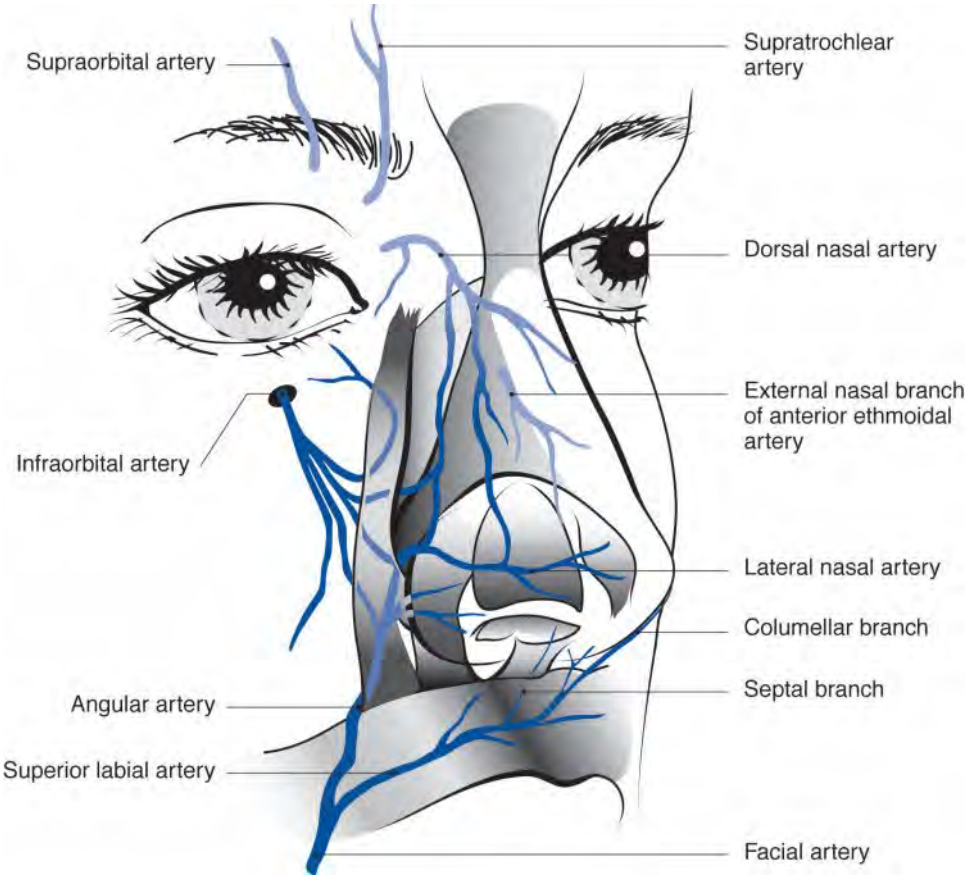


FIGURE 66-3. Arteries of the external nose. The arterial supply of the external nose comes from branches of the external carotid artery *(dark blue)* and the internal carotid artery *(light blue)*. (Adapted from Jewett B. Anatomic considerations. In Baker SR, editor. Principles of Nasal Reconstruction. St. Louis: Mosby; 2002; p. 18.)

ethmoidal arteries. The anterior ethmoidal artery supplies the anterosuperior part of the septum and the lateral nasal wall. The posterior ethmoid artery supplies the septum, lateral nasal wall, and superior turbinate.[2]

The internal maxillary artery branches include the sphenopalatine artery and the greater palatine artery. The sphenopalatine artery supplies most of the posterior part of the nasal septum, lateral wall of the nose, roof, and part of the nasal floor. The greater palatine artery supplies a portion of the anterior and inferior portion of the nasal septum (Figure 66-4).[2]

The surgically significant area for internal nasal bleeding is known as *Kiesselbach's plexus* (also termed *Little's area).* This is the area in the anteroinferior part of the nasal septum that is a common site of epistaxis. It is where the sphenopalatine, greater palatine, superior labial artery, and anterior ethmoid arteries anastamose (Figure 66-5).[2] The venous drainage of the nose is primarily from the facial and ophthalmic veins.

One concern during nasal surgery is the possibility of compromised blood flow to the nasal tip if the surgeon performs an external rhinoplasty. The blood supply to the nasal tip has been analyzed by lymphoscintigraphic studies, cadaver dissections, and histologic sections.[3,4] The conclusion is that the primary blood supply to the nasal tip comes from the bilateral lateral nasal arteries that course in a plane superficial to the alar cartilages in the subdermal plexus approximately 2 to 3 mm above the alar groove. Thus, a columellar incision does not compromise tip blood supply. Also, there are no significant veins and minimal lymphatics in the columellar region.[3,4] Some surgeons believe that external rhinoplasty remains more edematous for longer postoperative periods than an endonasal rhinoplasty.

Bone and Cartilage

The structure of the nose consists of the paired nasal bones as well as the frontal process of the maxilla. The bone is thickest near the junction with the frontal bone and tapers as it joins with the upper lateral cartilages.

The upper lateral cartilages are in intimate contact with the nasal bones and underlie the nasal bones for approximately 6 to 8 mm. The connection between the nasal bones and the upper lateral cartilages should not be violated because this may disrupt the internal nasal valve, causing nasal obstruction and asymmetry. The internal nasal valve is formed by the junction of the upper lateral cartilages and the nasal septum.

The lower lateral cartilages compose the lower third of the nose and connect to the upper lateral cartilages in a union described as the *scroll.* There are various configurations of the scroll.[5,6] The scroll is described as interlocked (52%), overlapping (20%), end to end (17%), or opposed (11%) (Figure 66-6). The scroll provides significant support to the nasal tip. When performing an endonasal rhinoplasty, this

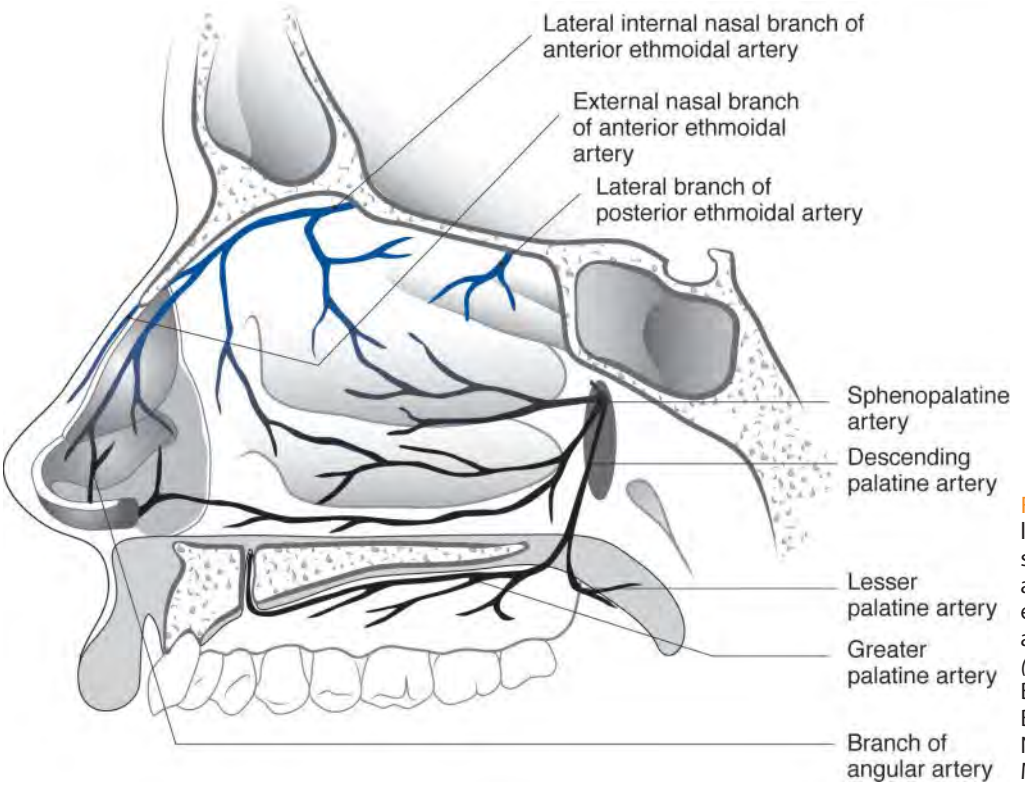


FIGURE 66-4. Arteries of the lateral nasal wall. The arterial supply of the lateral nasal wall arises from branches of the external carotid artery *(black)* and the internal carotid artery *(blue)*. (Adapted from Jewett B. Anatomic considerations. In Baker SR, editor. Principles of Nasal Reconstruction. St. Louis: Mosby; 2002; p. 23.)

area is violated by the intercartilaginous incision (Figures 66-7 to 66-9). The lower lateral cartilage is divided into medial and lateral crura. The medial crura are in intimate contact with the nasal septum and provide tip support. The lateral crura extend superiorly and form dense fibroareolar tissue attachments with the pyriform aperture. The intermediate crus is the diverging of the medial crus before turning to become the lateral crus proper. The highest point of the intermediate crus is an important surgical landmark known as the *tip-defining point* (Figure 66-10).

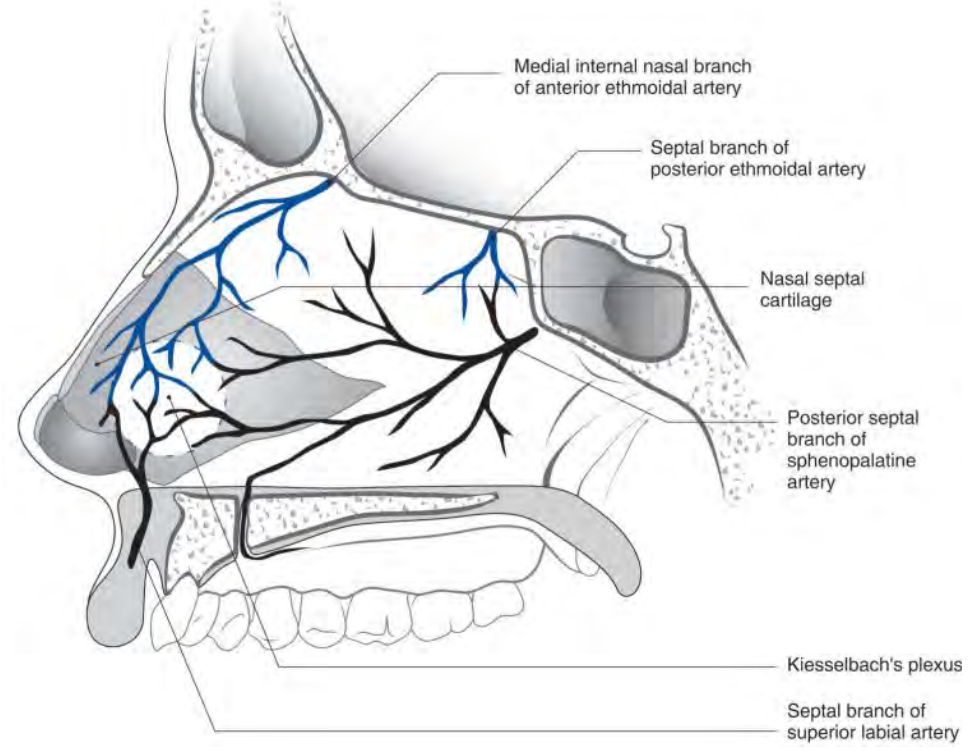


FIGURE 66-5. Arteries of the nasal septum. The arterial supply of the nasal septum arises from branches of the external carotid artery *(black)* and the internal carotid artery *(blue)*. Kiesselbach's plexus is formed by the sphenopalatine artery, greater palatine artery, superior labial artery, and anterior ethmoid arteries. It is a common site of epistaxis. (Adapted from Jewett B. Anatomic considerations. In Baker SR, editor. Principles of Nasal Reconstruction. St. Louis: Mosby; 2002; p. 23.)

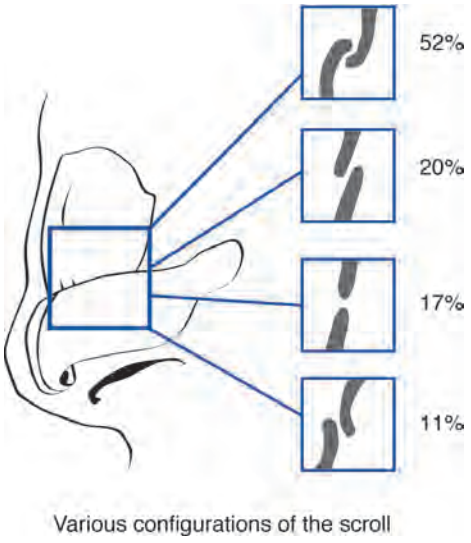


FIGURE 66-6. Configurations of the scroll. The relationship of the upper lateral and lower lateral cartilages is termed the scroll. Anatomic studies have identified four common configurations: interlocked (52%), overlapping (20%), end to end (17%), and opposed (11%). (Adapted from Lam SM, Williams LE. Anatomic considerations in aesthetic rhinoplasty. Facial Plast Surg 2002;18:209–214.)

The nasal septum is formed by both bone and cartilage. The ethmoid and vomer provide bony support posteriorly. The quadrangular cartilage provides support anteriorly (Figure 66-11).

Support for the nasal tip is classified into major and minor divisions. The major tip support comes from the size, shape, and strength of the lower lateral cartilages, the attachment of the medial crura of the lower lateral cartilage to the caudal septum, and the fibrous attachment of the lower lateral cartilage to the upper lateral cartilage. The minor tip support comes from the nasal spine, the membranous septum, the cartilaginous dorsum, the sesamoid complexes, the interdomal ligaments, and the alar attachments to the skin (Table 66-2).[5]



FIGURE 66-7. Partial transfixion. The partial transfixion incision through the membranous septum and short of the medial crural foot pads.



FIGURE 66-8. Intercartilaginous incision. The intercartilaginous incision, between the upper and the lower cartilage, allows access to the nasal dorsum. Note the incision does not violate the nasal valve.

Nerves

The sensory nerve supply to the skin of the external nose is provided by the ophthalmic and maxillary divisions of the trigeminal nerve. Branches of the supratrochlear and infratrochlear nerves supply the skin in the region of the radix and rhinion. The lower half of the nose is supplied by the infraorbital nerve and the external nasal branch of the anterior ethmoidal nerve (a branch of the nasociliary nerve that arises



FIGURE 66-9. Connecting intercartilaginous and partial transfixion incisions. The intercartilaginous incision extends along the upper edge of the lateral crus to connect with the transfixion incision. This will provide access for a septoplasty during internal rhinoplasty.

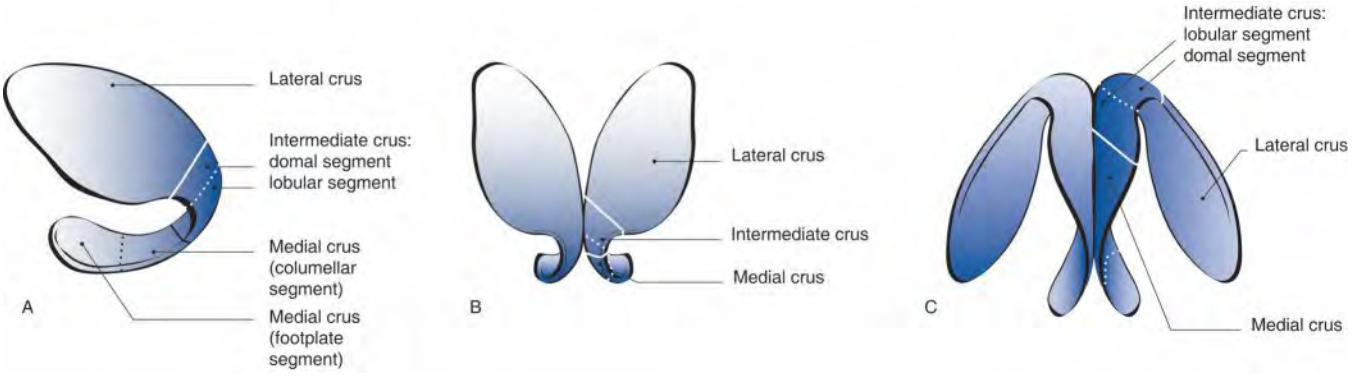


FIGURE 66-10. **A–C,** Anatomy of the lower lateral cartilages. The lower lateral cartilages are often described as having a lateral crus, a medial crus, and an intermediate crus. The intermediate crus is the most projected portion of the lower lateral cartilages and these form two of the tip-defining points seen on nasal tip analysis. **(A–C,** Adapted from Jewett B. Anatomic considerations. In: Baker SR, editor. Principles of Nasal Reconstruction. St. Louis: Mosby; 2002; p. 21.)

from the ophthalmic branch of the trigeminal nerve) (Figure 66-12).

The main sensory nerve supply to the nasal septum comes from the internal nasal nerve (a branch of the anterior ethmoidal nerve) and the nasopalatine nerve (Figure 66-13). The lateral nasal wall sensation is supplied by the anterior ethmoidal nerve, branches of the pterygopalatine ganglion, branches of the greater palatine nerve, the infraorbital nerve, and the anterior superior alveolar nerve.

Parasympathetic innervation is derived from branches of the pterygopalatine ganglion, which are derived from cranial nerve VII. Some sympathetic branches reach the nasal cavity via the nasociliary nerve.[2,7]

Nasal Valve

The airflow through the nose is regulated by the internal and external nasal valves. The external nasal valve comprises the lower lateral cartilage and the nasal septum and floor. Collapse of the external nasal valve can sometimes be noted when the nares become occluded on even gentle inspiration. This problem is seen in patients with narrow nostrils, a projecting nasal tip, and thin alar sidewalls. External nasal valve collapse is usually seen in patients who have had previous rhinoplasty surgery and excessive trimming of the cephalic portion of the lower lateral cartilages. It is also seen with increased age and in facial nerve paralysis. The external nasal valve collapse can be corrected by deprojecting the overprojected nose, realigning the lateral crura into a more caudal orientation, and placing alar batten grafts to provide structural support and prevent collapse.[8]

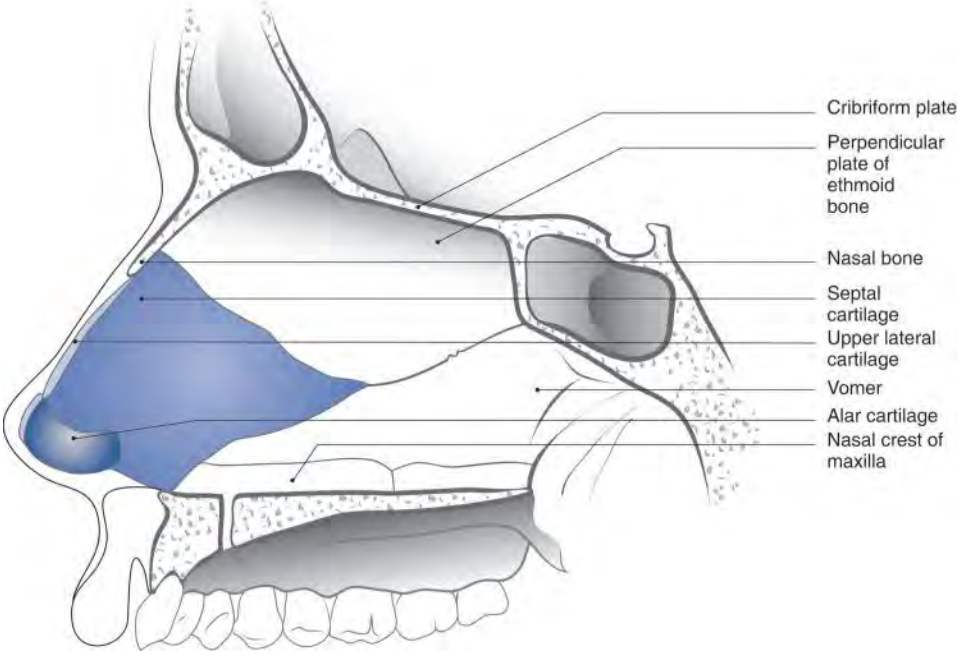


FIGURE 66-11. Anatomy of the nasal septum. The nasal septum is composed of the perpendicular plate of the ethmoid, the vomer, and the quadrangular cartilage. (Adapted from Jewett B. Anatomic considerations. In Baker SR, editor. Principles of Nasal Reconstruction. St. Louis: Mosby; 2002; p. 22.)

TABLE 66-2. **Tip Support Mechanisms**

The three major tip support mechanisms are
1. The size, shape, and strength of the lower lateral cartilages.
2. The attachment of the medial crura to the caudal septum..
3. The attachment of the lower lateral cartilages to the upper lateral cartilages.

The minor tip support mechanisms are

1. The interdomal ligament.
2. The sesamoid complex extending the support of the lateral crura to the piriform aperture.
3. The attachment of the alar cartilages to the overlying skin.
4. Cartilaginous septal dorsum.
5. Nasal spine.
6. The membranous septum.

The internal nasal valve is formed by the junction of the septum with the upper lateral cartilages. The angle formed should be a minimum of 10 to 15 degrees to maintain patency. Deviation of the nasal septum or separation of the upper lateral cartilages from the nasal bones can lead to obstruction. This problem is also seen after rhinoplasty if the patient has had weakening of the upper and lower lateral cartilages. These patients often have a pinched appearance in the supra-alar region. The Cottle test is used to evaluate obstruction at the internal valve by using a finger to distract the check and lateral wall of the nose, thereby opening the valve. If nasal airflow is dramatically improved, then the internal valve may require correction. These patients often have symptomatic relief by the use of external taping devices. Surgical correction involves the placement of spreader grafts between the septum and the upper lateral cartilages to increase the angle at this junction.[8–10]

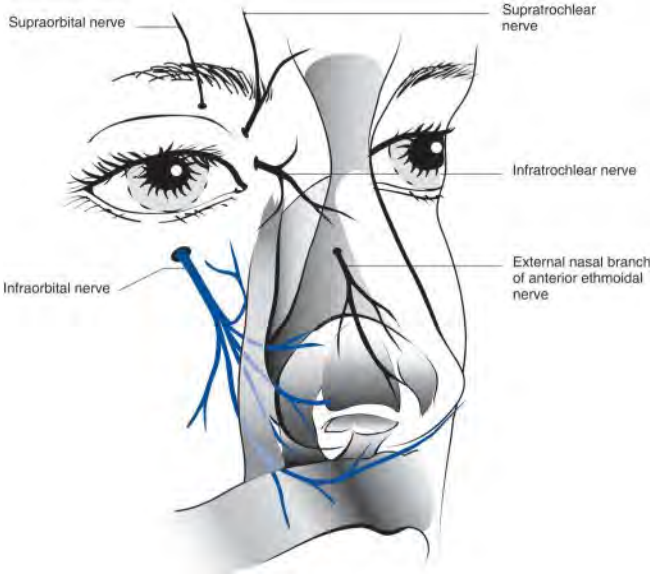


FIGURE 66-12. Sensory nerves of the external nose. The sensory innervation of the nose is derived from the V1 (ophthalmic: *black)* and from V2 (maxillary: *blue)* divisions of the trigeminal nerve. (Adapted from Jewett B. Anatomic considerations. In Baker SR, editor. Principles of Nasal Reconstruction. St. Louis: Mosby; 2002; p. 19.)

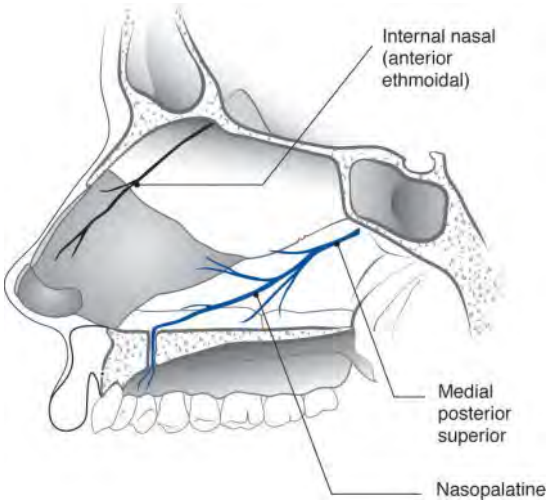


FIGURE 66-13. Sensory nerves of the nasal septum. The main sensory nerve supply comes from the internal nasal nerve (a branch of the anterior ethmoidal nerve V1 *(black)* and the nasopalatine nerve V2 *(blue).* (Adapted from Jewett B. Anatomic considerations. In Baker SR, editor. Principles of Nasal Reconstruction. St. Louis: Mosby; 2002; p. 19.)

COSMETIC EVALUATION

The cosmetic evaluation begins in the same way as with any examination, by eliciting the chief complaint of the patient. The patient should be given a mirror and a cotton-tipped applicator to point out specific cosmetic concerns. Following this, a thorough medical history should be obtained. Specific attention should be directed toward obtaining a history of nasal trauma, nasal obstruction, previous nasal surgery, and medications (including over-the-counter and herbal medications).

Psychiatric Stability

In addition to analyzing the nose, the surgeon needs to assess whether the patient is psychologically prepared for a cosmetic procedure. Patients should have realistic expectations and motivations. A patient who is internally motivated (e.g., wishes to improve self-esteem) to have the procedure is a better candidate than one who desires the procedure for external reasons (e.g., spouse wants them to have it done).[11,12]

The surgeon should beware of patients who are indecisive, rude, uncooperative, or depressed, who have unrealistic expectations, or who have significant personality disorders because they may never be satisfied. Other warning signs of poor patients are those who overly flatter, are talkative, consider themselves to be a very important patient, have minimal or no deformity, are surgeon shoppers, are price hagglers, or are involved in litigation. Most importantly, do not operate on a patient that you do not like.[11–14]



FIGURE 66-14. Preoperative rhinoplasty. **A,** Preoperative frontal view shows the width of the nose and alar base. **B,** Preoperative lateral view shows the nasal profile and dorsum in relation to the nasofrontal angle and nasolabial angle. **C,** Preoperative three quarter, or oblique, view is most natural and often revealing for harmony of the orbital rims and gull wings that flow into the nasal dorsum. **D,** Preoperative basal view is taken either from above or from below the patient and is a good view of tip and base morphology.

General Facial Analysis

Before performing a specific analysis of the nose, a global assessment of the face and its proportions should be done. Refer to Chapter 53 for additional information on facial analysis in orthognathic surgery.

Nasal Analysis

The nasal examination should be performed in a systematic manner so that the proper diagnosis is obtained (Figures 66-14 and 66-15).

GENERAL ASSESSMENT

Skin

The skin should be assessed for its thickness, mobility, and sebaceous gland content. Any pigmentations or scars should also be noted. Thick skin does not redrape well after rhinoplasty.

Symmetry

Any gross asymmetries in all views should be noted.

Lateral View

Nasofrontal Angle

The *nasofrontal angle* is defined as the angle formed by lines that are tangential to the glabella and the nasal dorsum and intersect through the radix as seen on a profile view. The normal angle is between 125 and 135 degrees (Figure 66-16).

The position of the radix should then be assessed in terms of its anteroposterior and vertical positions from a profile view. The radix should lie in a vertical plane somewhere between the lash line and the supratarsal folds. In addition, it should be 4 to 9 mm anterior to the corneal plane (see Figure 66-16).



FIGURE 66-15. Postoperative rhinoplasty. **A,** Postoperative frontal view shows the change in the width of the nose. This is the patient's most critical analysis. **B,** Postoperative lateral view shows the change in dorsal reduction and tip position. **C,** Postoperative three quarter, or oblique, view demonstrates the symmetry and graceful balance of the nose with the face. **D,** Postoperative basal view shows the width of the nose and any tip deviation from the dorsal midline.

Nasal Dorsum

In women, the nasal dorsum should lie approximately 2 mm posterior to a line drawn from the radix to the nasal tip. In men, the nasal dorsum should lie on this line or slightly in front of it (see Figure 66-16).

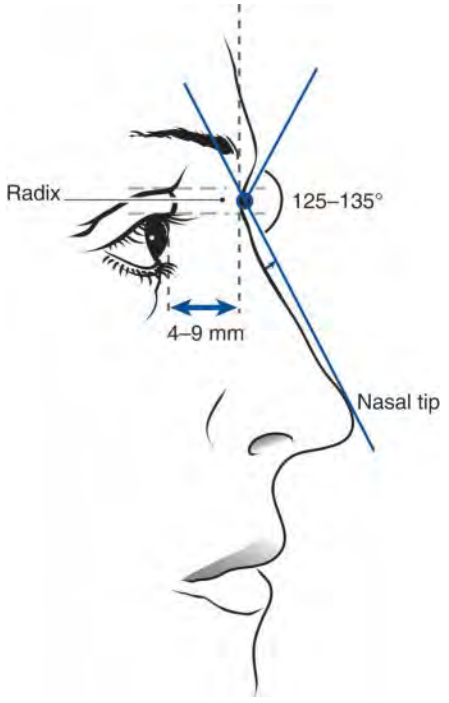


FIGURE 66-16. Position of the nasal dorsum and radix. The nasal dorsum is typically 2 mm behind a line drawn from the radix to nasal tip in women. In men, the nasal dorsum typically lies on this line. The radix should lie between the upper eyelid margin and the supratarsal folds in a vertical plane and approximately 4 to 9 mm anterior to the corneal plane. (Adapted from Austermann K. Rhinoplasty: planning techniques and complications. In Booth PW, Hausamen JE, editors. Maxillofacial Surgery. New York: Churchill Livingstone; 1999; p. 1380.)

The length of the nose (radix to tip) can be measured clinically or on photographs taken during the initial examination. The ideal nasal length should approximate the distance from stomion to menton if the lower facial height is proportionate to the middle facial height (glabella to subnasale). If the lower face height is not proportionate, it is best to estimate the nasal length as 0.67 times the middle facial height.

Nasal Tip Definition

The nose should have four tip-defining points that when drawn on the nose in the frontal view appear as two equilateral triangles (Figure 66-17). These points include the

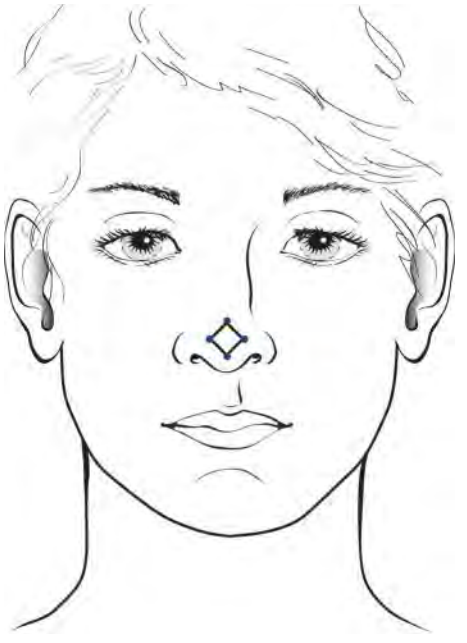


FIGURE 66-17. Nasal tip-defining points. A nose should have four tip-defining points. These are defined by the supratip, columellar-lobular angle, and the tip-defining points of each intermediate crus of the lower lateral cartilages.

supratip break, the columellar-lobular angle, and the two tip-defining points (the most projected portion of the nasal tip).

Nasal Tip Projection

Nasal tip projection can be defined as the distance that the tip (pronasale) projects anterior in the facial plane.[15] Perception of nasal tip projection can be influenced by may factors: upper lip length, nasolabial angle, nasofrontal angle, dorsal hump, and chin projection. There are several methods to determine whether the nasal tip projection is adequate. Most cosmetic rhinoplasty procedures are designed to preserve tip projection.

The simplest method to remember is Simons' method, which states that the lip-to-tip ratio is 1:1. Essentially the length of the upper lip (from subnasale to labrale superioris) should equal the nasal projection (measured from subnasale to pronasale). This method may be invalid because of the wide variation in lip lengths.[16]

The Goode method is another way of determining nasal projection. Using the Goode method, a line is drawn from the radix to the nasal tip. A second line is drawn from the radix to the alar columellar junction. A third line is drawn perpendicular to this and passes through the nasal tip. Goode's analysis states that if the nasofacial angle is between 36 and 40 degrees, then the length of the perpendicular line passing through the nasal tip should be 0.55 to 0.6 of the length of the nasal dorsum (Figure 66-18).[16]

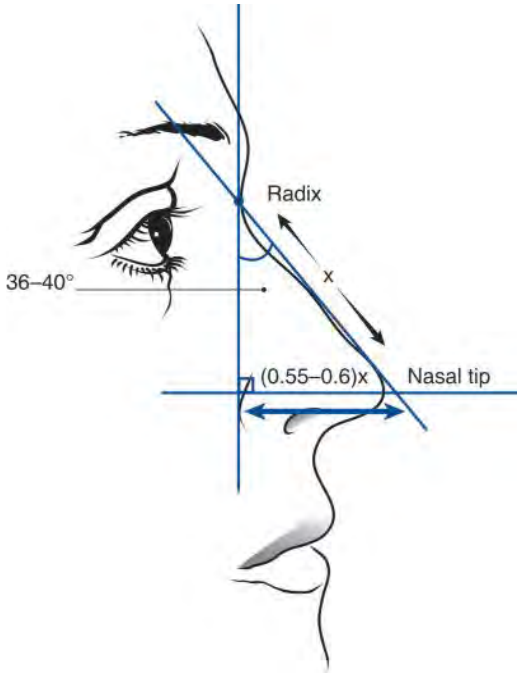


FIGURE 66-18. Goode's method of nasal projection. This method is sometimes used to determine adequacy of nasal projection. If the nasofrontal angle is between 36 degrees and 40 degrees, then the length of a perpendicular line through the nasal tip should be 0.55 to 0.6 the length of the nasal dorsum. x = nasal length. (Adapted from Austermann K, Rhinoplasty: planning techniques and complications. In Booth PW, Hausamen JE, editors. Maxillofacial Surgery. New York: Churchill Livingstone; 1999; p. 1380.)

Gunter and Rohrich[17] describe another technique of assessing nasal tip projection. If the nasal dorsal length is appropriate, the tip projection should be 0.67 times the ideal nasal length. The ideal nasal length should be equal to the distance from stomion to menton or 1.6 times the distance from the nasal tip to stomion. The tip projection is measured from the alar facial junction to the nasal tip.[17] This method is subject to a great deal of facial variation.

In addition, a vertical line drawn from the most projected portion of the upper lip should divide the nose in two equal halves between the alar facial groove and the nasal tip. If the anterior portion is greater than 60%, then the nose is likely to be overprojected (Figure 66-19).[17]

Nasal Tip Rotation

The nasal tip rotation is evaluated by the nasolabial angle and the columellar-lobular angle. *Nasolabial angle* is defined as the angle formed by lines that are tangential to the columella of the nose and the philtrum of the lip and intersect at the subnasale. In women, this should be approximately 95 to 110 degrees, whereas in men, this should be 90 to 95 degrees. Lip position may be dependent on tooth position. The *columellar-lobular angle* is defined as the angle formed by the intersection of a line tangential to the columella and a line tangential to the infratip lobule. This angle is normally between 30 and 45 degrees.

Tip Support

The strength of the cartilage in the tip of the nose is apparent when one presses on the tip. A nose with poor support may require cartilaginous struts to counteract the inherently weakened tip from the rhinoplasty. The effect of facial animation

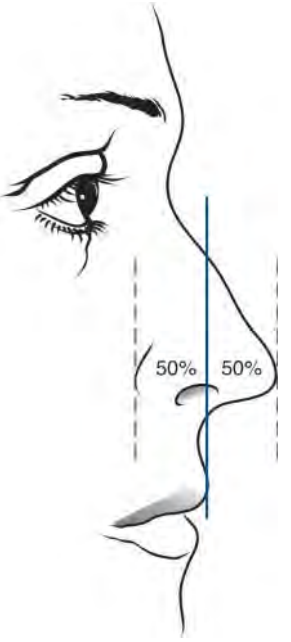


FIGURE 66-19. Nasal projection. A vertical line through the most projected part of the upper lip should divide the nose into two equal parts. If the nasal tip is greater than 60%, then the nose may be overprojected.

should also be noted. Some patients have overactive depressor septi nasi muscles, which result in a drooping nasal tip on smiling. The columella show on a lateral view should be 3 to 4 mm below the inferior alar rim.[13]

Frontal View

Width of Nasal Dorsum

The width of the nasal body and tip should be approximately 80% of the alar base width. This is assuming that the alar base is in proper anatomic proportions. The alar base width should approximate the intercanthal distance. If the width of the nasal dorsum is significantly greater than 80%, then lateral nasal osteotomies should be considered. The eyebrows should gracefully flow into the nasal dorsum analogous to a gull wing in flight.

The alar rims and columella should also be a gently curving line that appears as a bird in flight.

Alar Width

The alar base width should approximate the intercanthal distance. Seldom is the nasal width less than the intercanthal dimension.

Basal View

From a basal view, the columella-to-lobule ratio should be 2:1. Nostril size and shape should also be noted. An aesthetic nostril is teardrop-shaped, but there is a great amount of ethnic variation (Figure 66-20).

Oblique View

The oblique view is most natural and sometimes more revealing than standard photographs. It demonstrates the flow of subunits and facial harmony. The three-quarters view is how we usually see each other in routine interaction.

Functional Considerations

Although the patient desires cosmetic correction of the nose, the functional significance of the nose should be closely considered. Nasal airflow through both the internal and the external nasal valves should be evaluated. The septum should be evaluated for deviation and perforations. The septum is often a good site for harvesting autogenous cartilage for grafting. The turbinates should be evaluated for hypertrophy. Rhinoscopy with a nasal speculum can be performed both before and after the administration of a topical decongestant.

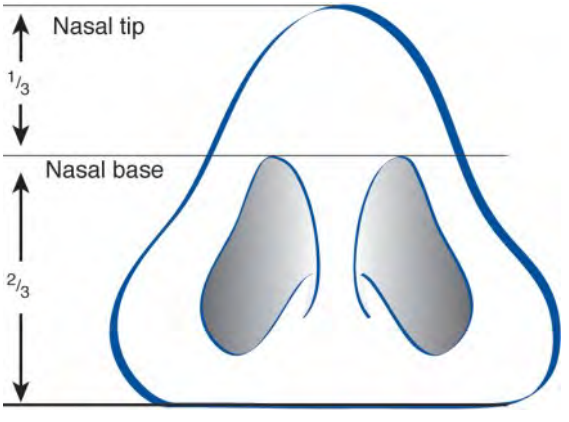


FIGURE 66-20. Columella-to-lobule ratio. The columella-to-lobule ratio should be 2:1.

Photographs

The examination is not complete without standardized facial photographs. The standard facial photographs should include frontal, right, and left lateral views; right and left oblique views; and a high and low basal view. Close-up views are taken if warranted. The photographs are beneficial from a medicolegal standpoint, and they also allow the surgeon to study the nose in more detail and to develop a surgical plan.

ANESTHESIA

Proper anesthesia of the nose is important to ensure minimal distortion of the tissues as well as to provide adequate hemostasis. Before injecting the nose, cottonoids or cotton-tipped applicators soaked in 4% cocaine or oxymetazoline are placed in each nostril to constrict the mucous membranes of the turbinates. If the rhinoplasty is to be performed under sedation, cocaine is preferred because of its anesthetic properties. If the procedure is performed under general anesthesia, oxymetazoline is sufficient.

Three cottonoids are placed in each nostril: one along the middle turbinate, one along the superior nasal vault, and one along the inferomedial septum.

Local anesthesia is achieved with 2% lidocaine with 1:100,000 epinephrine. In an endonasal rhinoplasty, the following areas are injected:

- 0.5 mL deposited at the junction of each upper and lower lateral cartilage (intercartilaginous area)
- 0.5 mL deposited in the region of each marginal incision
- 3 mL along the nasal dorsum and lateral nasal bones (hugging periosteum)
- 1 mL along the nasal septum
- 0.5 mL at each alar base
- 1 mL at each infraorbital nerve
- 1 mL at the nasal tip

For external rhinoplasty, the following additional area is injected:

- 1 mL to the columella

INCISIONS/SEQUENCING

Multiple incision techniques are used to gain access to the cartilage and bone support of the nose.

Complete Transfixion

This incision provides access to the caudal septum, medial crura, and nasal spine. The incision is made with a no. 15 blade, beginning just caudal to the superior caudal end of the nasal septum. The incision extends inferiorly through the membranous septum, following the cephalic margin of the medial crura (Figure 66-21A; see also Figure 66-7). It results in ptosis and deprojection of the nose.

Partial Transfixion

This incision is similar to the complete transfixion incision except that it stops at the level of the medial footpads of the lower lateral cartilages. The advantage of this incision is that the attachments of the medial footpads of the lower lateral cartilages to the caudal septum are not disrupted (see Figures 66-7 and 66-21B).

Hemitransfixion

This incision is a complete transfixion incision that is performed on only one side of the membranous septum. It does not traverse both mucosal surfaces, and therefore, some attachments of the medial crura to the caudal septum are maintained. Access to the nasal septum is good with this incision; however, delivery of the lower lateral cartilage on the side opposite to the incision is difficult (see Figures 66-7 and 66-21C).

Killian Incision

This incision is seldom used in rhinoplasty. It is a useful incision to gain access to the nasal septum if only a septoplasty is to be performed. The incision is made several millimeters cephalad to the caudal edge of the septum. It can be extended onto the nasal floor if needed.

