PETERSON'S
PRINCIPLES OF
ORAL AND
MAXILLOFACIAL
SURGERY
Second Edition

Michael Miloro
Editor
G. E. Ghali • Peter E. Larsen • Peter D. Waite
Associate Editors

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Notice: The authors and publisher have made every effort to ensure that the patient care recommended herein, including choice of drugs and drug dosages, is in accord with the accepted standard and practice at the time of publication. However, since research and regulation constantly change clinical standards, the reader is urged to check the product information sheet included in the package of each drug, which includes recommended doses, warnings, and contraindications. This is particularly important with new or infrequently used drugs. Any treatment regimen, particularly one involving medication, involves inherent risk that must be weighed on a case-by-case basis against the benefits anticipated. The reader is cautioned that the purpose of this book is to inform and enlighten; the information contained herein is not intended as, and should not be employed as, a substitute for individual diagnosis and treatment.
To Beth and Macy, my two reasons for being, for your love and support. To Pete, my teacher, for making me a better surgeon and person.

Michael Miloro

To my wife, Hope, for being my best friend and the love of my life. To my parents, Elias and Linda, and my brother Fred, for their support, inspiration, devotion, and love.

G. E. Ghali

To my wife, Patty, and my sons, Michael, Matthew, and Mark. You are the most important people in my life, yet always understand and are patient with my absence. To my father who inspired me to enter medicine. Lastly, to my former and current residents who teach me every day.

Peter Larsen

To my wife, Sallie, and my children, Allison, Eric, and Jon. To my father who inspired my interest in oral and maxillofacial surgery and to my residents who have continued to teach me.

Peter Waite
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**PART 9: FACIAL ESTHETIC SURGERY**

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The Second Edition of Peterson's Principles of Oral and Maxillofacial Surgery, reflects the efforts of many people made in a very short period of time. The time from the decision to undertake a second edition until publication release totaled less than 2 years. This is a monumental accomplishment considering the current state of affairs in the specialty of oral and maxillofacial surgery and the difficulties in pursuing scholarly activity, even for the academic practitioner. Although it is certainly not a simple task to assemble an author list as extensive as the one in this text, it was perhaps made easier because editors and authors were inspired by feelings of tribute to Larry Peterson to deliver on short notice.

When Larry Peterson decided to publish the first edition of this book over a decade ago, he recognized the need in our specialty for a comprehensive and complete reference textbook in oral and maxillofacial surgery that was practical and readable. Oral and maxillofacial surgery encompasses an ever-expanding range of diverse topics that makes it unique among the medical and dental specialties. There was no concise textbook that dealt with the full scope of the specialty that was available for residents and surgeons to use as a reference for clinical practice. The textbook Contemporary Oral and Maxillofacial Surgery appropriately covers the requisite information for the dental student and general dental practitioner, but Peterson's Principles of Oral and Maxillofacial Surgery provides an organized and systematic approach to the specialty for residents and clinicians practicing full-scope oral and maxillofacial surgery. The first edition of this text was the only reference of its kind. It is now continued with the second edition, which is unique in many respects, among them the inclusion of contributions from more than 100 oral surgeons and other dental and medical specialists, 500 pieces of original artwork, and a CD-ROM.

The clear purpose of this textbook is to provide a concise, authoritative, easy-to-read, currently referenced, contemporary survey of the 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 the individual practitioner, the material contained in this text is definitely within the scope of the specialty. This textbook should be considered a reference for the oral and maxillofacial surgeon during residency and into clinical practice. It will be an excellent resource for examination preparation purposes as well; in fact, the first edition was adopted in some European countries as a required textbook for oral surgery board certification.

As with the first edition, the authors, primarily oral and maxillofacial surgeons, were chosen because of their broad experience and expertise in each specific area of the specialty. The contributions from these national and international authors certainly reflect their knowledge and specialization. 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 is free from commercial and personal bias. If additional information is required, references have been provided so that other specialty textbooks may be consulted. Considering the rapid advancements and developments in the fields of medicine and surgery, a nearly 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 text will reflect these changes in clinical practice.

This text would not have been possible without the help and support of many people, including Ghali, Pete, and Peter; the outstanding authors who contributed their practice-defining knowledge; and the group at BC Decker Inc, including Catherine Travelle, Susan Cooper, and Paula Presutti, who sent a seemingly endless number of e-mails in an attempt to ensure deadlines were met. Certainly a debt of gratitude is owed to Brian Decker for his vision, dedication, and commitment to publish this textbook.

Peterson's Principles of Oral and Maxillofacial Surgery is the authoritative textbook for the specialty of oral and maxillofacial surgery.