Intercartilaginous Incision

This incision is made at the junction of the upper and lower lateral cartilages. The nare is elevated superiorly with a double skin hook. A no. 15 blade should pass below the lower lateral cartilage and above the upper lateral cartilages. This incision is typically made after a transfixion incision. The

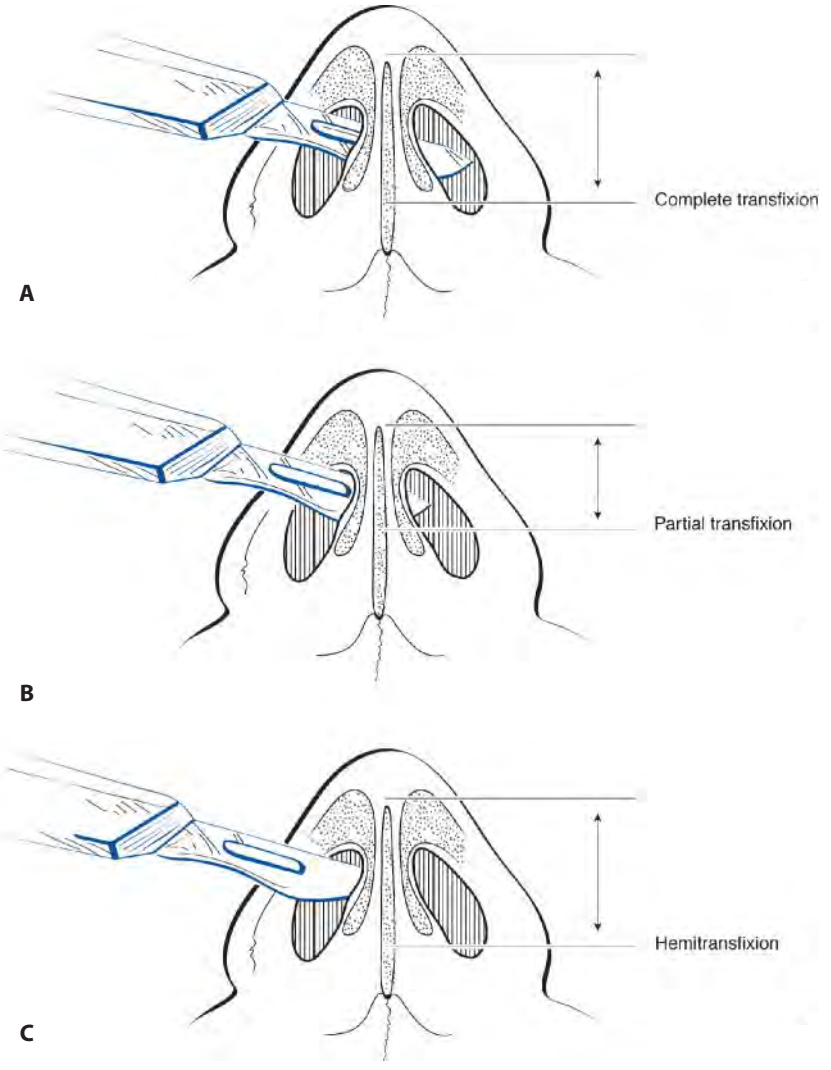


FIGURE 66-21. Transfixion incisions. **A,** A complete transfixion incision is made caudal to both the medial crura and through the membranous septum. **B,** A partial transfixion incision is similar except the incision stops short of the medial footpads of the medial crura. **C,** A hemitransfixion incision is a complete transfixion incision that is performed on only one side; therefore, the other medial crura and footpad are not violated.

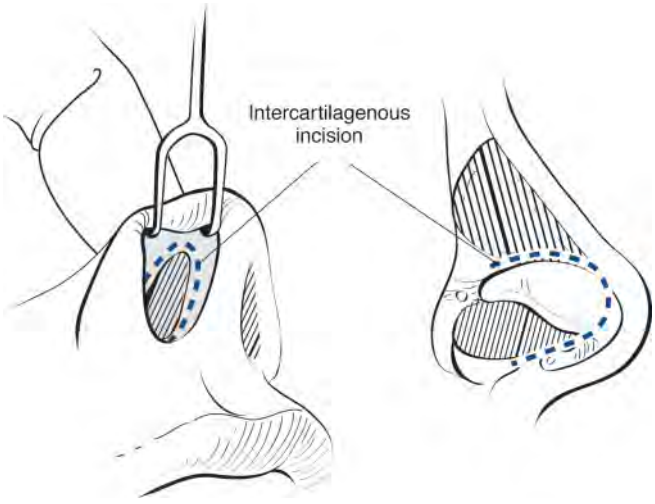


FIGURE 66-22. Intercartilaginous incisions. The intercartilaginous incision is made at the junction of the upper and lower lateral cartilages. The blade should pass below the lower lateral and above the upper lateral cartilage. (Adapted from Alexander R. Fundamental terms, considerations, and approaches in rhinoplasty. In Waite PD, editor. Atlas of the Oral and Maxillofacial Surgery Clinics of North America: Rhinoplasty. Philadelphia: WB Saunders; 1995; p. 19.)

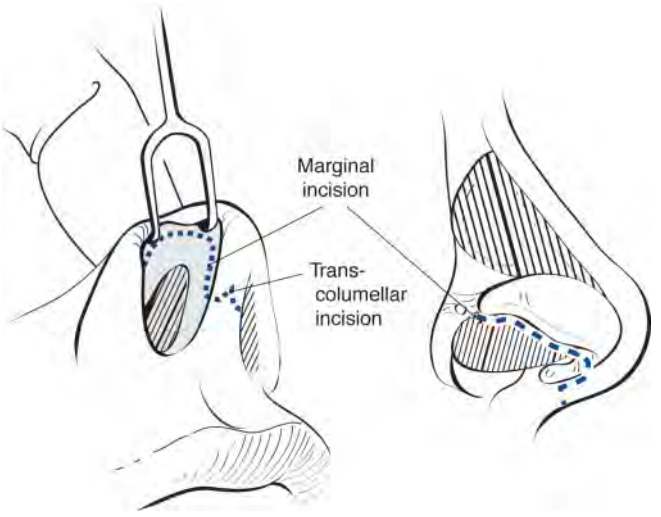


FIGURE 66-23. Marginal incision. This incision is made parallel to the caudal edge of the lower lateral cartilage. This incision can be combined with bilateral intercartilaginous incisions for a cartilage delivery technique in endonasal rhinoplasty or combined with a transcolumellar incision for an external rhinoplasty. (Adapted from Alexander R. Fundamental terms, considerations, and approaches in rhinoplasty. In Waite PD, editor. Atlas of the Oral and Maxillofacial Surgery Clinics of North America: Rhinoplasty. Philadelphia: WB Saunders; 1995; p. 19.)

intercartilaginous incision is then connected to the transfixion incision (Figure 66-22; see also Figures 66-8 and 66-9).

Intracartilaginous Incision

This incision is made through both the vestibular nasal mucosa and a portion of the lower lateral cartilages. This incision is similar to the intercartilaginous incision except that it is made 3 to 5 mm posterior to the junction of the upper and lower lateral cartilages. This incision in effect performs a complete cephalic strip of the lower lateral cartilages without the need for delivering the cartilage. The disadvantage is that the lower lateral cartilage is not directly visualized and it may, therefore, be difficul to achieve symmetry between the right and the left sides.

Rim/Marginal Incision

This incision parallels the caudal edges of the lower lateral cartilages. The incision is used in combination with an intercartilaginous incision in an endonasal rhinoplasty. The two incisions allow the lower lateral cartilage to be delivered and visualized. This allows the surgeon to more accurately trim the cartilage if needed. In an open rhinoplasty, this incision is combined with a transcolumellar incision in order to gain access to the lower lateral cartilage and nasal dorsum (Figure 66-23).

Transcolumellar Incision

This incision is made through the thinnest portion of the columella at a level just superior to the flaring of the medial crura. The incision can be made with a notched V in the center of the columella or as a "stair step." This will break up the scar and assist in closure. This incision is connected with a marginal incision bilaterally for open rhinoplasty (see Figure 66-23).

The two principle techniques are the endonasal and external rhinoplasty. Each of these techniques are described in general terms, in the order in which the authors perform them. Other surgeons may perform the sequence in a different order (Tables 66-3 and 66-4).

SEPTOPLASTY

In rhinoplasty surgery, there are several reasons to access the nasal septum: (1) to correct nasal airflow obstruction, (2) to

TABLE 66-3. **Surgical Sequence for Endonasal Rhinoplasty**

1. Local anesthesia
2. Partial transfixion incision (see Figure 66-7)
3. Intercartilaginous incision (join with partial transfixion) (see Figures 66-8, 66-9, 66-21, and 66-22)
4. Septoplasty (if needed) (see Figures 66-24 and 66-25)
5. Dorsal reduction (see Figures 66-28 to 66-30)
6. Lateral nasal osteotomies (see Figure 66-31)
7. Marginal incision (see Figure 66-23)
8. Delivery of lower lateral cartilages (see Figure 66-37)
9. Tip modification (i.e., cephalic strips/cartilage grafting/suture techniques)
10. Alar base modification (see Figure 66-41)
11. Closure, taping, and splinting

TABLE 66-4. **Surgical Sequence for External Rhinoplasty**

1. Local anesthesia
2. Bilateral marginal incisions (see Figure 66-23)
3. Columellar incision (see Figure 66-23)
4. Skeletonization of upper and lower lateral cartilages and nasal dorsum
5. Dorsal reduction
6. Dome division if access is needed to the septum for septoplasty or graft harvest
7. Septoplasty (if needed)
8. Turbinate reduction
9. Lateral nasal osteotomies
10. Tip modification (i.e., cephalic strips/cartilage grafting/suture techniques)
11. Alar base modification
12. Closure, taping, and splinting

assist in the correction of asymmetries, and (3) to harvest cartilage for tip grafting.

Access to the nasal septum in an endonasal approach is through a partial-transfixion incision, which is connected to bilateral intercartilaginous incisions. The partial-transfixion incision can be extended to the nasal floor on the side on which the septoplasty is to be performed. After completing the incisions, the caudal aspect of the nasal septum is exposed by dissecting the mucoperichondrium from one side. Two tunnels will be developed, one superior and the other inferior, that will ultimately be joined so that wide exposure of the septum is obtained.[18] Intially sharp dissection is done with a no. 15 blade or scissors to expose a portion of the caudal septum. The perichondrium is gently scored using a no. 15 blade. A dental amalgam condenser is then used in a sweeping motion to develop a plane between the perichondrium and the nasal septum (Figure 66-24). Once this plane of dissection is started, a Freer or Cottle elevator can be used to complete the septal envelope (Figure 66-25). The mucoperichondrium is tightly bound at the junction of the septum and the maxillary crest.



FIGURE 66-24. Identifying perichondrium. The perichondrium is elevated with a dental amalgam condenser. One will notice a slight blue-gray cartilage and a distinct plane of dissection.



FIGURE 66-25. Elevation of mucoperichondrium. The Cottle elevator is specifically designed to elevate the nasal envelope without perforation.

Once the septum is exposed, it can be treated in one of four ways: (1) resection, (2) morselization, (3) segmental transection, and (4) swinging door flaps.[18] Submucosal resection allows a significant portion of cartilage to be harvested for grafting. At least 1 cm should be maintained superiorly and anteriorly in an L-shaped configuration to provide support for the nose (Figure 66-26). In order to resect the cartilage, a Cottle elevator is used to cut the cartilage. Fomon scissors may be used to make the superior and inferior cuts through the bony septum. The cartilage can also be removed with a Ballenger swivel blade. If no cartilage is needed for the rhinoplasty, the resected cartilage can be morselized and replaced. Morselization can be performed in situ. Another technique for aligning the septum is through a segmental transection. In this technique, the mucoperichondrium is elevated on one side of the septum. Cross-hatching with a no. 15 blade is performed to weaken the cartilage (Figure 66-27). The mucoperichondrium on the other side of the septum provides support. Some 4-0 gut mattress sutures can be positioned through the septum to assist in realignment. A septal splint is placed for 1 week. Finally, a swinging door type flap can be used to reposition a large segment of flat cartilage that is improperly angulated. The mucoperichondrium is elevated on one side. Through and through incisions are made on either side of the deviated cartilage. The cartilage is also separated from the maxillary crest so that it can hinge into a more normal position. Septal splints may be required for 1 week. In all septal procedures, a 4-0 gut on a straight needle is routinely used to perform a mattress suture through the septum and mucosa. This decreases the likelihood of a septal hematoma formation and circumvents the need for nasal packs.

Tears in the septal mucosa are not uncommon. However, it is not problematic as long as the tears are only on one side of the septum. Unilateral tears require no elaborate closure.

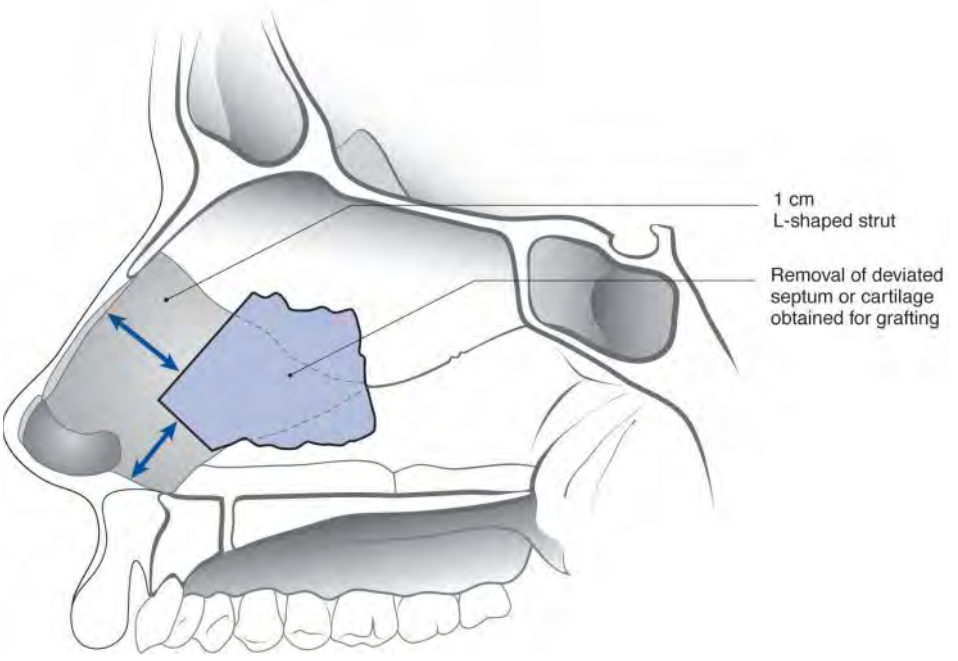


FIGURE 66-26. Resection of cartilage/bone from the nasal septum. This may be done to harvest cartilage for grafting procedures or for removal of grossly deviated septum. It is important to maintain 1 cm dorsally and caudally for nasal support.

If the tear is through and through, at least one side should be closed. This is best done with a 5-0 chromic gut suture.

TURBINECTOMY

Although the focus of this chapter is the cosmetic rhinoplasty, some mention needs to be made on maintaining function. Inferior turbinate hypertrophy is a problem that can result in nasal obstruction after cosmetic rhinoplasty, if the problem is not recognized preoperatively Hypertrophy of the inferior turbinates is the most common cause of nasal airway obstruction.[19,20] Hypertrophy can be caused by numerous factors. Most commonly, it is related to allergic symptoms. Hypertrophy caused by allergy should be managed medically with antihistamines and topical corticosteroids. If this fails, surgical management can be considered.[21] In cases of a deviated nasal septum, the turbinate on the side at which the nasal passage is enlarged can become hypertrophic with time. In patients with anatomic enlargement of the turbinate, the problem needs to be recognized so that the nasal passage does not become obstructed when the septum is straightened.

Management of inferior turbinate hypertrophy is controversial and outside the scope of this chapter. The surgical procedures used to treat this problem have included corticosteroid injection, turbinate outfracture, electrocautery, cryosurgery, laser reduction, partial turbinate resection, total turbinate resection, submucous turbinate resection, and vidian neurectomy[20–24] Each of these procedures has various advantages and disadvantages; the procedure chosen depends on the patient. The most common complications from turbinate surgery are hemorrhage, atrophic rhinitis, and ozena.

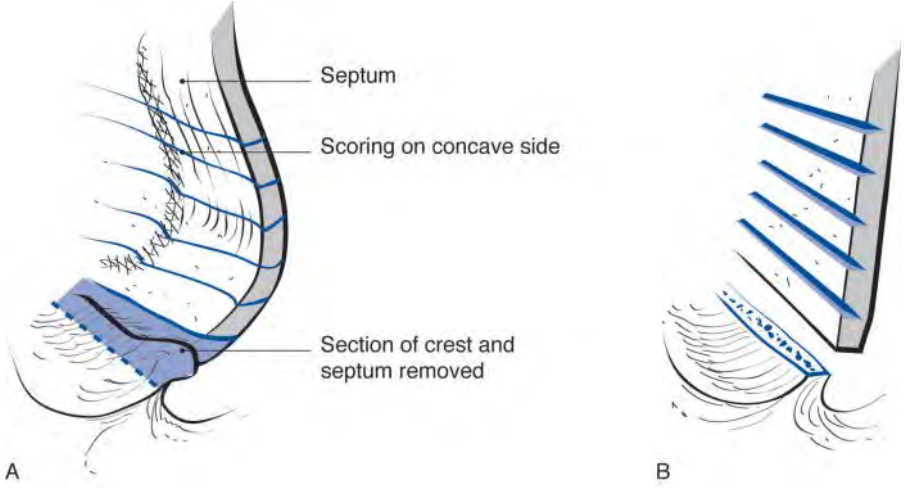


FIGURE 66-27. Septal repositioning. A deviated nasal septum can be repositioned by removing the obstruction inferiorly **(A)** and cross-hatching the cartilage to allow the deviated portion to be repositioned **(B). (A** and **B,** Adapted from Robinson RC. Functional septorhinoplasty. In Waite PD, editor. Atlas of the Oral and Maxillofacial Surgery Clinics of North America: Rhinoplasty. Philadelphia: WB Saunders; 1995; p. 35.)

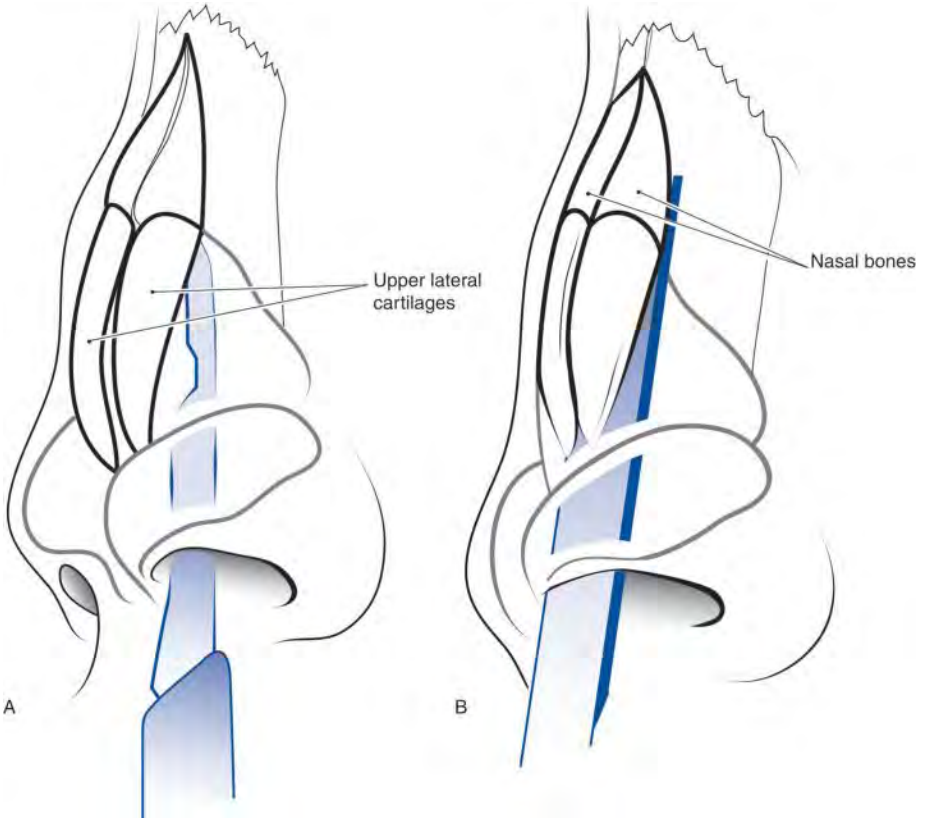


FIGURE 66-28. Removal of a dorsal hump. **A,** The dorsal hump is removed by first using a scalpel to incise through the upper lateral cartilages. **B,** Next, a Rubin osteotome is used to reduce the bony prominence. Care is needed to keep the osteotome from being directed too far posteriorly thereby overreducing the dorsum. **(A** and **B,** Adapted from Austermann K, Rhinoplasty: planning techniques and complications. In Booth PW, Hausamen JE, editors. Maxillofacial Surgery. New York: Churchill Livingstone; 1999; p. 1389.)

NASAL DORSUM

Reduction

One of the most dramatic changes that can be achieved in rhinoplasty surgery is correction of a dorsal hump. There are many ways to remove the hump. Some surgeons use a scalpel and osteotome; others use rasps; and a few use power rasps. The authors recommend to first incise the cartilaginous convexity below the nasal bones and then to use a Rubin osteotome to remove the bony hump (Figures 66-28 to 66-31). Care must be taken to keep the osteotome directed superficially, because it can deflect downward and result in overreduction. After removing the gross hump, sequential rasping can be used for refinement. After removal of any significant dorsal hump, the patient is left with an open roof deformity. This must be closed with lateral nasal osteotomies (see Figure 66-31).

Augmentation

Augmentation is indicated when there has been excessive reduction from previous rhinoplasty or from a post-traumatic defect. Several techniques are used to augment the nasal dorsum.

Autogenous Augmentation

In the setting of acute trauma, cranial bone grafts can be used to provide support. These are cantilevered off the frontal bone with a miniplate. The graft must be properly shaped so that it provides support but does not distort the shape of the nose.[25–27] Rib cartilage can also be harvested for augmentation of the nasal dorsum. Silicone sizers can be used to estimate the size and shape of graft needed. Once the graft is harvested, a 0.035-inch K-wire can be placed in the center of the graft to stabilize it. Rib grafts have a tendency to distort with time and the K-wire may help limit this tendency.[28]



FIGURE 66-29. Dorsal reduction. An Aufricht retractor lifts the dorsal drape and can protect the skin during hump reduction. A no. 15 blade is used to incise the cartilaginous dorsum. Working through this incision, an osteotome or rasp is used to reduce the bone of the dorsum.



FIGURE 66-30. Dorsal reduction. The dorsum should be about two thirds of the cartilage and one third of the bone.

For a less aggressive augmentation, autogenous cartilage harvested from the nasal septum can be used. This can be layered and sutured together. It is then placed through traditional rhinoplasty incisions.[29–31]

Alloplastic Augmentation

Another technique is to use cadaveric dermis along the nasal dorsum. The advantage here is that no harvesting is required and the material is pliable. However, the resorption of this material is unpredictable. Other implantable materials include silicone and expanded polytetrafluoroethylene (ePTFE) implants. These can be contoured to the appropriate size intraoperatively. The issue with implants is that the grafts can extrude or become infected. Meticulous placement is essential.[31–34]



FIGURE 66-31. Lateral nasal osteotomies. Removal of a large dorsal hump will often leave a flat open roof deformity; this can be reduced by lateral nasal osteotomies with an invert chisel saw, or rasp.

Osteotomies

Osteotomies are performed after the nasal reduction has been performed. The purposes of lateral nasal osteotomies include reduction of the open nasal roof, correction of deviated nasal bones, and narrowing of a wide nasal base (see Figure 66-31).

The two principal types of nasal osteotomy are lateral and medial. The lateral nasal osteotomy can be performed at different levels. It typically begins low on the piriform rim and can end either high or low in its relationship to the nasal bones. Thus, the osteotomy is often termed a *low-to-low osteotomy* or a *low-to-high osteotomy.* These osteotomies can be performed via an internal or external technique. Regardless of which technique is used, limited periosteal dissection is favored so that support is provided to the nasal bones. Medial osteotomies are seldom needed but can be used to obtain a controlled fracture in patients with thick nasal bones or when a low-to-low technique is used. Also, regardless of the osteotomy technique, the osteotomies should not be carried above the intercanthal line. The bone above this point becomes much thicker and mobilization becomes difficult. Care should be taken when performing medial osteotomies, because the thicker portion of the nasal bone can be included in the lateral osteotomy segment and result in widening of the upper nasal dorsum. This is termed a *rocker deformity.*

Lateral nasal osteotomies are not always required to close an open roof deformity after dorsal hump reduction. Some surgeons believe it is better to place spreader grafts in those patients with short nasal bones so that compromise of the internal nasal valve does not occur. If an osteotomy is performed in a patient with shorter nasal bones, a low-to-high technique is preferred.

NASAL TIP

Understanding the mechanisms of nasal tip support is critical when performing rhinoplasty. The surgeon must understand both the desired and the undesired changes that occur from the surgical approach or technique.[35]

The three major tip support mechanisms include

1. The size, shape, and strength of the lower lateral cartilages.
2. The attachment of the medial crura to the caudal septum.
3. The attachment of the lower lateral cartilages to the upper lateral cartilages.

The minor tip support mechanisms include

1. The interdomal ligament.
2. The sesamoid complex, extending the support of the lateral crura to the piriform aperture.
3. The attachment of the alar cartilages to the overlying skin.
4. The cartilaginous septal dorsum.
5. The nasal spine.
6. The membranous septum.[36,37]

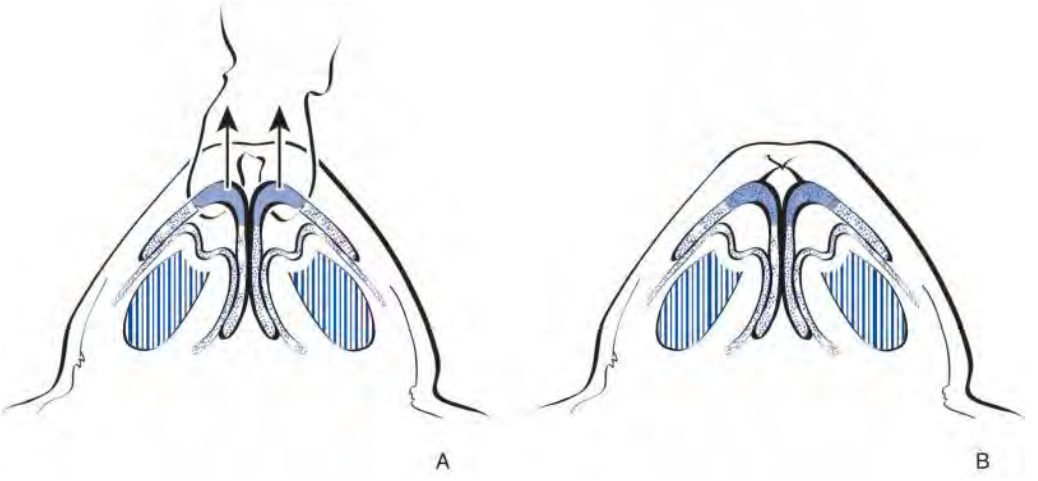


FIGURE 66-32. Lateral crural steal. **A** and **B,** A horizontal mattress suture is placed in the lateral crura in order to increase nasal projection and narrow the nasal tip. **(A** and **B,** Adapted from Taylor CO. Surgery of the nasal tip. In Waite PD, editor. Atlas of the Oral and Maxillofacial Surgery Clinics of North America: Rhinoplasty. Philadelphia: WB Saunders; 1995; p. 61.)

Certain surgical procedures can affect tip support. For example, a complete transfixion incision will disrupt the fibrous attachments of the caudal septum to the medial crura, thus leaving little support for the nasal tip. Suturing techniques and cartilage strut grafts may be necessary to reestablish support if this incision is performed. Intercartilaginous incisions, which are useful to gain access to the nasal dorsum, interrupt the ligamentous connections of the upper and lower lateral cartilages. This can result in cephalic tip rotation, which may or may not be desirable. A cephalic strip procedure creates even further disruption and rotation of the lower lateral cartilages. Most often, tip rhinoplasty is designed to refine and decrease the tip lobule while maintaining or even increasing rotation and projection.

The cartilaginous support of the nasal tip is often described in terms of a tripod concept.[38,39] The medial crura of both the lower lateral cartilages together form one strut of the tripod, and each of the lateral crura of the lower lateral cartilages forms a strut. By selectively shortening or lengthening any of these struts, the tip position can be altered.

The tip position changes are referred to in terms of both projection and rotation. *Tip projection* is the distance from the tip of the nose to the alar-facial junction. Increasing tip projection is one of the most difficult procedures to perform in rhinoplasty surgery. Nasal tip projection can be increased by both grafting and nongrafting techniques.

Tip Projection

Increasing Tip Projection

Nongrafting techniques to increase nasal projection include

1. Suturing of divergent medial crura: For this technique to be effective, there must be diverging medial crura. Intervening soft tissue may require excision before suturing with mattress sutures.[40]
2. Lateral crural steal: The lower lateral cartilage is skeletonized and the lateral crura cartilages are sutured with a mattress suture so that the lateral crura now contributes to the medial crura (Figure 66-32). This results in increased projection and some rotation as well.[41,42]

Grafting techniques to increase projection include

1. Collumellar strut: This technique involves the placement of a strut of septal cartilage between the feet of the medial crura and abutted against the nasal spine. The medial crura are elevated superiorly with double skin hooks and the cartilage strut is sutured to the medial crura via mattress sutures. Only a minor amount of tip projection can be increased with this method.
2. Peck graft: This is an onlay graft in the region of the nasal tip. Layers of cartilage are placed in the domal region to increase projection. The graft material is either conchal or septal cartilage. The cartilage is secured to the dome by sutures. This technique can increase projection by 2 to 6 mm (Figure 66-33).

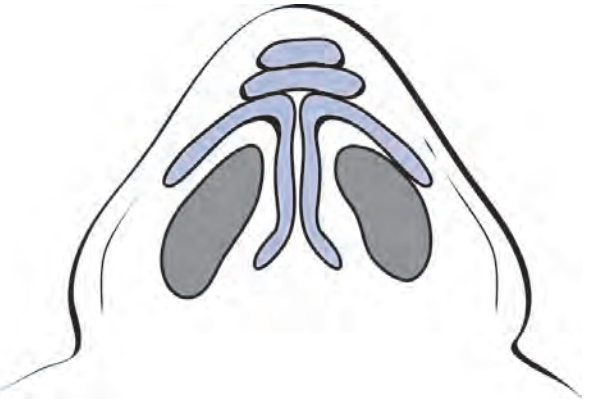


FIGURE 66-33. Peck graft. This involves the placement of layers of cartilage grafts in the region of the nasal tip to increase nasal projection. (Adapted from Taylor CO. Surgery of the nasal tip. In Waite PD, editor. Atlas of the Oral and Maxillofacial Surgery Clinics of North America: Rhinoplasty. Philadelphia: WB Saunders; 1995; p. 62.)

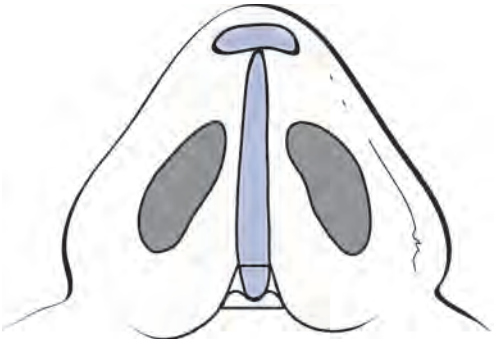


FIGURE 66-34. Umbrella graft. This is essentially a columellar strut graft placed between the medial crura combined with a tip graft. This technique improves support of the medial crura as well as increases nasal projection. (Adapted from Taylor CO. Surgery of the nasal tip. In Waite PD, editor. Atlas of the Oral and Maxillofacial Surgery Clinics of North America: Rhinoplasty. Philadelphia: WB Saunders; 1995; p. 61.)

3. Umbrella graft: This technique involves the creation of a cartilaginous structure that resembles the appearance of an umbrella. It is useful when both tip projection and support of weak medial crura are required. The umbrella graft is constructed from harvested septal, ear, or rib cartilage. It is then sutured in position so that the "handle" of the umbrella is between the medial crura and the "canopy" of the umbrella rests atop the dome. The canopy portion can be modified to incorporate the Peck graft technique by stacking layers of cartilage (Figure 66-34).[43]
4. Shield graft: This graft was first described by Sheen.[30] A piece of septal cartilage is shaped to form a trapezoidal configuration measuring 6 to 8 mm superiorly and 5 mm inferiorly The graft is usually 10 to 12 mm long and is beveled so that the corners are blunted. The graft is placed in a pocket through an endonasal approach or sutured in position via an open approach. The superior and lateral aspect of the graft forms the tip-defining points (Figure 66-35).[30]

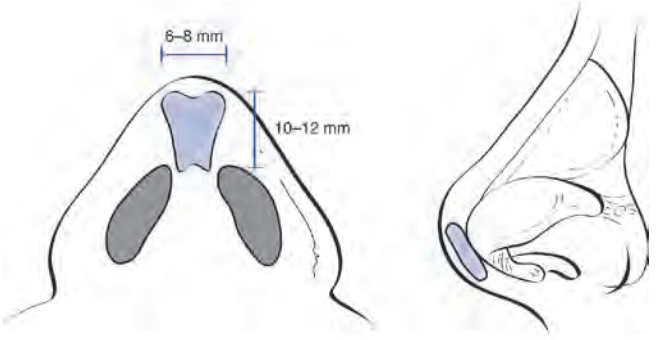


FIGURE 66-35. Shield graft. **A** and **B,** This is a grafting technique used to redefine the tip-defining points of the nose. The graft is typically 6 to 8 mm wide superiorly, 5 mm wide inferiorly, and 10 to 12 mm long. **(A** and **B,** Adapted from Taylor CO. Surgery of the nasal tip. In Waite PD, editor. Atlas of the Oral and Maxillofacial Surgery Clinics of North America: Rhinoplasty. Philadelphia: WB Saunders; 1995; p. 62.)

Decreasing Tip Projection

Decreasing tip projection involves reduction of the tip-supporting mechanisms. Achieving acceptable results when decreasing projection can be difficult because nasal definition can be lost.[44] If the nasal projection needs to be decreased, be certain to first confirm that the problem is not the result of an optical illusion caused by a low radix position. If the problem is a low radix, a dorsal radix graft is the appropriate treatment.

Methods to decrease projection include

1. Complete transfixion incision: As discussed previously, a complete transfixion incision will decrease tip support. Intercartilaginous incisions or cephalic strips will also weaken the tip support but will increase tip rotation.
2. Lower the septal angle: If the septum is providing significant support for the nasal tip, the septal angle must be lowered. This is done by excision of a portion of the caudal septum. In addition, the medial crura can be separated from the caudal septum to decrease projection.
3. Crural excision: To dramatically decrease tip projection, the medial and lateral crura may need to be sectioned, overlapped, and sutured into a new position with less projection. This technique maintains the natural shape of the tip at the domes (Figure 66-36). Excision of a segment cartilage in the domes and suturing them back together can be done, but it will change the shape of the nasal tip.

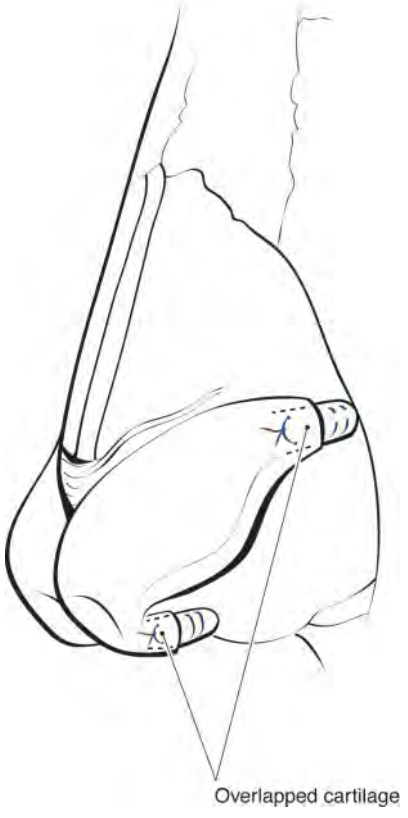


FIGURE 66-36. Crural excision. This is used when the nasal tip needs dramatic deprojection. A portion of the lateral crura is excised and the ends are sutured back together.

Sometimes, after decreasing the nasal projection, the patient may have flaring of the ala and increased infratip columellar show. This can be treated with an alar base resection but this should be used judiciously.

Tip Rotation

Increasing Tip Rotation

Understanding the tripod concept and tip-supporting mechanisms is important when determining which of the following methods to use to increase tip rotation.

1. Removal of dorsal hump: A subtle way to increase rotation of the tip is to reduce a dorsal hump if present.
2. Resection of the caudal septum: A small triangular piece of caudal septum can be removed. The base of this triangular shape is at the nasal dorsum.
3. Cephalic strips from lower lateral cartilages: A complete strip of cephalic cartilage from the lower lateral cartilages will result in increased tip rotation. Even an intercartilaginous incision will result in some tip rotation (Figure 66-37).
4. Shorten the lateral crura.
5. Shield graft: A shield graft gives the illusion of increased tip rotation.
6. Augmentation of premaxilla: Placement of cartilage or ePTFE in the premaxilla region below the anterior nasal spine will also give the illusion of increased tip rotation.

Decreasing Tip Rotation

Decreasing tip rotation is done by two methods:

1. Trim the caudal septum near the anterior nasal spine.
2. Augment the nasal dorsum: this creates the illusion of decreased tip rotation.



FIGURE 66-37. Delivery of lower lateral cartilage. The lower lateral cartilage is best delivered by a marginal incision or exposed through an open rhinoplasty for direct visualization and surgical manipulation. Tip refinement is improved in this case by complete tip reduction to reduce the volume of the tip.

Tip Shape

In addition to changing the tip position, the tip shape must also be considered. Historically, changes to the nasal tip were performed by selective cartilage excision and reapproximation. The Goldman tip is an example of such a technique. The current trend is to preserve and reorient existing cartilage and place cartilaginous grafts if required.[45] Excessive grafting can be unpredictable in the long run.

Although cartilage preservation is emphasized, there is still sometimes a need to remove cartilage. There are three principal techniques of cartilage excision in the nasal tip region: a complete strip technique, a weakened complete strip technique, and an interrupted strip technique. A greater resection generally results in more dramatic tip narrowing and rotation.

Complete strip techniques involve the removal of a complete piece of cartilage from the cephalic end of the lower lateral cartilages (Figure 66-38; see also Figure 66-37). This procedure is thought to be more stable because it leaves an intact strip of the inferior border of the lower lateral cartilage. Aggressive resection can result in loss of tip support, alar notching, alar retraction, and the appearance of increased collumellar show. Most surgeons feel that a minimum width of 6 mm is required to maintain the structural integrity of the lower lateral cartilage.

The weakened complete strip technique involves the removal of a complete cephalic strip followed by weakening of the cartilage by selective morselization of the medial and lateral crura with a scalpel blade.

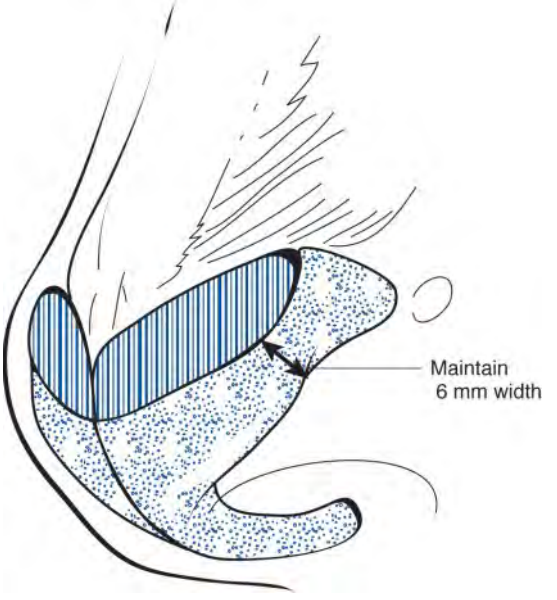


FIGURE 66-38. Complete strip technique. This involves the excision of a strip of cartilage on the cephalic portion of the lower lateral cartilage. This will result in increased tip rotation. It is important to maintain a minimum of 6 mm width of cartilage for structural support of the nose. (Adapted from Taylor CO. Surgery of the nasal tip. In Waite PD, editor. Atlas of the Oral and Maxillofacial Surgery Clinics of North America: Rhinoplasty. Philadelphia: WB Saunders; 1995; p. 58.)

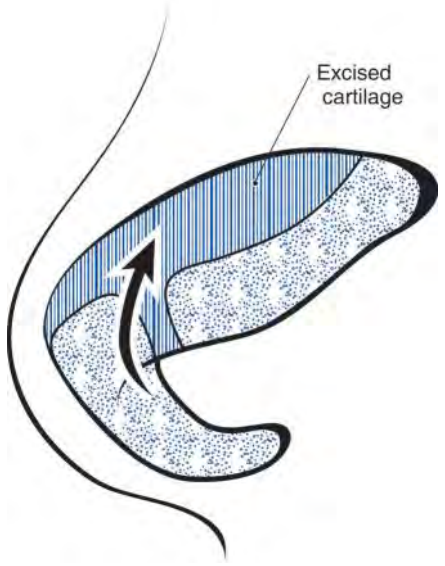


FIGURE 66-39. Interrupted strip technique. This is similar to the complete strip except the remaining cartilage is also divided in a vertical fashion. This allows for even greater tip rotation, as indicated by the *arrow;* however, it can result in a pinched nasal tip and functional problems. The cartilage can be weakened by scoring it in a vertical fashion and this is termed a *weakened complete strip technique.* (Adapted from Taylor CO. Surgery of the nasal tip. In Waite PD, editor. Atlas of the Oral and Maxillofacial Surgery Clinics of North America: Rhinoplasty. Philadelphia: WB Saunders; 1995; p. 59.)

An interrupted strip involves division of the lateral crura from the dome (Figure 66-39). This technique provides greater rotation than a complete strip but can also result in complications, including loss of tip support, alar notching, and alar retraction. In addition, the nasal tip can develop a pinched appearance. The classic Goldman tip is an example of an interrupted strip technique (Figure 66-40). In this technique, the lateral crura are divided lateral to the tip-defining points. The medial segments are sutured together, which results initially in increased tip projection. The lateral crural segments are left alone as independent units. This procedure is no longer commonly used because of problems with tip asymmetry, pinched appearance of the nasal tip, and long-term tip ptosis.

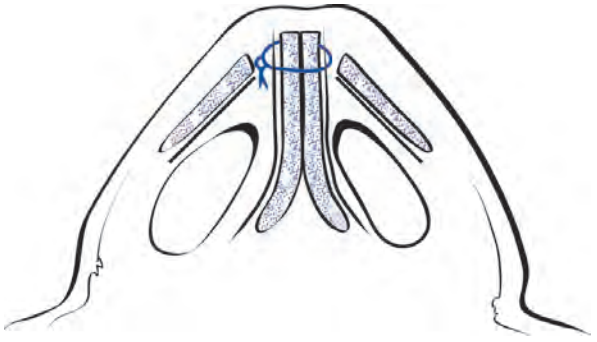


FIGURE 66-40. Goldman tip. This is an interrupted strip technique in which the lateral crura are divided lateral to the tip-defining points. The medial segments are then sutured together to increase nasal tip projection and to narrow the nasal tip. (Adapted from Willis AE, Costa LE. Surgical management of the nasal base. In Waite PD, editor. Atlas of the Oral and Maxillofacial Surgery Clinics of North America: Rhinoplasty. Philadelphia: WB Saunders; 1995; p. 61.)

For patients with a broad nasal tip, transdomal suturing techniques are often used to narrow the tip. Volume reduction is performed first if needed by cartilage excision as described previously. Next, excision of excessive interdomal soft tissue is performed. A 4-0 polydioxanone transdomal suture is placed in a horizontal mattress fashion to narrow and reorient the alar cartilages. The advantage of this technique is that the suturing can be done multiple times until the surgeon is satisfied with the result. In addition, the long-term results of this technique have been favorable.[46–48]

NASAL BASE ALAR REDUCTION

The alar base should approximate the intercanthal distance and be no more than 1 to 2 mm wider than this. The nostrils should have a symmetrical appearance. Asymmetry of the nostril is often due to a deviated nasal septum and this should be reevaluated before consideration of an alar base resection.

The primary procedure to reduce the alar base width is an alar base resection. Alar modification is often considered in cases in which the nose has to be deprojected or to balance the anatomy in certain ethnic types. It is mandatory to be conservative when performing alar reduction because it is difficult to correct an overreduction. If there is any doubt, the surgeon should delay the alar base reduction until a later date.[49]

The procedure is performed by excising a small wedge of vestibular mucosa and skin. The angulation can be adjusted so that greater reduction of the outer perimeter of the ala is reduced and only limited reduction of the internal perimeter is performed.[49] The excision should be conservative and will rarely be greater than 3 mm in width (Figure 66-41).

POSTOPERATIVE MANAGEMENT

After performing the rhinoplasty, the surgeon must decide whether intranasal stents or packing is necessary. We generally do not place nasal packing. If the septum requires additional support during healing, silicone stents are placed. These stents are also used if there are mucosal tears or if a turbinectomy was performed. The stents help reduce the incidence of synechiae formation. The stents are secured to each other by a 3-0 silk suture passed through the columella and are typically left in place for 1 week.

Next, the nasal dorsum is splinted. Benzoin or mastisol is painted on the nasal dorsum and #fr1/4>-inch brown paper tape is applied. After placement of the tape, the splint is applied. A metal Denver splint or thermoplastic splint is contoured and applied. Additional paper tape can be placed over the splint.

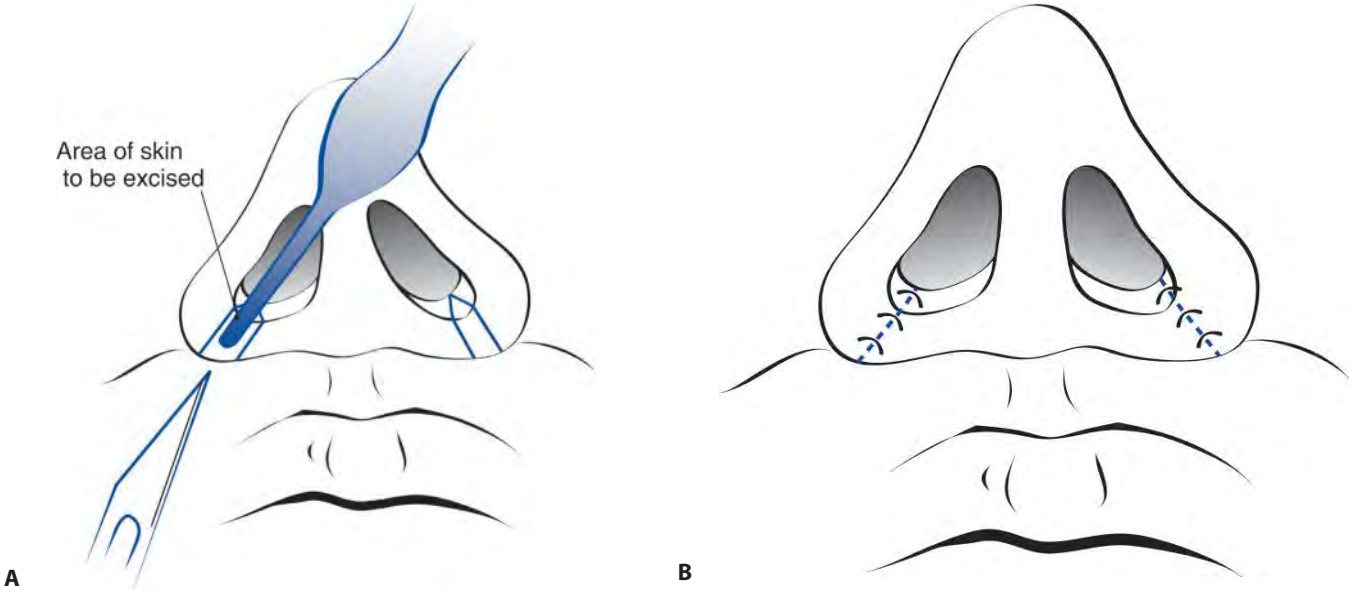


FIGURE 66-41. Alar base reduction (Weir's excision). **A,** This is used to narrow an overly wide nostril. **B,** A small amount of vestibular mucosa and skin is excised and sutured together. The excision is usually 1 to 2 mm wide.

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CHAPTER 67

Rhytidectomy

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Face-lifting has received significant attention over the past several decades owing to increasing patient demands for a more youthful appearance. The face undergoes harmonious changes in the facial skeleton, deep soft tissue elements, and skin texture during the aging process. Dissection of cadavers has identified facial ligaments, muscle expansions, and dissection planes that give us a better understanding of facial aging and rejuvenation. Perhaps more important, it has also been the catalyst for the evolution of a variety of face-lifting techniques. The goal of facial rejuvenation should be to address all components of aging, leaving the patient with a younger-appearing face and a long-lasting result. If this is accomplished, the patient's face and neck will continue to age harmoniously.[1]

Critical evaluation of early techniques and a clear understanding of surgical anatomy have provided insight into the perils and pitfalls of surgical rejuvenation of the face and have resulted in the complexity of various rhytidectomy techniques. Today, rhytidectomy is one of the most frequently performed aesthetic surgical procedures in the United States.

Numerous techniques are currently used for performing face-lifts but no general agreement as to which of these techniques is most effective[2]; facial aesthetic surgeons have discussed the advantages and disadvantages of superficial and deep face-lifts for many years. A clear consensus is difficult because patient variables such as past medical history, anatomy, genetic background, social history (e.g., smoking, alcohol), motivation to have aesthetic surgery, and environment make it virtually impossible to perform a blinded long-term prospective clinical study. Evaluation of facial aesthetic surgery is also difficult because most procedures yield satisfactory results initially, often producing enough improvement to be accepted as a good result.

HISTORY

Although doubt still exists as to who performed the first face-lift, most authorities date the procedure to the early part of the 20th century[3–13] Historically, rhytidectomy procedures may be divided into three main categories: skin excision, subcutaneous undermining, and superficial musculoaponeurotic system (SMAS) manipulation. Early rhytidectomy procedures were limited to skin excision and wound closure without any appreciable subcutaneous undermining.[3,4,8–10,14] Beginning in the late 1920s, the conventional face-lift operation, consisting of skin dissection with subcutaneous undermining, was established.[13]

The subcutaneous rhytidectomy was the preferred technique for many years. A small amount of subcutaneous tissue is elevated with the skin and is simply redraped, leaving the patient with less redundancy. However, this technique does not address underlying skeletal deformities, ptotic deep soft tissue structures, or changes in skin texture. Therefore, it usually results in a more unnatural look and increases the likelihood of complications, particularly skin slough.

In an attempt to improve the results obtained with the subcutaneous face-lift, several clinicians described techniques to correct platysmal banding and submental lipomatosis.[15–18] The third historic category came with the advent of SMAS manipulation. Multiple surgeons have described various techniques involving the SMAS and platysma to enhance cervicofacial rhytidectomy.[1,19–35] In 1974, Skoog[19] described a procedure based on surgical anatomy. At that time, the subdermal plane was accepted by most to be the anatomic limit for face-lifting and rejuvenation. Skoog's technique redraped the skin and platysma together, leaving the patient with a more youthful jaw line.

Subsequently Mitz and Peyronie's description of the SMAS[21] provided an anatomic basis for restoration of

TABLE 67-1. **Generations of Rhytidectomy**

I	Subcutaneous dissection only with variable skin undermining
II	Subcutaneous dissection + SMAS plication or imbrication
III	Subcutaneous dissection + SMAS plication or imbrication + deep midface section dissection
IV	Composite dissection

SMAS = superficial musculoaponeurotic system.

the face. Hamra[35] (initially with the deep-plane and later with the composite rhytidectomy) and, later, Owsley[36] (with the multiplanar/multivector approach) modified and improved Skoog's technique by performing a more complete release of the nasolabial fold. Ramirez[37] showed that after subperiosteal release, the soft tissues of the cheek, forehead, jowls, lateral canthus, and eyebrows can be restored to their youthful relationship with the underlying skeleton. Finally, Watson and colleagues[38] described a technique similar to that of Owsley but combined it with laser resurfacing. This technique involved a larger plane of subdermal or subcutaneous undermining than that described by Hamra.

Four generations of rhytidectomy techniques are recognized (Table 67-1).[39] Current literature has popularized more complex procedures including the deep-plane and composite rhytidectomies.[1,34,35,40] These methods have incorporated multiplanar dissections and craniofacial techniques in an attempt to gain better control of the midface soft tissues. Whether these techniques provide longer-lasting results remains to be shown.[1,34,35,39–42] Multiple authors have cautioned that the more complex deep-plane and composite rhytidectomies typically carry increased morbidity.[1,41,43–49]

PATIENT EVALUATION

When a rhytidectomy is being contemplated, the treatment requirements of the surgeon must be balanced with the desires of the patient (Table 67-2).[50] These requirements are critical for proper patient selection. The patient evaluation must include general medical and psychological considerations in addition to physical facial features. Neglect of any aspect of the patient evaluation can lead to future problems.[51]

A thorough medical evaluation must be completed before surgery. Medical illnesses such as diabetes, hypertension, hypothyroidism, and asthma must be appropriately treated and controlled preoperatively. Medical evaluation is essential for detecting conditions that may adversely affect a patient's ability to tolerate an anesthetic or that may compromise the final surgical result. Appropriate consultations regarding cardiovascular disease, pulmonary disease, coagulopathies, and other active medical problems should be obtained preoperatively. Current medication profiles should be elicited, including aspirin and aspirin-containing compounds, nonsteroidal anti-inflammatory drugs, and herbal drugs, as well as high doses of vitamin E. Examination of prior surgical incisions, such as those resulting from previous thyroidectomies or parotidectomies, is useful in evaluating the wound-healing capacity.

TABLE 67-2. **Rhytidectomy Requirements**

Patient	Surgeon
Minimal morbidity risk	Safe and predictably consistent outcomes
Long-lasting results	Reasonable operative time
Quick recovery	Reasonable cost to patients
Affordable	Reasonable postoperative recovery period
Performed on outpatient basis; will be ambulatory	Adaptable for revision procedures
	Teachable to residents and fellows

Tobacco and alcohol use, as well as the use of illicit substances, may increase the incidence of surgical complications. Cigarette smoking, diabetes mellitus, and previous head and neck radiation therapy may also impede healing and should be recognized preoperatively. Patients should be told that cigarette smoking significantly increases the likelihood of skin flap necrosis, poor healing, and unsightly scars.[52,53] Studies have demonstrated a 12-fold greater risk of skin slough in smokers than in nonsmokers.[54,55] Animal studies involving skin flap experiments have also supported this conclusion.[56,57]

A review of the patient's psychological history should focus on motivation for surgery and outcome expectations. Patients seeking surgery based on recent emotional events or with unrealistic expectations should be further counseled and the procedure postponed or cancelled.[58,59]

The goal of face-lifting is to correct anatomic changes to the face and neck that have occurred as a result of the normal aging process. Patients considered for rhytidectomy present with various degrees of age-related alterations to the facial soft tissues. Ideally, face-lift candidates are 45 to 55 years of age, are in good health, are of normal weight, possess a good bone structure, and possess a thin neck and a deep cervicomental angle. Physical examination of the patient includes a detailed regional evaluation of the face. A face-lift is capable of correcting specific anatomic regions, and a complete and detailed description of the patient's physical characteristics is important. A systematic evaluation allows the surgeon to assess the areas that can be improved with an isolated rhytidectomy and to determine whether other ancillary procedures will be beneficial. In addition, a detailed regional evaluation provides an opportunity for the surgeon and the patient to understand the limitations of the rhytidectomy; the surgeon can refer to the evaluation when explaining the expected outcome.

Evaluation of the upper facial one third includes the forehead and upper and lower eyelids. In general, these areas are not affected by the standard rhytidectomy and are, therefore, not addressed in this review. Evaluation of the middle facial one third includes the face and ears. The quality of the skin should be noted, including thickness, redundancy, previous scarring, fine or coarse rhytids, and the location of the hairline. Patients with significant elastosis or actinic damage do

TABLE 67-3. **Dedo Classification of Facial Profiles**

Classification	Comments
I. Normal	Well-defined cervicomental angle. Good muscle tone No submental fat
II. Cervical skin laxity	Obtuse cervicomental angle owing to relaxed skin
III. Submental fat accumulation	Requires submental lipectomy
IV. Platysma muscle banding	Requires muscle clipping, plication, or imbrication
V. Retrognathia/ micrognathia	Requires genioplasty or orthognathic surgery
VI. Low hyoid	Difficult to alter Important to inform patient of limitation

Adapted from Dedo DD. A preoperative classification of the neck for cervicofacial rhytidectomy. Laryngoscope 1980;90:1894–1896.

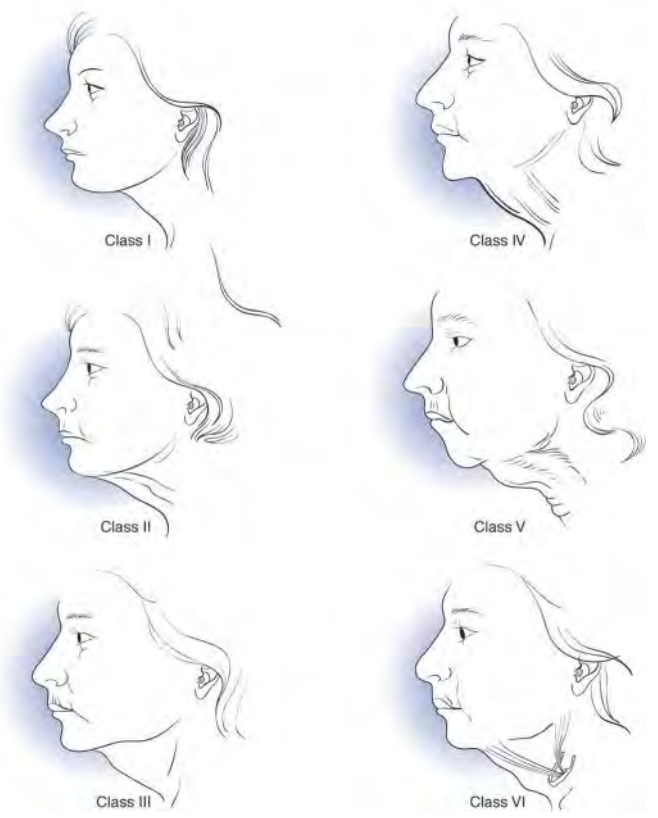


FIGURE 67-1. Six classes of facial profiles by Dedo. (Adapted from Dedo DD. A preoperative classification of the neck for cervicofacial rhytidectomy. Laryngoscope 1980;90:1894–1896.)

not obtain and retain the same quality of results as do patients possessing good skin quality. Assessment of the earlobe shape and position is critical, and any deviations from normality should be pointed out to the patient preoperatively. Documentation of the nasolabial folds, including length, depth, and symmetry, as well as an assessment of the degree of jowls should be noted. Presence of perioral rhytids is important because they are not corrected with rhytidectomy procedures.

The evaluation of the lower facial one third includes the chin, jaw line, and neck. A useful preoperative classification of the neck for cervicofacial rhytidectomy divides the neck profile into six distinct classes (Table 67-3).[60] Using this classification, the clinician can identify each patient's specific abnormality and choose the most appropriate procedure for optimal results (Figure 67-1). Skin redundancy, platysmal banding, cervicomental angle, and submental fat accumulation are important to note in this region. In addition, the degree of ptosis of the submandibular glands should be assessed and recorded at this time.

Preoperative photographs provide invaluable medicolegal documentation as well as an opportunity to review patient characteristics before and during surgery. The surgeon should obtain the photographs of the full face in repose and smiling, right and left profiles, and right and left three-quarter views. Closeups of the forehead, eyebrows, and periorbital and perioral regions should be taken, depending on the particular deformity.

An essential component of the patient evaluation includes the preoperative visit. It is an excellent opportunity to educate the patient regarding various aspects of the procedure, postoperative care, and expectations. It is wise to review any and all patient instructions during the preoperative visit including medications, skin cleaning, and makeup. Patients must discontinue all aspirin-containing compounds, nonsteroidal anti-inflammatory medications, and vitamin E at least 2 weeks before surgery. Germicidal shampoo and skin cleaners are used several days before the scheduled date of the procedure to reduce bacterial counts. The patient should wash his or her hair and face and remove all makeup the night before surgery. A written copy of postoperative instructions should be given to and reviewed with the patient (see Appendix).

A description of the expected convalescence is appropriate during the preoperative visit. In general, 10 to 14 days is a reasonable time period to wait for ecchymosis to resolve sufficiently to allow camouflage with makeup. Patients should not expect to return to work or social activities before this time. They should be educated regarding expectations in the early postoperative period because those unaware of the slow evolution of results will quickly become frustrated. It should be clearly explained that appearance will continue to improve over a period of several months.

SURGICAL TECHNIQUE

Superficial-Plane Rhytidectomy

Once appropriate monitors are placed and the patient is adequately sedated, proposed incision lines and anticipated areas of undermining are marked with a skin marker while the patient is seated in an upright position. The face, neck, and hair are prepared with an appropriate antiseptic surgical scrub. Skin preparations should include the full length of the

planned incision and all exposed skin within the surgical field.

Our preference is to perform rhytidectomies under local anesthesia with appropriate sedation and analgesia. Suitable agents include intravenous fentanyl, midazolam, ketamine, and/or propofol. Local anesthesia is provided along the incision line with a 2% lidocaine solution with 1:100,000 epinephrine via a dental syringe. Hydrodissection is performed within the planned plane of dissection with a tumescent anesthetic technique (Figure 67-2).[61–66]

Although multiple techniques exist for mixing the tumescent solution, our preference is to mix 20 mL of a 2% lidocaine with 1:100,000 epinephrine solution with 180 mL of normal saline, creating a solution of 0.2% lidocaine with a 1:1,000,000 epinephrine concentration. This mixture is administered through four trocar sites: temporal, infralobular, mastoid, and submental (Figure 67-3). The solution is deposited subcutaneously in the supra-SMAS plane.

Hydrodissection should extend 1 cm beyond the proposed undermining mark that delineates the anticipated extent of flap development. Approximately 75 mL of the tumescent anesthetic solution is deposited per side, with an additional 50 mL deposited in the submental region. The anesthetic is allowed to work for 8 to 10 minutes before proceeding.

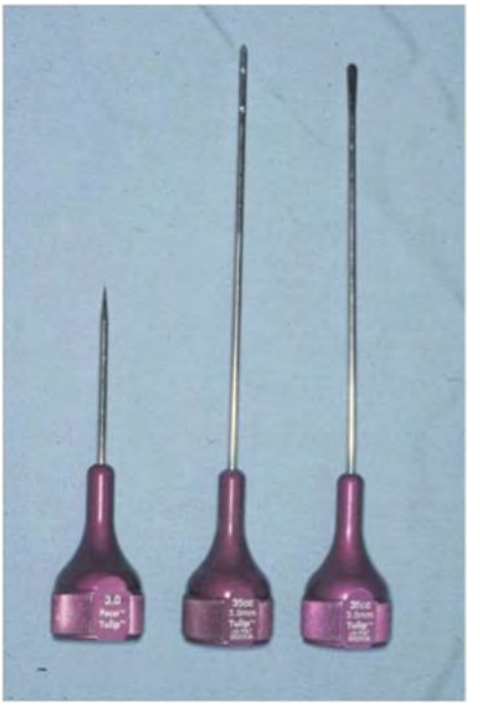


FIGURE 67-2. From left to right: procar/trocar, infiltrator, and dissector used for the tumescent technique.



FIGURE 67-3. Four trocar sites are typically used. **A,** Temporal. **B,** Infralobular. **C,** Mastoid. **D,** Submental.



FIGURE 67-4. Cervicofacial dissection in the supra-SMAS (superficial musculoaponeurotic system) plane without suction.

Typically, the contralateral side is not infiltrated until just before initiating closure on the first side.

Following deposition of the anesthetic solution, blunt cannula dissection of the cervicofacial and submental regions is performed. The cervicofacial dissection is generally performed without suction (Figure 67-4), whereas the submental dissection is performed under suction (Figure 67-5). The submental dissection, using the blunt cannula, is accomplished before the initiation of the cervicofacial dissection. As previously mentioned, the planned incisions should be marked with the patient seated in an upright position, and before administration of the local anesthetic, to prevent distortion of local anatomy. Numerous modifications of the standard face-lift incision are available, but several general principles should be followed closely.[67–70]

It is not necessary to shave the hair in the incision area. If necessary, the hair may be braided along the proposed incision, with the temporal extension placed no more than 2 cm within the hair and parallel to the hairline. Some surgeons recommend a pretrichial incision to prevent the posterosuperior migration of the temporal line.



FIGURE 67-5. Submental dissection in the supraplatysmal plane under suction.



FIGURE 67-6. Temporal, preauricular, and infralobular components of the typical rhytidectomy incision.

The incision should be extended inferiorly toward the root of the helix, anterior to the curve of the crus helicis, following the margin of the tragus, and proceed anteriorly just above the base of the incisura intertragica (Figure 67-6). Preservation of the incisura helps to prevent distortion of the tragus postoperatively, providing a more aesthetic scar. The incision curves inferiorly 1 to 2 mm below the junction of the lobule with the cheek, rising superiorly onto the back of the conchal bowl approximately 3 to 5 mm and reaching the level of the postauricular sulcus or superior crus of the antihelix. The incision is then directed posteroinferiorly approximately 4 to 5 cm into the scalp of the retromastoid region (Figure 67-7).

If significant cervical skin redundancy is noted, the incision may parallel the posterior hairline for several centimeters before being directed into the hair-bearing skin. This maneuver can prevent a step deformity in the posterior hairline when significant cervical skin is excised. Care must be taken to maintain approximately a 90-degree angle at the reflection of the posterior flap to prevent skin slough at the tip of the flap.

Several considerations for incision design are important in the male patient.[18,71] In the temporal region, the planned incision is affected by the patient's hair pattern. Individuals with thick temporal hair can tolerate a standard incision. Those with thinning hair, temporal recession, or significant male pattern baldness require a modification of the incision design.

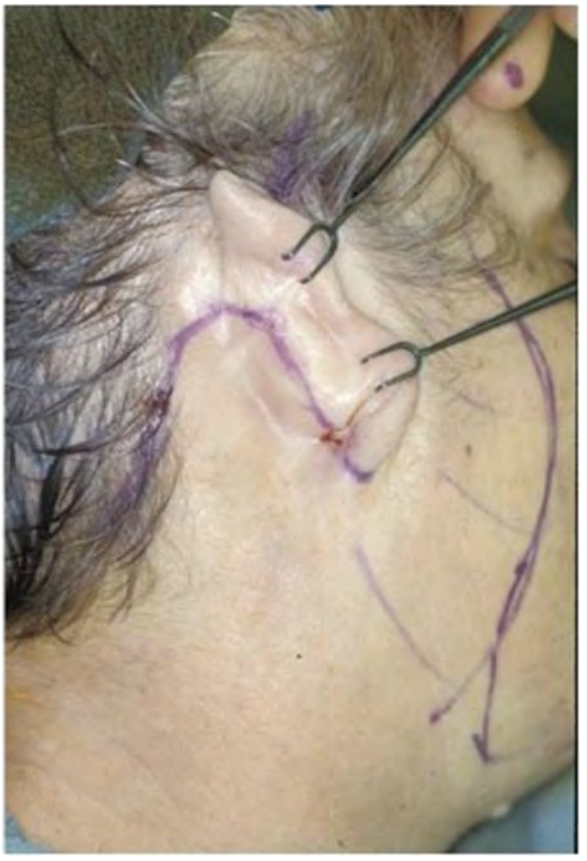


FIGURE 67-7. Postauricular and mastoid components of the typical rhytidectomy incision.

In the preauricular region, the male sideburn must be taken into consideration and the non–hair-bearing skin anterior to the ear must be left intact to prevent an unnatural appearance. Therefore, the incision should extend in a linear fashion, following a natural skin crease adjacent and parallel to the sideburn. This is in contrast to the curved incision used in female patients.

Last, the posterior extension of the incision is placed along the margin of the postauricular hairline. Although this placement has the potential to be slightly more noticeable, the importance of preventing posterior displacement or a step deformity to the hairline must be taken into account. A final consideration in the male patient is the inevitable transfer of hair-bearing skin into the postauricular region or into the ear canal itself.

Management of the cervical region, if indicated, is begun with the placement of a transverse incision in a submental skin crease as an extension of the submental trocar puncture (Figure 67-8). This incision should not be placed in the dominant crease of the "double-chinned" deformity, because scarring may result during the healing process and accentuate the crease. The incision should be approximately 2 cm in length and placed just inferior to the dominant crease.

Dissection in the submental region proceeds in a subcutaneous plane and joins the lateral subcutaneous neck planes



FIGURE 67-8. Submental incision placed to address platysmal banding.

subsequent to the submental dissection. Excess subcutaneous fat may be excised with sharp scissors or a cannula, if indicated. Lipectomy in the submental region should be done cautiously. Overly enthusiastic removal of fat in this region can lead to an atrophic appearance of the cervical facial tissues or a "cobra" neck deformity.[17]

Next, the anterior borders of the platysma bands are identified, and excess subcutaneous tissue is removed. The medial borders should be released along their deep surface from the submental region inferiorly to the level of the thyroid cartilage. The medial borders are then repositioned in the midline, and any overlapping tissue is excised. Plication of the medial platysma borders proceeds from the submental region to the level of the thyroid cartilage with a 2-0 slow-resorbing suture (Figure 67-9).

A horizontal myotomy of the inferior aspect of the platysma may be beneficial in accentuating the cervicomental angle and relieving tension along the anterior surface of the neck. Partial horizontal transection is frequently all that is required and can be performed with sharp scissors at the inferiormost aspect of the dissection of the medial borders. If complete transection is indicated, the lateral aspect of the platysma can be excised through the lateral neck face-lift flap.[72–75]

Flap Development

Flap development is initiated by undermining 1 cm along the entire length of the face-lift incision. This is accomplished in a subdermal plane with a blade or sharp scissors, using skin hooks for retraction (Figure 67-10). On the underside of the flap, maintain approximately 3 to 4 mm of subcutaneous fat to preserve the subdermal vasculature.

In the temporal region, the depth of the flap should be carried through the temporoparietal fascia (subgaleal) down to the loose areolar tissue overlying the deep temporal fascia. Dissection in this plane creates a thicker flap, providing increased protection from any ischemic injury that would damage hair follicles and create subsequent alopecia. The temporal dissection is extended in this plane using a combination of blunt and sharp dissection.

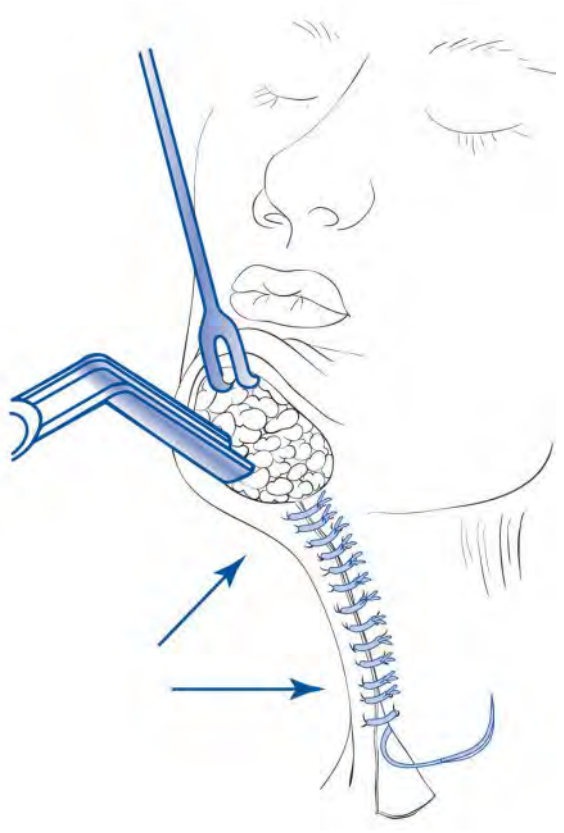


FIGURE 67-9. Platysmal plication accomplished.

Blunt-tipped scissors should be used in a push-cutting motion into the residual tunnels that were developed secondary to the previous blunt cannula dissection (Figure 67-11). Rees T-clamps are used for countertraction and aid in the dissection. Undermining in this plane can be safely carried superiorly to the level of the lateral canthus.

At this point, the dissection is carried inferiorly and medially across the cheek in the subcutaneous plane. This zone of transition from the sub-SMAS (deep to the temporoparietal fascia) to the subcutaneous plane of dissection corresponds



FIGURE 67-10. Flap development initiated by 1 cm undermining via a blade or sharp dissection scissors.



FIGURE 67-11. Scissors are used in a push-cutting motion with the tips up.

to the mesotemporalis that contains the superficial temporal artery and the frontal branch of the facial nerve.[76–79] The facial nerve is located anterior and inferior to the frontal branch of the superficial temporal artery, so preservation of this vessel during dissection helps to protect this important nerve.

The extent of undermining necessary depends on the patient. Younger patients without excessive laxity of the skin require only 4 to 5 cm of undermining, but older patients with redundant tissue and severe jowling may require undermining to within 1 cm of the oral commissure. When platysma banding is present, the cervical dissection is carried inferiorly to the level of the thyroid cartilage and should communicate with the dissection from the contralateral side.

In the postauricular region, the flap should be developed in the subcutaneous plane below the earlobe to protect the great auricular nerve. This nerve is the one most commonly injured during rhytidectomy procedures (Figure 67-12).[80,81] The great auricular nerve runs just deep to the superficial fascia overlying the sternocleidomastoid muscle and supplies sensation below and behind the ear. With the head turned 45 degrees toward the contralateral side, the great auricular

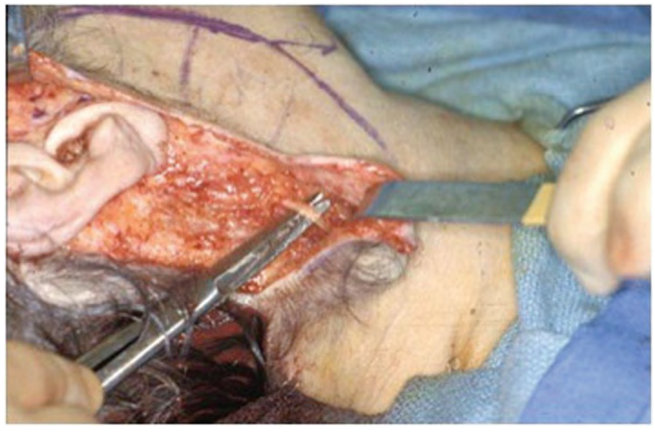


FIGURE 67-12. Great auricular nerve demonstrated.

nerve consistently crosses the middle of the sternocleidomastoid muscle at a level 6.5 cm below the caudal edge of the bony external auditory canal (Erb's point).[80] Maintaining subcutaneous dissection below the earlobe helps protect the great auricular nerve, but above the level of the earlobe, the greater auricular is not at risk and dissection may be carried deeper.[81] Meticulous hemostasis is then accomplished with bipolar cautery. Overzealous use of electrocautery should be avoided on the skin-flap side to reduce the risk of ischemic injury and flap necrosis.

Next, the underlying SMAS in the cheek region is manipulated. Many of the age-related changes in the facial region are due to ptosis of the underlying fat. Correction of these changes is best obtained by repositioning this tissue along direction lines (vectors) different from those used for the skin flap. Independent bidirectional suspension of the SMAS and the skin flap reposition the ptotic facial tissues, providing longer-lasting results and reducing an unnatural appearance.

SMAS manipulation can be by plication, imbrication, or a combination of these techniques; there are proponents of each method. *Plication* is a technique whereby the SMAS is folded upon itself to obtain the desired repositioning, and *imbrication* is a technique in which the SMAS is incised or excised so that the distal portion is repositioned to overlap the proximal tissue (Figure 67-13).

SMAS plication is accomplished with a 2-0 slow-resorbing suture on a tapered needle (Figure 67-14). All knots are buried to prevent irritation to the skin flap or palpability. Two key sutures are placed initially, with the first extending from the fascia overlying the angle of the mandible to the fascia immediately inferior to the tragus. The second

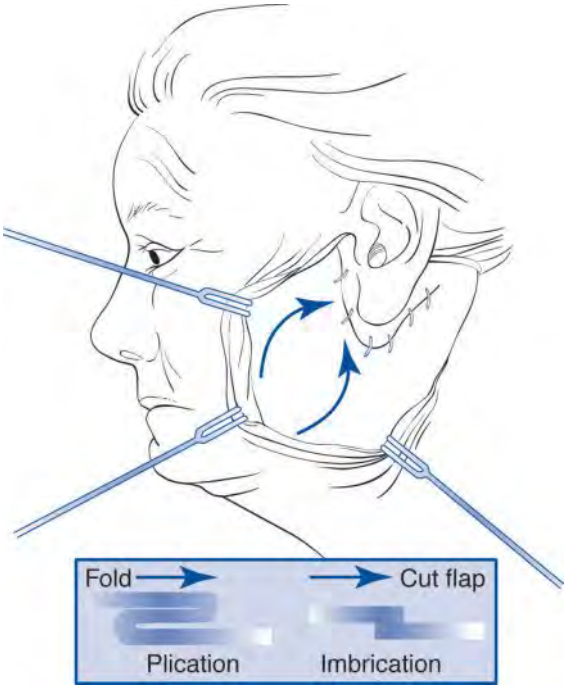


FIGURE 67-13. Illustration of plication versus imbrication.



FIGURE 67-14. The area of plication is assessed.

suture is placed from the fascia lateral to the oral commissure to the fascia immediately superior to the tragus. Several additional sutures may be placed, if needed, in the preauricular and postauricular areas. This suture placement provides a posterosuperior repositioning of the ptotic tissues.

Imbrication requires elevation of a sub-SMAS flap. An incision is made horizontally just inferior to the zygomatic arch and vertically posterior to the angle of the mandible. Landmarks for the incision include the zygomatic arch, tragus, platysma muscle, and mandible (Figure 67-15). The horizontal incision is made approximately 1 cm below and parallel to the zygomatic arch to prevent damage to the frontal branch of the facial nerve. The middle portion of the tragus may be used as a reference for staying below the zygomatic arch.

The incision is carried forward 2 to 3 cm. The vertical incision descends inferiorly along the posterior border of the platysma several centimeters below the angle of the mandible. It is important to keep the incision posterior to the angle of the mandible to prevent damage to the marginal mandibular branch of the facial nerve. The SMAS is then elevated for 2 to 3 cm in a sub-SMAS plane (Figure 67-16). The flap is



FIGURE 67-15. The area of proposed resection in preparation for imbrication is marked.



FIGURE 67-16. After SMAS flap elevation, 2-0 resorbable suture on a tapered needle is used for buried knots to secure the imbrication.



FIGURE 67-17. After the placement of key staples, flap trimming is accomplished with a blade or Iris scissors.

redraped posterosuperiorly and sutured using the technique previously described for plication.

Redraping of the skin flap is performed with the patient's head in a neutral position. Extension or flexion of the neck influences the amount of skin excised and may adversely affect the outcome. Key suspension staples or sutures are placed before trimming the skin flap. In general, the skin flap is redraped in a posterosuperior direction with an emphasis on the posterior direction. The majority of the needed superior suspension is accomplished by SMAS manipulation. Care should be taken to prevent a misdirection of facial rhytids and a distortion of the temporal hairline. Careful assessment of appearance should be made before suturing the flap in place.[82]

The first key skin-suspension suture (or staple) is placed in the temporal region just above the ear. The flap is then grasped, and the appropriate vector is determined and held in place while the flap is trimmed and the staple placed. The second staple is placed in the postauricular region at the most posterior and superior aspect of the flap. Careful attention should be given to proper inset of the ear. In a nonoperated ear, the long axis of the ear lobe hangs 10 to 15 degrees posterior to the long axis of the ear proper. This relationship must be maintained to prevent obvious deformities of the ear.

Skin Closure

Trimming of excess skin is performed with a blade or Iris scissors (Figure 67-17). It is important to be aware of the amount of skin to be excised in the temporal region to prevent distortion of the hairline. The distance from the lateral canthus to the anterior margin of the temporal hairline should be recorded preoperatively to serve as a reference for skin excision. Skin closure in the hair-bearing scalp (i.e., temporal and mastoid regions) is performed with subdermal 3-0 resorbable sutures. Staples may be used to approximate skin margins of the temporal and mastoid scalp. Our preference is to provide a layered closure to minimize tension on the most superficial aspect of the skin.[82] The immediate postauricular region is closed with a 4-0 plain gut suture, without the need for deep sutures. In the preauricular region, a 4-0 resorbable suture is placed followed by approximation of the skin edges with a 6-0 or 7-0 nylon running suture. The deep layers of the submental incision are closed with 4-0 resorbable suture, and 6-0 nylon is used for the skin edges.

The decision of whether to place drains must be made on an individual basis, depending on how much oozing or edema is present. In our experience, drains are rarely needed. Antibacterial ointment and gauze dressings should be placed along the incision lines. Gauze is also placed preauricularly and postauricularly as well as in the submental region. The entire face should then be wrapped, taking care to prevent excessive tightness of the dressing because it can lead to ischemia of the flaps. In addition, the appropriate positioning of the ears under the dressing should be noted. Dressings should not be manipulated for evaluation until the first postoperative day. Most complications in rhytidectomy occur early in the postoperative period (i.e., 24–36 hr); close follow-up is, therefore, essential.[83–87] Some surgeons prefer to use no dressings postoperatively.[88] The degree of edema and ecchymosis postoperatively is frequently underestimated and can be quite shocking to the patient, despite the surgeon's best attempts to educate her or him. The patient's hair should be gently cleansed and rinsed for additional comfort. Incision lines should be cleansed daily with a 1:1 solution of hydrogen peroxide and water. Antibacterial ointment should be used until all sutures are removed.

The preauricular sutures are removed after 4 to 5 days, as are the staples in the temporal and mastoid regions after 10 days. Patients should be instructed not to wash their hair until all sutures have been removed and then to wash only gently with baby shampoo. Written suggestions for the avoidance of ultraviolet light and high heat from hair dryers and for the use of sunblock and incision massage should be given to the patient and repeated orally in the early postoperative visits. The effects of the procedure are still striking for years after surgery (Figure 67-18).



FIGURE 67-18. Typical result achieved with a superficial-plane rhytidectomy including SMAS imbrications and upper blepharoplasties. **A** and **B,** Preoperative photographs. **C** and **D,** Photographs taken 2 years postoperatively.

COMPLICATIONS

Based on the literature, complication rates tend to be lower for superficial rhytidectomy techniques than for the more complex composite and deep-plane techniques.[1,34,35,39,46,47,49,50] Major types of surgical complications include hematoma, facial nerve injury, skin loss, scarring, alopecia, auricular deformities, and temporal hairline deformities.

Major Complications

Hematoma

Hematoma formation is the most common major complication that results from rhytidectomy. It occurs postoperatively in roughly 2% to 4% of patients within the first 48 hours; the cause varies.[89] Early recognition is essential because untreated hematomas are associated with an increased risk of skin slough (Figure 67-19). Typical signs and symptoms of hematoma include increased facial pain, tightening of dressings, ecchymosis of the buccal mucosa and lips, and bulging of the lips. Hematomas are best treated with early exploration of the wound, with the most common finding being diffuse oozing under the skin flap. Removal of clots is indicated, followed rarely by bipolar cautery or suture ligation of larger vessels (Figure 67-20). Pressure dressings and drains may be used to complement these techniques but are no substitute for meticulous hemostasis. Factors associated with increased risk of



FIGURE 67-19. Hematoma present in early postoperative period.



FIGURE 67-20. Early exploration and clot evacuation are essential in preventing skin slough. Clot being removed from left temporal region.

hematoma formation include preoperative hypertension, intraoperative hypotension, and postoperative hypertension.

A review of 500 face-lifts demonstrated a 2.6% incidence of hematoma formation; however, in this same population, patients with preoperative hypertension had a 9.2% incidence of hematoma formation.[90] Preoperative hypertension in this review was associated with a 2.6-fold increase in the risk of hematoma formation. Conversely, intraoperative hypotension was associated with increased hematoma formation, probably as a result of rebound hypertension. In a review of 806 face-lifts, Rees and Aston[91] reported 23 hematomas (2.8%), with 20 of the 23 associated with controlled intraoperative hypotension. Maintenance of a normotensive state during surgery was associated with a decreased incidence of hematomas.

In another review of 1236 face lifts by Rees and coworkers,[92] a 1.9% incidence of hematoma formation was noted. The incidence drops to approximately 1% in normotensive general anesthetic states. Postoperative hypertension also has been associated with an increased risk of hematoma formation.[93] The average maximum blood pressure postoperatively in the preceding study was 152/98, a relatively minor elevation in blood pressure. Kamer and Kushnick[94] described an increase in hematoma formation when using propofol as the intraoperative anesthetic agent. With propofol, the overall incidence of hematoma formation was 4.2%. A combination of valium, demerol, and brevital had only a 2% incidence of hematoma formation, however. Finally, recent studies show a decrease in the incidence of hematoma formation with the use of fibrin glue sprayed under the flap before closure.[95]

Patient cooperation is helpful in the prevention of hematomas. Instructions for the reduction of hematoma formation include eliminating aspirin, aspirin-containing compounds, nonsteroidal anti-inflammatory drugs, and vitamin E for at least 2 weeks preoperatively and avoiding exertion and bending for 2 weeks postoperatively. The importance of patient compliance should be stressed in the preoperative visits.