MICHAEL MILORO, DMD, MD
Dr. Larry J. ("Pete") Peterson is easily the smartest person I have ever known, and I do not mean with regard to medicine and surgery alone. Pete certainly forgot more information in his life than most people ever know. He made everyone around him want to be better than they were, and he helped them to reach their potential. *Peterson’s Principles of Oral and Maxillofacial Surgery*, Second Edition, is dedicated to this man. Unfortunately, the majority of readers will never have had the opportunity to meet him and to experience his imposing presence. The fact that this book will continue to educate many surgeons for years to come would have pleased him very much since his greatest passion in life was, perhaps, teaching.

Pete obtained his doctor of dental surgery degree at the University of Missouri, Kansas City, in 1968. He completed his training in oral and maxillofacial surgery at Georgetown University, where he also received his masters of science degree. Pete served on the faculty at the Medical College of Georgia and, subsequently, at the University of Connecticut as the director of Oral and Maxillofacial Surgery Residency Training. However, he is best known for his academic accomplishments at Ohio State University, where he served as chairman of Oral and Maxillofacial Surgery, Pathology, and Anesthesiology from 1982 through 1999. To experience the full range of our specialty, Pete entered private practice in 1999 and continued in that area until his death on August 7, 2002.

Pete’s professional and personal accomplishments and his contributions to our specialty are innumerable. In 1993 Pete assumed the role of editor-in-chief of *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics*, upon the retirement of Dr. Robert Shira. Pete demanded excellence in the manuscript submissions and maintained high standards for this journal during his tenure. Pete also edited *Contemporary Oral and Maxillofacial Surgery*, which, like its predecessor from his mentor Dr. Gustav O. Kruger, defined dental undergraduate education in oral and maxillofacial surgery nationwide. Pete’s dedication to education was further demonstrated in his role as chair of the American Association of Oral and Maxillofacial Surgeons Committee on Residency Education and Training. He lectured and published extensively both nationally and internationally, with a particular emphasis on the topics of odontogenic infections and dental implantology, and his contributions to the literature are many and varied.

Pete was a loving husband and father and enjoyed life to the fullest at each and every opportunity. To Pete, life was a journey. The answer to any problem was inconsequential; the long arduous path from question to answer was the only purpose for the question in the first place. Dr. Peter Larsen and I had the privilege of working closely with Pete and experiencing his talents and benefiting from his wisdom and guidance at Ohio State University for several years. We had the unique opportunity to observe Pete in and out of the hospital—the phrase “work hard, play hard” epitomizes the Peterson philosophy. Peter Larsen remembered Pete at his funeral; here is a portion of that eulogy:
When I tried to decide what to say about this amazing man, I started by making a list. What I discovered was a man of what I like to call “wonderful contradiction.”

Pete was perhaps one of the most successful men I have known, yet he would have listed his Eagle Scout Award as being more important than many of the prestigious professional honors he received.

He was our most vigorous critic and yet our strongest advocate.

He was the teacher of teachers but also the perpetual student.

He was not an OSU alumnus but bled scarlet and gray.

He demanded hard work but taught me that it isn’t really work if you love what you do.

He was a teacher who, when honored, thanked his students for teaching him.

Although surrounded by personal success, he found the greatest satisfaction in the success of others.

He was our boss but was more comfortable as our partner in a raft on the New River.

He would argue with you, not to get you to agree, but to get you to disagree and defend.

He trained many to reach great financial success but placed the reward gained by teaching higher than any financial reward.

He had much of which to boast and be proud, but instead practiced humility.

He was perhaps the smartest man I have ever known but was always first to admit when you had a good idea, and was gracious enough not to point out that he had thought of it himself, perhaps even years prior.

I never heard him speak on a topic when I was not totally impressed with the insight and knowledge he seemed to have, but he was often more content listening to what others had to say.

He was more interested in finding the truth than about being right himself.

He was 15 years older than me but looked younger.

He would often tell residents, much to their dismay, I might add, that it is not the answer that is important, but the question.

Many of his accomplishments could easily be ranked on a 1-to-10 scale as a “10.” Yet, I can still hear him say, “There is no such thing as a ’10.’”

He had the same enthusiasm for a giant rope swing as he did for a new operation.

He knew more than many of the speakers at the lectures he attended, but he always took notes.

He built what is perhaps the best Oral and Maxillofacial Surgery Department in the country, but, for me, his finest hour as our leader was when he tenderly took care of Vicki, Arden Hegtvedt’s wife, when Arden died.

He was a man most deserving of a long and wonderful life, yet we are here today because this wonderful life has been tragically cut short.

If, as said by William James, “the greatest use of life is to spend it for something that will outlast it,” then Pete spent his life well. For, as I look around, I see scores of us who owe so much of what we are to this one life well spent.

Pete died too young, and he will be missed, but through this textbook his teachings will continue.

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Part 1

PRINCIPLES OF MEDICINE, SURGERY, AND ANESTHESIA
Wound Healing

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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 following injury. Although healing culminates uneventfully in most instances, a variety of intrinsic and extrinsic factors can impede 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 appraise and selectively use the growing array 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 depends 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. On the other hand 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; however, similar patterns of biochemical and cellular events occur in virtually every other tissue. 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. Vasconstriction of the injured vasculature is the spontaneous tissue reaction to staunch 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. Additionally 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 β (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 vasconstriction 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 to 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 persisting neutrophils, including tumor necrosis factor α (TNF-α) and interleukins (IL-1α, IL-1β), continue to stimulate the inflammatory response for extended periods.\(^{[3]}\)

 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 wound microdébridement initiated by the neutrophils. They 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 (eg, matrix metalloproteinases and collagenases), inducing formation of new extracellular matrix, and modulating angiogenesis and fibroplasia through local production of cytokines such as thrombospondin-1 and IL-1β. The centrality of

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**FIGURE 1-1** Immediately following 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 dermal fibroblasts through growth factors, MMPs, and members of the TGF-β family. FGF = fibroblast growth factor; MMP = matrix metalloproteinase; PDGF = platelet-derived growth factor; TGF-β = transforming growth factor beta. Adapted from Bissell MJ and Radisky D.\(^{[3]}\)
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 post injury 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). Starting as early as the third day post injury 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 extracellular matrix (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 proportion 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 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 about 80% of the original strength. Homeostasis of scar collagen and ECM is regulated to a large extent by serine proteases and matrix metalloproteinases (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. Neurapraxia 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 relates 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. 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 proximal 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 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/d. 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 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 perios- teum. Necrotic material at the fracture site elicits an immediate and intense acute inflammatory response which 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. Comprised 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.

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. 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 (~2 weeks) die and are removed by phagocytes. The majority (up to 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 (~2,000 microstrains) are considered physiologic and necessary for cell differentiation and callus remodeling, high strain levels (> 2,000 microstrains) begin to adversely affect osteoblastic differentiation and bone matrix formation. 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. 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 containing 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 setting 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 that begin to penetrate towards 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 following tooth extraction. Due 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. In such instances 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 to 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.\(^\text{16}\) 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.\(^\text{17}\) Following 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 transplant is signaled by the pink appearance of the graft, which appears between the third and 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 re-innervation depends on how accessible the neurilemmal sheaths are to the entering nerve fibers. The clinical performance of the grafts depends on their relative thickness. As split-thickness grafts are thinner than full-thickness grafts, they are 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. Nevertheless 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 although 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 where the bacterial burden of replicating microorganisms actually impairs healing.\(^\text{18}\) Experimental studies have demonstrated that regardless of the type of infecting microorganism, wound infection occurs when there are more than 1 \(\times\) 10^5 organisms per gram of tissue.\(^\text{19,20}\) Beyond relative numbers, the pathogenicity of the infecting microorganisms as well as host response factors 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 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 extracellular matrix components, including glycosaminoglycans and collagen Type I. 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, on the other hand, 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 evolution of granulation tissue into scar. Dysregulation in apoptosis results in excessive scarring, inflammation, and an overproduction of extracellular matrix components. Both keloids and hypertrophic scars demonstrate sustained elevation of growth factors including TGF-β, platelet-derived growth factor, IL-1, and IGF-I. The growth factors, in turn, increase the numbers of local fibroblasts and prompt excessive production of collagen and extracellular matrix. Additionally, 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 extracellular matrix 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.