Facial Nerve Injury

The facial nerve branch injured most often varies with different reports in the literature.[85,96] In the author's experience, the marginal mandibular and temporal (frontal) branches of the facial nerve are injured most often. These two branches are the most superficial and have less crossover anastomosis than other branches of the facial nerve. Injury to the marginal mandibular branch invariably occurs during extension of the subcutaneous dissection anteriorly to the lateral chin region, where the nerve courses superficial to the depressors of the mouth. Less commonly, it may be damaged during dissection in the region of the mandibular angle (Figure 67-21). Injury to the temporal branch frequently occurs during temporal dissection because the plane is in transition from subgaleal to subcutaneous levels. Damage to these structures may result in temporary or permanent motor deficits to the respective muscles of facial expression.

Compiled reviews of face-lifting have demonstrated a 0.7% (50 of 6500) incidence of temporary injury and a 0.1% (7 of 6500) incidence of permanent injury.[76] Facial nerve injury is less likely with SMAS placation techniques versus SMAS imbrication or more complex composite and deep-plane techniques.[48,82] Rates of facial nerve injury with sub-SMAS dissections have been reported as high as 16%.[97] Prevention of facial nerve injury is achieved through a complete understanding of face-lift anatomy and the usual course and common variations of the facial nerve and meticulous attention to detail during the procedure. Most surgeons would agree that injury to the facial nerve is the most devastating complication associated with rhytidectomy; fortunately, permanent injury to the facial nerve is rare.[1,39,44,46,47,48,76,97]



FIGURE 67-21. Patient demonstrates typical findings associated with unilateral injury to the marginal mandibular branch of the facial nerve.

Skin Slough

Skin slough is the result of vascular compromise to the involved soft tissue flap. It most commonly occurs in the postauricular and mastoid regions because of the thinness of the skin and it being farthest from the blood supply of the cervicofacial skin flap (Figure 67-22). Fortunately, small sloughs in this region are concealed by the hair and ear. The incidence of postoperative skin slough is approximately 2% to 3%.[85]

Delay in the evacuation of hematomas is a major cause of skin slough and underlies the importance of early detection. Hematomas of significant size prevent the reestablishment of nutrient flow to the skin flap from the richly vascularized underlying tissues and cause tension within the flap, which creates further ischemia. When skin necrosis does occur, a black eschar forms that separates at approximately 1 week, and the wound then heals by secondary intention. Reassurance should be given to the patient because most areas of minor skin necrosis heal without major sequelae. A minimum of 3 to



FIGURE 67-22. Most common site for skin slough located in the postauricular region.

6 months should elapse before any major surgical revision is undertaken.

Local ischemia may result from various factors including diabetes mellitus and tobacco use. Without a doubt, cigarette smoking increases the risk of skin slough.[52–57] Nicotine has been implicated as a factor that predisposes patients to skin necrosis by affecting several aspects of wound healing, including alteration of epithelialization, impairment of the inflammatory phase of healing, and compromise of small blood vessel flow.[98] In evaluating the effects of tobacco on face-lifts, Rees and associates[52] demonstrated a 12-fold greater risk of skin slough in smokers compared with nonsmokers. Webster and colleagues[53] advocated a more conservative undermining (short flap technique) in face-lift patients who smoke. It is the author's opinion that abstinence from smoking for at least 2 weeks—and preferably 1 month—is recommended before operating on a patient who has not stopped smoking. Improper tissue manipulation and flap design are easily preventable causes of skin slough that result from inexperience. Delicate handling of skin flaps is essential during elevation to prevent a compromised result. Skin flaps should be designed to maintain an adequate thickness of subcutaneous tissue on their deep surface (3–4 mm) and, in the postauricular region, to maintain a 90-degree angle at the tip of the flap.

Neurosensory Disturbances

Neurosensory disturbances, particularly transient numbness or hypesthesia, are common in the early postoperative period as a result of elevation of the skin flap and interruption of small sensory nerves. As a result, a temporary, diffuse cutaneous numbness that lasts 4 to 6 weeks is not uncommon. During this time, patients should be warned about possible injury to numb skin from razors, curling irons, and hair dryers. The most common nerve injured during rhytidectomy is the great auricular nerve. Injury to this nerve is the result of an improper plane of dissection over the sternocleidomastoid muscle. Studies have shown the great auricular nerve to cross the middle portion of the sternocleidomastoid muscle approximately 6.5 cm below the caudal edge of the bony external auditory canal, which courses cephalad just deep to the SMAS.[80,81] Injury to the great auricular nerve occurs when the postauricular dissection is carried too deep, thereby violating the strict subcutaneous plane in this region.

Minor Complications

The significance of a complication may vary according to the patient's ability to deal with the complication. A particular patient may be frantic over a mild postauricular skin slough, whereas another patient may demonstrate minimal to no anxiety associated with marginal mandibular nerve weakness. Although the authors do not discuss minor complications in detail, complications that occur after rhytidectomy may include widened scars, auricular deformities, edema or ecchymosis, loss of temporal hair tuft or temporal alopecia, and sialoceles.[89,96,99] When complications occur, the surgeon must acknowledge them to the patient and take appropriate measures to correct the problem. Availability of the surgeon in the postoperative period is the key to successful management of most postoperative complications.

SUMMARY

Since the early 1980s, numerous rhytidectomy techniques have been described to improve the results of facial rejuvenation. When contemplating facial rejuvenation surgery, the treatment requirements of the surgeon must be balanced with the desires of the patient. These requirements are critical in defining the advantages of one technique of face-lifting over another. As we prepare our patients for aesthetic surgery, we must consider all the variables discussed and individualize each treatment plan. Not all patients should undergo a deep-plane face-lift; however, many will benefit from release of the retaining ligaments and a repositioning of tissues in multiple vectors.

In the realm of oral and maxillofacial surgery, the rhytidectomy procedure finds great application as an adjunct to traditional skeletal surgery. It seeks to reverse the effects of gravity and relaxation of the facial skin and fascia by resuspending the facial units and eliminating excess skin and subcutaneous tissue. Although a wide variety of techniques are reported for face-lifting, this chapter has attempted to provide a broad overview of the superficial-plane rhytidectomy technique. By tailoring the approach to the specific needs of each patient, based on a thorough knowledge of surgical anatomy, the maxillofacial surgeon should achieve consistently good results with minimum morbidity.

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Appendix Postoperative Rhytidectomy Instructions

IMMEDIATELY UPON ARRIVING HOME

Head elevation: Lie down with your head and back elevated on two pillows. You must sleep in this position for 1 week.

Dressings: Do not remove bandages. These will be removed at the office on your first postsurgery visit.

Ice packs: Place ice packs (ice in freezer bags or packages of frozen peas) over the cheek areas on and off over a period of 24 hours. *Do not* put ice on after 24 hours unless you are told to do so.

Swelling: Ice packs will keep swelling and bruising to a minimum.

Bruising: Bruising often lasts 7 to 14 days.

Medication: Take pain medication *only if needed* and with food or crackers.

Diet: Upon arriving home from surgery, begin with clear liquids until fully awake; then begin regular food intake with soft foods.

Suture care: Keep all sutures clean with a peroxide-water solution. Keep sutures covered with antibiotic ointment at all times. Clean three to five times per day.

ONE DAY OR MORE AFTER SURGERY

Moist heat: Ice packs are to be discontinued 24 hours after surgery. *Wait 12 hours;* then you may begin moist heat. Use a moist washcloth between an electric heating pad and your face. Do not use heat continuously; for example, use for 30 minutes and then off for 30 minutes. *Do not* set the heating pad higher than *medium* at any time, regardless of how cool it feels to you.

Activity: Stay up as much as possible. Avoid bending over or lifting heavy objects for 1 week. Strenuous activities should be limited for 2 to 3 weeks.

Work: Most people plan to return to work in 2 weeks. This depends on how you feel about being seen with bruising. Most of the bruising can be masked with makeup if you prefer to return earlier.

Makeup: Cosmetics may be applied on the sixth day. Ask about special coverup products for bruising. Mint green coverstick followed by a flesh-tone foundation will cover most bruises.

Bathing: You may bathe or shower, but keep the bandages dry. When bandages are removed, gently wash the facial areas.

Hair care: You may wash your hair on the fifth day after surgery. *Do not bend over* to wash your hair; this may cause bleeding or swelling to occur. Use medium heat on your hair dryer. High heat or hot rollers should not be used for 7 to 10 days. You may use color on your hair in 3 weeks.

Diet: Eat regular but soft meals. You will need to take vitamins and minerals to help with the healing. We will be glad to give you vitamin and mineral information.

Sun: Protect your facial skin from excessive sun exposure for 1 month after surgery.

PLEASE REPORT ANY OF THE FOLLOWING TO OUR OFFICE

Excessive pain or bleeding
Itching or rash around stitches
Oral temperature > 100°F (37.8°C)
Excessive swelling/bruising, fatigue, or depression

68

CHAPTER

Forehead and Brow Procedures

Angelo Cuzalina, MD, DDS

Upper facial cosmetic surgery has enjoyed an unprecedented increase in popularity over the past decade. The yearning of baby boomers to look and feel rejuvenated has led to new endoscopic techniques aimed at creating a more youthful and natural appearance with shorter recovery periods than existed in past decades.[1–3] Yet some older techniques, such as an open coronal browlift, are still used routinely by some surgeons with wonderful results. Variations on the hairline or trichophytic open brow lift may be the perfect treatment for a patient with an extremely high highline and brow ptosis. The ultimate goal of improving a person's appearance remains unchanged. Society shapes our views of what looks attractive, and no mathematical formula can ever be used to determine an ideal eyebrow position (Figure 68-1). Each individual has his or her own unique perception of facial beauty. For most people, the upper face and eyes impart more emotion than does any other part of the human body; it is clear that rejuvenation of this vital area can provide an aesthetically pleasing result.

Aesthetic concerns of the forehead and brow regions of the face affect a wide range of age groups. Unlike the standard lower face and neck rhytidectomy, which more commonly affects patients after the age of 45 years, cosmetic concerns in the upper third of the face may be evident for patients in their 20s and 30s owing to genetic predisposition. The forehead and brow area must be entirely evaluated for a wide range of interlacing diagnoses. Matching the problem(s) to the ideal rejuvenation technique(s) is essential for maximum aesthetic benefits. Thinning skin and laxity due to age and gravity encompass only a portion of the forehead and brow dilemmas that must be addressed when planning rejuvenation procedures (Figure 68-2).

The aging process typically leads to forehead and brow ptosis on almost every patient; however, it is important to distinguish whether the ptosis in the forehead and brow region is owing to problems with brow position, upper eyelid laxity, or a combination of the two (Figure 68-3). Other problems such as dynamic lines caused by muscle activity in the glabellar region, variable hairline patterns, bony abnormalities, and asymmetries, as well as skin texture itself, must also be assessed in relation to one another. Achieving the patient's desired expectation depends not only on sound surgical skill and judgment but also critically on communication between the surgeon and the patient. Truthful disclosure of what can reasonably be attained is prudent and helps to prevent patient dissatisfaction.

Rejuvenation of the upper third of the face is one of the most rewarding and fulfilling procedures a surgeon can offer to select patients. Specific elevation and correction of lateral hooding can be appear natural and still impart a tremendous improvement in the patient's overall beauty and youthful appearance (Figure 68-4). The goal of this chapter is to review the upper third of facial anatomy specific to forehead and brow rejuvenation techniques and to discuss a variety of the most common techniques for rejuvenating the forehead and brow region.

ANATOMIC AND AESTHETIC CONSIDERATIONS

It is generally accepted that a youthful forehead is roughly one third of the overall facial height.[4–9] Essentially, the distance from the hairline to the glabella is equal to the distance from the glabella to the point at the base of the columella or subnasale (Figure 68-5). A youthful-appearing eyebrow is different for men and women. The female eyebrow should be arched with the highest point of the brow on a sagittal line from the lateral canthus.[10,11] The entire brow itself should be above the orbital rim. In general, the medial brow of the female is located ideally 1 to 3 mm above the orbital rim and the lateral third of the brow 5 to 10 mm above the rim.[12] This is in contrast to a typical male eyebrow that should lie at or only slightly above the orbital rim in a more horizontal or uniform arch fashion (Figure 68-6). Elevating the lateral

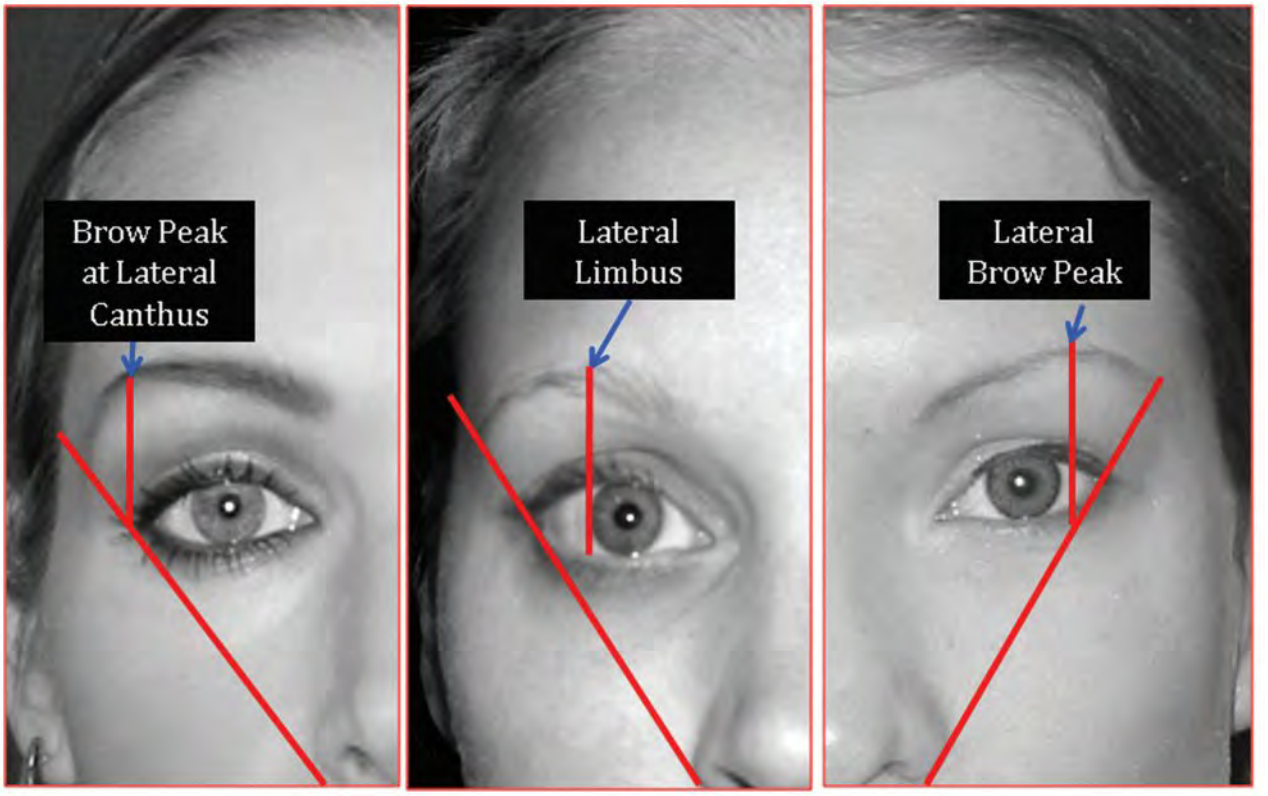


FIGURE 68-1. Three different types of aesthetically pleasing foreheads and eyebrow position. The tail of the eyebrow is located along the alar-canthal line. The greatest brow arch is seen in the lateral third between the lateral limbus and the canthus of the eye. The outer half of the brow is "ideally" located 5 to 10 mm above the orbital rim in females.

third of the male eyebrow disproportionately more than the remaining brow will create a feminine appearance.

The detailed anatomy of individual areas has been well described in the literature and often relates to the specific procedure being performed.[13–25] Therefore, the following anatomic discussion is simplified by separating the specific regions into bony landmarks, muscle and fascial anatomy, vessel and nerve anatomy, and specific endoscopic anatomy; each anatomic region is addressed individually as it relates to specific surgical procedures.

Bony Landmarks

Bony landmarks of the forehead and brow region can be focused all around the frontal bone, which makes up the highest percentage of the upper third of the face. The connections (suture lines such as the nasofrontal, zygomaticofrontal, and coronal) are important landmarks because they can be clinically relevant for limits of dissection and can help surgeons determine their location during dissection. For instance, the zygomaticofrontal suture line is an ideal location to end most basic brow lift dissections (Figure 68-7). Additional dissection can be performed if midface lifting is also planned or if the patient desires more elevation at the lateral canthal region. Overaggressive dissection here in many patients can create an unnatural cat's-eye appearance, particularly if too much tissue is elevated medially along the suture line and lateral canthus. Likewise, the nasofrontal suture line is a nice landmark to note during dissection for a few reasons. First, dissection usually needs to proceed only a few millimeters below this suture level onto the nasal bones for adequate release. Second, the paired procerus muscles can be identified here and transection performed if required. Third, depending on the level of horizontal transection in this area, the nasofrontal angle point of take-off can be altered slightly if desired. Last, nasal tip rotation can be achieved if wanted, especially with significant dissection below the nasofrontal suture line.

Another general bony landmark is the orbital rim, which limits inferior dissection but must be well visualized and free of periosteal attachments to lift the brow and brow fat pads for long-term results. Important muscle and fascial attachments are also located at the level of the orbital rim medially and laterally. The tenacious temporal fusion line (zone of fixation) that exists along the temporal ridge must be identified during dissection.[26,27] It is also important to know its location preoperatively so that proper incision placement can be made to facilitate a clean dissection under this area that enhances visualization endoscopically (Figure 68-8).

Bony thickness varies in different areas of the skull. In addition, venous lakes present on the inside surface of the skull tend to be more centralized around the sagittal suture

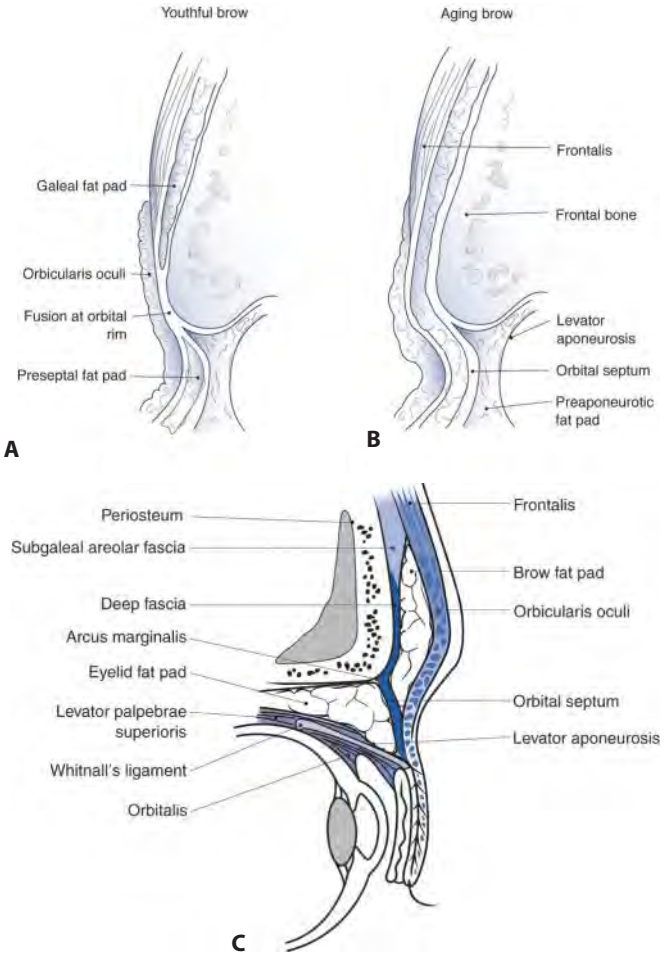


FIGURE 68-2. **A,** The youthful brow is elevated proportionately and has densely adherent periorbital fascia and muscle. **B,** Brow descent due to aging and the associated loss of fascial integrity, along with orbital fat prolapse. **C,** Cross-section of the brow near the midpupillary position.

line. If bone tunnels or screws are planned for fixation purposes, the midline should be avoided, if possible, because of the sagittal sinus as well as higher-density venous lakes in this area (Figure 68-9). Thickness does increase posteriorly near the occiput, but screw or bone tunnel fixation here is more challenging and is not required. Caution must be taken also to avoid lateral placement because of thinness of the lateral skull and the middle meningeal arteries. Knowledge of average thickness for a given location and internal anatomy indicates that the safest location for bone tunnels or screws is located along a parasagittal line approximately at the midpupil or lateral limbus line and just anterior to the coronal suture (see Figure 68-9).

Muscle and Fascial Anatomy

Paired muscles of the forehead and brow region are often thought of as elevators and depressors. Although several depressor muscles can pull the brow down or obliquely, the only true elevator of the forehead, the frontalis, moves upward to raise the brow. This movement, along with some static tone, maintains brow position but also can lead to horizontal creases over time. The frontalis originates from the deep galeal plane (galea aponeurotica that connects to the occipitalis posteriorly). It inserts into the orbital portion of the orbicularis oculi, which inserts into the dermis immediately below the eyebrow. Its lateral extension fuses into the dense collection of fascia almost 1 cm wide, called the *zone of adherence*, which extends along the superior temporal line and ends inferiorly just above the zygomaticofrontal suture.

The fascial attachments, known as the *orbital ligament* (see Figure 68-7), are the inferior termination point of the zone of adherence near the orbital rim where connective tissue fibers of the temporoparietal fascia are fixated to the bone at the superolateral orbital rim (Figure 68-10). Lateral

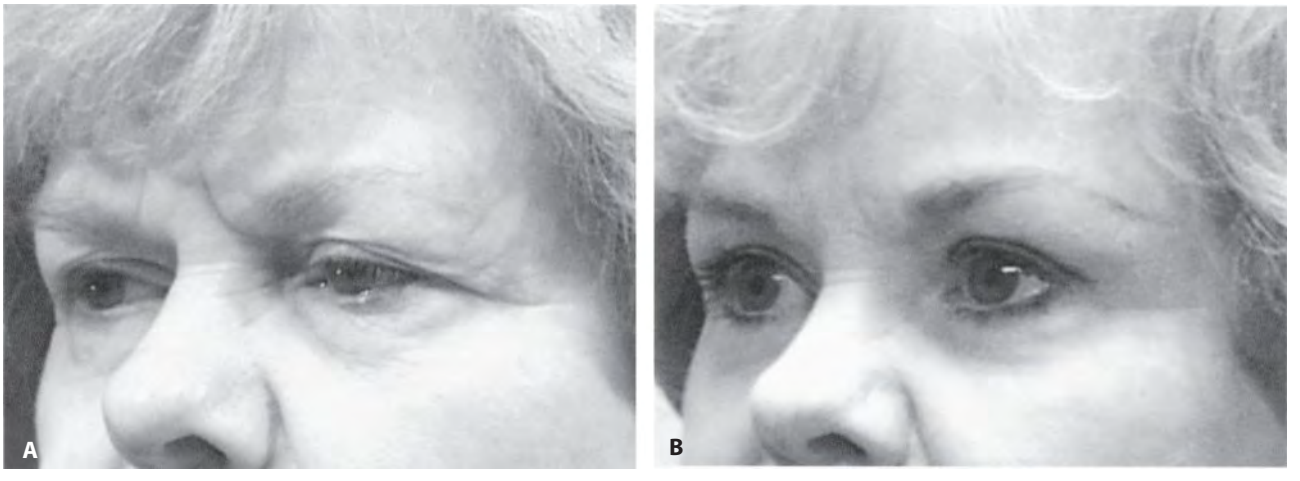


FIGURE 68-3. **A,** Rejuvenation of the upper third of the face must address whether the problem is limited to brow ptosis, eyelid ptosis, or a combination of both, as seen in the patient on the left. Skin texture must also be evaluated. **B,** One month after a coronal brow lift, upper blepharoplasties, and full-face laser resurfacing.



FIGURE 68-4. **A,** Preoperative view of patient with classic lateral hooding brow ptosis and only "pseudo" upper eyelid laxity or ptosis. **B,** One week after endoscopic forehead and brow lift only. (Slight overcorrection is noted in this early period.) **C,** Correction of lateral hooding with isolated brow lift after 1 month.



FIGURE 68-5. **A,** Example of ideal facial proportions based on vertical facial thirds and horizontal proportions approximately the width of the eye or one fifth of the facial width. **B,** Preoperative. **C,** Six weeks after endoscopic forehead and brow lift along with laser skin resurfacing.

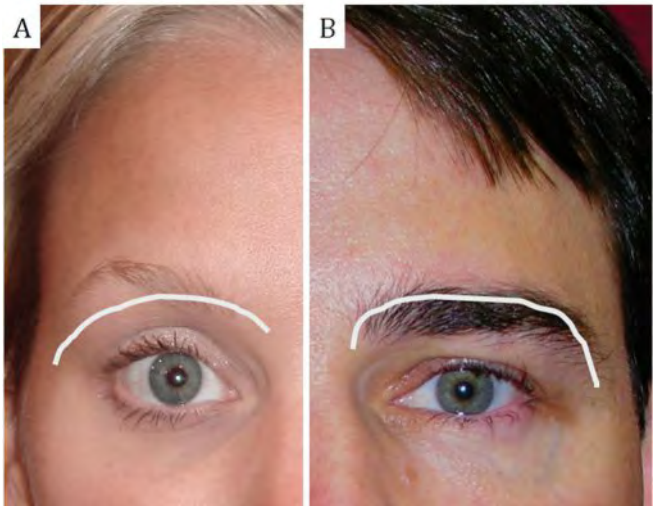


FIGURE 68-6. **A,** Female brow shown with a nicely accentuated arch in the lateral third well above the orbital rim. **B,** The average male brow position is level with the orbital rim with a symmetrical arch form.

and posterior along a near-horizontal line from the orbital ligament is the orbicularis-temporal ligament, which is the transverse fusion zone of fibers from the lateral orbicularis, the temporoparietal fascia, and the temporalis fascia. These are important clinical anatomic areas because freeing the zones of adherence is necessary to achieve long-term results with lift procedures. However, care is required in this region to avoid overzealous stretching and injury to the facial nerve.

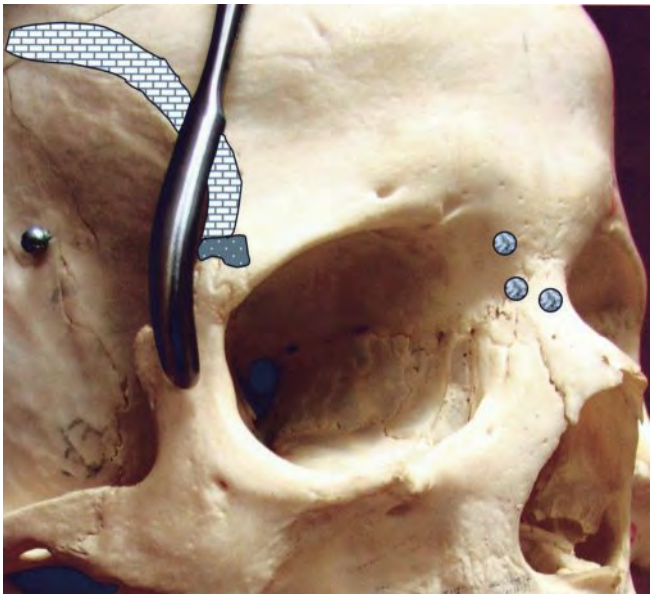


FIGURE 68-7. Periosteal elevator shown at a more aggressive level of dissection to elevate the lateral canthus slightly, if desired. Fascial and muscle attachments are labeled. Elevation at this level detaches only the superficial layer of the lateral canthal tendon. (The deep portion of the lateral canthus is 5 mm within the orbital rim attached to Whitnall's tubercle.)

The acronym *SCALP* applies for the standard layers in the forehead: *s*kin, sub*c*utaneous tissue, *a*poneurosis (the thick galeal fascia), *l*oose areolar (subgaleal) plane, and *p*eriosteum[28–30]; however, the galeal fascia fuses into the frontalis muscle and its midline fascial attachments at this level. This allows a sliding movement over the scalp with contraction of the muscle. The frontalis and galea together can also be thought of as an extension of the temporoparietal fascia in the temporal region as well as the superficial musculoaponeurotic system (SMAS) below the level of the zygomatic arch.[31–33] The temporoparietal fascia appears somewhat loose or spongy clinically and houses the temporal nerve within its undersurface.

Many other paired forehead and brow muscles thought of as depressors are present along the brow to facilitate facial expression.[34–41] The two most well known are the procerus and the corrugator supercilii, which are present in the glabella (Figure 68-11). The procerus muscles are paired superiorly but fuse inferiorly into one muscle belly that originates from the nasal bones and cartilage. Superiorly, procerus fibers insert into the medial frontalis and the overlying dermis. The procerus is responsible for depression and frowning in the midline, which often creates a horizontal crease ("bunny lines") across the upper portion of the nose. The corrugator supercilii are depressors that act obliquely across the glabella and produce the classic vertical lines seen when squinting (Figure 68-12). The corrugator originates from the frontal bone just above the nasal bones and inserts in the dermis of the medial brow. The corrugator has two heads, the oblique and the transverse, that act to pull the medial brow in respective locations. Together, the paired procerus muscles and corrugator are the main depressors of the medial brow and are the most common muscles treated with botulinum toxin type A to help alleviate frown lines in the glabella. These same two muscles are also most often transected during a brow or forehead lift to achieve a smoother and longer-lasting result (Figure 68-13).

Another depressor muscle of importance is the depressor supercilii, which originates on the frontal process of the maxilla just below the corrugator supercilii and inserts in the medial frontalis fibers and dermis just above the medial brow. Because it lies superficial to the corrugator, it can be easily paralyzed inadvertently by botulinum toxin. It is also important to note because it lies behind the corrugator and can be transected by aggressive dissection through the corrugator during a brow lift. Although patients with a very low medial brow position may occasionally benefit from this maneuver, it often gives rise to overelevation of the medial brow after surgery, which causes the patient to look somewhat surprised (Figure 68-14). Superficial to the depressor supercilii is the orbital portion of the orbicularis oculi that inserts into portions of the adjacent depressors, the superficial surface of the inferior frontalis, as well as the dermis below the brow.[42,43] The orbital portion of the orbicularis muscle originates in part from the medial canthal tendon and adjacent bone. Deep to all the depressors is the galeal fat pad,

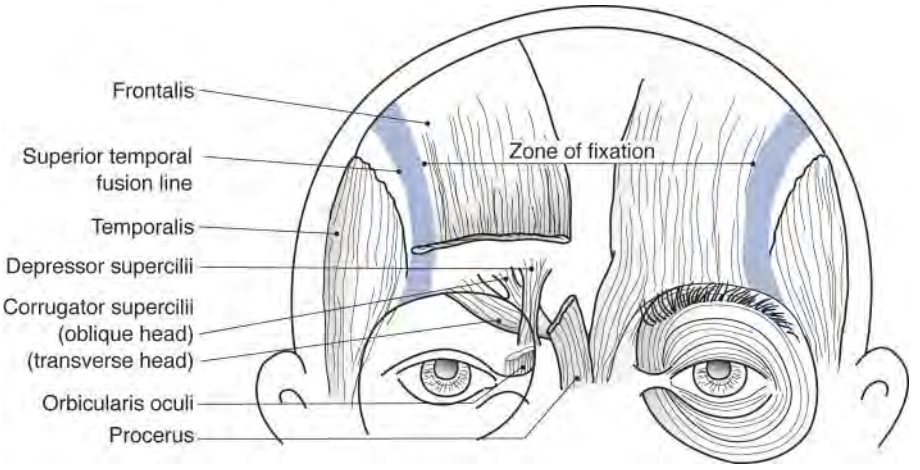


FIGURE 68-8. Cutaway portions of the frontalis muscles, procerus, and orbicularis oculi on one side demonstrate the relationship to the deeper depressors of the brow (corrugator supercilii and depressor supercilii). The zone of fixation *(blue)* runs medial to the superior temporal fusion line.

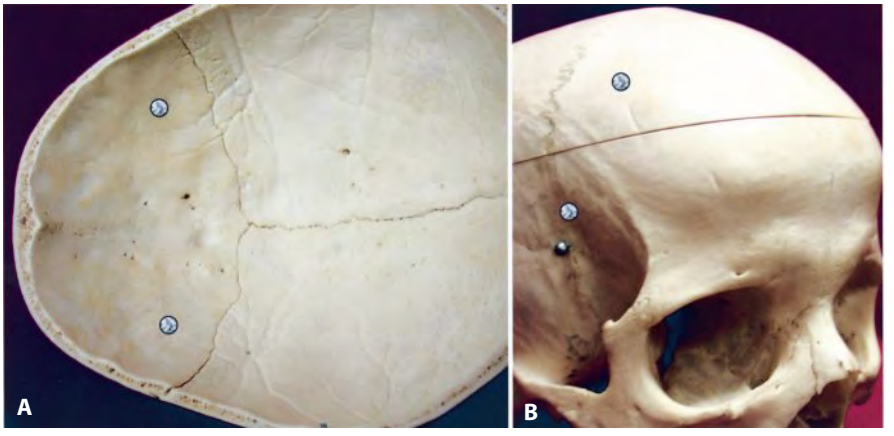


FIGURE 68-9. **A,** Inside view of the calvarium of the skull demonstrates the high density of venous lakes near the midline and associated structures. **B,** The ideal location placement for bone screws or tunnels based on ideal vector of lift and anatomic limitations.

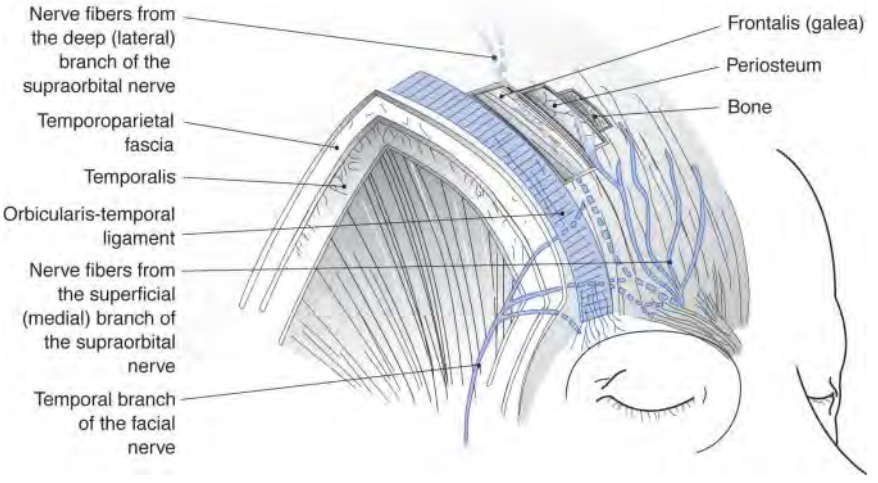


FIGURE 68-10. Layers of fascia are seen on each side of the zone of fixation *(blue)*. The layers must be elevated and connected to a uniform sliding plane surgically to achieve pleasing and long-lasting brow lift results while not damaging the associated motor and sensory nerves.

which lies immediately below the transverse head of the corrugator and helps in identification of muscular landmarks.[44] The galeal fat is usually exposed clinically instantly after transection through the periosteum along the orbital rim (Figure 68-15).

Finally, paired temporalis muscles are located in each temporal fossa, where they originate and then insert on the coronoid process of the mandible. The importance of these muscles during upper facial rejuvenation chiefly pertains to their overlying fascia, which can be used to delineate surgical planes and aid in fixation. The spongy temporoparietal fascia is superficial to the dense and shiny white temporalis fascia. The temporalis fascia adheres to the temporalis muscles below and splits into a superficial and deep layer in the lower

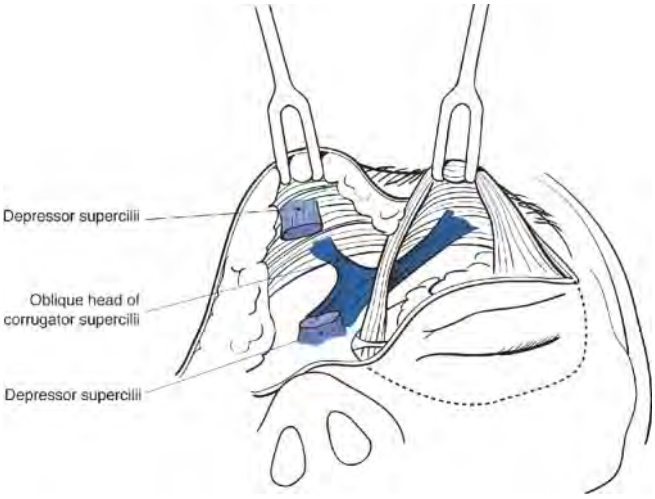


FIGURE 68-11. The oblique and transverse heads of the corrugator supercilii are seen behind the stump of the depressor supercilii. Both heads of the corrugator muscles and the orbicularis oculi insert into the dermis below the brow.

half of the fossa. For consistency, the superficial layer of deep temporalis fascia (which really describes only that portion of deep temporalis fascia at the level of the split and below) is subsequently referred to simply as *temporalis fascia*. In essence, this term will be used to describe any of this deep thick fascial layer that is seen clinically from the temporal crest down to the zygomatic arch (Figure 68-16).

One method of fixation during brow lifting is the use of suture to fixate the temporoparietal fascia from below a skin incision to the dense and adherent temporalis fascia above the incision to elevate the lateral brow. Some surgeons advocate removing a window of temporalis fascia and exposing the underlying temporalis muscle in hopes of creating scarification in this region and improving fixation longevity.[12]

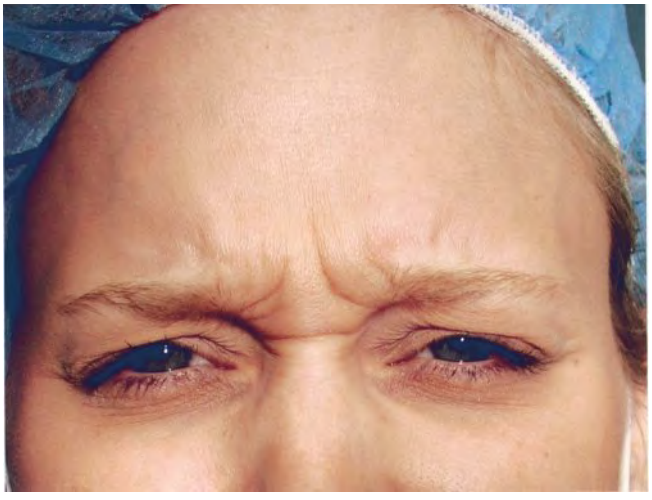


FIGURE 68-12. Frown lines of the glabella are produced by the actions of the corrugator supercilii to produce the classic vertical wrinkles, whereas the actions of the more vertically arranged fibers of the procerus muscle produce the horizontal wrinkles seen across the bridge of the nose.

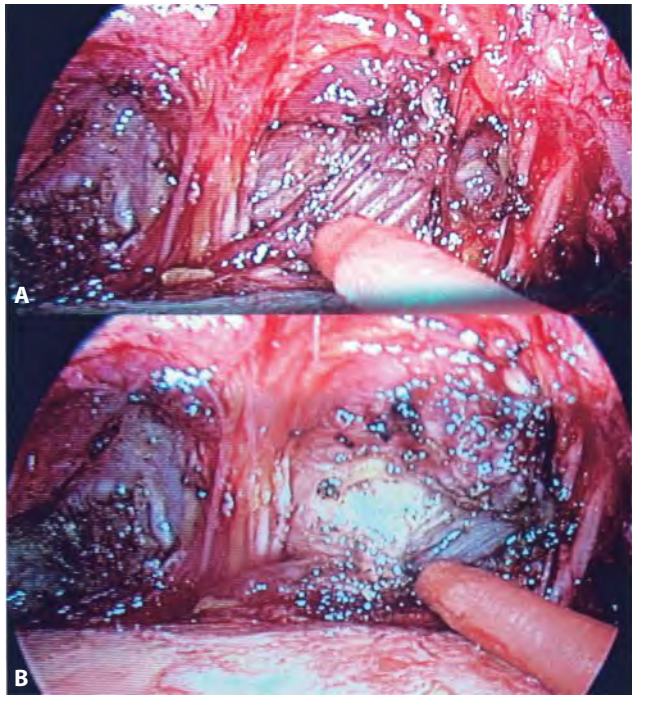


FIGURE 68-13. Endoscopic views of the right side of the forehead. **A,** Location of the corrugator supercilii relative to the supraorbital nerve immediately before it is transected with a needle tip cautery. **B,** Following transection through the belly of the corrugator supercilii.

Vessel and Nerve Anatomy

Blood supply to the upper face and scalp is plentiful and comes from multiple sources. Several major vessels of the



FIGURE 68-14. Before **(A)** and after **(B)** photos following endoscopic forehead and brow lifting demonstrate good elevation of the lateral hooding but overresection of the medial depressors in the area indicated *(arrow)*. This can result in a surprised look, especially when the patient elevates the brow, as shown.

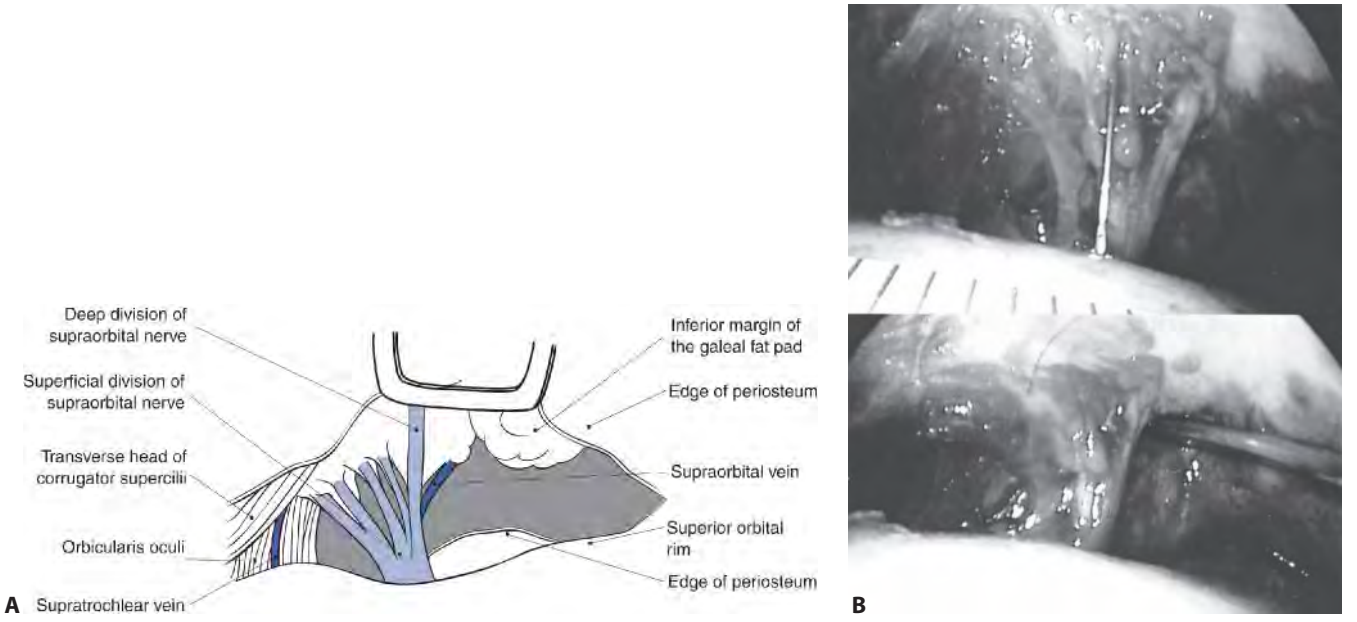


FIGURE 68-15. **A,** Right-sided forehead landmarks. **B,** Endoscopic view of the right supraorbital nerve and vessels. The first view is seen with a 27-gauge needle over the nerve trunk after it is placed through the skin of the brow level with the patient's medial limbus (iris).

upper face originate from the external carotid artery including the superficial temporal artery and the facial artery. These give rise to the blood supply in the medial canthal region via the angular artery and in the lateral canthal region by way of the frontal or anterior branch of the superficial temporal artery. The internal carotid artery gives way to the middle meningeal artery and the ophthalmic artery. The ophthalmic artery then gives rise to the supraorbital and supratrochlear arteries, which exit their respective foramina and supply the majority of the forehead and midscalp with blood. The terminal arterial branches of the upper face have major anastomoses with adjacent vessels (Figure 68-17).

Venous drainage of the upper face follows the respective arterial supply but can be somewhat more variable. However, one particular vein, known as the *sentinel vein* (medial zygomaticotemporal vein), runs perpendicular through the temporalis fascia connecting the superficial and middle temporal veins (Figure 68-18).[45] The sentinel vein can most often be found approximately 1 cm lateral or posterior to the zygomaticofrontal suture line (Figures 68-19 and 68-20). It is clinically significant during endoscopic procedures because, if injured, it can result in impaired field visualization and significant bruising.

Nerve supply parallels arterial supply to some degree. The supratrochlear and supraorbital nerves, which are responsible for the majority of sensation in the forehead, exit via the same foramina or general location as the supraorbital and supratrochlear blood vessels. The sensory nerves originate from the first division of the trigeminal nerve. The supraorbital nerve has two divisions after exiting its foramen: the deep (or lateral) division supplies the more lateral and posterior portion of the forehead and scalp and the superficial

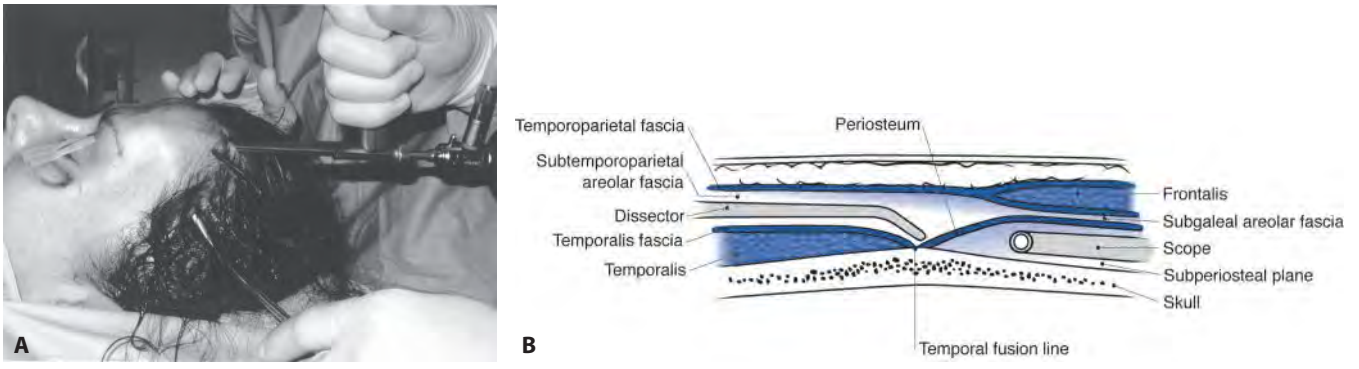


FIGURE 68-16. **A** and **B,** Endoscopic dissection must connect the tissue planes on each side of the temporal crest. Various approaches may be used as long as the anatomic planes seen here are sufficiently understood to allow proper tissue release, a clean endoscopic view, and protection of the facial nerve.

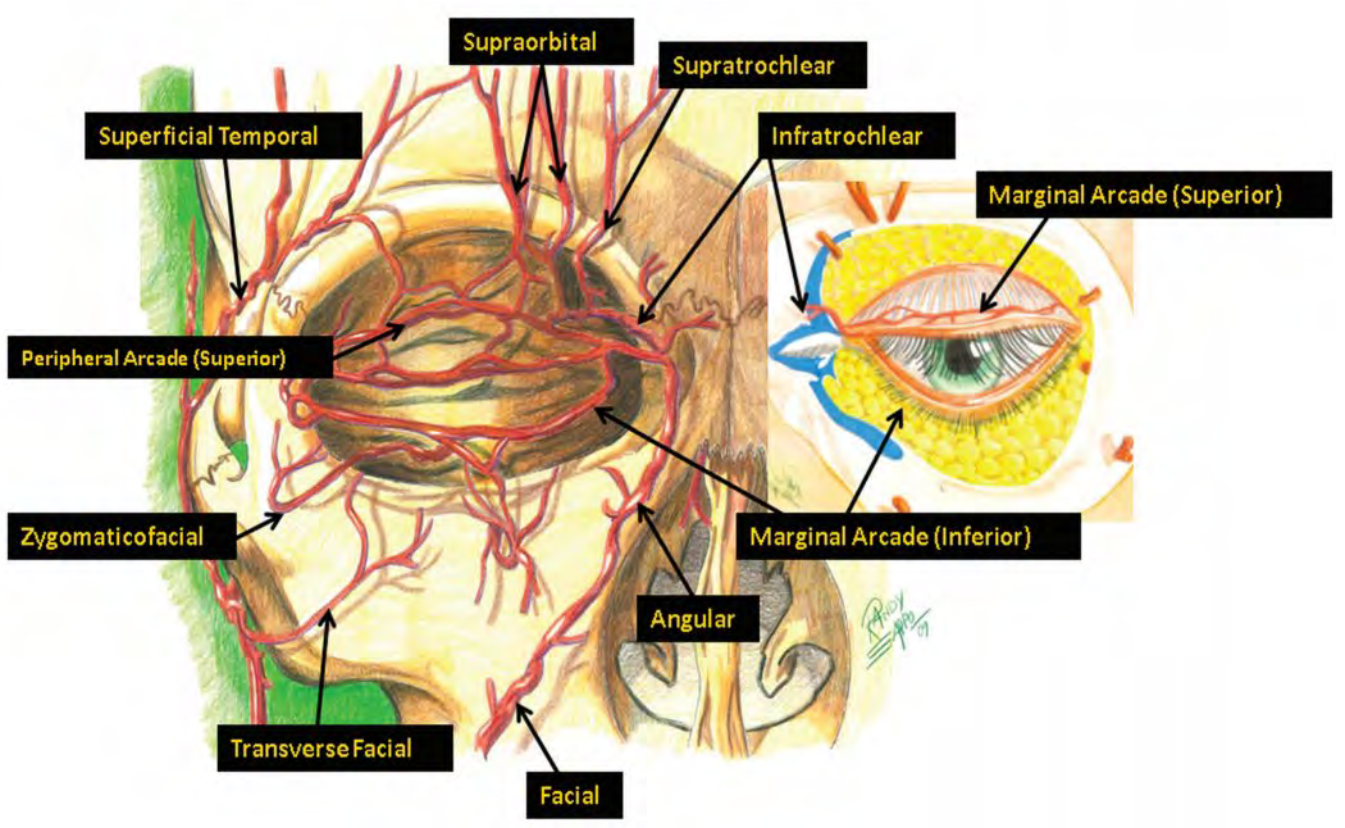


FIGURE 68-17. Periorbital arterial vessels demonstrate the excellent anastomotic blood supply for the periorbital and forehead regions. Most of the vessels have accompanying sensory or motor nerves running parallel with their course.

(or medial) division pierces the frontalis and runs superficially to the muscle, supplying sensation to the forehead along the midpupil line (Figure 68-21). The location of the supraorbital nerve's exit is relatively consistent. The supraorbital foramen or notch is typically found within 1 mm of a line drawn in a sagittal plane tangential to the medial limbus (Figure 68-22).[46] The deep division has been known to exit as often as 10% from another foramen that can be as high as 1.5 cm above the orbital rim. However, this was may be inaccurate. A review of 300 endoscopic brow lifts did not demonstrate any obvious supraorbital nerve over 0.5 cm from the rim. Further study of 40 cadaveric head dissections also failed to demonstrate any significant size foramen beyond 0.5 cm above the orbital rim at the level of the supraorbital notch or foramen. Microsize foramen can be seen in various locations on the forehead skull but are most consistent with tiny perforating vessels and would not support any notable sensation.

The supratrochlear nerves exit from around the orbital rim at an average of 9 mm medial to the exit of the supraorbital nerve.[46] The nerves supply sensation to the midforehead with some overlap from the supraorbital nerves. Infratrochlear nerves, also from division I of the trigeminal nerve, exit just below the supratrochlear nerves around the medial orbital rim to supply sensation to the upper nose and medial orbit. Zygomaticofrontal and zygomaticotemporal nerves are from

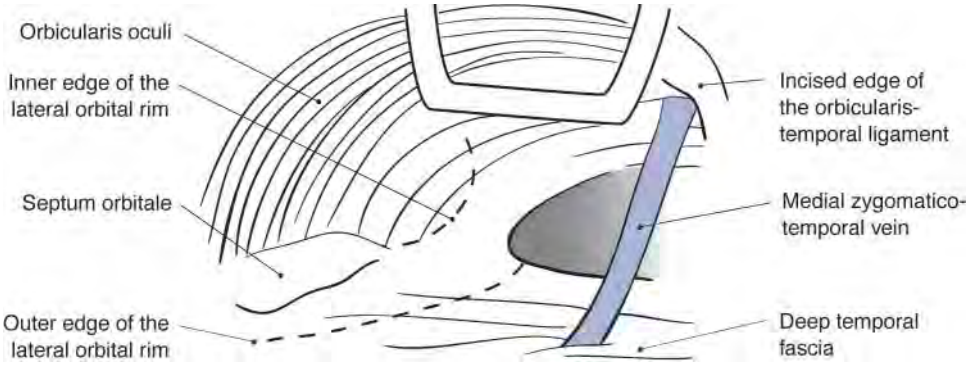


FIGURE 68-18. Dissection below the patient's right temporal crest with release of the orbicularis-temporal ligament. The medial zygomaticotemporal (sentinel) vein seen here pierces the temporalis fascia approximately 1 cm posterior to the zygomaticofrontal suture line.

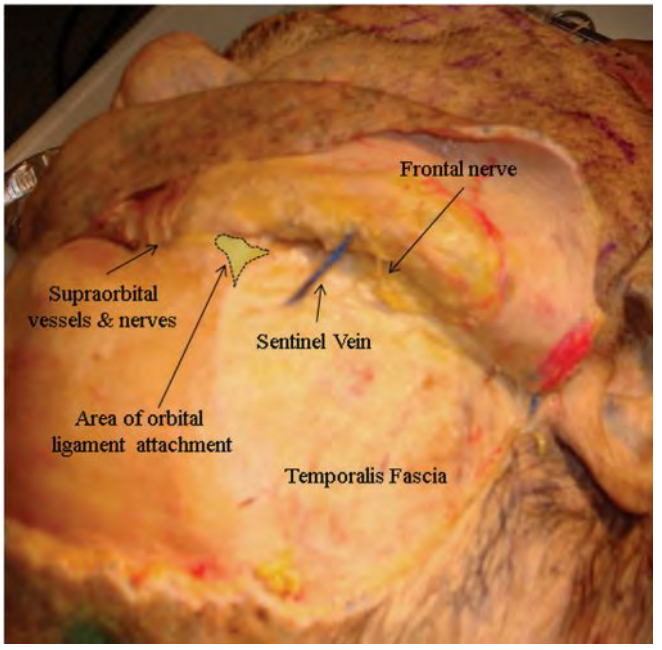


FIGURE 68-19. The sentinel vein, show here in *blue latex,* runs perpendicular to the temporalis fascia. The cadaver dissection also demonstrates the location of the temporal nerve, which runs obliquely just superficially to the sentinel vein in the deep temporoparietal fascia.

the orbital rim and zygomatic arch. Elevation of the deep tissues in this "safe zone" is essentially performed blindly through each of the small scalp incisions. Incisions and specific tissue release and fixation techniques are highly variable among surgeons.[52–59] The author prefers to dissect within the second division of the trigeminal nerve. They exit their respective small foramina and supply sensation to the lateral orbit and temporal regions of the face.

The facial nerve supplies motor innervation to the forehead and glabella.[47–51] The frontal (or temporal) branch of the facial nerve supplies the frontalis muscle, the superior portion of the orbicularis oculi, the superior portion of the procerus, and the transverse head of the corrugator supercilii. The zygomatic branch of the facial nerve supplies the medial head of the orbicularis oculi, the oblique head of the corrugator supercilii, the inferior portion of the procerus, and the depressor supercilii (Figure 68-23).

The auriculotemporal nerve, from the third division of the trigeminal nerve, supplies sensation in front of the ear to the temporal skin above the zygomatic arch and along the course of the superficial artery. It may be confused clinically during a facelift with the frontal branch of the facial nerve. It can, however, be distinguished from the facial motor nerve because it runs within 1 cm anterior to the tragus of the ear and parallel to the superficial temporal artery. The much more significant frontal branch of the facial nerve runs an average of 2 cm anterior to the tragus when crossing the zygomatic arch. The temporal branch of the facial nerve crosses the arch at an oblique angle at an average of 2 cm posterior to the orbital rim. The depth of the temporal nerve is just below the SMAS at the arch and below the temporoparietal fascia immediately above the arch (Figure 68-24).

Initial dissection must be performed to gain adequate space for the endoscopic equipment. This early dissection is performed in the posterior forehead and temporal regions; endoscopy-guided dissection is used for the last 1 cm above

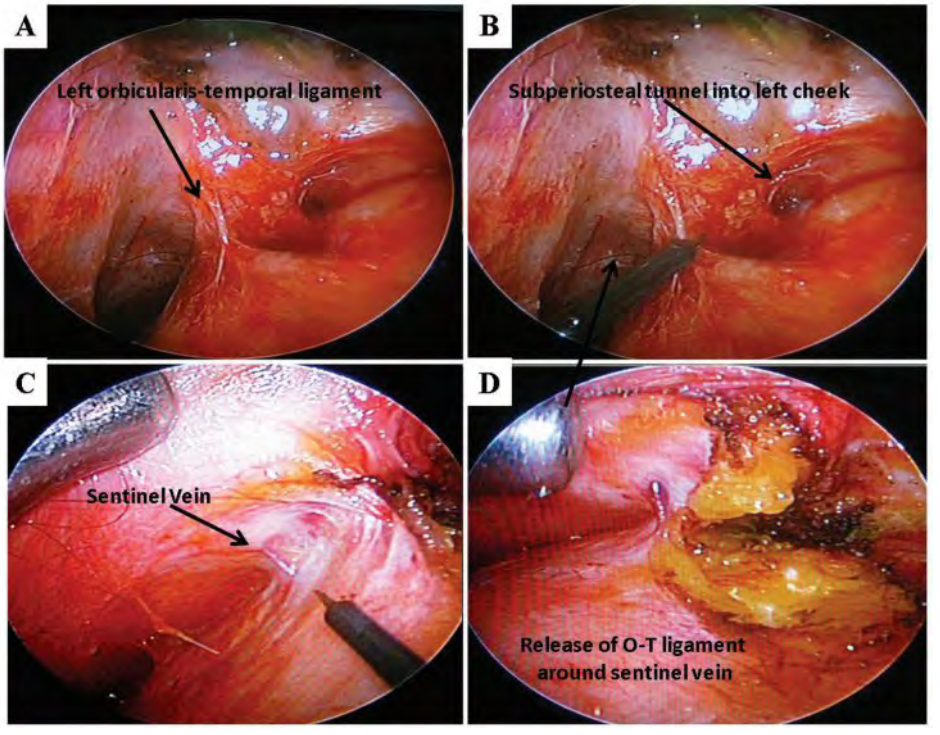


FIGURE 68-20. **A** and **B,** The left orbicularis-temporal (O-T) ligament, also known as the *orbital ligament,* immediately before release via needle tip cautery. Medial to O-T is a subperiosteal tunnel created by a larger elevator past the zygomaticofrontal suture and down into the left cheek. The orbicularis oculi muscle can be severed inside the tunnel. **C** and **D,** Exposure of the sentinel vein before and after complete release or the orbital ligament.

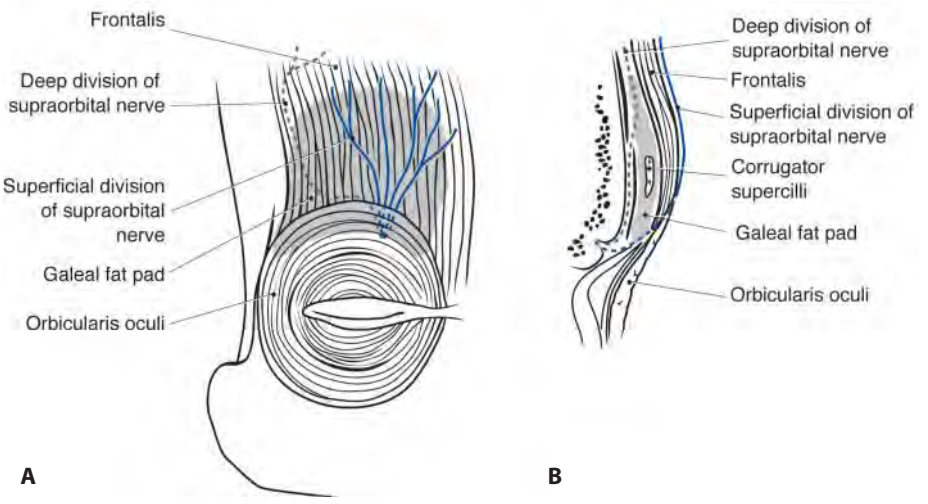


FIGURE 68-21. **A** and **B,** Distribution of the superficial (medial) and deep (lateral) divisions of the supraorbital nerve.

a completely subperiosteal plane medially to the temporal crest and in the plane immediately above the temporalis fascia below the temporal line on each side. Subperiosteal dissection in the lateral forehead helps to avoid injury to the deep or lateral division of the supraorbital nerve, which runs in the subgaleal plane near the zone of fixation. Some surgeons begin their dissection in a subgaleal plane in the posterior scalp.[59,60] Regardless, a space is created in the safer posterior areas of the scalp to allow room for placement of an endoscope, which aids dissection in the more risky areas of the forehead.

The first anatomic landmark the surgeon must consider is the zone of fixation along the superior temporal crest. Its inferior edge is found near the superior lateral orbital rim. A convergence of fibers from the periosteum, galea, temporalis, and temporoparietal fascia interlace and fuse to form the zone of adherence, in much the same way the layers of tissue planes come together at the level of the zygomatic arch. The zone of fixation can be elevated bluntly at the hairline level and a couple of centimeters below, but as the surgeon approaches the lateral brow beginning approximately 1 cm above brow level, use of an endoscope aids dissection. At this point, the ligament has branches of the temporal nerve within it, and care must be taken to remain against the bone and temporalis fascia below to avoid nerve injury. Another fibrous attachment, the orbicularis-temporal ligament, is also

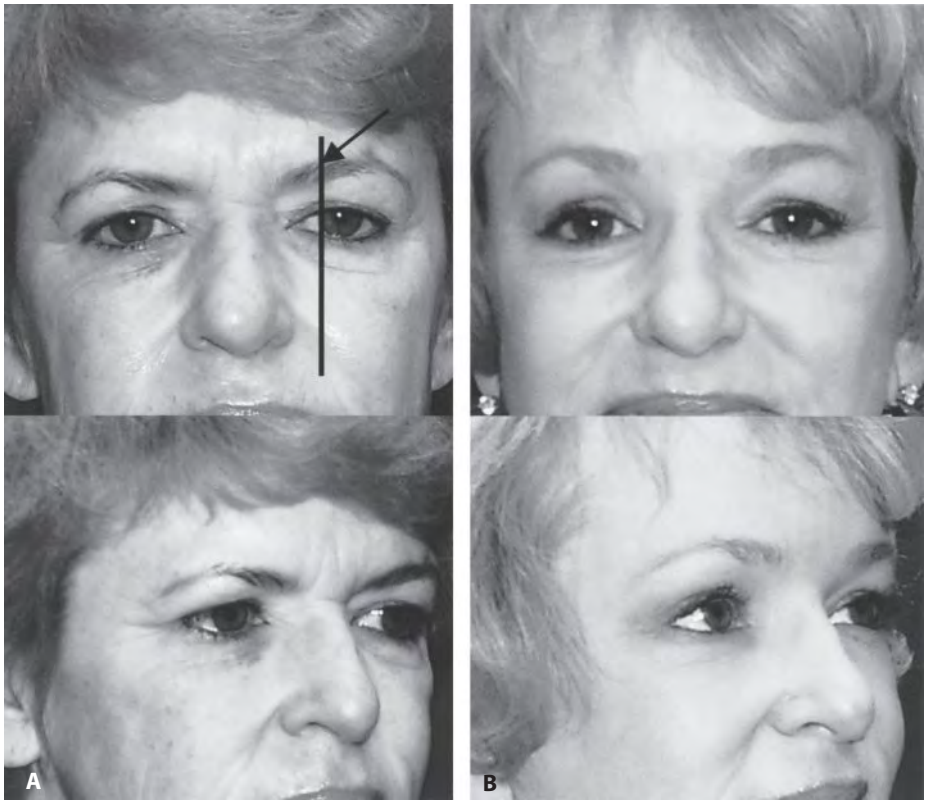


FIGURE 68-22. **A,** Preoperative photograph demonstrates the location of the supraorbital vessels by a line drawn vertically from the medial iris. **B,** One and a half years after an endoscopic forehead and brow lift. No blepharoplasty was ever performed.

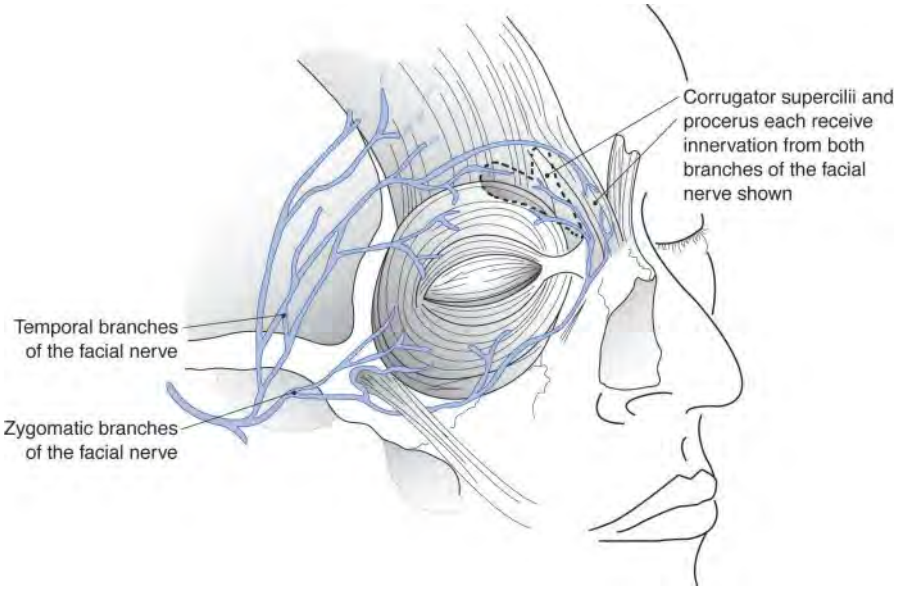


FIGURE 68-23. Motor nerve supply to the forehead depressor muscle comes from both the temporal and the zygomatic branches of the facial nerve.

present here and contains motor nerve fibers (see Figure 68-18); it is the decussation of fibers from the temporoparietal fascia and of the temporal fascia that extends laterally from the orbital ligament. The zone of adherence becomes even more tenacious as the orbital ligament (see Figure 68-7) at the orbital rim level is approached. Slow meticulous dissection is required at this point to avoid nerve injury as well as injury to the sentinel vein that is located within the orbicularis-temporal ligament approximately 1 cm laterally to the zygomaticofrontal suture. Careful dissection exposes an intact sentinel vein that can be seen piercing through the temporal fascia at a perpendicular angle and entering the temporoparietal fascia above (see Figure 68-18).

Dissection above the orbital rims in the subperiosteal plane should expose the entire superior orbital rim from each zygomaticofrontal suture. The curvature of the rims should be visualized so that transection through the periosteum can be made at the level of the rims. The nasofrontal suture may not always be seen but can be felt by the periosteal elevator used to lift tissue. When transecting through the periosteum across the entire orbital rim, subgaleal fat is often encountered initially, except when the transection is directly behind

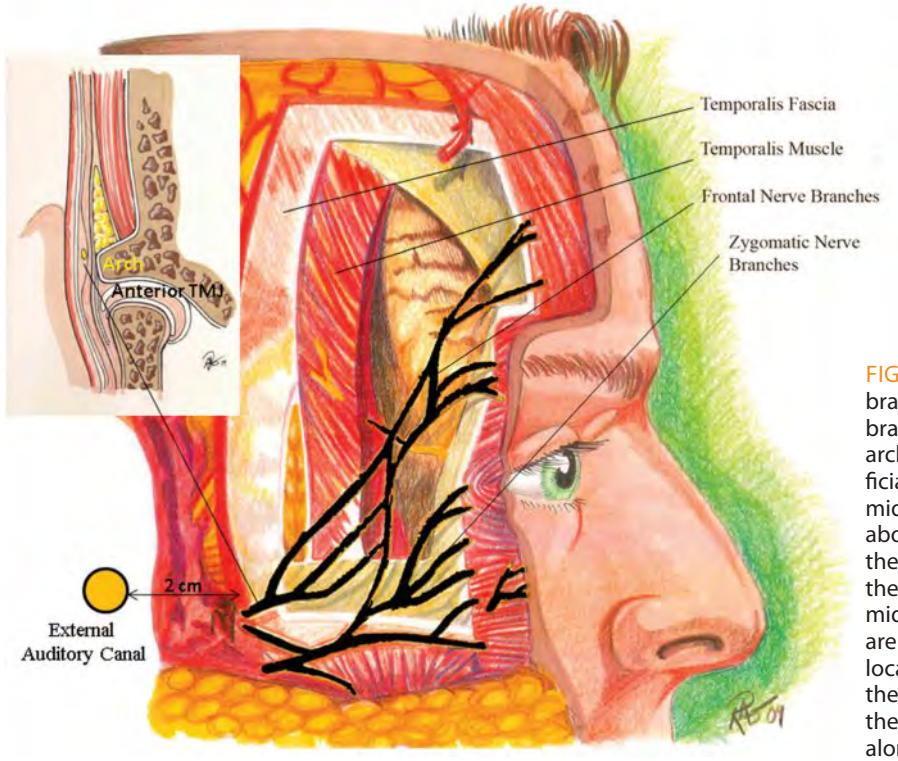


FIGURE 68-24. The temporal and zygomatic branches of the facial nerve divide into multiple branches by the time they reach the zygomatic arch. The nerve branches are deep to the superficial musculoaponeurotic system (SMAS) in the midface and deep to the temporoparietal fascia above the arch. In facelift and forehead lifting, the surgeon must be either above or below the nerve branches and be most careful in the midzygomatic arch region because the tissues are all very condensed around the nerve in this location. Typically, there is a relatively safe area the first 2 cm in front of the ear canal because the frontal nerve is not found in this region along the arch.

the supraorbital nerve at the rim level where the deep (or lateral) division of the nerve is closely adherent to periosteum (see Figure 68-15). Preoperatively marking a point on the brow at a level tangential to the medial limbus iris helps the surgeon to easily identify the location of the supraorbital vessels and nerves.[46] Dissection through the periosteum in this region should be performed slowly and superficially to avoid injury to these structures (Figure 68-25). The transverse head of the corrugator supercilii is seen at the orbital rim level behind the supraorbital vessels and nerves. The corrugator supercilii can be carefully transected or partially excised.[61] Medially, the oblique head of the corrugator is encountered, and by a transection through this portion of muscle, the supratrochlear nerve and depressor supercilii muscle may be seen and protected from injury. Medially, in the glabella, the procerus muscle, which is variable in thickness, is seen. Care should be taken to avoid overaggressive muscle resection in thin patients because this can result in an atrophic defect in the glabella. Deeper dissection toward the skin level under the brow will lead to the orbicularis oculi but is typically not necessary to gain the desired effect (except with regard to the lateral orbicularis, where limited transection may improve lateral brow elevation).[62,63] In addition, one or more incisions through the periosteum at higher levels under the frontalis muscle in the midline can be performed but this is required only if deep horizontal lines are present.[64] It is more important to gain complete release of the retaining lateral ligaments, transection of those muscles causing glabellar lines, and adequate separation of the periosteum along the orbital rim to get the elevation of brow and forehead tissues for the most pleasing and long-term aesthetic result.[65–75]

PREOPERATIVE EVALUATION AND SURGICAL PREPARATION

Determining whether a patient will benefit from a brow or forehead lift and which procedure will work best is critical to avoid disappointing the patient. Commonly, the novice surgeon notices only horizontal forehead lines as an indication for a brow lift. Unfortunately, this is much less of a problem for most patients than is a low lateral brow position (hooding) or glabellar crease (see Figure 68-3). As discussed previously, the ideal female brow position is above the orbital rim at a level that varies among individuals. An average distance of 5 to 10 mm of brow elevation above the rim in the lateral third generally looks most pleasing. Men require a straight-up elevation of the entire brow to avoid feminizing their appearance by overelevation of the lateral brow. In addition, men may benefit more from a standard upper blepharoplasty and local transpalpebral brow lift if the brow ptosis is minimal. As with any cosmetic surgery, a decision regarding the risks and benefits must be made and must conform to the patient's desires. Patient education is required so that they know the risks as well as what can *realistically* be achieved (Figure 68-26). Even with fairly aggressive muscle resection and forehead elevation, patients often form new dynamic lines in the upper face after surgery. A *subcutaneous browlift*, which maintains a surgical plane above the frontalis muscle, may be required if heavy horizontal forehead lines exist and the patient wants them significatly reduced in appearance. Comparatively, subgaleal and subperiosteal dissection planes during forehead and brow lifting allow treatment of the depressors muscles but do not allow major affective treatment of the frontalis muscle and horizontal forehead creases

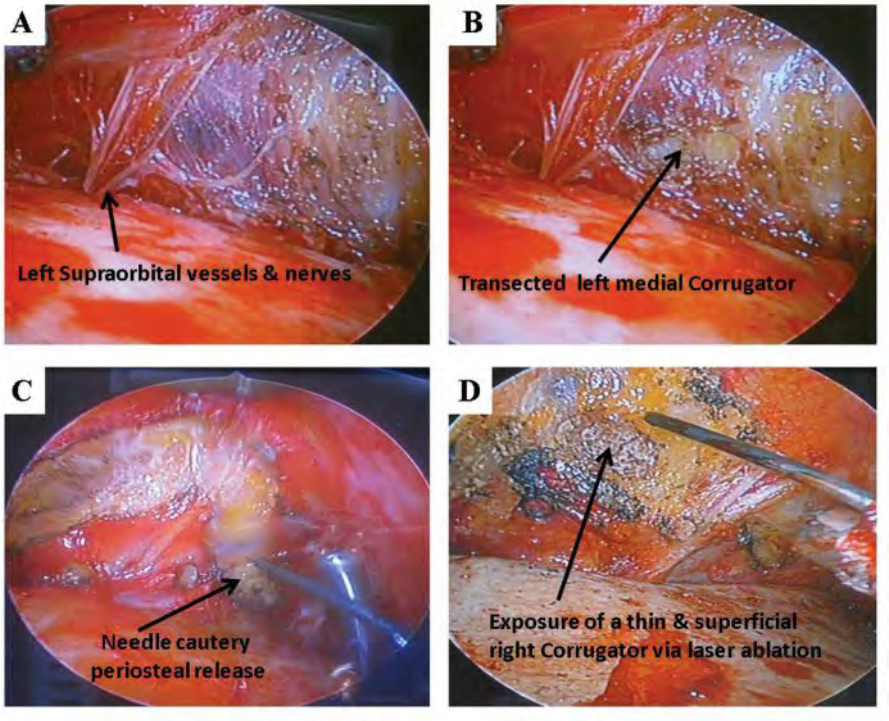


FIGURE 68-25. **A** and **B,** The left supraorbital vessels and nerves immediately adjacent to the medial portion of the oblique head of the corrugator muscle. **A,** An intact, large corrugator. **B,** The same muscle immediately after needle tip cautery has transected the midbelly of this muscle. **C,** Periosteal release is made 1.5 cm above the rim at the supraorbital neurovascular belly to prevent accidental trunk transection. All other regions of the brow are released directly at the arcus marginalis. **D,** The right corrugator muscle is thin and very superficial. Laser ablation of periosteum and brow fat medial to the nerve is performed to locate this sometimes small muscle for treatment.



FIGURE 68-26. **A,** Because of both brow ptosis and upper eyelid laxity, the patient required upper blepharoplasties as well as endoscopic forehead and brow lifting to achieve the results she desired. **B,** The patient before and after only blepharoplasty and full-face laser skin resurfacing. She has multiple problems including asymmetry of the brows due to a blepharospasm on the left side, eyelid asymmetry and severe laxity, pseudoelevation of the brows due to frontalis compensation for severe eyelid ptosis, and severe actinic skin damage. She is not a good candidate for simultaneous brow lifting because a change in brow position will likely occur after the removal of the eyelid ptosis. She is a good candidate for botulinum toxin therapy on her left side.

(Figure 68-27). The depressor muscles and their resulting creases in the glabellar region can be adequately treated from a subperiosteal, subgaleal, or subcutaneous plane. Lateral crow's-feet owing to the action of the orbicularis oculi when smiling may appear improved after a brow lift because the muscle is unfolded. However, they are not completely eliminated by brow lifting alone, and the patient must understand that botulinum toxin therapy may be required to treat these particular lines on an ongoing basis.[76]

In addition to lines on the forehead, lines in the glabella, brow ptosis, and the condition of the patient's skin must also be evaluated. Intrinsic skin and collagen damage from the effects of sun, age, and smoking are not treated by lifting alone. Topical skin care (e.g., retinoic acid, microdermabrasion, pulsed-light therapy, sunblocks) along with possible surgical resurfacing must be considered.[77–79] In general, the forehead can be treated safely with chemical peels or laser skin resurfacing into the dermal level simultaneously with brow lifting procedures, provided the lifting is performed with a subgaleal or subperiosteal technique rather than a subcutaneous one. Finally, bony irregularities or hypertrophic bony orbital rims can be evaluated for treatment by means of a cephalometric radiograph or computed tomography (CT) scan as required. Bony contouring can be performed on a limited basis endoscopically, but a major reduction for significant bone hypertrophy such as a frontal boss is best treated with an open (coronal) approach. The amount of bone reduction is limited by the pneumatization of the frontal sinus, which is best evaluated by CT. Although treatment planning for placement of bone tunnels does not

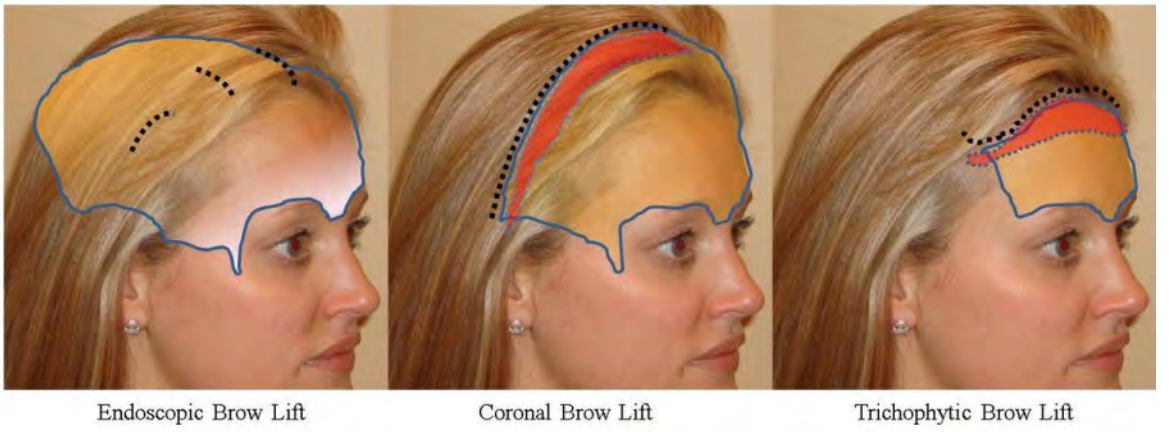


FIGURE 68-27. The three most classic forms of brow lifting are presented using *dashed lines* to demonstrate the incision used for each technique. The *shaded areas* demonstrate the extent of dissection typically required for each technique. Interestingly, the endoscopic brow lift actually requires more undermining to allow tissue redraping because no direct scalp excision is performed compared with the other techniques.

require a preoperative CT, a standard cephalometric radiograph may help to reassure the surgeon regarding the thickness of corticocancellous bone available.

As with any surgical procedure, appropriate preoperative laboratory and other indicated tests must be performed. Written instructions are given to the patient regarding pre- and postoperative care, including instructions for shampooing hair with antibacterial soap or other antiseptic shampoo and avoidance of the use of hair spray or other hair products immediately before surgery. The patient should be thoroughly instructed on the critical need to avoid all medications that may cause platelet dysfunction 10 days before surgery (including aspirin and other nonsteroidal anti-inflammatory drugs, vitamin E, and many over-the-counter herbal supplements). Endoscopic techniques require a very dry operating field that necessitates strict avoidance of these medications as well as proper preoperative injection of vasoconstrictive agents.

Before induction of anesthesia, photographs are taken and the patient is marked while awake and sitting up. Following the introduction of general anesthesia or intravenous sedation, the patient is prepared and carefully injected with local anesthetic with epinephrine. The author prefers to use a local anesthetic with 1:100,000 epinephrine along the entire orbital rim and a tumescent anesthesia solution (250 mL of normal saline mixed with 1 mL of 1:1,000 epinephrine and 20 mL of 2% lidocaine) in the remaining upper forehead, temple, and posterior scalp. Careful injection in the desired tissue planes helps to avoid the formation of a hematoma during the injection and allows for a nearly bloodless procedure. Minor shaving of hair along the marked incision lines is performed if desired immediately before the final preparation and draping of the area.

CORONAL FOREHEAD AND BROW LIFT

Still one of the most common approaches for forehead and brow lifting, the classic coronal lift involves an incision across the entire forehead from ear to ear, staying well behind the hairline.[80–88] Dissection is typically in the subgaleal or subperiosteal plane and then connects to the subtemporoparietal plane laterally. This gives great exposure of the entire orbital rims for bony osteoplasty, if required, and treatment of muscles that require resection including the depressors (corrugator and procerus) as well as the frontalis. Heavy horizontal forehead creases can be addressed with this technique by way of either midline myotomies or minor midline thinning of the frontalis. Major resection of the frontalis should be avoided to prevent postoperative irregularities and strange facial expressions during frontalis movement. The lateral frontalis should be avoided to prevent nerve damage, ptosis, and other irregularities.

Regrettably, the coronal lift also has the disadvantages of a long incision and a significant elevation of the hairline. Patients with a high hairline are not good candidates for this technique because a significant amount of scalp excision is required. Many surgeons believe this scalp excision is a reasonable trade-off because they feel that the technique gives a more lasting approach than do newer endoscopic techniques. If performed correctly, the endoscopic technique can be as long-lasting and possibly more precise than open brow lifting techniques. Care must be taken with the coronal lift to avoid elevating the medial brow too much and creating a very high hairline. Roughly, to gain 1 cm of brow elevation, 1.5 to 2 cm of scalp must be excised with this technique. The amount of tissue excised is not a precise determinant of the amount of brow elevation obtained. Scoring of the underlying fascia and muscle resection can cause the tissue to stretch oddly, making prediction of the exact brow elevation difficult.

The benefits of the coronal lift include great exposure and relatively easy dissection. It can also be used to extend the procedure into a deep-plane facelift by dissection over the zygomatic arches and onto the zygoma and masseter. This much more aggressive lift gives excellent elevation of the midface but greatly increases postoperative edema and the potential for motor nerve damage. The extended technique should be attempted only by an experienced surgeon,[89–93] and careful consideration should be given to alternative treatments. Comparatively, the basic coronal lift is an easier procedure for the novice surgeon. When selecting this tried-and-true method, one should take into account the disadvantages, including the lengthy scar and possible hair loss, significant scalp anesthesia, and a significantly elevated hairline.

TRICHOPHYTIC OR PRETRICHIAL FOREHEAD AND BROW LIFT

Although trichophytic and pretrichial lifts are sometimes thought to be the same procedure, the *pretrichial* lift actually involves an incision in front of the hairline. With this procedure, hair does not grow anterior to the incision, leaving a visible scar in front of the hairline. In contrast, in the *trichophytic* lift, although still at the frontal hairline, the incision is placed just behind the hairline. This incision is beveled so that follicles in front of the initial skin incision survive and hair grows anterior to the incision to better camouflage the resulting scar. It should be noted that many surgeons use these terms interchangeably. Even better than the trichophytic lift is the irregular trichophytic hairline, which not only employs a beveled incision but also creates a wavy pattern along the hairline for a more natural postoperative appearance compared with a straight-line scar.