**Tissue Trauma**

Minimizing surgical 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 as 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 interfere 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. Additionally, hematomas can result in necrosis of overlying flaps. However, hemostatic techniques must not be used too aggressively during surgery as 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. 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 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 incite varying degrees of foreign body reaction and adversely impact 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. 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. Relative hypoxia in the region of injury stimulates a fibroblastic response and helps mobilize other cellular elements of repair. 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 extracellular matrix. 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. 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 affect wound healing. Smoking is a common contributor to decreased tissue oxygenation. After every cigarette the peripheral vasoconstriction 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. 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. The wound ischemia and impaired recruitment of cells resulting from the small vessel occlusive disease renders 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 1,200 and 1,800, and levels below 800 are considered severe total lymphocyte deficits. Patients with debilitated immune response include human immunodeficiency virus (HIV)-infected patients in advanced stages of the disease, patients on immunosuppressive therapy, and those taking high-dose steroids for extended periods. Studies indicate that HIV-infected patients with CD4 counts of less than 50 cells/mm³ are at significant risk of poor wound outcome. Although newer immunosuppressive drugs, such as cyclosporine, have no apparent effect on wound healing, other medications can retard the healing process both in rate and quality by altering both 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. 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 glucocorticosteroids 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. The cellular and molecular responses to tissue irradiation are immediate, dose dependent, and can cause both early and late consequences. 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 constituent cells gradually recover their proliferative abilities. However, these early symptoms may not be apparent in some tissues such as bone, where 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. Collagen hyalinizes and the tissues become increasingly fibrotic and hypoxic due to obliteratorive 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 following 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. Due to the relative hypoxia at the irradiated site, tissue with intact blood supply needs to be brought in to provide both oxygen and the cells necessary for inflammation and healing. The progressive obliteration of blood vessels makes bone particularly vulnerable. Following 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 (PO₂) of 5 to 20 mm Hg, leads to anaerobic cellular metabolism, increase in tissue lactate, and a decrease in pH, all of which inhibit wound healing. HBO therapy entails the patient lying in a hyperbaric chamber and breathing 100% oxygen at 2.0 to 2.4 atmospheres 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 \( P_O_2 \) ultimately reaches 800 to 1,100 mm Hg. 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.\(^{65}\) Animal therapy can be an effective adjunct in the treatment of osteomyelitis and soft tissue infections.\(^{66,67}\) 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.\(^{68,69}\)

**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.\(^{37}\) 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.\(^{38}\) 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.\(^{39}\) 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 \(< 2\) 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.\(^{40,41}\) 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 about 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.\(^{42}\) 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 \( \alpha \)-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 scurvy. 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.\(^{43}\) Copper is essential for the John molecular basis of wound healing, especially in molecules that have a high content of copper. Ascorbic acid 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.\(^{44}\) Numerous enzymes are zinc dependent, particularly DNA polymerase and reverse transcriptase. On the other hand, 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.\(^{45,46}\)

**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 extracellular matrix synthesis. 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 PDGF, angiogenesis factor, epidermal growth factor (EGF), TGF, 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, and when and at what dose. To date only a single growth factor, recombinant human platelet-derived growth factor-BB (PDGF-BB), has been approved by the United States Food and Drug Administration for the treatment of cutaneous ulcers, specifically diabetic foot ulcers. Results from several controlled clinical trials show that PDGF-BB gel was effective in healing diabetic ulcers in lower extremities and significantly decreased healing time when compared to the placebo group. 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. 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 following injury.

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. Insulin-like growth factors have demonstrated similar neurotrophic properties. Although most of the investigations hitherto 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 for their ability to promote the formation of new bone. Of the multiple osteoinductive cytokines, the bone morphogenetic proteins (BMPs) belonging to the TGF-β superfamily have received the greatest attention. 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 (osteogenesis). Yasko and colleagues 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 colleagues 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.

**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. 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. 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 extracellular matrix. 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 use 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. Similarly bone marrow cells transfected ex vivo with hBMP-2 cDNA have been shown to heal femoral defects. Using osteoprogenitor cells for the expression of bone-promoting osteogenic factors enables the cells to not only express 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. The human skin substitutes available 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. 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. 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.

References

Wound Healing


Oral-maxillofacial surgery frequently causes temporary but clinically significant alteration of the anatomy and physiology of the upper aerodigestive tract, but has minor direct impact on vital organ systems. Therefore, the surgery itself is generally safe to perform even on relatively unhealthy individuals. However, the physiologic stresses produced by surgery and the anesthetic techniques necessary for these procedures can lead to serious morbidity and mortality. This is especially true in patients with various organs on the brink of decompensation due to disease or comorbid conditions.

This chapter presents the common medical situations that can compromise the successful outcome of oral or maxillofacial surgery. Emphasis is given to the means of detecting health problems preoperatively and preparing patients with various medical disorders so that complications in the perioperative period are avoided or minimized. The liberal use of medical consultations is highly recommended for all situations in which a surgeon has concerns for the medical well-being of a surgical patient.

Most commonly oral-maxillofacial surgery is performed on healthy patients. A quick screen of health conditions may give additional data in the evaluation of the healthy patient. A preoperative patient questionnaire has been used in determining whether any further risk should be ascertained. The questions in Table 2-1 have been valuable in preoperative patient evaluation.

In addition to this group of questions, other questionnaire-type screening tools can be valuable. Exercise capacity, such as the 6-minute walk test, use of medications and herbal supplements, and age can be important determinants of perioperative risks. Exercise tolerance has been shown to predict long-term mortality as well as short-term perioperative risks. All patients should be questioned regarding their exercise tolerance with a question such as, “If I asked you to walk as far as you could, how far would that be?” This may be answered as a function of time or distance. It is helpful to ask, “When was the last time you walked that far?” If there is a limitation of exercise, then ask, “What is the reason for the limitation?” It may be due to orthopedic or other musculoskeletal problems that limit exercise, or cardiac or pulmonary insufficiency.

Medication use is important, and with the use of a plethora of over-the-counter medications and dietary supplements,
specific questioning is in order. Aspirin or other nonsteroidal anti-inflammatory drug use may exacerbate bleeding during major surgery. Some herbal supplements are known to increase the risk of bleeding as well.4

Finally age can be used as a surrogate for underlying disease or decreased reserve. There are no absolute cutoffs for age in estimation of risk; age of 70 years is used as a benchmark for a separate risk factor in surgical mortality. Laboratory testing may be helpful in a small subset of patients. Routine testing requirements may vary from operative center, office, or hospital, but in general there is often overtesting and under-review of the results. If guidelines at a particular center have been established, it is important to use a checklist of the tests, including their results. Many of these tests are arbitrary and not supported by evidence-based research. However, it is not unreasonable to establish a schedule of routine testing in unselected patients. While most young and apparently healthy patients do not need any preoperative laboratory testing, unselected adults over the age of 40 years may benefit from a preoperative hematocrit and tests of renal function and blood glucose. A blood count may reveal anemia or serve as a benchmark when excessive blood loss or anemia is found after surgery. Glucose determination is helpful in those patients with diabetes or obesity, and serves as a useful screening tool for diabetes in the general population.5

The preoperative evaluation of healthy patients should include the following6,7:

1. A screening questionnaire for all patients (see Table 2-1)
2. A history of exercise tolerance for all patients
3. Blood pressure and pulse for all patients
4. History and physical examination if one of the above is abnormal, in patients over 60 years, or in those undergoing major surgery
5. Pregnancy test for women who may be pregnant
6. Hematocrit for surgery with expected major blood loss
7. Serum creatinine concentration if undergoing major surgery, hypotension is expected, nephrotoxic drugs will be used, or the patient is over age 50 years
8. Electrocardiogram (ECG) recommendations as above, unless obtained within the previous month
9. Chest radiograph for patients over 60 years, or for those with suspected cardiac or pulmonary disease, if such imaging has not been performed within the past 6 months
10. Other tests only if the clinical evaluation suggests a likelihood of disease

Cardiac Disease
Cardiac disease is common in the North American and other populations, and the patient is usually well aware of any existing cardiac problem. Thus, it is essential to screen for cardiovascular disease, and recent interventions have shown the ability to greatly reduce perioperative risks in patients with known or suspected cardiac disease.

Preservation of cardiac health is an essential element of any perioperative protocol. The proper match of oxygen supply to oxygen use in myocardial tissue is the key to maintaining normal contractility and electrical activity. In the patient with a healthy heart and lungs, the myocardium is protected in the perioperative period by avoiding hypovolemia, ensuring adequate oxygen-carrying capacity of the blood, keeping serum electrolytes within physiologic limits, and supplying the lungs with adequate oxygen. Cardiac output also depends on properly functioning valves. Finally the load against which the ventricles must work should stay within reasonable limits to preserve optimal myocardial function.

Several cardiac conditions can exist preoperatively that have the potential to compromise the heart’s ability to maintain adequate blood pressure intra- or postoperatively. These conditions include coronary artery disease, valvular disease, various processes predisposing the heart to congestive failure, and abnormalities of electrical impulse generation or conduction. In the discussion of the four conditions that follows, emphasis is on the means of assessing the degree of cardiac compromise and reserve, of improving the situation preoperatively, and of managing the condition perioperatively.

Coronary Artery Disease
The two principal processes that cause an insufficient blood supply to the myocardium are coronary artery obstruction and spasm. Myocardial ischemia will occur when the supply of oxygen is inadequate to meet the demand for oxygen. Myocardial oxygen need is increased when the heart has increased rate or mass, or is forced to work against an increased afterload that increases end-diastolic wall tension. In these situations symptoms of ischemia will occur if oxygen supply to the myocardium cannot be increased because the coronary arteries are critically narrowed by fixed atheromatous lesions and/or spasm; clinically this is manifested by exercise-induced angina pectoris.