Regardless of the specific incision design, the ultimate advantages of the trichophytic forehead and brow lift include great exposure (similar to that with the coronal approach) and the ability to lower a high forehead. Unlike the classic coronal lift, bare forehead skin is excised from the hairline. In addition, lateral incisions and dissection are usually limited with this technique unless required. Incision design can even improve hair thinning in the temporoparietal areas by excising the area of hair loss and bringing forward areas of dense hair-bearing scalp. The posterior scalp and hairline can

be brought forward to lower a high forehead by almost any amount. The more lowering that is desired, the more posterior is the dissection and release. Limited or no posterior dissection can be performed if the hairline is to remain at the same level.

The forward dissection is the technique that varies the most among surgeons. A totally subperiosteal technique versus a subgaleal technique is an option. A subcutaneous technique has become more popular, particularly when the depressors in the lower brow are less concerning than the horizontal forehead creases.[94] Staying superficial to the frontalis breaks the dermal insertions that create deep horizontal rhytids. The subcutaneous lift is occasionally combined with deep dissection to treat glabellar lines as well as horizontal lines in the forehead.

Overall, the trichophytic technique of forehead and brow lifting is an invaluable tool for any surgeon performing facial cosmetic surgery. When a patient presents with a high forehead and low brow position, the trichophytic approach is the procedure of choice to correct both problems. The main disadvantage is the potential for a visible incision despite best efforts. All prospective patients considering this technique must be informed of the chance that there may be a visible scar at the hairline. Surprisingly, when presented with the potential problems and given the choice, many patients prefer to undergo an endoscopic approach with a slight elevation in hairline rather than risk a visible hairline scar. Still, the patient with an extremely high hairline is often thrilled with the lower hairline obtainable only with the trichophytic approach. Attention to detail and gentle soft tissue management are essential to attaining a natural hairline and hidden scar with this popular technique.

ENDOSCOPIC FOREHEAD AND BROW LIFT

Early attempts at endoscopic surgery began over a century ago with Nietze's description of a crude cystoscope. A few decades ago, endoscopic surgery progressed through use in upper gastrointestinal examinations and then intra-abdominal surgery. However, facial endoscopic cosmetic surgery did not blossom until the early 1990s. Over the past decade, the endoscopic forehead and brow lift procedure has been considered by many to be the state-of-the-art technique for upper facial rejuvenation.[95–97] It is versatile and can be combined with many other procedures. The most noted benefits of the endoscopic technique are the smaller scars hidden in the hairline and selective brow elevation without the need for removal of any hair or skin (Figure 68-28).

The technique involves several incisions placed strategically behind the hairline to gain access for early blunt dissection and insertion of the endoscope and tissue retractor. Other incisions can be used as ports for dissecting tools such as periosteal elevators, electrocautery, lasers, tissue graspers, and suction instruments. Among surgeons a variety of incision (port) designs are used. Fixation points are usually placed at these incision sites; therefore, the author prefers five separate 2.5-cm-long incisions placed for easy access but mostly for ideal fixation placement. Each of the five incisions begins approximately 1 cm posterior to the hairline. One is placed in the midline in the sagittal plane and two in the parasagittal plane tangential to the lateral third of the brow (where maximum lift is typically desired in females). This same incision can be moved slightly medially in male patients to give a more even brow elevation. The midline incision plus the two parasagittal incisions are aligned vertically to avoid unnecessary transection of sensory nerves originating from the supraorbital nerves below. The two parasagittal incisions are placed medial to the temporal crest to gain access to skull bone rather than the more lateral temporalis fascia. Bone is the strongest fixation tissue available and ideally should be used thus.[98–100]

It is important to access the subperiosteal plane easily for a clean future endoscopic view. Accidental placement of the parasagittal incisions too far laterally over the zone of fixation or temporalis muscle makes pocket development difficult and obscures future endoscopic visualization. Moreover, the parasagittal incisions are located in a thick area of the frontal bone where there is a low density of venous lakes. Placing the incision here helps to prevent accidental intracranial injury during bone tunnel creation or placement of bone screws.

Lastly, two temporal incisions are made, one on each side of the head, for direct access to the thick temporal fascia. These incisions are placed perpendicular to the desired



FIGURE 68-28. Sequential appearance after endoscopic forehead and brow lifting (eyelid and skin resurfacing procedures were also performed). Slight overelevation of the brow is noted for 6 days after surgery, as expected. The brow position remains very stable from 2 weeks to 7 years after the surgery. Adequate tissue release is the key to long-term stability of brow lifting.

elevation vector from the lateral canthal region. Coincidently, the temporal incision parallels the course of the temporal branch of the facial nerve that is located 2 to 3 cm inferior to this incision. It also parallels the superficial temporal artery and vein. Arranging the three medial incisions on a vertical axis and the two temporal incisions in an oblique position to parallel the nerve and blood supply in each area can reduce interference with sensation and vascular supply to the scalp.

Dissection is performed through the above incisions down through periosteum medial to the temporal crest and down to the temporalis fascia lateral to the crest. Some surgeons may elect to use a subgaleal rather than a subperiosteal placement of the incision medially. Total subperiosteal dissection medial to the temporal lines rather than subgaleal dissection leads to better fixation and long-term stabilization (see Figure 68-28).

Blunt and blind dissection can be carried out after reaching the subperiosteal and subtemporoparietal planes through the five incisions. Finger dissection and long curved endoscopic periosteal elevators are used to lift the tissue anteriorly to a point 2 cm above the orbital rims and zygomatic arch. Posteriorly, blunt dissection should elevate the temporal tissues a few centimeters behind the ear, where the temporal fossa becomes self-limiting. The subperiosteal dissection above needs to elevate the scalp at least 10 cm posteriorly but can extend as far back as the lambdoid suture. Once these areas are freed, a connection can be made from the temporal region to the subperiosteal dissection through the upper portion of the zone of fixation at the temporal crest by finger dissection (Figure 68-29). Blind release of the more inferior portion of the temporal line where the facial nerve crosses should be avoided. Endoscope-guided dissection here helps to prevent nerve injury. Using finger dissection, the upper zone of fixation is broken through, proceeding from the temporal incision toward the medial scalp, rather than vice versa, to prevent creation of a false tunnel in the spongy or foamy temporoparietal fascia. False tunnels along the temporal crest create problems when the endoscope is inserted through the parasagittal port to visualize the lateral forehead; the tunnels force the placement of the endoscope in a more superficial plane within the temporoparietal fascia, which greatly increases the chance of nerve injury. Therefore, it is critical to stay firmly against the periosteum and the temporalis fascia when initially elevating the scalp and forehead.

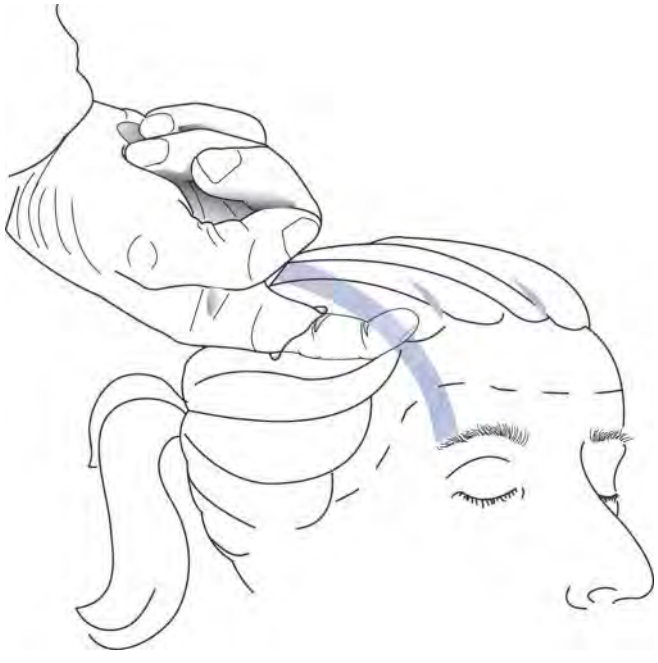


FIGURE 68-29. Blind finger dissection is performed initially, avoiding overzealous dissection inferiorly. Dissection proceeds from the subtemporoparietal plane laterally to the already elevated subperiosteal plane medially. The opposite direction of elevation (medial to lateral) may produce false tunnels in the temporoparietal tissue, which impair future endoscopic vision.

Following blunt elevation of the scalp from each incision for complete flap elevation, the endoscope is normally inserted through one of the three more medial incisions. Poor initial blunt dissection makes the initial endoscopic dissection feel very tight, and care must be taken not to perforate the skin by excessive retraction. Medial dissection over the nasofrontal suture and orbital rims is performed under direct endoscopic vision with a curved and smooth elevator to avoid inadvertent tearing of the periosteum. The periosteum may be thin in some patients in whom a straighter elevator may be used to transect the periosteum at the level of the rim (arcus marginalis). However, the entire rolled edge of the orbital rim must be visualized before proceeding with periosteal incision (Figure 68-30). Typically, the periosteum is more precisely incised with a needle tip cautery or laser set at low power. The supraorbital nerves and vessels as described earlier are at a level tangential to the medial limbus and are immediately behind (superficial to) the periosteum from the internal endoscopic view.[46,101] This necessitates meticulous cautery dissection here to avoid injury to these structures (see Figure 66-25). Suction placed by an assistant from another port is required to maintain a clear view when using cautery or laser. Temporal incisions work well for suction ports during dissection over the rims because the endoscope and cautery take up most of the room through any of the middle three incision sites. With clear and near bloodless dissection at this point, transection can be performed through the corrugator supercilii and procerus. If unwanted bleeding is encountered and cannot be controlled easily with pinpoint accurate cautery, pressure should be applied externally over the rim until improved visualization allows for control of bleeding without nerve damage.

Vertical rhytids in the glabella created by the corrugators can be improved greatly by transection through these muscles. Likewise, horizontal glabellar lines are treated by transection of the procerus muscle that creates these particular facial wrinkles. Some surgeons advocate more aggressive surgical avulsion of these muscles with endoscopic biopsy forceps. Aggressive muscle removal may lead to a more permanent treatment of glabellar lines compared with isolated transection only, but it should be avoided in most cases owing

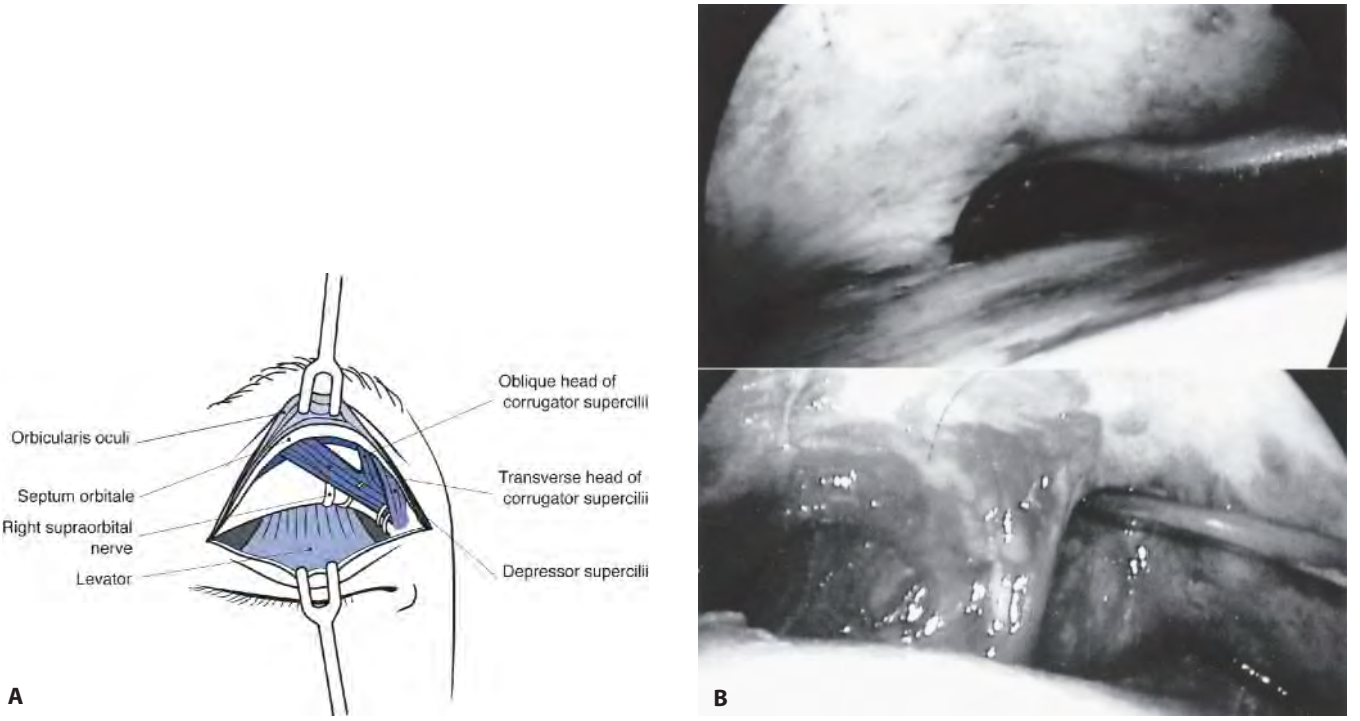


FIGURE 68-30. **A,** The orbital rim and local depressor muscle as seen from a transblepharoplasty incision. **B,** Endoscopic photographs show the rolled border of orbital rim before periosteal release in the first view and the supraorbital nerve and vein in the next view after excising through the periosteum.

to an increased risk of significant postoperative irregularities and abnormal facial expression. As a rule, patients prefer a more natural appearance with some minor return of frown lines to risking a bizarre facial expression and glabellar depression.

Once the periosteum is completely freed across the orbital rims and appropriate muscles have been treated, the cut periosteal edges are spread apart (periosteal elevators work well for this) by at least 1 cm to aid the release at the arcus marginalis. This allows significant and long-term brow elevation. Next, the lateral orbital rim must be exposed in the subperiosteal plane after careful release below the zone of fixation and orbital ligament. Dissection along the anterior and inferior aspects of the temporal crest must be performed cautiously to avoid temporal nerve injury. Overzealous retraction of the dense tissue here that contains the nerve can result in nerve damage. Staying snuggly against periosteum and the temporalis fascia helps to prevent nerve damage and produces a much cleaner dissection. Slowly creating a distinct plane of dissection down to the zygomaticofrontal suture line and avoiding excess retraction help to prevent unwanted bleeding from the sentinel vein (zygomaticotemporal vein), which needs not be sacrificed for a standard endoscopic forehead and brow lift.

Dissection for a standard endoscopic brow lift should not proceed all the way to the zygomatic arch but should stop approximately 1 cm above this level. If an extended midface lift is planned and there is a desire to elevate tissue over the zygomatic arch itself, dissection must go below the superficial layer of deep temporal fascia just above the arch. Abbreviated midface lifts performed simultaneously with endoscopic brow lifts may simply stay in the subperiosteal plane along the lateral orbital rim and avoid the more risky full-arch release. The beauty of the classic endoscopic brow lift is its versatility and the ease with which additional procedures can be combined simultaneously with this elegant cosmetic surgery. For instance, the temporal incision of an endoscopic forehead lift can easily be extended inferiorly to meet up with the preauricular incision from a standard lower facelift. Also, midfacelifting (with intraoral dissection) can connect the intraoral subperiosteal dissection over the zygoma to the subperiosteal plane from the endoscopic brow lift through a tunnel near the lateral orbital rim (Figure 68-31).

After all dissection is complete, appropriate elevation and fixation is required (Figure 68-32). Many techniques have been described such as tissue suture only, bone screws and plates, resorbable screws, bone tunnels, local skin excision, temporalis muscle exposure for added scarification, tissue glue, and tight head wraps.[102] Regardless of any specific fixation technique, the key to long-term fixation is adequate lower forehead tissue release during endoscopic dissection. Failure to adequately release internal tissue results in a relapse of brow ptosis, even with heavy fixation and the appearance of a "nice" lift during surgery.

Once complete internal release of the forehead is obtained, the specific lifting vectors must be determined for the most pleasing aesthetic effect. The lateral third of the female brow



FIGURE 68-31. Before **(A)** and after **(B)** an endoscopic forehead, brow, and midface lift. *Arrows* represent vectors of lift. Fixation is performed at the level of the hairline through the temporal and parasagittal incisions shown.

is elevated to the greatest extent, which is up to 1 cm above the orbital rim. The medial brow should be only slightly above the rim level and definitely below the middle and lateral brow levels to avoid a surprised or bewildered expression (see Figure 68-14). Typically, the glabellar region is elevated on its own without the need for midline fixation, which helps to avoid overelevation medially. The lateral third of the brow is lifted straight up and fixated at the level of the hairline. The galeal tissue is typically secured to bone at this point, while the lateral brow is held at the desired height or 1 to 2 mm above the desired level.[12] Very little relapse occurs with proper technique and averages only 1 to 2 mm after 2 weeks. Measurements can also be made with clear circular templates from the pupil to the brow to help improve symmetry. The brow position remains very stable after this early recovery period (see Figure 68-28). A question remains as to the time required for complete fixation of the periosteum. Some animal studies suggest a full 12 weeks are required for what is termed *full histologic periosteal refixation.*[103] However, some clinical evidence suggests that adequate fixation occurs in as little as 7 days. An example is the common fixation technique used by many surgeons who place a single transcutaneous bone screw at each parasagittal incision, which is removed after only 1 week. The 1-week fixation technique has been used with success for many years. It has been suggested that longer bony fixation may provide longer-term retention and less early relapse that some have considered normal. The key to long-term fixation seems for now to be determined usually by proper tissue dissection and release (Figure 68-33).

Although there are many fixation techniques, the use of bone tunnels at the parasagittal incisions appears to be one of the best methods for fixating the galea and periosteum near the hairline to a bone tunnel created posteriorly under the incision using a single heavy suture (see Figure 68-32). Fixation of the lateral tail of the brow is performed at each temporal incision, where an isolated heavy suture plicates the temporoparietal fascia in a posterior and superior vector to the thick temporalis fascia. Optional creation of a small window of exposed temporalis muscle in this area may aid in internal scar formation and fixation. The vector of lift at this outer tail of the brow follows a line drawn at an angle from the outer nasal ala that passes just beside the lateral canthus (see Figure 68-31).

Final closure of the hair-bearing scalp incisions can be performed with skin staples only with excellent scar formation because no skin is excised and no pressure exists at the

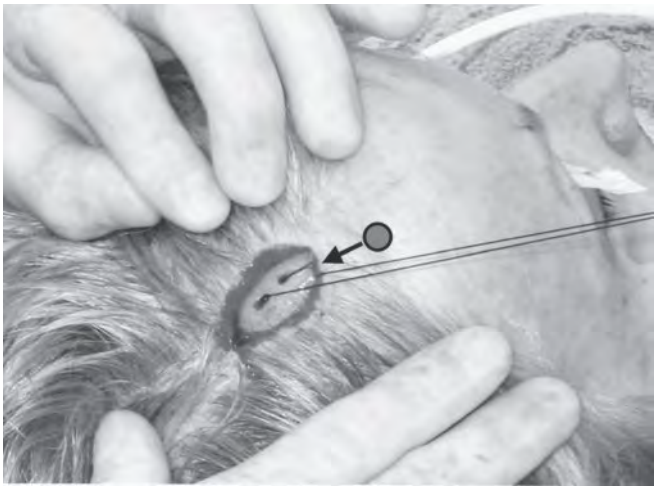


FIGURE 68-32. Example of bone tunnel fixation shown at the site of the right parasagittal incision. The *anterior circle* represents the position of suture placement through the galea, which elevates the lateral brow toward the bone tunnel.



FIGURE 68-33. The patient before an endoscopic brow lift **(left)** and 3 years after the lift **(right).** Extensive release of internal tissues was performed along the entire orbital rims superiorly with transection of the depressor muscles to obtain a very stable lift over time.

incision sites. Redundant tissue (forehead skin) created by an average of 1 cm of brow elevation is easily distributed evenly over the posterior 15 to 20 cm of elevated scalp, which essentially absorbs or redistributes this excess tissue with few to no signs of bunching. Because of this phenomenon, the endoscopic forehead and brow lift tends to elevate the hairline only a very small amount compared with the open skin excising coronal technique.

Interestingly, in a survey performed in 1998 of American Society of Plastic Surgeons members, of the total 6951 brow lifts performed by 570 members who returned the questionnaire, 3534 involved a coronal technique and incision and 3417 were performed endoscopically The most noted difference was the higher risk of hair loss with the coronal technique; however, both techniques enjoyed very low overall complication rates.

TEMPORAL LIFT

The rationale for lifting from a temporal approach alone is the potential to lift the outer brow and midface through a relatively conservative incision and with minimal dissection. Realistically, the procedure has a few potential problems that have kept the procedure from becoming extremely popular. The most worriesome problem is that the vector of dissection runs precisely perpendicular to the path of the frontal nerve making the potential for motor nerve injury possibly higher than other techniques. Also, the attempt to achieve significant lift in this tenacious area of tissue adhesion can be challenging and result in early relapse or inadequate lifting. Albeit challenging, a well-performed temporal lift may be the ideal procedure for the right patient.

The ideal patient for an isolate temporal brow lift has lateral brow hooding and mild midface ptosis but very little problems in the forehead or glabellar regions. For this patient, a temporal incision is made perpendicular to the desired vector of lift followed by dissection directly on top of temporalis fascia or in a very superficial subcutaneous plane (Figure 68-34A). Deep dissection below the nerve is inherently safer but requires more release of retaining ligament to obtain adequate lateral brow and ckeek lift. A subcutaneous flap above the frontal nerve is more likely to damage hair follicles or nerves, but obtaining adequate release for brow elevation is much easier and can be performed with the use of endoscopes or lighted retractors. Selection of the specific technique is operator dependent and based on the surgeon's comfort level. If adequate tissue release is achieved through the compact surgical region, good results can be obtained (see Figure 68-34B).

DIRECT BROW LIFT

The direct brow lift involves excision of an ellipse of skin adjacent to and just above the eyebrow (see Figure 68-34). A beveled incision is used to parallel the hair follicles of the brow or so that some follicles remain at the base of the bevel

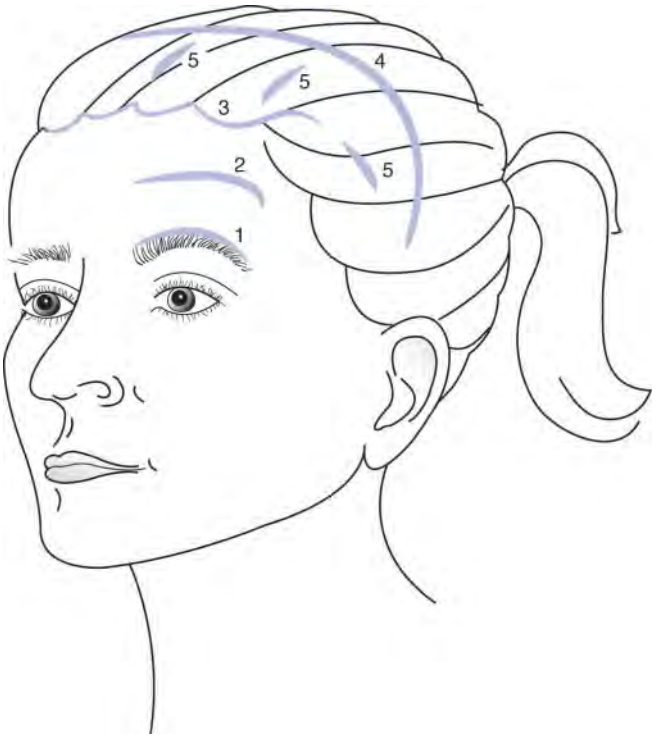


FIGURE 68-34. Representative incisions for typical brow lifting procedures: (1) direct brow lift, (2) midforehead lift, (3) trichophytic brow lift, (4) coronal brow lift, and (5) endoscopic brow lift.

to grow later above the scar. The dissection remains in the subcutaneous plane to avoid muscle or nerve injury.

Advantages of the direct brow lift are that it is a simple procedure (with an easy two-layer closure), it can be performed under local anesthesia, and it can treat brow position asymmetries. It remains a good alternative technique that may be an excellent option for an elderly patient who has severe brow ptosis and heavy wrinkles but cannot tolerate more extensive surgery and would benefit from a short procedure under local anesthesia. The main disadvantage is the potentially visible scar immediately above the brow.

MIDFOREHEAD AND BROW LIFT

Incisions made in the middle or upper forehead regions have similar advantages and disadvantages to the direct brow lift.[104–107] The incisions are made on each side of the forehead in an elliptical fashion so that the resulting scar follows a horizontal line already present in the forehead. Although this is probably the least used of all the techniques described, it may be a practical alternative for the elderly patient with thin eyebrows and deep horizontal rhytids who requires a short procedure under local anesthesia.

TRANSPALPEBRAL AND OTHER LOCAL BROW PROCEDURES

There has been a significant increase in the movement toward minimally invasive techniques to perform cosmetic

surgery. New techniques for forehead and brow rejuvenation fill the literature and offer potentially exciting methods to gain aesthetic improvement with less risk than with current procedures. A few such procedures include lateral brow lifting with temporal incisions only, denervation techniques through small punctures around the brow, and direct approaches through an upper blepharoplasty incision. It should be noted that, although procedures such as making small punctures to destroy medial portions of facial nerve innervating medial depressors may seem minimally invasive, they are certainly not without risk.

Many of the "minimally invasive" procedures take advantage of the proximity of the local depressor muscle. For instance, the transpalpebral or transblepharoplasty approach for forehead rejuvenation gains access to the local depressors through an upper eyelid incision.[108] Dissection through this incision involves a short distance to the corrugator supercilii, the procerus, and depressor supercilii of the glabella, which can each be selectively transected from this incision to reduced unwanted wrinkles and elevate the medial brow (see Figure 68-11). Likewise, the orbicularis can be incised and subperiosteal dissection performed above the orbital rim to elevate the lateral brow through this same local incision. Suture plication of the periosteum above the rim may further elevate the lateral brow.

Another adjunctive technique in the upper third of the face is that of fat grafting in areas of age-related fat atrophy. Fat can essentially be grafted anywhere; however, caution is required in the glabellar region where occasional local necrosis can occur from fat infiltration. This also occurs occasionally after collagen injections in the same region.[109] There are a great number of alternative techniques, and each must be evaluated for safety, efficacy, and longevity on an individual basis.

BOTULINUM TOXIN–ASSISTED BROW LIFT

Botulinum toxin has been used for nearly 2 decades to improve the aesthetic appearance of the upper third of the face by reducing wrinkles of the forehead (horizontal lines), glabella (frown and bunny lines), and lateral orbital crow's-feet (laugh lines).[110] More recently, it has been used specifically to elevate certain regions of the brow to obtain a "chemical brow lift."[111] The depressor muscles are paralyzed with botulinum toxin not only to reduce the wrinkles they create but also to allow the frontalis muscle to elevate the brow farther because of the decrease in muscular antagonism. By decreasing the tone and downward pull of the orbicularis immediately below the brow, the lateral third of the eyebrow elevates approximately 2 to 4 mm from the result of botulinum toxin placed in the upper crow's-feet area. Such treatment of depressor muscles in the glabellar region can help elevate the medial brow. Of course, as with surgical brow lifting, overcorrection in the medial brow may result in an abnormal facial expression.

Dosages used vary with individuals. Botox comes in a 100-unit vial to be mixed with 1 to 10 mL normal saline. The more dilute solutions (6–10 mL/100 units) begin to lose efficacy and can distort the tissue, whereas high concentration mixtures (1–2 mL/100 units) may be wasteful and imprecise. Regardless of dilution, the total dosage in units of botulinum toxin and its proper placement determine the outcome. For most individuals, 5 to 10 units is all that is required for each lateral crow's-foot region. However, the larger muscles of the glabella (procerus and corrugators) require at least 15 units of the toxin and up to 50 units for maximum results. Appropriate dosage in the glabella is the most variable. Treatment of horizontal forehead lines typically requires between 15 and 25 units. It should be noted that simultaneous treatment of horizontal forehead lines from the frontalis may decrease or eliminate brow elevation that otherwise may have been created by botulinum toxin treatment of the depressor muscles. Moreover, excessive toxin treatment of horizontal lines close to the eyebrows (within 1 cm) should often be avoided owing to the risk of true ptosis of the forehead, brow, and upper eyelids.

Botulinum toxin has also been recommended to aid long-term stability of the surgical forehead and brow lift. The theory involved is that control of the downward pull of the depressors (by temporarily paralyzing them chemically) gives the periosteum time to attach securely in an elevated position. The injection can be done during surgery, but there is an increased risk of eyelid ptosis and an unwanted delay because botulinum toxin typically takes 3 to 5 days to take full effect. Therefore, ideally botulinum toxin is injected 1 to 2 weeks before surgery. Regardless of any benefit this may give to long-term surgical fixation, the resulting reduction in wrinkles of the forehead and glabella and in crow's-feet is almost always popular with patients, even though the results last for only 3 to 6 months.

ADJUNCTIVE PROCEDURES: SKIN CARE AND MICROPIGMENTATION

A variety of procedures can be used for the superficial treatment of poor skin texture and are covered more completely in Chapter 70. For complete rejuvenation of the upper third of the face, skin resurfacing techniques may be required to address aging problems, especially those related to sun exposure, that cannot be adequately treated with lifting methods alone.

Prior to any resurfacing procedure such as laser skin resurfacing, chemical peels, or dermabrasion, the patient should be treated with topical skin medications to decrease the risk of scarring and pigment problems. Retinoic acid–type preparations used for ideally 6 weeks before resurfacing and 4% hydroquinone for patients with darker skin tones (Fitzpatrick 3 or higher) are two possibilities (see Chapter 70). Simultaneous resurfacing procedures can be accomplished with brow lifting, provided the surgical plane of dissection is subperiosteal or subgaleal and not subcutaneous.

Another adjunctive procedure growing in popularity is medical micropigmentation. The use of new skin pigments

that do not contain iron oxide has improved the appearance of tattoos placed to enhance a thin eyebrow or as permanently applied eyeliner. The ink is relatively permanent but often requires touch-ups owing to some fading over the first 3 to 5 years. Patients who have poor hand motor skills can greatly benefit from this procedure. A certified technician under a doctor's supervision usually performs the micropigmentation. However, consultation with a surgeon before micropigmentation is important because placement of a permanent brow tattoo in a more elevated position may create problems if the patient desires a surgical brow lift later. Therefore, if a patient is seeking brow lifting in addition to the micropigmentation, it is advisable to perform the surgical brow lift before the permanent makeup if feasible.

POSTOPERATIVE CARE

Following surgical forehead and brow lifting, a compression bandage is applied using a material such as Coban or Coflex. The pressure helps to limit edema and hematoma formation while possibly improving fixation. Typically, a drain is not required if a very dry field has been maintained. The patient should be instructed to limit activity and to use cold compresses over the eyes and brows. Head elevation is also recommended for the first several days. Avoidance of antiplatelet drugs preoperatively, a careful surgical technique, and the immediate postoperative use of cold compresses, elevation, and limited strenuous activity significantly decrease postoperative healing time.

The relatively snug postoperative dressing may be removed on postoperative day 1 to visually inspect the surgical site for any problems. A less constrictive Velcro-type head wrap can then be used to allow patient comfort and easy removal for showering. Patients are allowed to gently shampoo their hair after 24 hours but must be cautioned to avoid water pressure directly over any incision sites. Each incision is then cleaned twice a day with a dilute peroxide solution, and a thin layer of antibiotic ointment is applied for the first week. Staples are removed at the end of 1 week. Chemical treatments of hair such as "perms" should be delayed for at least 2 weeks to avoid possible hair loss as a reaction to the harsh chemicals. Hot curling irons or other similar devices must be used with caution because areas of scalp anesthesia may be present for months and can predispose a patient to an accidental self-inflicted burn.

COMPLICATIONS

Fortunately, major complications are rare with properly performed forehead and brow rejuvenation procedures. Good patient selection, diligent preoperative planning, meticulous surgical technique, and thorough postoperative care are all required to help limit the chance for complications.[112–115] Minor complications can always occur despite a surgeon's best efforts. No matter how minor the problem, the patient must be treated with concern and compassion. Typically, patients who undergo cosmetic surgery are expecting to look better as soon as possible and are not always as tolerant of perioperative problems as are trauma patients. Extensive edema and ecchymoses are not normally considered complications but may warrant appropriate reassurance and even simple suggestions to hasten recovery when feasible. Suggestions regarding makeup from a well-trained staff member may greatly improve a postoperative patient's mood when shown how to better hide persistent erythema or ecchymosis.

True complications include poor scar appearance, wound dehiscence, hematoma, skin sloughs or perforations, asymmetries, sensory disturbances, facial paralysis, eyelid ptosis, corneal abrasions, dry eye syndrome, hair loss (alopecia), infection, relapse, irregular facial expressions, and contour irregularities. Of all these potential problems, permanent facial paralysis and major tissue loss are the most devastating. Fortunately, these particular complications are rare (<0.3%, which is less than that for a standard lower facelift). Regardless, it is critical to know the precise anatomy and to avoid improper or excessive retraction, overzealous cautery, and overthinning of the flaps when transecting the depressors. In addition, hematomas must be diagnosed and treated without delay.

Some problems such as corneal abrasions can be very concerning to the patient owing to the severe pain and can be nearly eliminated by proper technique and perioperative attention to detail. For instance, an eye lubricant should always be used. Also, thought should be given to the placement of temporary tape strips, such as Steri-Strips, over the eyelids or a tarsorrhaphy suture to help prevent inadvertent scratching of the cornea by gauze or tubing, for example, during the procedure (see Figure 68-16). All severe pain requires immediate evaluation, and suspected abrasion should be treated by appropriate ophthalmic drops for pain and patching of the affected eye for 12 to 24 hours. Appropriate ophthalmologic consultation is required for persistent or uncontrollable eye pain, persistent dry eye symptoms, or unusual changes in vision. Minor blurred vision for the first 12 hours is not unusual owing to chemosis and use of ophthalmic ointments.

Alopecia and sensory disturbances can be bothersome to the patient and often are not permanent. The problem is the inability to predict whether the numbness a patient has will partially, fully, or not go away, and just how soon it might be alleviated. With proper technique, an endoscopic forehead and brow lift has a high rate of sensory nerve recovery, but full recovery may take several months and require patient reassurance. Although exact numbers are not known, empirical observation of the last 300 endoscopic brow lifts suggests that sensory disturbances are an occasional early concern but an unusual complaint after 6 to 12 months. Most patients have early sensation of the forehead but numbness in the posterior scalp supplied by the deep branch of the supraorbital nerve. This is typically not a major concern to the patient and slowly improves over a period of 8 months. Alopecia, connversely, is a significant concern, especially if it persists longer than 6 to



FIGURE 68-35. The patient 3 months after a trichophytic brow lift. She hoped to lift her brows and at the same time lower her high hairline. **A** and **C,** She developed severe telogen affluvium type alopecia with hair loss in a very sporadic pattern. **B** and **D,** Fortunately, she had complete hair regrowth, by 1 year postoperative, as seen on the right with hair growth in front of the beveled incision to create a nice scar behind the hairline.

12 months. Hair may return after an average 4- to 8-month dormancy period of the hair follicle. (Figure 68-35). The "shock" period of hair loss is called *telogen affluvium* and can be dramatic, but fortunately, it is temporary. However, excessive tension on the flaps, rough handling of wound margins, or excessive use of cautery near follicles may lead to permanent hair loss that requires treatment.[116]

Proper planning, technique, and postoperative care helps to reduce the incidence of complications. Immediate and appropriate treatment along with sincere concern for the patient's well-being should help to reduce the chance of the situation worsening or the patient being dissatisfied.

SUMMARY AND CONCLUSIONS

An explosion in the number of rejuvenation techniques for the upper face in the past decade, lead by the use of endoscopes and botulinum toxin, has revolutionized the treatment of aging in this area. Cosmetic surgery treatment of the upper third of the face is frequently an essential component for complete facial rejuvenation. Procedures are highly variable and can offer improvement to both young and old. Matching the problems to the ideal rejuvenation techniques is essential for maximum aesthetic benefits. Even the best surgical technique can result in inadequate or even poor results if improper patient selection or incorrect diagnoses are made; for this reason, the forehead and brow area must be evaluated critically for a wide range of interlacing diagnoses.

Specific skin problems vary with a patient's age and sex, but gravity remains consistent and nonselective; therefore, the only issues regarding the occurrence of brow ptosis are when it will occur and how severe it will be. Wrinkles are also inevitable but may be dynamic or static in nature. Thanks to botulinum toxin, the previously difficult treatment of dynamic upper facial lines can be effected at low risk with a simple injection. The common and consistent finding of brow ptosis, especially in the lateral third of the brow, may now be selectively treated endoscopically to achieve a more youthful appearance. Society's idea of beauty at any one moment in time will ultimately help to guide the patient and surgeon to choose where the brow should be placed as opposed to merely raising it higher. True rejuvenation is

likely more complex and involves multiple modalities and even tissue replacement such as fat grafting. Fat grafting is poised to be one of the most rejuvenating procedures of the face if more consistent techniques develop. The use of stem cells to enhance fat grafting results will likely become the norm at some point in the future. Only time and persistence will prove what best restores youth to the upper face.

Facial cosmetic surgery continues to rise in popularity exponentially. The aging population wants to feel and look more youthful but nonetheless demands to remain natural looking. Today's discerning patient is often very knowledgeable on the subject of the cosmetic surgery options and may insist on a specific technique. The advice of a well-trained surgeon and diagnostician may make or break the ultimate result and prevent a cosmetic disaster. It is vital that the surgeon refuse to perform treatment that is not in the best interest of the patient. Cosmetic surgery is a luxury and is an optional procedure, no matter how much of an emergency it seems to the patient. At the end of the day, it is the surgeon's responsibility to provide the patient with the best and safest options available to achieve realistic goals.

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69

CHAPTER

Otoplastic Surgery for the Protruding Ear

Todd G. Owsley, DDS, MD

Auricular deformities, in particular prominent or protruding ears, are a common congenital anomaly, affecting nearly 5% of the white population.[1] Congenital in nature, children are most likely to suffer the consequences of the deformity in the form of ridicule by their peers. Protruding ears, commonly referred to as *prominauris*, can be predictably treated for children before they enter grade school and, thus, help them avoid the emotional trauma caused by the ridicule. Otoplastic surgery is primarily performed on children and can be a valuable service for the patient and satisfying for the surgeon.

It is important for the surgeon to understand the history of various surgical techniques to develop a predictable and successful technique to address the problem of protruding ears. Dieffenbach, in 1845, is credited with the first otoplastic technique to correct a prominent auricle.[2] Ely, in 1881,[3] authored the first case report describing correction of prominent ears in a 12-year-old boy who was being teased at school. Since that report, over 180 surgical techniques have been described in the literature for the correction of protruding ears.

When clinically evaluating the facial complex, the ears are often overlooked. If protruding ears are present, reduction otoplasty as an adjunctive or isolated procedure can be performed predictably and often with satisfying results. A thorough understanding of the embryology and development of the human auricle along with the resultant external anatomy of the ear is of paramount importance in developing a predictable and stable technique to deal with the common auricular deformities.

EMBRYOLOGY OF THE AURICLE

Malformations of the auricle are common, occurring in 1 out of 12,500 births.[4] They can occur alone or in combination with a syndrome affecting the head and neck structures. The embryogenesis of the auricle exemplifies in miniature the precise and logical progression so characteristic of the developing human form. The external ear development during the 3rd to 12th weeks of embryonic life is complex. The precursors to the auricle present in days 36 to 38 of intrauterine life, developing first from the first branchial groove where the first (mandibular) and second (hyoid) branchial arches are present (Figure 69-1). Both arches give rise to the auricular hillocks often referred to as the *auricular tubercles of His*.[4] Numbers 1, 2, and 3 arise from the caudal border of the mandibular arch, and numbers 4, 5, and 6 are formed from the cephalic border of the hyoid arch. The auricular hillocks present in their most prominent and characteristic form by intrauterine day 41. During this same stage, the groove between the mandibular and the hyoid arches (hyomandibular groove) widens and deepens by the increased growth of the hillocks. This groove eventually forms the external auditory canal and concha. By days 43 to 45, the hillocks have migrated and coalesced to form the auricle.[4] During this union, the mesenchyme of the hyoid arch increases substantially relative to the mandibular arch to contribute 85% of the external adult ear. Hillocks 2 and 3 from the mandibular arch lose their individuality and fuse to form the helical crus. Later, hillocks 4 and 5 from the hyoid arch merge and alter their configuration as they give rise to the helix and antihelical fold. Hillock 1 remains prominent and becomes the tragus, and hillock 6 becomes the antitragus.[4]

SURGICAL ANATOMY

The majority of the growth of the pinna is completed by an early age. The average child has 85% ear development by 3 years of age. The ear is nearly fully grown by the age of

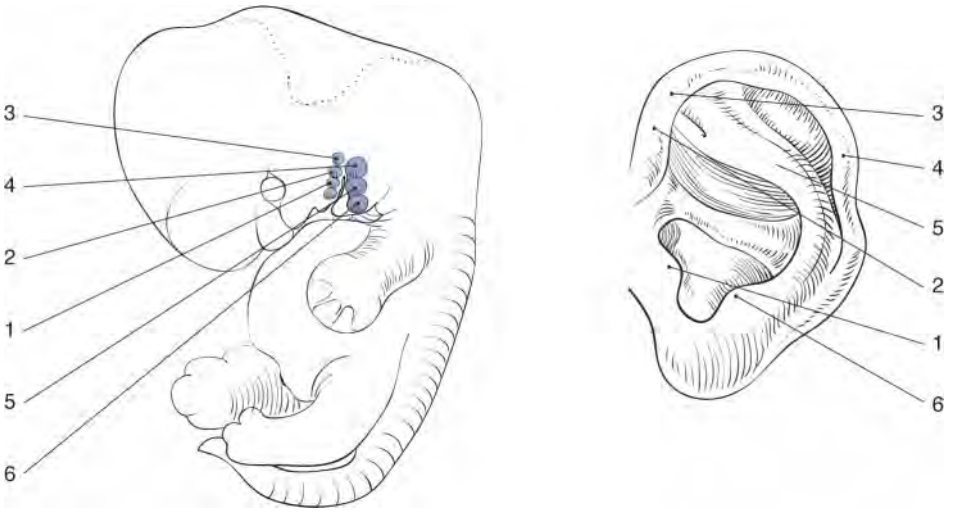


FIGURE 69-1. Development of the auricle from the first and second branchial arches. The *numbers in the left diagram* correspond to the structures in the right diagram. (Adapted from Owsley T. Otoplastic surgery for the protruding ear. In Fonseca RJ, editor. Oral and Maxillofacial Surgery. Vol 6. Philadelphia: WB Saunders; 2000; pp. 408–418.)