Coronary artery disease is one of the most studied diseases in humans. Over the past several years new paradigms regarding coronary artery disease have emerged and have been validated. The idea of a hard plaque slowly encircling the lumen of a coronary artery until occlusion has occurred has been replaced by the concept of plaque rupture. Many plaques in the lumen of the coronary vessels are considered to be soft, with a membrane or thin cell layer covering a highly thrombogenic lipid core. This membrane may rupture even in small lesions, exposing thrombogenic materials into the blood. This sets up an immediate clotting cascade resulting in thrombus formation, occluding the vessel
and precipitating myocardial infarction or unstable angina.8,9

Coronary artery disease includes the progression of an endothelial lesion from a fatty streak to an occlusive lesion or plaque rupture as noted above. Several risk factors for coronary artery disease have been identified, including family history of early coronary disease (under age 65 yr), male gender, diabetes mellitus, and elevated cholesterol, including total cholesterol and/or low-density lipoprotein (LDL) cholesterol. High levels of LDL cholesterol, low levels of high-density lipoprotein cholesterol, hypertension, and cigarette smoking are the most predictive risk factors of coronary artery disease. Additional risk factors such as elevated levels of homocysteine, C-reactive protein, myeloperoxidase and others are being evaluated.10 Interestingly a large percentage of patients with first-time myocardial infarction do not have known risk factors for coronary artery disease.11,12

As noted above, a plaque may progress to cause a limitation of flow of blood through the coronary artery to the myocardial tissue. Myocardial ischemia produces decreased myocardial contractility rapidly leading to systemic hypotension and pulmonary vascular congestion. The limitation of flow leads to the symptom of angina. Patients may complain of a squeezing, choking, or tight feeling in the substernal region radiating to the throat, jaw, shoulders, or arms. The patient may also experience dyspnea, diaphoresis, and nausea. Anginal symptoms will dissipate soon after the provoking activity ceases or after transmucosal nitroglycerin is administered. Infarction symptoms will usually persist despite nitroglycerin use or rest.

It is important to ask patients suspected of having coronary artery disease if they have discomfort with exertion, rather than focusing on pain. A patient may give a history of dyspnea and chest tightness, among other symptoms, after exertion, eating a heavy meal, or entering a cold environment. Typically these symptoms are reproducible. Patients who have angina symptoms that are progressive with less precipitating forces, angina with increasing frequency, or angina at rest are considered to have unstable angina and require evaluation by a qualified cardiovascular specialist.

There are no standard physical signs of coronary artery insufficiency so preoperative screening relies on historic information and electrocardiography. A cardiovascular examination may show evidence of vascular or valvular disease, or some degree of cardiac decompensation. Symptoms of compromised coronary or carotid arteries should be sought preoperatively in all adult males, as well as in menopausal and postmenopausal females.

**Physical Examination** The physical examination in patients with coronary artery disease is frequently unrevealing. The history is the most important determinant of risk. However, a cardiovascular examination may show evidence of vascular disease, valvular disease, or evidence of cardiac decompensation.

Patients with findings of peripheral vascular disease should be considered at high risk for underlying coronary artery disease. On heart examination an S4 may be present, reflecting reduced compliance in an ischemic myocardium. Auscultation of the neck, periumbilical area of the abdomen, and inguinal areas should be used to detect bruits. In addition, pedal pulses and inguinal pulses should be checked. Diminished or absent pulses, cool feet, and skin changes such as hair loss in the ankles and feet may indicate peripheral vascular disease. Specific questioning about problems occurring during physical activity or postprandially should be included. It must be remembered that many patients with first time myocardial infarction have no known risk factors.

A resting ECG should be done within a month of a planned elective general anesthetic and surgery in all males age 35 years and older, all females age 45 years and older, and all other patients with a history suggestive of cardiac disease.13 More elaborate routine cardiac testing is unwarranted. Although it is unlikely to see resting ECG changes suggestive of acute ischemia, old silent infarcts (representing 20 to 60% of all infarctions) or conduction blocks due to coronary disease may be detected.14 It should be noted that 30% of patients with a history of myocardial infarction have a normal resting ECG.15 ECG after controlled treadmill exercise is a more sensitive means of detecting ischemic tendencies as evidenced by ST depression or T-wave inversion. Patients with a past history of cardiac disease should have preoperative posteroanterior and lateral chest radiographs to detect early signs of congestive heart failure. Finally a thallium stress test can be used, but only in the case of an equivocal treadmill test, or coronary angiography can be performed to identify areas of narrowing, which predispose the patient to perioperative myocardial ischemia if clinical indications for angiography are present.

All patients with a documented history of angina may have an increased risk of perioperative infarction. This risk varies with the severity of the coronary disease and the degree of physiologic stress in the perioperative period. Patients with stable angina have only a slightly raised risk during anesthesia and surgery compared to the normal population. Angina that is worsening with respect to frequency, duration, response to medication, or ease of production is, by definition, unstable angina. Surgery in such a situation should only proceed if required emergently. Patients with stable but poorly controlled angina need medical intervention to improve their cardiac status before most elective surgery.