7 to 8 years. The ear height continues to grow into adulthood, but the width and distance of the ear from the scalp change little after 10 years of age. It is important to note that each individual's ears often vary in size and shape.[5] The average adult ear is approximately 6.5 cm in length and 3.5 cm in width. In the normal ear, the auricle lies between horizontal lines drawn from the upper rim of the orbit and the nasal spine. The normal posterior wall of the conchal bowl is set at an angle of approximately 90 degree to the mastoid.[6] A second 90-degree angle is formed as the antihelical fold and is called the *scaphaconchal angle*. These two angles in combination with the curvature of the helix set the auricle adjacent to the scalp at approximately 25 to 35 degrees and is called the *auriculocephalic angle* (Figure 69-2).[7] In otoplastic surgery, when correcting prominent ears, three important anatomic pearls can be used intraoperatively to assess the final result. The helical rim should be seen just lateral to the most lateral presence of the antihelix from the frontal view. The distance measured between the helical rim and the mastoid area is slightly less than 2 cm. Finally, the distance between the skull and the uppermost aspect of the helix is approximately 1 cm (Figure 69-3).

The auricular cartilage is a unique and delicate structure that is intricately shaped with multiple elevations and depressions providing both skeletal support and form to the adult ear. The cartilage of the auricle is a single piece of yellow (elastic) fibrocartilage with a complicated relief on the anterior, concave side and a smooth, posterior convex side. Cartilage thickness is fairly uniform throughout. The cartilage is covered on both surfaces by a thin, firm, adherent layer of perichondrium. The anterior lateral surface of the cartilage is covered with a fine, thin skin, closely adherent to the cartilaginous framework. Subcutaneous fat is practically nonexistent, but a diffuse subdermal vascular plane exists that supports flap viability.[8] The posterior surface of the cartilage framework is draped with a less adherent skin that contains two layers of fat and a larger subdermal plexus of arteries, veins, and nerves.

A helical border terminates anteriorly in a crus, commonly called the *radix*, which lies almost horizontally above the external auditory meatus. The antihelix crowning the posterior conchal wall separates and diverges into both a superior and an anterior crus enclosing the triangularis fossa. Between the helix and the antihelix lies a long, deep furrow called the *scaphoid fossa*. The conchal cavity, composed of the cymba (superior) and cavum (inferior) concha, arises from the floor, which is approximately 8 mm deeper than

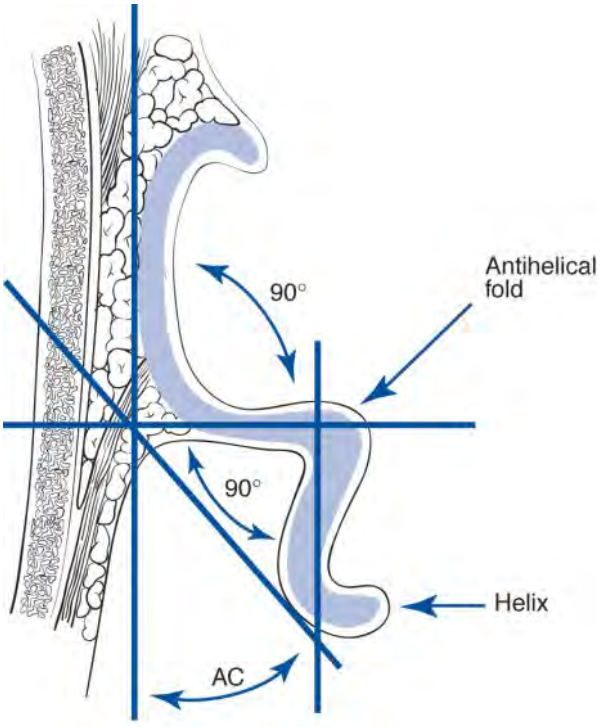


FIGURE 69-2. An axial section at the level of the helical crus demonstrates the ideal auriculocephalic angle (AC) of 30 degrees. (Adapted from Tanzer RC. Congenital deformities. In Converse J, editor. Reconstructive Plastic Surgery. Vol 4. 2nd ed. Philadelphia: WB Saunders; 1977.)

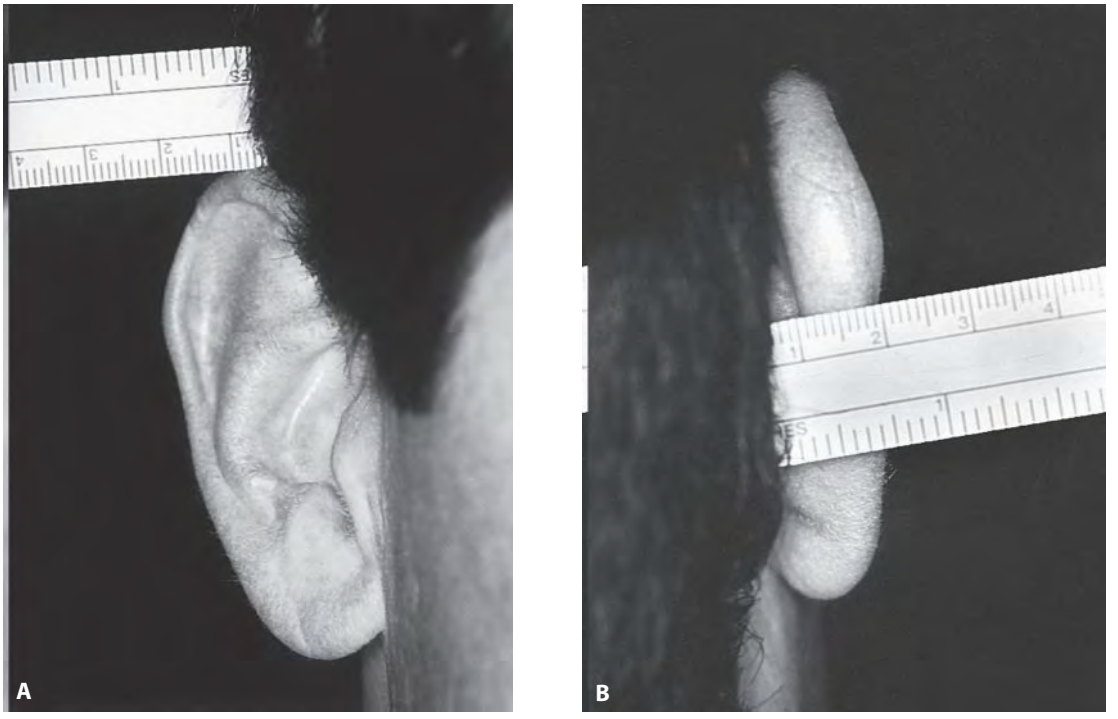


FIGURE 69-3. **A** and **B,** Anatomic "pearls," useful intraoperatively. (**A** and **B,** Reproduced with permission from Owsley T. Otoplastic surgery for the protruding ear. In Fonseca RJ, editor. Oral and Maxillofacial Surgery. Vol 6. Philadelphia: WB Saunders; 2000; pp. 408–418.)

the overlying tragus and antitragus. The inferior tip of the helical cartilage is referred to as the *cauda* or *tail*. Extending from this inferiorly is the lobule, hanging devoid of skeleton (Figure 69-4).

BLOOD SUPPLY

The arterial blood supply to the ear is derived principally from two main branches of the external carotid artery: superficial temporal artery and posterior auricular artery. The superficial temporal artery emerges from the parotid capsule, 1 cm in front of the ear deep to the veins and below the anterior auricular muscle. It gives off the superior, medial, and inferior branches supplying the anterior and anterolateral surface of the auricle (Figure 69-5A). The posterior surface is dominantly supplied by the posterior auricular artery, which travels parallel to the postauricular crease upward crossing below the great auricular nerve and under the posterior auricular muscle. Awareness of this relationship is important to avoid damage to the artery or nerve during surgery. The posterior auricular artery gives off three branches—superior, medial, and inferior—providing a greater volume of blood to the postauricular ear than its anterior counterparts. These same vessels perforate the auricular cartilage over a large surface of the anterior ear and anastomose with the branches of the superficial temporal artery. The external ears have a tremendous blood supply, allowing multiple surgical approaches or salvage of the ear after traumatic avulsion.

Venous drainage of the ear via the complementary veins is into the external jugular vein. Lymphatic drainage is into three surrounding areas via the complex and extensive fine network of lymphatic vessels.

NERVE SUPPLY

The sensory nerve supply is primarily from the anterior and posterior branches of the great auricular nerve. The nerve is an important surgical landmark traveling 8 mm posterior to the postauricular crease. When dissecting in this area, care must be taken to avoid damage to the nerve that can result in near-complete anesthesia to the ear. Less important contributions of sensation are made by the auriculotemporal and lesser occipital nerves to the conchal cavity and external auditory meatus (see Figure 69-5B). Regional anesthesia of the auricle is readily accomplished by instilling anesthetic solution along its base anteriorly and posteriorly. Supplemental anesthesia may be needed at the posterior wall of the external auditory meatus supplied by the auricular branches of the vagus nerve (Arnold's nerve).

DEFORMITIES

Ear deformities are common and variable owing to the complex embryologic engineering that takes place in the development of the auricle, as described previously. Many classification systems of ear deformities have been attempted.

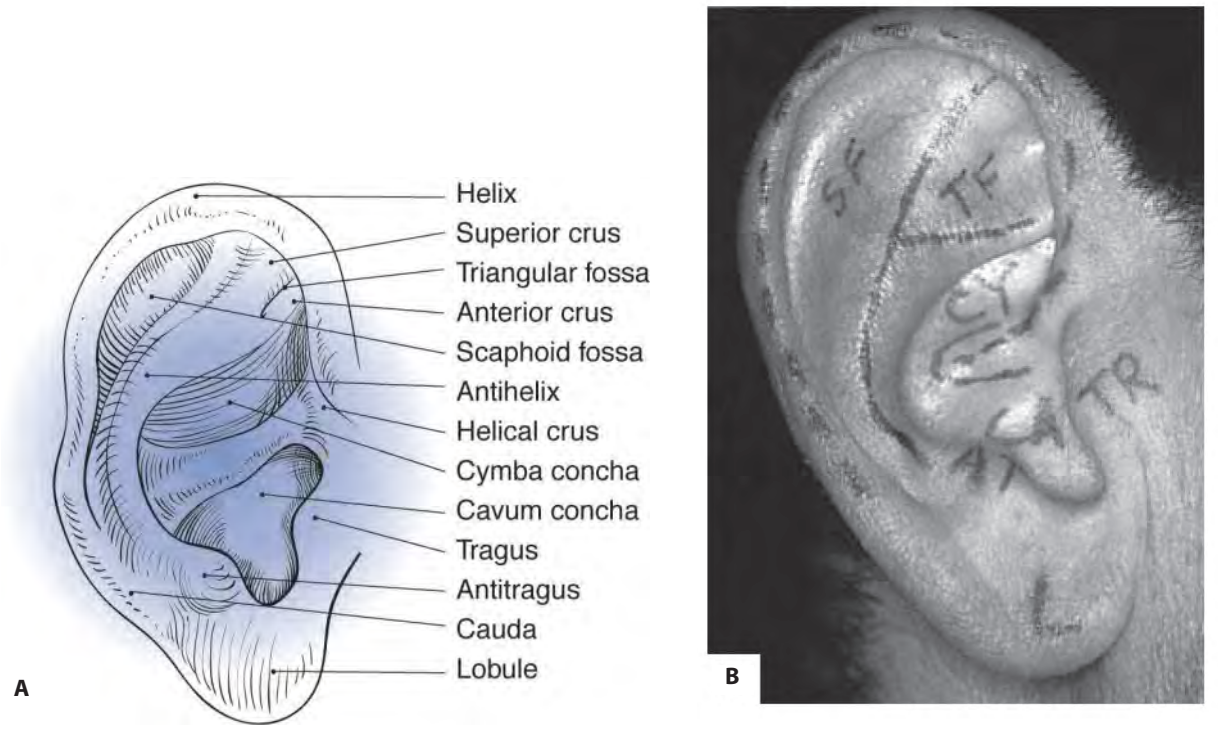
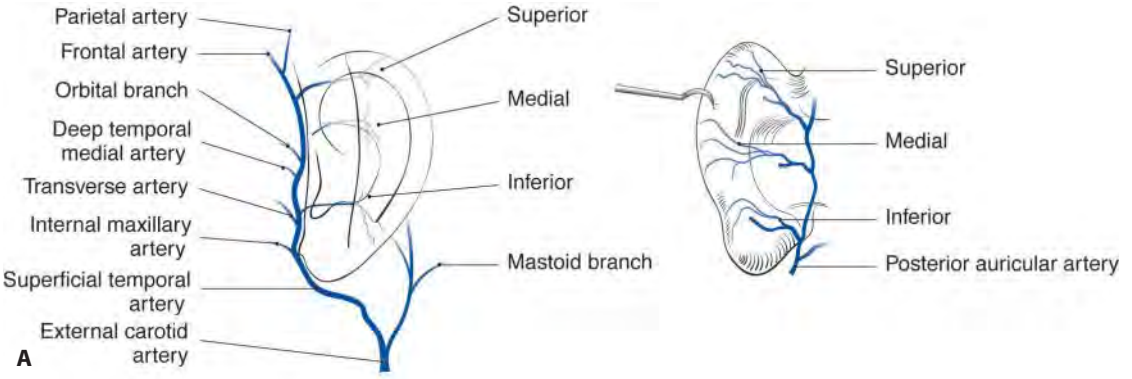


FIGURE 69-4. **A** and **B,** Anatomy of the external ear.



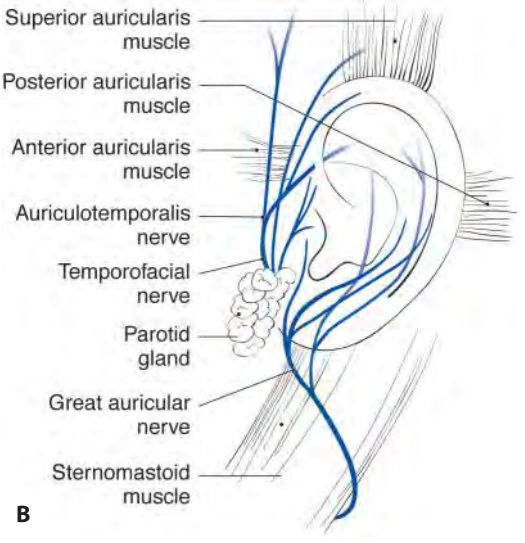


FIGURE 69-5. **A,** Arterial blood supply. **B,** Nerve supply. (**A** and **B,** Adapted from Owsley T. Otoplastic surgery for the protruding ear. In Fonseca RJ, editor. Oral and Maxillofacial Surgery. Vol 6. Philadelphia: WB Saunders; 2000; pp. 408–418.)

Tanzer[9] classifies congenital ear defects, correlating embryologic development with a surgical approach for correction of the deformity. Marx[10] has a well-accepted classification system subdividing microtia into three groups according to severity. Rogers[6] simplifies classification of congenital ear defects, dividing them into four groups according to various stages of arrested development: microtia, lop ear, cup ear, and protruding ear. In this chapter addressing the prominent ear, the term *protruding* is used as a general term to refer to any ear that is more prominent than is considered "normal." It is beyond the scope of this chapter to classify other congenital abnormalities and their correction.

Protruding Ear

In patients with protruding ears, there are two major deformities that individually or in combination account for the majority of the abnormalities. Several lesser deformities are seen that can accentuate the abnormality as well. The most common is a poorly developed antihelical fold that can involve both the superior and the anterior crura. This eliminates a definition between the conchal cavity and the scapha, resulting in the lateral projection of the upper portion of the helix. The second abnormality is the formation of excessive conchal cartilage, in particular the posterior conchal wall. This causes significant protrusion of the auricle. It is not uncommon to recognize some degree of both abnormalities present producing the protruding auricle.

Other potential deformities are a protruding earlobe, irregularities along the helix including an unrolled margin of the helical rim, and more recently described, an anteriomedially displaced insertion of the postauricular muscle. Of importance, this deformity is often bilateral and can be associated with ossicular deformities and a hearing deficit.[11]

Obviously, precise recognition of the cause or causes of the protruding ear is crucial in formulating the surgical technique to be employed in correction of the abnormality.

SURGICAL CORRECTION

Perhaps the most important indication for surgical correction of prominent ears is to eliminate the psychosocial effects that the ear defect produces. Macgregor[12] and others have well documented the irreversible social and psychological consequences that facial anomalies, especially of the ears, can inflict. The ridicule by peers begins as early as age 4 to 5 years during development, described as the body image concept. This leads to problems with social adjustment, self-image deficit, and ultimately, behavioral disorders. Therefore, most patients present at an early age for surgical correction. Most surgeons agree that surgical correction of the ear can be performed safely between 4 years of age and the beginning of school attendance to avoid the ridicule, becoming one of the most common elective procedures performed in young children. Adults also present to correct a life-long cosmetic defect.

SURGICAL TECHNIQUES

Many otoplasty techniques described in the surgical literature are similar and have evolved over an 80-year experience. Many techniques address only the conchal hypertrophy without mention of the antihelical fold. The described techniques generally can be subdivided into a "suture-only" technique, a cartilage splitting or weakening technique, or a combination of the two.

Furnas, in 1968,[13] described his suture-only technique for the correction of the prominent ear deformity. This technique consisted of a postauricular approach, removing the skin, connective tissue, and vestigial posterior auricular muscle and its ligament down to the underlying mastoid fascia. A total of two or three nonresorbable sutures are placed through conchal cartilage and mastoid fascia and tightened to the new desired retracted position of the ears. This remains a commonly used technique to set back the ears in patients with conchal hypertrophy alone. Problems associated with this technique have been relapse secondary to the cartilaginous memory, a shallow conchal bowl, and irregularities created in folding the conchal floor. Another difficult problem is displacing the conchal bowl anteriorly, creating narrowing of the external auditory canal.

The Converse–Wood-Smith technique[14] is used to correct and create an antihelical fold using a cartilage cutting and suture method. Several full-thickness cuts through cartilage are placed in the scaphoid fossa. The cartilage is then folded on itself, described as "tubing," and permanent horizontal mattress sutures are placed, forming the new antihelical fold. In addition, if excessive conchal cartilage is present, it can be addressed with a cartilage incision added at the rim of the bowl to reduce the bowl and retract the ear. A new antihelical fold is formed, however, typically with sharp cartilaginous ridges seen through the thin anterior auricular skin.

This chapter describes two techniques that address the two fundamental abnormalities in protruding ears and have produced consistent, satisfying cosmetic results, essentially eliminating the fear of relapse. The Davis method addresses the protruding ear caused by a hypertrophic posterior wall of the conchal bowl.[15] The Mustarde method is used to create a new antihelical fold[16] (Figure 69-6).

The refinement of other abnormalities such as the prominent earlobe are also discussed.

Davis Method

The Davis method, for the correction of conchal hypertrophy, is a cartilage excision technique performed in a step-wise fashion.[8,15]

1. Marking the cartilage excision: Initially, the area of cartilage to be removed is marked on the skin of the anterior surface of the conchal bowl. Methylene blue transfixion tattoos are preferred to mark the posterior surface of the conchal cavity. The line lies just below the lower crus border, carries forward into the cymbal fossa, and continues around as an "arrowhead" to preserve a well-defined



FIGURE 69-6. **A–D,** Preoperative and postoperative photographs of an 11-year-old girl who presented for correction of protruding ears. Her diagnosis was a combination of conchal hypertrophy and lack of antihelical fold formation bilaterally. The Davis and Mustarde techniques were used on both ears. **E–H,** Lateral views of both ears demonstrate a natural-appearing, new antihelical fold and a deepened conchal bowl.

helical radix. The tracing should continue forward against the posterior edge of the external auditory canal. From that location, it curves posteriorly into the cavum concha, continuing onto the posterior conchal wall, leaving 8 mm of posterior conchal wall height measured from the conchal scaphal junction. This should complete the circle, which should appear "kidney bean" shaped including the entire conchal bowl. The exact height of the posterior wall is important and must be measured for each case individually (Figure 69-7A-C).

2. Removal of skin and cartilage: Before beginning the actual surgical procedure, local anesthesia is used for hydrodissection of the tightly adhered anterior auricular skin along the conchal bowl. This simplifies the dissection of cartilage from the overlying skin. The concha is exposed through a postauricular elliptical skin excision. The width of the ellipse is only to remove the predicted excess skin that is produced with ear retropositioning. The closure of the incision should be passive. This area is predisposed to hypertrophic or keloidal scarring when closed under tension. Once having removed the postauricular skin, the conchal cartilage is visualized along with the previously placed tattoo marks. The marks are used as a guide for incising through the cartilage, taking care to avoid perforation of the anterior skin. The dissection is then carried onto the anterior conchal cartilage surface subperichondrially. The hydrodissection along with a small, sharp Freer elevator allows separation between the skin and the cartilage. The cartilage is then removed, which includes the entire conchal bowl with the exception of the 8 mm left along the posterior conchal wall. The ear is then placed passively onto the mastoid surface, and the new projection of the helical rim is observed and carefully measured. Any defective prominence can be revised with further cartilage removal. The postauricular muscle and underlying connective tissue are removed down to the underlying mastoid fascia to allow for passive skin draping of the conchal floor, producing a natural-appearing, deepened conchal bowl. Any excessive postauricular skin can be removed at this time (see Figure 69-7D–F).
3. Ear fixation: The ear is fixed in its new position with three or four mattress transfixion sutures of 3-0 silk that perforate the skin anteriorly, anchor deeply into the postauricular muscle stump, and pass back through the anterior skin. The mattress sutures are then gently tied over a dental cotton roll, moistened with a triple antibiotic ointment, which has been placed into the cymbal and caval fossa, making sure to place one end of the cotton roll into the external auditory canal to avoid postoperative stenosis. The sutures and cotton roll hold the ear in place during healing, stretch and flatten the skin uniformly over the conchal floor, and give depth to the conchal bowl. The postauricular incision is then closed in a running fashion with a resorbable suture, leaving a small opening inferiorly for drainage. The cotton roll dressing is left in place for a minimum of 1 week for optimal healing (see Figure 69-7G–I)

Mustarde Method

The Mustarde method, first described in 1959, is indicated for the prominent ear with a poorly formed or lack of an antihelical fold.[16] This cartilage weakening technique relies on precisely placed horizontal mattress sutures, creating a new antihelical fold. It is rarely performed alone and used commonly in combination with the Davis method described previously.

1. Antihelical fold markings: The scapha is folded back against the underlying scalp by applying digital pressure on the superior helical rim, which creates an antihelical fold. The crest of the fold is marked with a surgical marker. To prepare for placement of the mattress sutures, marks are placed parallel to the crest at least 7 mm apart to avoid creating too narrow a fold. The lateral marks placed on the skin are transferred to the underlying cartilage with a hypodermic needle dipped in methylene blue (Figure 69-8A and B).
2. Dissection and cartilage weakening: Local anesthetic solution is infiltrated along the scaphal fossa beneath the anterior auricular skin, hydrodissecting the skin from the underlying cartilage. This is to facilitate the anterior dissection and mattress suture placement. The postauricular skin is then removed in an identical fashion as that described with the Davis method. Once having identified the marks placed on the posterior surface of the scaphoid cartilage, a Freer elevator is passed through a small horizontal incision created through the cartilage at the most inferior aspect of the new antihelical fold. The anterior auricular skin is then dissected from the underlying cartilage corresponding to the crest of the new antihelical fold. Through the tunnel dissected along the anterior surface of the cartilage, the body of the new antihelical fold is weakened to facilitate folding and remove the inherent memory. Many methods have been described; however, cartilage weakening can be performed adequately using a Brown-Adson forceps, a nasal rasp, or a dermabrader with a small diamond fraise (see Figure 69-8C–E).
3. Suture placement: Nonresorbable, horizontal mattress sutures (4-0 Mersilene) are placed. The sutures are all placed through the medial perichondrium, cartilage, and lateral perichondrium, being careful not to include the anterior skin. The sutures should be placed perpendicularly across the antihelical fold, so that upon tightening, a well-rounded, antihelical fold will be created. The sutures are tightened under direct observation and adjusted accordingly to form the new antihelical fold (see Figure 69-8F and G).
4. Dressing: The dressing is important and is placed to provide adequate pressure to obliterate dead space and avoid a postoperative hematoma formation. A carefully layered dressing is placed over the anterior and opposing posterior surfaces of the scaphoid region with a ¼-inch petrolatum gauze, followed by 4 × 4-inch fluffs, held secure with a pressure-type facial dressing (see Figure 69-8H and I).

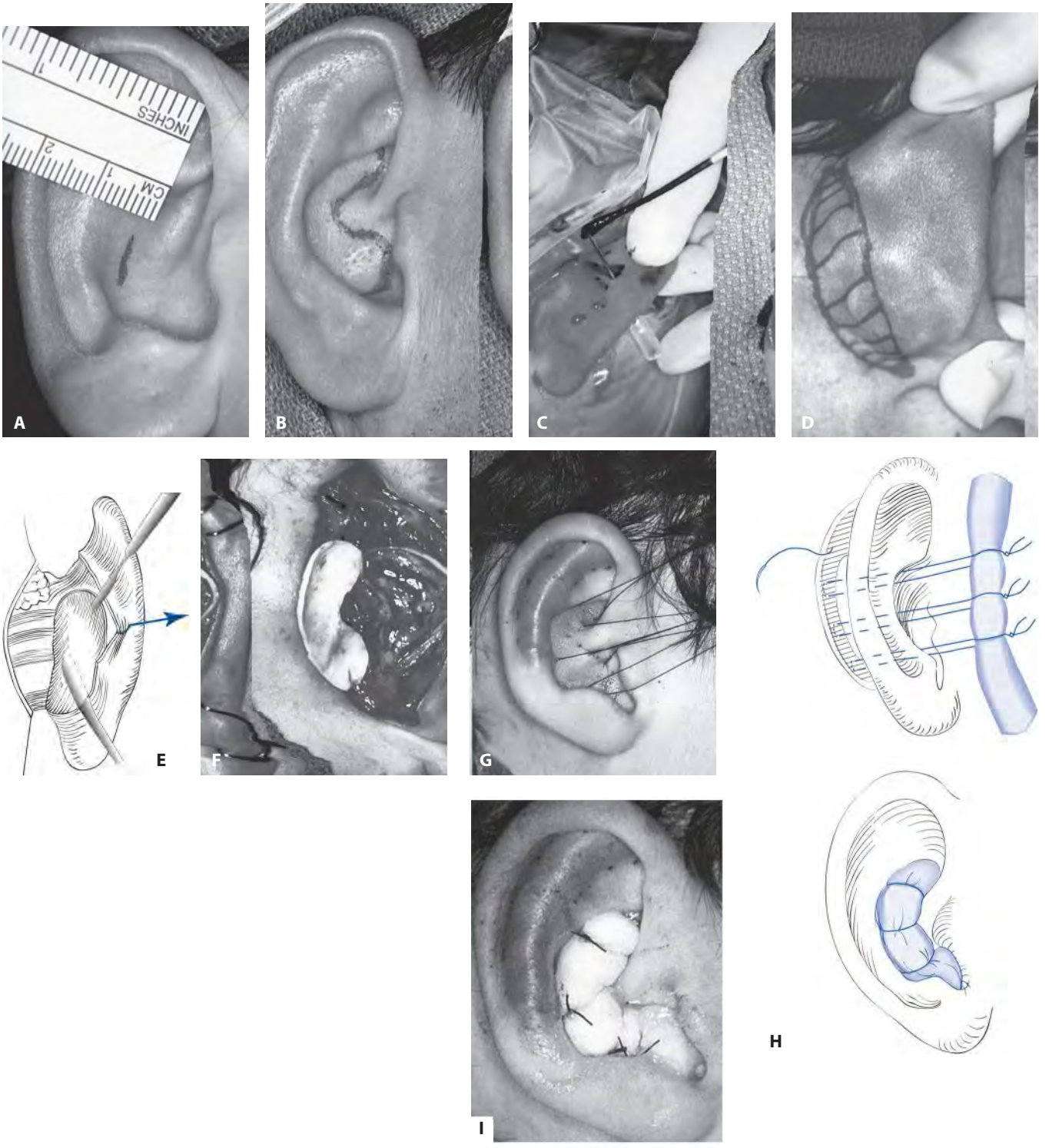


FIGURE 69-7. Davis method. **A,** Marking the height of the posterior conchal wall that will remain. **B,** Marking the conchal bowl to be excised. **C,** Transferring the marking to the underlying cartilage with methylene blue. **D,** Initial amount of skin to be removed in an elliptical fashion. **E** and **F,** Postauricular view of excised cartilage. **G–I,** Through-and-through fixation sutures anchored to the postauricular muscle and mastoid fascia used to secure the cotton bolster. (**A–D, F, G,** and **I,** Reproduced with permission from Owsley T. Otoplastic surgery for the protruding ear. In Fonseca RJ, editor. Oral and Maxillofacial Surgery. Vol 6. Philadelphia: WB Saunders; 2000; pp. 408–418; **E** and **H,** adapted from Owsley T. Otoplastic surgery for the protruding ear. In Fonseca RJ, editor. Oral and Maxillofacial Surgery. Vol 6. Philadelphia: WB Saunders; 2000; pp. 408–418.)

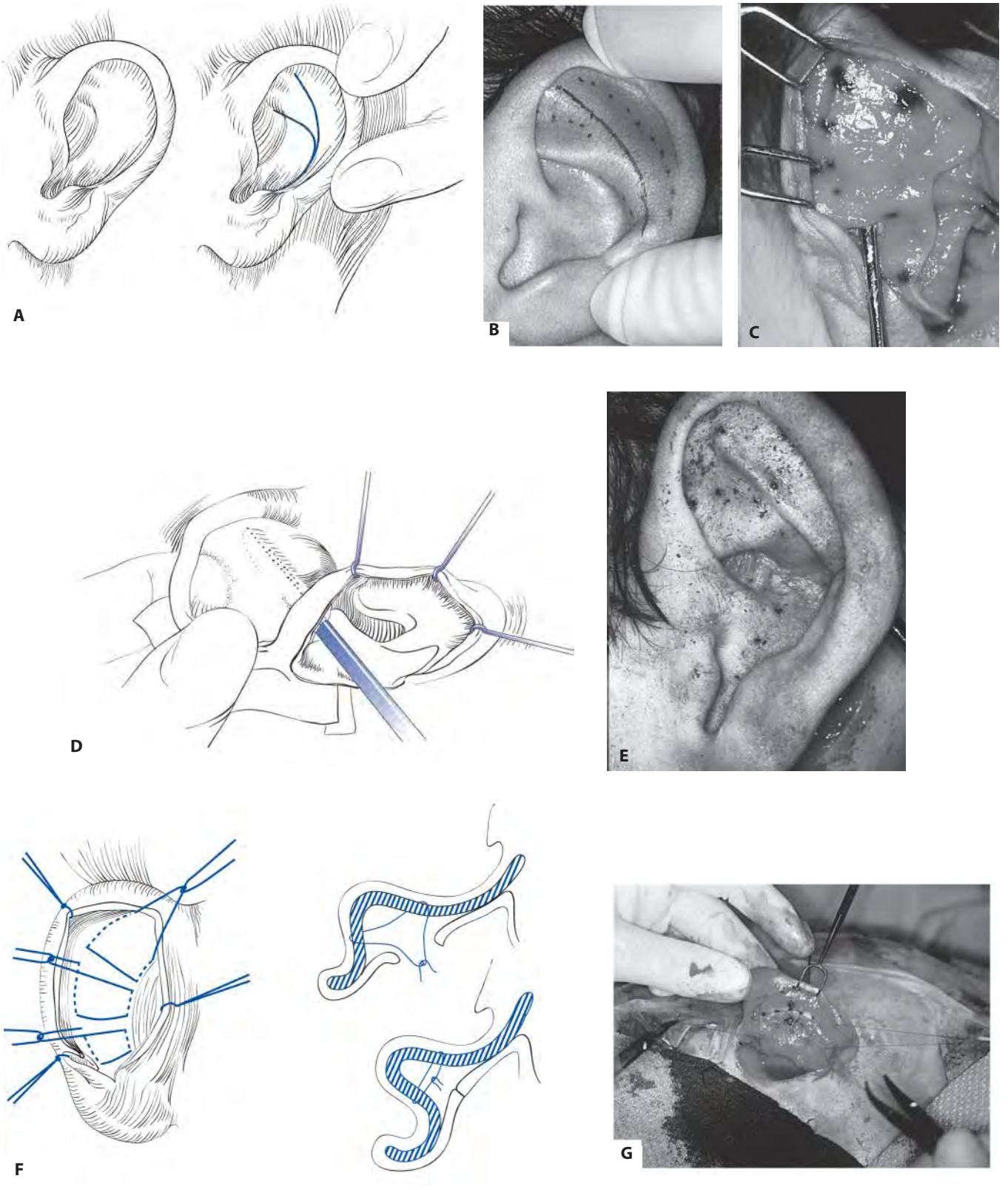


FIGURE 69-8. Mustarde technique. **A** and **B,** Creating and marking the desired antihelical fold by folding back the helix with digital pressure. Lines, parallel to the crest of the fold, are marked and transferred to the underlying cartilage and used for suture placement. **C–E,** Dissection of the scaphoid fossa beneath the anterior skin for weakening and creation of the desired antihelical fold. **F** and **G,** Placement of the horizontal mattress sutures to create a new antihelical fold. *(continued)*





FIGURE 69-8. *(continued)* **H** and **I,** Placement of petrolatum gauze and fluffs as an important pressure dressing. (**A, D,** and **F,** Adapted from Owsley T. Otoplastic surgery for the protruding ear. In Fonseca RJ, editor. Oral and Maxillofacial Surgery. Vol 6. Philadelphia: WB Saunders; 2000; pp. 408–418; **B, C, E,** and **G–I,** reproduced with permission from Owsley T. Otoplastic surgery for the protruding ear. In Fonseca RJ, editor. Oral and Maxillofacial Surgery. Vol 6. Philadelphia: WB Saunders; 2000; pp. 408–418.)

CORRECTION OF THE PROTRUDING EARLOBE

A protruding earlobe often accompanies a protruding ear. If so, it must be identified and corrected simultaneously. If the lobule of the ear protrudes, an extension of the posterior auricular incision is drawn with a surgical marker in the shape of a V onto the earlobe. Finger pressure on the freshly marked lobe, compressing it to the mastoid skin, produces a mirror image imprint forming a W-shaped (fish-tail) portion of skin to be excised. After this portion is excised and hemostasis achieved, closure is accomplished with a 4-0 plain gut suture. The two V-shaped incisions are brought together, reducing the protruded state of the lobule (Figure 69-9).[14]

COMPLICATIONS

The incidence of complications after reduction otoplasty is quite low.[17] The major complications to be avoided are infection and keloid formation. Immediate complications include pressure necrosis from an overly tight dressing and hematoma formation. Delayed or long-term complications include hypertrophic or keloid scar formation, recurrence of the ear deformity, neurosensory deficits, and unaesthetic results.

Hematoma

Postoperative hematoma formation, with an incidence of 2% to 4%, is the most common problem that requires immediate and aggressive intervention.[17] It is most often related to inadequate hemostasis achieved at the time of surgery. Other factors in hematoma formation include an overly tight wound closure without drainage at the base of the wound, postoperative trauma, hypertension, and a preexisting bleeding dyscrasia. Persistent pain beneath the dressing or significant bleeding through and around the dressing suggests hematoma formation and demands prompt inspection. The presence of a hematoma is indicated by a tense and bluish swelling beneath the auricular skin, most often in the retroauricular space. Management includes suture removal, blood clot evacuation, hemostasis, and reclosure of the wound with a large pressure dressing reapplied. Large doses of antibiotics are advisable to prevent perichondritis. If this problem remains untreated, it can result in fibrosis, perichondritis, and cauliflower ear deformity.

Perichondritis

Wound infection in otoplasty occurs in the early postoperative period and is usually a sequela to an undetected or inadequately treated hematoma. Symptoms include pain, erythema, fever, and discharge that may or may not be present. Treatment includes high doses of antibiotics after appropriate wound cultures. Common bacteria include *Staphylococcus aureus*, *Escherichia coli*, and *Pseudomonas aeruginosa*. Adequate drainage is achieved by opening all sutures and carefully irrigating necrotic debris from the wound. All correction sutures must be removed and the cosmetic deformity addressed at a later date. The complication can be devastating, causing massive cartilage destruction with a severe deformity resulting, even with aggressive therapy. Prophylactic antibiotics have not been scientifically proved to be beneficial but are often used in the preoperative and postoperative period to avoid this devastating complication.

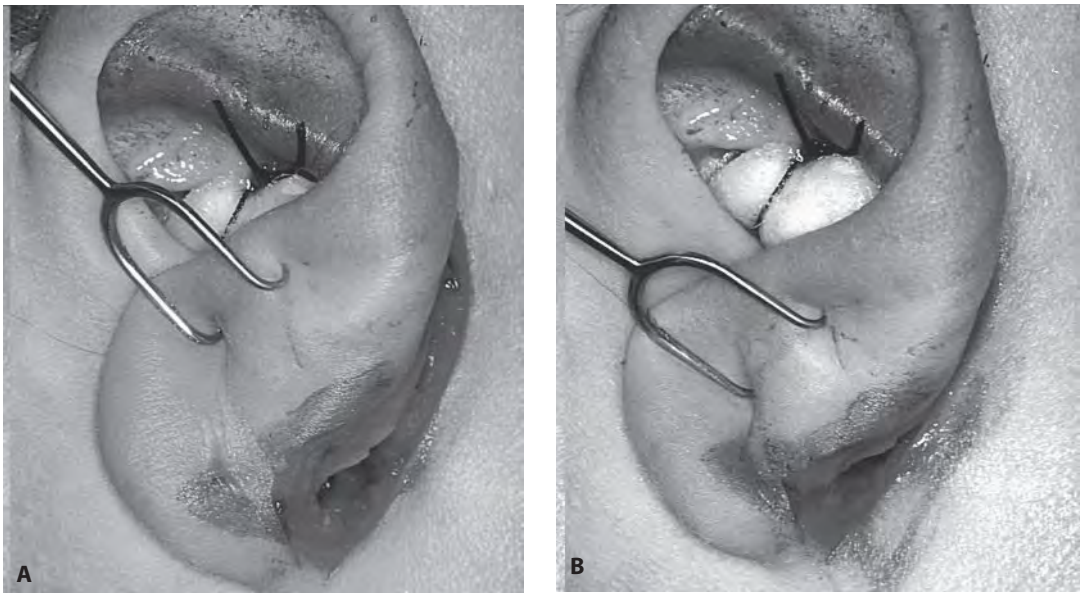


FIGURE 69-9. **A** and **B,** Marking of skin to correct a protruding lobule. Areas marked are excised in "fishtail" fashion and closed. (**A** and **B,** Reproduced with permission from Owsley T. Otoplastic surgery for the protruding ear. In Fonseca RJ, editor. Oral and Maxillofacial Surgery. Vol 6. Philadelphia: WB Saunders; 2000; pp. 408–418.)

Keloid and Hypertrophic Scar Formation

The closure line of the skin incision in the postauricular region is susceptible to scar formation, especially closed under tension. It is most commonly seen in younger patients and patients with deeply pigmented skin. Keloid formation, one of the most frustrating of all postoperative complications, requires aggressive therapy. In the early stages of keloid formation, intralesional triamcinolone acetonide is injected weekly until regression or significant improvement is evident. Most hypertrophic scars improve with steroid infiltration, but some keloids can progress into significant unaesthetic lesions. Low-dose radiation, although potentially dangerous, may provide the only effective means of control of some keloids. The more advanced lesions require surgical excision combined with radiation and delayed skin grafting of the irradiated area, with final aid from intralesional triamcinolone. The risk can be minimized by ensuring that the skin incision is closed passively.

Aesthetic Complications

Inadequate correction of the ear deformity is the most common untoward result of otoplasty, often more obvious to the surgeon than to the patient. Calder and Nassan[18] described at least one complication or residual deformity in 16.6% of all the patients who underwent otoplasty using all techniques. Recurrence of the ear deformity is a more common complication of reduction otoplasty, but is less likely to happen after excising a portion of the cartilage as well as a segment of skin, as described in the Davis method. Depending on sutures alone for achieving correction carries a greater risk of recurrence.

Telephone Ear Deformity

Telephone ear deformity occurs when the root of the helix and the ear lobule remain protruded while the middle half or third of the ear is set back against the head. This is more common in a large ear with a wide scapha. The incidence has been reported to be 3%.[19] Reverse telephone ear reveals a pronounced conchal bowl with respect to the lower and upper poles. These deformities can be avoided by carefully checking the position of the helical root, the upper helical rim, and the lobule at the completion of surgery.

Scapha Buckling

Scapha buckling or a transverse fold can develop in the Mustarde technique. This deformity can be avoided by placing the horizontal mattress sutures closer together where the scapha is widest, combined with adequate anterior scoring or weakening.

Narrowed Meatus

A constricted external auditory meatus can occur if the conchal bowl is rotated anteriorly in setting the ear back in any technique in which the conchal bowl is not excised. This problem is eliminated in the Davis method in which the floor of the conchal bowl is excised. Care must also be taken in placing the inferior end of the cotton roll bolster dressing into the external auditory canal to avoid stenosis.

SUMMARY

Reduction otoplasty carries few complications and can provide satisfying results for both the patient and the surgeon in the majority of cases. As in all cosmetic procedures, proper patient selection is imperative. Accurate preoperative assessment of the individual deformities and the appropriate choice of a surgical correction will minimize unfavorable aesthetic results. The single greatest cause of an unfavorable result in this procedure is inaccurate diagnosis.