The American College of Cardiology has produced a listing of major, intermediate and minor cardiovascular risk factors and matched these with a listing of higher-risk operations. These risks are then entered
into a straightforward algorithm directed to decisions on invasive testing, noninvasive testing, intervention or progression to surgery (Table 2-2 and Figure 2-1). Risk reduction strategies have also evolved, with reduced emphasis on preoperative testing. The newest risk reduction strategy includes the use of β-blockade in patients with known coronary artery disease or with risk factors for coronary artery disease.17

Patients with stable, well-controlled angina, or who have delayed surgery after an uncomplicated myocardial infarction for a period dictated by their cardiologist, can usually undergo elective maxillofacial procedures safely if intraoperative hyper- or hypotension is avoided. Although some studies indicate the risk of infarction increases with the duration of surgery, this has only been well documented in the case of major thoracic or upper abdominal procedures.18,19 In general, nonurgent surgery should be postponed for at least 6 weeks after myocardial infarction. Patients who need nonurgent surgery in this 6-week window should be co-managed by a cardiologist. Modern day general anesthesia may actually be protective of the myocardium, because supraphysiologic levels of oxygen are administered and cardiac work is minimized through maintenance of muscle relaxation, sympathetic nervous system antagonism, blood pressure control, and prompt dysrhythmia recognition and management. To assist with these goals consideration should be given to radial artery cannulation for blood gas and pH measurement and precise blood pressure monitoring. The presence of signs of chronic congestive failure following a myocardial infarction increases operative risk, as is discussed later in this chapter.

The risk of general anesthesia after a recent myocardial infarction is due to possible extension of the earlier myocardial infarction and the development of cardiac dysrhythmias. A target-like zone is described in myocardial infarction, with the center being infarcted tissue. It is a zone surrounding this infarcted tissue that is considered to be stunned or vulnerable. This zone is the area into which the myocardial infarction may extend and from which dysrhythmias can be generated. After the 6-week window has passed, the patient can be evaluated as any other coronary artery disease patient.20

Patients with coronary artery disease have their greatest risk of cardiac problems in the early postoperative period. The cardiorespiratory system is no longer controlled by general anesthesia, and the normal stresses that occur in the early recovery period exist. There is usually a need for increased cardiac output, which the diseased heart may not be able to deliver or tolerate, and ischemia can result. Therefore, these patients need frequent cardiopulmonary physical examinations and close monitoring of vital signs, urine output, jugular venous pressure, and electrolytes. An immediate postoperative ECG should be obtained in patients with a history of coronary artery disease, particularly if they have any of the following:

- Unexplained hypotensive or syncopal episode
- Signs of heart failure
- Dysrhythmias
- Angina

### Table 2-2 Clinical Predictors of Increased Perioperative Cardiovascular Risk (Myocardial Infarction, Heart Failure, Death)

<table>
<thead>
<tr>
<th>Category</th>
<th>Major</th>
<th>Intermediate</th>
<th>Minor</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Unstable coronary syndromes</td>
<td>Mild angina pectoris (Canadian Class I or II)</td>
<td>Advanced age</td>
</tr>
<tr>
<td></td>
<td>Acute or recent myocardial infarction* with evidence of important ischemic risk by clinical symptoms or noninvasive study</td>
<td>Previous myocardial infarction by history or pathological Q waves</td>
<td>Abnormal electrocardiogram (left ventricular hypertrophy, left bundle-branch block, ST-T abnormalities)</td>
</tr>
<tr>
<td></td>
<td>Unstable or severe angina (Canadian Class III or IV)</td>
<td>Compensated or prior heart failure</td>
<td>Rhythm other than sinus (eg, atrial fibrillation)</td>
</tr>
<tr>
<td></td>
<td>Decompensated heart failure</td>
<td>Diabetes mellitus (particularly insulin-dependent)</td>
<td>Low functional capacity (eg, inability to climb one flight of stairs with a bag of groceries)</td>
</tr>
<tr>
<td></td>
<td>Significant dysrhythmias</td>
<td>Renal insufficiency</td>
<td>History of stroke</td>
</tr>
<tr>
<td></td>
<td>High-grade atrioventricular block</td>
<td></td>
<td>Uncontrolled systemic hypertension</td>
</tr>
<tr>
<td></td>
<td>Symptomatic ventricular dysrhythmias in the presence of underlying heart disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Supraventricular arrhythmias with uncontrolled ventricular rate</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Severe valvular disease</td>
<td></td>
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</tbody>
</table>

*The American College of Cardiology National Database Library defines recent myocardial infarction as greater than 7 days but less than or equal to 1 month (30 days); acute myocardial infarction is within 7 days.