The surgeon must understand the normal external anatomy of the ear and learn to recognize the pathologic characteristics of the abnormal ear. Having accurately assessed the deformity, the surgeon needs to be familiar with the various surgical approaches available to correct them. Finally, it is important to have a working knowledge of the potential complications of otoplasty and their prevention and treatment.

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70

CHAPTER

Adjunctive Facial Aesthetic Procedures

Joseph Niamtu, DMD

In the lifetime of almost everybody reading this book, there has been a significant paradigm shift in cosmetic facial surgery and it is a minimally invasive revolution. Since the mid-1990s, cosmetic facial surgery has ushered in "lunchtime" procedures such as neurotoxins, facial fillers, fractional lasers, and anatomic facial implants. Advances and refinement of chemical peeling, fat transfer, and skin care have also been made. Although many surgical practices perform these procedures, many clinicians from various specialties perform minimally invasive procedures (MIPs) as the bulk of their practices. MIPs interface well with all aspects and ages of cosmetic patients. Younger patients can benefit solely from MIPs, middle-aged patients may stave off surgery for a number of years with MIPs, and older patients can amplify surgical results with MIPs.

Another reason for this popularity is the fact that cosmetic facial surgery involves primarily females. Many females are in the workplace and have expendable income from employment, but cannot take off extended time for recovery; hence, they demand MIPs. MIPs have very distinct advantages and disadvantages. Neurotoxins and fillers, for instance, are very predictable and efficient. Other MIPs such as radiofrequency skin tightening and fractional lasers are frequently overmarketed by industry and the media, and the expectations often outweigh the results. The adage "if it sounds too good to be true, it probably is" must always be kept in mind with MIPs. In general, you get what you pay for, in terms of recovery. Many procedures that promise maximal effect with minimal recovery are often disappointing.

NEUROTOXINS

If there is a "real deal" in MIPs, it is neurotoxin technology. Before the mid-1990s, no predictable procedures existed to stop unwanted mimetic muscular movement and associated skin rhytids. Botulinum toxin A (BTA) is one of the seven serotypes of toxins produced by *Clostridia botulinum*. This toxin was originally used in humans to treat blepharospasm and strabismus. Treated patients coincidently noticed rhytid effacement on the treated side. This led to experimentation with these toxins for purely cosmetic use, and by the early 1980s, off-label cosmetic use of BTA was well under way. By 2002, Botox (BTA) was U.S. Food and Drug Administration (FDA) approved for glabellar wrinkles but was also used for numerous off-label wrinkle and medical treatments. In 2009, Dysport, another formulation of BTA, was FDA approved for glabellar wrinkles. At the time of the writing of this text, numerous other BTA preparations are in the pipeline for FDA approval and will surely become part of the armamentarium of the oral and maxillofacial surgeon (OMS). With their years of training in facial anatomy and experience with facial injections, OMSs can easily incorporate the cosmetic use of BTA in their practice.

BTA produces temporary muscle paralysis by preventing the release of acetylcholine at the neuromuscular junction. When selectively injected into the muscles of the head and neck, various cosmetic and functional changes are predictably achieved.

The most common regions for BTA injection are the glabella, frontalis, and lateral canthal regions. Additional muscles treated for cosmetic or functional reasons include the nasalis, levator labia superioris ala que nasi, orbicularis oculi, orbicularis oris, masseter, mentalis, and platysma.[1]

Because BTA will exert a temporary paralysis on any voluntary muscle, precise dosage and injection are paramount. In the upper face, levator palpebrae superioris injection remains the most feared complication. Keeping the toxin injections 10 mm above the bony orbital rim is a requisite to

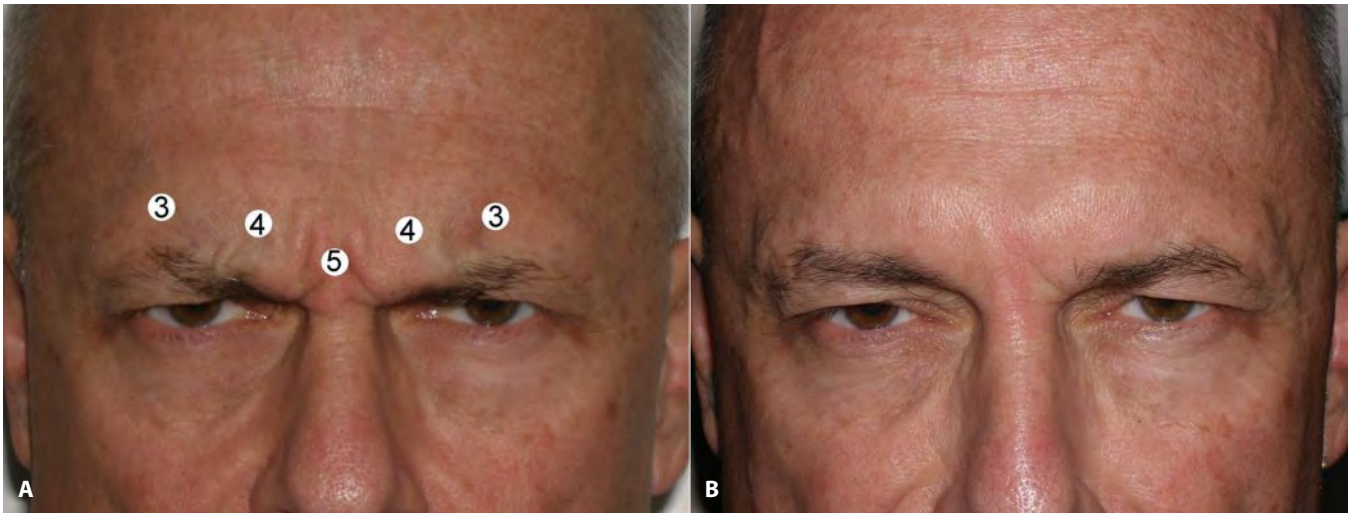


FIGURE 70-1. **A,** The *white dots* indicate common injection points for glabellar treatment with the respective number of Botox units for each area. **B,** The author 10 days after 20 units of Botox was injected into the glabella.

prevent upper eyelid ptosis. Novice injectors should actually measure and mark injection points on the brow and lateral canthal regions to remain 10 mm away from the orbital rim as not to inadvertently affect the levator palpebrae superioris or lateral extraocular musculature.

Although variable in some patients, most injection patterns and dosages are similar. Figure 70-1 shows the common injection points for the glabella, Figure 70-2 for the frontalis, and Figure 70-3 for the lateral canthal regions. For optimum treatment in females, the author recommend 5 Botox units or 15 Dysport units for each glabellar injection point. The frontalis, being a thinner muscle, is generally adequately treated with 3-unit injections, as is the lateral canthus regions. Males and patients older than 65 years old frequently require more units per area and the treatment may have a shorter longevity. After injection, BTA generally exerts its effects within 72 hours and paralysis averages 90 days. The glabellar region includes injection in the procerus muscle, both corrugator supercilii muscles, and the lateral orbicularis oculi regions.

INJECTABLE FACIAL FILLERS

Another paradigm shift has occurred since the turn of the last century with injectable fillers. Since the year 2000, bovine collagen was the most popular and only FDA-approved facial filler. Being from an animal source, allergy testing was required and results were very transient, often 2 to 3 months. Our European colleagues during this time had numerous choices of safe and predictable fillers. In 2003, Restylane (nonanimal stabilized hyaluronic acid) was approved by the FDA for the treatment of nasolabial folds. This natural polysaccharide was not only nonanimal but, owing to cross-linking

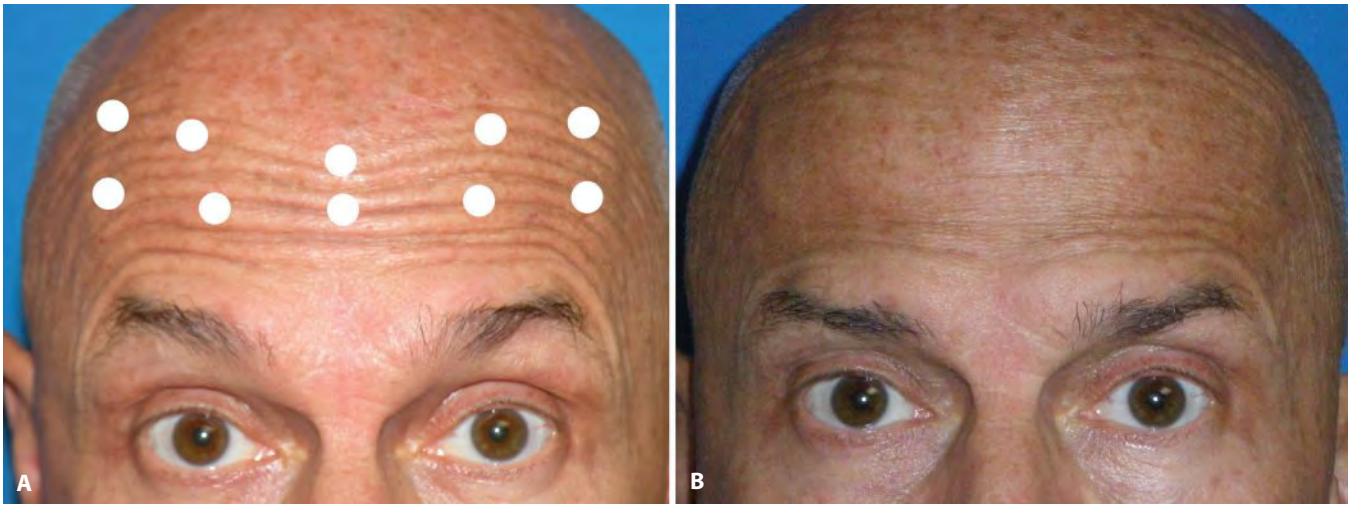


FIGURE 70-2. **A,** The *dots* show injection points on the author's frontalis region. The dose and position vary from patient to patient, but generally 2 to 3 units per injection point is adequate. **B,** The author 10 days after 25 units was injected into the frontalis region.

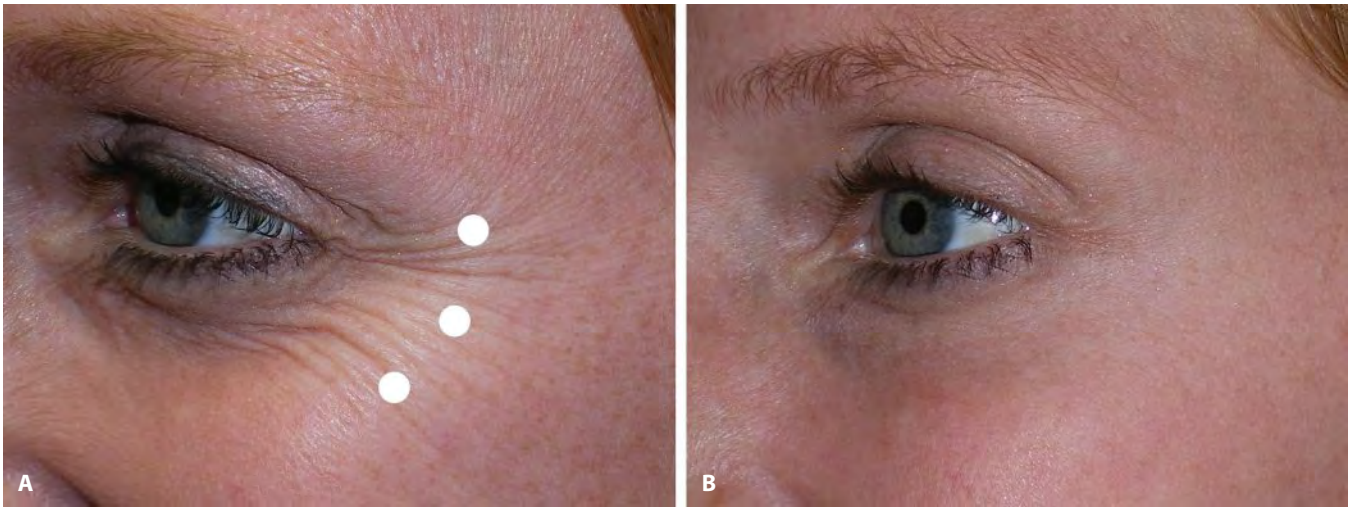


FIGURE 70-3. **A,** Common injection points for the lateral canthal region. Generally, 3 units of Botox is injected at three regions in a semilunar shape with care to stay 1 cm lateral to the lateral orbital rim. **B,** The same patient 1 week after injection of 10 units of Botox on that side.

technology, lasted much longer that bovine collagen. Also, allergy testing was not required, which had been a major drawback for impetuous cosmetic patients and the "I want it now" generation.

The most common regions for filler injection in the author's practice are nasolabial folds, lips, vertical lipstick lines, perioral regions, and various skin wrinkles, in decreasing order.

Choice of Filler

Numerous FDA fillers are approved for on- and off-label use. The hyaluronic acid fillers (Juvederm Ultra, Juvederm Ultra Plus, Restylane, and Perlane) are far and away the most commonly injected products. These hyaluronic acid fillers are clear gels and can persist from 6 to 12 months, depending upon the injection site. Other common FDA-approved temporary fillers include hydroxylapatite (Radiesse), and porcine collagen (Evolence). L-Polylactic acid (Sculprta) and silicone oil (Silikon 1000) are stimulatory fillers in that they induce a foreign body reaction that induces collagen encapsulation and, hence, further augmentation. Sculptra may persist for up to 2 years and silicone can be permanent. Artefil is a filler consisting of polymethylmethacrylate microspheres in a collagen matrix and is also considered permanent filler. At the time of this publication, both Evolence and Artefil have closed operations in the United States, but will more than likely reappear under a new company.

Injecting the Nasolabial Folds

This author most frequently injects hyaluronic acid fillers in the nasolabial folds. It is easy to inject, feels natural, and can be reversed with hyaluronidase. The author's technique involves the intradermal placement of the filler in the depth of the nasolabial fold (NLF) from the alar base to the oral commissure (or end of the fold).[2] Care must used to make sure the filler does not migrate laterally upon injection or the fold can be made larger. If the filler appears to be migrating laterally, the needle is removed and placed in a more medial position. It is imperative that the injector see the actual improvement of the wrinkle or fold while injecting. If the wrinkle is not improving as it is being injected, the needle is likely too deep and the filler is lateralizing. Figure 70-4 shows a hyaluronic acid filler being injected in the dermis of the nasolabial fold. Figure 70-5 shows a before and 14-day after injection picture on a patient treated with two syringes of Juvederm Ultra.

Injecting the Lips

Lip augmentation is more technique-sensitive and more difficult to learn than treating skin folds or wrinkles. Younger



FIGURE 70-4. An intradermal injection of hyaluronic acid filler is shown on the nasolabial fold.



FIGURE 70-5. This patient is shown before and 2 weeks after injection of 0.8 mL of hyaluronic acid filler to each nasolabial fold.

patients can benefit with simple volumization of the deep lip. Older patients may require "white roll" outline, deep volumization, and vertical lipstick line fill, which presents a greater challenge.

Deep Lip Augmentation for Volume and Pout

Many young patients and virtually all females older than 40 can benefit from lip volumization. "Putting some air in the tires" can enhance facial beauty by a simple procedure. The lips are first anesthetized with intraoral perisulcular local anesthetic infiltration.[3] Next, the filler is injected at the level of the wet/dry line with the needle positioned in the mid lip. Filler is injected as the needle is withdrawn and the treatment is tapered to augment the center of the lip more than the lateral portions. Most younger patients can benefit from 0.4 to 0.5 mL of hyaluronic acid filler per lip, whereas older patients may require up to 1 mL per lip. Massage is performed with petroleum jelly to smooth and contour the gel to a smooth and homogenous state. Figure 70-6 shows deep lip injection and Figure 70-7 shows before and after images of deep lip volume augmentation using 0.8 mL of hyaluronic acid filler in each lip.

Defining the White Roll

The configuration of the vermilion/cutaneous junction is referred to as *Cupid 's bow.* A light reflex exists in the aesthetic lip known as the "white roll." This treatment is usually imperative in older patients who have lost definition and outline and in younger patients with underdeveloped lip border anatomy.

Injection of the white roll involves the augmentation or reconstruction of Cupid 's bow and its angular anatomy in the upper lip. The white roll of the lower lip is curvilinear in nature. In either lip, this area is injected by placing the needle in the potential space that exists between the mucosa and the orbicularis oris muscle. In the correct plane, the filler will flow freely and the filler is injected while withdrawing the needle. White roll augmentation in younger patients may involve only the central two thirds of the lip and gently tapers laterally. In older patients with loss of vertical dimension, the white roll may require extension to the commissures. Figure 70-8 shows hyaluronic acid filler being injected in the white roll region of the lip. Figure 70-9 shows a patient treated with white roll and deep volume injection.

In addition to the aforementioned areas, augmentation of the philtral columns, oral commissures, and vertical rhytids is also commonly performed.

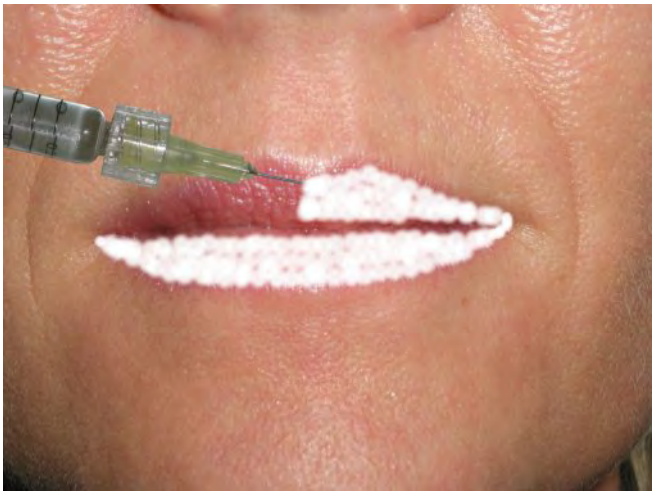


FIGURE 70-6. When performing deep lip volumization, the needle is inserted into the middle of the lip at the level of the wet/dry line and the filler is injected upon withdrawal. The injection is bilaterally tapered so the center of the lip has more volume than the lateral portions. The same procedure is repeated in the lower lip.



FIGURE 70-7. This patient is shown before and after deep injection volumization of the upper and lower lips.



FIGURE 70-8. White roll augmentation is performed by injecting a line of filler in the potential space between the mucosa and the orbicularis oris muscle to re-create or enhance the Cupid's bow effect.

CHEMICAL PEEL

Although laser resurfacing has changed the face (no pun intended) of treating wrinkles and dyschromias, chemical peel is a time-honored method for facial skin rejuvenation. Chemical peeling is safe when performed with adequate training, is inexpensive, and has a moderate recovery of up to 1 week.

Chemical peels are performed with a myriad of acids, and deep or aggressive peels can cause problems with burns, scars, and hypopigmentation. The depth of the peel is dependent upon the peeling agent used, the concentration of the agent, the number of coats applied, and the physical properties of the skin. This section deals exclusively with trichloroacetic acid (TCA) chemical peeling. Peels can be classified into superficial, medium, and deep depending on how deeply the acid penetrates the skin. Superficial chemical peels are limited to the epidermis. Medium-depth peels are generally considered to be those that penetrate to the level of the papillary dermis; deep peels penetrate into the reticular dermis.

TCA exerts its actions by coagulating skin proteins. The author purchases TCA that is 30% compounded by the weight/volume method. Although the author will in some applications use this directly out of the bottle, most frequently the author dilutes it to 20% or 30%. To prepare a 20% solution of TCA, 4 mL of 30% TCA is diluted with 2 mL of water. To compound a 15% TCA solution, 2 mL of 30% TCA is mixed with 2 mL of water.

The novice peeler should gain expertise with 15% TCA and light peels without anesthesia before attempting more aggressive peeling with sedation.



FIGURE 70-9. This patient is shown before and after hyaluronic acid augmentation of the deep lip and vermilion/cutaneous junction.

All peel patients should be pretreated with Retin-A and hydroquinone 4% for 2 to 4 weeks before the peel. Darker skin types require longer prepeel skin priming.[4] Prepeel priming allows more efficient absorption of the acid into the skin, faster reepithelialization, and decreased postinflammatory hyperpigmentation. All peel patients are covered with an antiviral and antibiotic medication beginning the day before surgery and for the next 5 to 7 days. Superficial peels do not require this premedication unless the patient has a history of recurring herpes simplex infection.

The best means of learning chemical peeling is to perform superficial procedures on a patient such as an employee or friend who can be observed every day for the first week. It is imperative to learn the various stages of peeling and healing. A single coat of 15% (2 mL of 30% TCA and 2 mL of water) can be applied with minimal discomfort. The 4 mL of mixture is applied to the skin using a makeup sponge, cotton ball, Q-tips, or gloved finger. As the 15% solution is applied, the skin will tingle and slightly burn, but the discomfort is tolerable. This should be the treatment on one's first patient. The skin will develop a patchy white frost in some areas that will disappear in 5 to 10 minutes (Figure 70-10). After the procedure, the patient can apply hypoallergenic moisturizer multiple times a day. There will not be much peeling, only a little flaking, and there will not be a significant visible clinical result.

After experience with doing this several times, the same procedure can be performed, but a second coat applied this time. The second coat will burn more than the first, but two coats of 15% TCA are generally tolerable. The second coat will produce a more distinct patchy frost that the pervious single-coat 15% TCA treatments. With this treatment, it is advisable to apply Vaseline to the face for the first 48 hours, then moisturizer for the next several days. The goal is to keep the skin moist continuously. Patients treated with two coats of 15% TCA will experience some distinct peeling at days 3 to 5, but this will be minimal compared with more aggressive peels.



FIGURE 70-10. This patient is shown 5 minutes after a single application of 15% trichloroacetic acid (TCA) to the cheeks and has a patchy white frost.

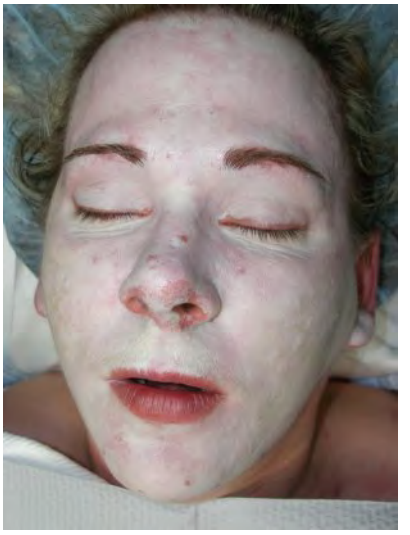


FIGURE 70-11. This patient has been treated with two or three coats of 30%; a homogenous white frost is evident 20 minutes into the procedure. The level of skin wounding with this extent of peel is the papillary dermis.

Generally, the entire volume of the mixture is applied as a single coat. The surgeon must then wait several minutes before the next coat to allow the action to ensue and the frosting to form. The end point of a superficial peel is a scant, patchy, white frost. The end point of a basal layer peel is a light white frost with a pink background, and the end point of the medium (or deeper level) peel is a dense homogenous white frost (Figure 70-11).

The surgeon should not allow ego or desire to overtake common sense in learning these procedures. Begin with very conservative peels and after doing 5 or 6 patients, move on to the next level. It should take, in the author's opinion, at least 10 cases of superficial peeling before the surgeon begins using three- or four-coat treatments. The time of healing will coincide with the depth of injury. Superficial peels will heal in 1 to 2 days, basilar peels in 3 to 4 days, and medium-depth peels to the papillary dermis will heal in about 7 days.

Although some patients can be treated without sedation with three and four coats of 15% TCA, it can become very uncomfortable, and with the intense anesthesia training of our specialty, there is no reason for any surgical patient to be uncomfortable. Again, the author strongly recommends that before attempting three- and four-coat 15% TCA peels, practitioners perform multiple cases with one- and two-coat treatments to completely understand the peeling and wounding and healing process. Thinner tissues such as the upper eyelids must be treated more judiciously. In addition, because the neck has far less pilosebaceous units, it is never peeled as aggressively as the face. Patients with pigmented skin are also treated with caution and, therefore, less aggressive peels.



FIGURE 70-12. This patient was treated with three coats of 20% TCA to improve her melasma. She is shown before and 1 month after peel.

Resurfacing pigmented or ethnic skin is an advanced procedure and should not be performed by the novice.[5]

After the practitioner is experienced with multiple-coat 15% TCA peels, advanced treatments can be done with two or three coats of 20% TCA or straight 30% TCA (Figure 70-12). Chemical peeling is very technique sensitive and more of an art form than a science; extreme caution must be used with higher concentrations of acid because disfiguring burn and scarring can occur. Purchasing a dedicated text on chemical peeling and observing and mentoring cases is the safest and most effective means of learning chemical peeling. The Obagi Blue Peel is also a safe and effective way to perform TCA peels and is good for the novice peeler.[6]

MIDFACE IMPLANTS

For years, some cosmetic surgeons make patients tighter but not younger. Much of this has to do with pulling excess skin over an atrophic skeleton. Contemporary cosmetic surgeons realize the importance of volume replacement as an integral part of cosmetic facial surgery.[7,8] Many options exist to augment the midface including cheek lifts, injectable fillers, fat transfer, and alloplastic implants. Midface lifting has a reputation for being transient, fat and fillers resorb, but cheek implants are a permanent, three-dimensional means of volumizing the midface. When discussing implants with prospective patients, it is reassuring to tell a patient that the result will be permanent, but if they are unhappy, the implant can be removed in 5 minutes.

The youthful face is replete with volumizing fat in the right places. Older patients undergo fat atrophy as well as skeletal and cutaneous changes and the cheeks at some point become the jowls, leaving an atrophic hollow in their place. Almost any patient older than 45 years can benefit from some type of midface augmentation, and younger patients with developmental midfacial deformities are also excellent candidates.

A myriad of implant sizes and shapes are available and this can be confusing for the inexperienced facial surgeon. The author basically categorizes the regions of midfacial volume deficiency to the submalar region, the malar region, or a combination of both. Patients with otherwise normally developed midface features will lose fat in the infraorbital and submalar regions. This is very common in both males and females. This is the earliest and most common cosmetic problem, and therefore, the majority of implants (90%) the author places are silicone submalar implants. The implants run a bit small, so the average female will require medium to large submalar implants and males may require extra large submalar implants.

For the patient who has adequate submalar fill, but requests solely malar augmentation, the malar shell implant configuration is convenient. This implant lies more superior and lateral and produces the "high cheekbone look." The author uses this implant the least, because most of the author's patients have submalar atrophy.

The second most common facial implant is the combined submalar shell implant. This implant is a hybrid of the submalar implant and the malar shell implant and serves to augment both regions simultaneously. The author probably uses this on 5% of his patients. These implants actually run large and the author frequently uses a medium or large combined submalar shell but almost always trims the borders to customize the implant. Figure 70-13 shows typical patients in need of the three types of implants mentioned; Figure 70-14 shows the three types of implants the author most commonly uses to treat the aging midface.

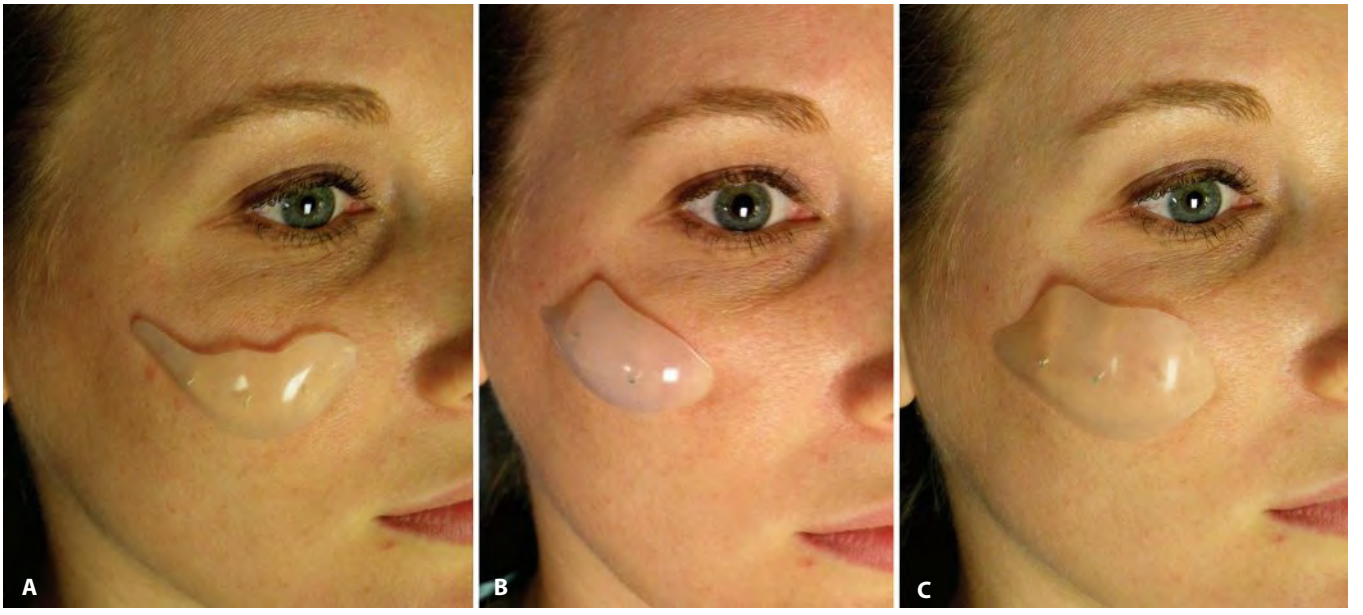


FIGURE 70-13. **A,** A patient with submalar deficiency. **B,** A patient with malar deficiency. **C,** A patient with deficiency in both the submalar and the malar regions.

The author prefers silicone implants because they are biocompatible, are easy to trim, will adapt to irregular contours, will not fracture with screw placement, and most important, are extremely easy to remove. Porous polyethylene implants can be a nightmare to remove owing to the extreme level of tissue ingrowth and frequently fracture and can cause much localized tissue damage when being removed.

Midface Implant Placement

Cheek implants can be placed with local anesthesia, although generally with intravenous sedation. The vast majority of cheek implants are concomitantly placed with face-lift surgery, although they are frequently placed as a stand-alone procedure.

About 5 mL of 2% lidocaine with 1:100,000 epinephrine is infiltrated percutaneously to the level of the periosteum along and slightly past the anticipated location of the implant. Another several milliliters of the anesthetic solution is infiltrated intraorally around the sulcus and the anticipated implant position.

A 1-cm horizontal incision is made in the sulcus above the canine tooth to the level of the periosteum. Subperiosteal dissection is then performed superiorly with care to preserve the infraorbital nerve. At this point, the dissection proceeds in an oblique direction and tapers off over the zygomatic arch. Submalar implants require the smallest amount of dissection, whereas the malar shell and combined submalar shell implants require more extensive dissection. It is not uncommon to dissect over the masseter tendon at its zygomatic attachment. The tendon is not disturbed because the implant can safely lie over the tendon. It is important that the entire dissection remains subperiosteal and the dissection pocket needs to be only slightly larger than the size of the implant. Larger dissections are unnecessary, encourage implant mobility, and extend recovery. Figure 70-15 shows the incision, maxillary and malar dissection, implant placement, and the implant in position.



FIGURE 70-14. **Left** to **right,** A submalar implant **(A)**, a malar shell implant **(B)**, and a submalar shell implant **(C)**.

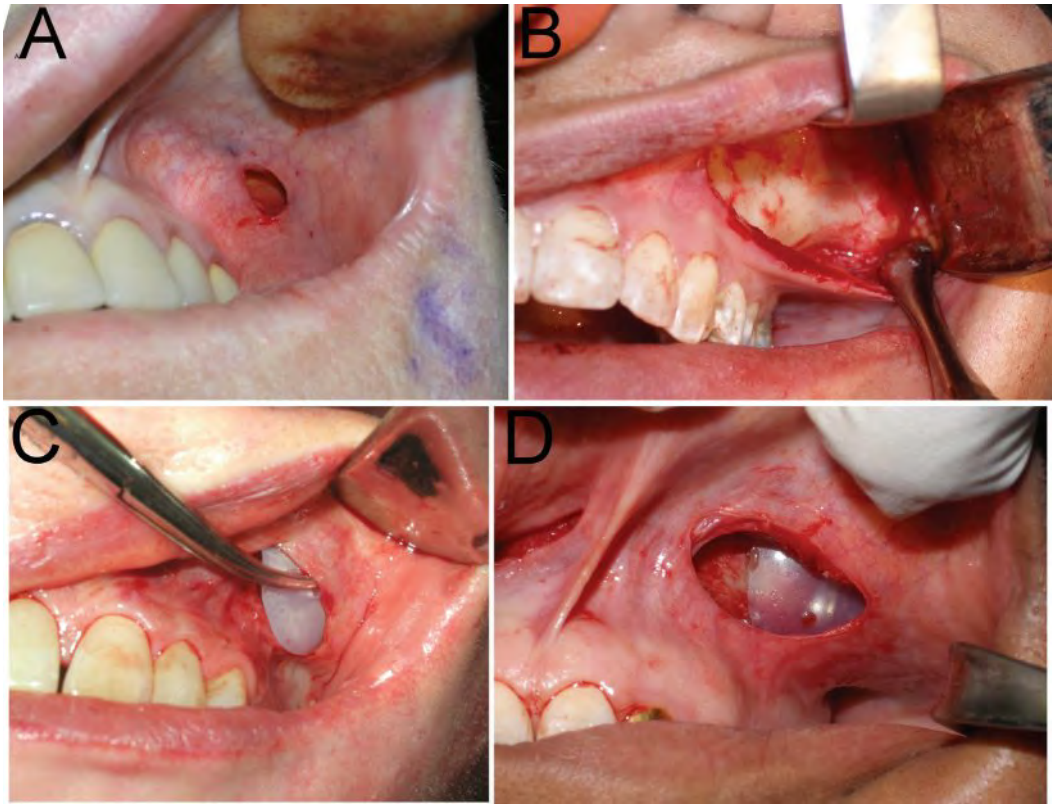


FIGURE 70-15. A 1-cm incision **(A)** is used to perform a subperiosteal dissection over the anterior maxilla and the malar region and tapering over the zygomatic area **(B)**. The implant is then inserted into the dissection pocket **(C)** and should lie passively in place **(D)**.

Most of these implants will lie somewhere in line with the second premolar tooth, and this can be used as an index point to position both sides equally. After the proper position is achieved, the implant is secured with a single fixation screw. The screw is preferably placed over the denser buttress bone. At this point, the pocket is irrigated with antibiotic solution and the incision closed with 4-0 gut suture.

Postoperatively, no dressing is required. The patient is asked to refrain from excessive oral function and animation, and the recovery is generally approximately 1 week, although some patients may have residual swelling for several more days. Owing to the disruption of the lip elevators during the dissection, the patient will exhibit a weak smile or pucker that returns over a 10- to 14-day period. These patients are generally covered with antibiotics beginning intraoperatively and for the next 5 days. Figures 70-16 and 70-17 show before and after cases of patients treated with silicone cheek implants.

FRACTIONAL LASER RESURFACING

The mid 1990s ushered in another cosmetic facial surgery paradigm shift—laser skin resurfacing (LSR). Before predictable laser technology, dermabrasion and deep chemical peel were the common treatment options, both of which were difficult to control and art ruled over science. Laser technology allowed precise ablation of tissue that was controllable in terms of microns, and each pass produced a controlled amount of damage. As with all new technology, many hard lessons were learned with LSR. Overtreatment and treatment of questionable areas produced significant scarring and hypopigmentation. The other drawback of LSR is the extended recovery. The recovery is formidable because the patient must heal with a raw face and reepithelialization can take up to 2 weeks. Extended erythema is also a drawback that can last for several months. The results, however, were unrivaled by any previous therapy, and to date, LSR treatment has overtaken the ability of aggressive CO_2 laser to simultaneously improve dyschromias, tighten deep wrinkles, and make new collagen. In terms of recovery, "you get what you pay for." If patients desire dramatic results, then 2 weeks of recovery is not too much to ask for the reversal of decades of aging. This author performs a high volume of traditional high fluence, high density, multipass CO_2 LSR; it remains one of the most dramatic procedures.

Owing to the demanding recovery and extended erythema associated with aggressive CO_2 laser, researchers have continued to look for a "friendlier" means of LSR. The erbium:yttrium-aluminum-garnet (Er:YAG) laser gained popularity in the mid-1990s and, although it enabled an easier recovery, the results were not as impressive as with traditional CO_2.



FIGURE 70-16. This patient underwent upper blepharoplasty, mini–face-lift, and bilateral silicone submalar cheek implants .

At the time of the writing of this text, fractional laser technology has become a popular option for facial rejuvenation. Fractional laser technology is very different from any previous laser treatment. Whereas traditional LSR treats the entire skin surface, fractional lasers do not treat the entire surface, but instead the laser beam is spaced and the result is treated microcolumns of lasered skin surrounded by untreated regions[9,10] (Figures 70-18 and 70-19). Traditional full-coverage ablative CO_2 laser treatment would be analogous to a painting a wall with a paint roller, in which the entire surface is "treated." Fractional laser resurfacing would be like painting a wall with polka dots, in which some of the wall is painted and the remainder is untouched. Because a portion of the skin is not treated, the healing is easier and faster than with ablative LSR.

The push for a gentler laser has been demanded not only by patients, who want noticeable results with shorter recovery, but also by doctors, who want a laser procedure that can



FIGURE 70-17. This patient was treated with a face-lift and medium submalar cheek implants and full-face CO_2 laser resurfacing.

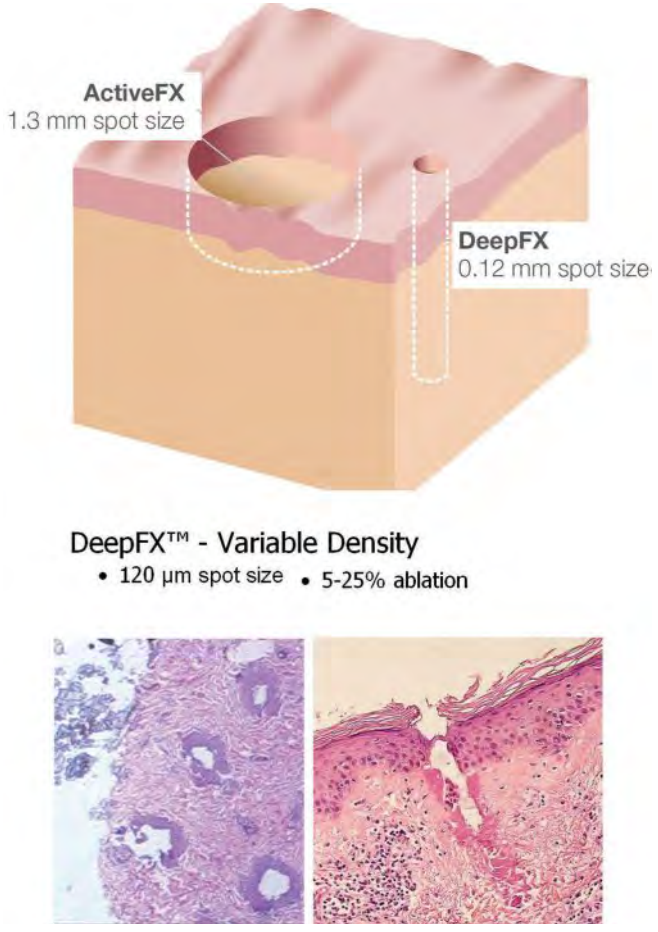


FIGURE 70-18. The effect of fractional laser skin resurfacing (LSR) in which laser microcolumns are surrounded by untreated areas of skin.



FIGURE 70-19. This patient is shown with the spaced treatment of nonablative CO_2 LSR known as Active FX (Lumenis Inc., Santa Clara, CA). The treated microcolumns of skin and the untreated areas are visible.

be performed without intravenous anesthesia or general anesthesia. This has been a good thing and a bad thing. It has been a good thing because fractional LSR has given us the ability to decrease recovery and prolonged erythema. It has, again in the author's opinion, been a bad thing because the results are not even close to those of traditional CO_2 LSR. Most practitioners who favor fractional LSR will perform three to five procedures to obtain significant results. The main theme of the newer technology was to decrease recovery; this has been done with most fractional LSR patients healing within 3 to 5 days. Having to do this three or four times, however, actually extends recovery time when compared with those of traditional LSR. For some surgeons, this may be a workable formula, but many patients expect bigger results. Some nonsurgical disciplines claim patients are very satisfied with the small changes seen from fractional LSR. Success with any procedure equals happy patients and low complications. Each type of surgeon has a different type of practice and what works in some practices may not be sufficient in others. Active FX fractional resurfacing is indicated for patients who will be satisfied with minor but noticeable results with a more user-friendly recovery. Figure 70-20 shows a patient treated with a single pass of Active FX fractional LSR. The areas of deeper pigmentation were treated with two or three passes.

Deep Fractional LSR

In the continuing search for a better laser treatment, deep fractional LSR is a newer technology. Similar to the Active FX treatment, the Deep FX (Lumenis, Inc., Santa Clara, CA) is a fractional laser with subtotal skin coverage. The difference is that the Active FX treatment penetrates the skin much deeper and can induce much more collagen production and, hence, more dramatic results. The spot size of the Deep FX is much smaller and the laser "drills" the microcolumns of energy from 400 to 900 µm into the dermis (Figure 70-21).



FIGURE 70-20. This patient was treated with a single pass of Active FX fractional LSR and the lentigos on the cheeks were treated with two additional passes.



FIGURE 70-21. The Deep FX is a more powerful and deeper-penetrating CO_2 treatment that enables treatment deeper into the dermis. Shown are the Deep FX laser handpiece and a patient 24 hours after a single pass of Deep FX resurfacing at 10 J/cm^2 and a density of 2.

Numerous clinicians are touting Deep FX's ability to improve wrinkles and acne scars, and encouraging work has been done with the treatment of burn scars using Deep FX laser technology. When performing Deep FX, the face is treated with one pass and areas of deeper wrinkling are treated with additional passes. Finally, a single pass of Active FX laser is performed over the entire face to blend the result. The Fraxel laser (Solta Medical, Heyward, CA) is another brand of laser that uses an erbium-doped system instead of CO_2.

CONCLUSION

OMSs are well positioned to incorporate cosmetic facial surgical procedures in their practices. Although traditional surgical procedures have been the mainstay of cosmetic facial surgery, MIPs have sprinted to the forefront of cosmetic treatment. The contemporary OMS understands the importance of MIPs, and they are a welcome and fun addition to our surgical practices.

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