†May include “stable” angina in patients who are unusually sedentary.

Adapted from Eagle KA et al.16
Medical Management of the Surgical Patient

Need for noncardiac surgery

Operating room

Postoperative risk stratification and risk factor management?

Coronary revascularization within 5 years?

Recent coronary symptoms or signs?

Recent coronary evaluation?

Recent coronary angiography or stress test?

Favorable result and no change in symptoms

Undesirable result or change in symptoms

Clinical predictors

Major clinical predictors*

Intermediate clinical predictors†

Minor or no clinical predictors‡

Consider delay or cancel noncardiac surgery

Consider coronary angiography

Go to step 6

Go to step 7

Operating room

Subsequent care dictated by findings and treatment results

Clinical predictors

Functional capacity

Surgical risk

Noninvasive testing

Invasive testing

Poor (< 4 METs)

Moderate or excellent (> 4 METs)

High surgical risk procedure

Intermediate surgical risk procedure

Low surgical risk procedure

Noninvasive testing

Operating room

Postoperative risk stratification and risk factor reduction

Consider coronary angiography

Subsequent care dictated by findings and treatment results

Low risk

High risk

FIGURE 2-1 Stepwise approach to preoperative cardiac assessment. Steps are discussed in the text. Note that subsequent care may include cancellation or delay of surgery, coronary revascularization followed by noncardiac surgery, or intensified care. MET = metabolic equivalent. *Major clinical predictors include unstable coronary syndromes, decompensated congestive heart failure, significant dysrhythmias, and severe valvular disease. †Intermediate clinical predictors include mild angina pectoris, prior myocardial infarction, compensated or prior congestive heart failure, diabetes mellitus, and renal insufficiency. ‡Minor clinical predictors include advanced age, abnormal electrocardiogram, rhythm other than sinus, low functional capacity, history of stroke, and uncontrolled systemic hypertension. Adapted from Eagle KA et al.16
Care in the postoperative period should be taken to maintain normal intravascular volume, avoid hyper-or hypotension, keep serum electrolytes in their physiologically normal ranges, manage patient anxiety and pain, give supplemental oxygen when needed, and resume preoperative cardiac medications. Signs of infections or pulmonary problems should be pursued aggressively.

**Left Ventricular Dysfunction** Left ventricular dysfunction can result from myocardial infarction or primary cardiomyopathy. Left ventricular dysfunction can be separated into systolic or diastolic dysfunction. Systolic dysfunction occurs after myocardial infarction or other direct muscle injury, causing either wall motion abnormalities or decreased cardiac output. Diastolic dysfunction results from stiffness or reduced compliance of the left ventricle.21

Concepts of preload, afterload, and compliance are useful to know when discussing left ventricular dysfunction. Preload is thought of as volume being presented to the right heart. The right heart is a low-pressure chamber, handling the influx of blood via the right atrium. Excess volume may be presented to the pulmonary vasculature, resulting in pulmonary congestion or pulmonary edema. Preload problems can occur from left heart failure causing fluid to back up into the pulmonary arterial tree, or may also be due to reduced compliance in the left ventricle. Rarely isolated right-sided ventricular failure occurs, such as from pulmonary hypertension or right ventricular infarction. Excess preload is usually managed with diuretic therapy or fluid restriction.

*Afterload* refers to the pressure in the aorta against which the left ventricle must pump. This arterial resistance or afterload may be increased in hypertension and aortic stenosis. Afterload may also be relative to the pumping capacity of the left ventricle; hence normal blood pressures may impair a failing heart. Afterload reduction using vasodilators, especially angiotensin-converting enzyme (ACE) inhibitors, is an important treatment in heart failure, certain valvular abnormalities, and hypertension. For instance, afterload reduction in systolic dysfunction reduces the work of the left ventricle against the normal arterial pressure. This reduces demand on the heart. Compliance refers to the ability of the heart to distend. Reduced compliance in the left ventricle is described as a stiffness or alteration in the diastolic filling of the left ventricle. If the left ventricle does not fill properly during the cardiac cycle, pulmonary congestion can occur, even though the apparent forward flow of blood is not impaired.

Left ventricular systolic dysfunction can be tolerated within the reserve capacity of the individual, or may manifest itself as congestive heart failure. As noted above it can be due to insults, such as myocardial infarction, viral myocarditis, or direct trauma to the heart. In addition there may be global dysfunction due to more widespread ischemia, idiopathic cardiomyopathy, or valvular abnormalities.

Symptoms suggesting congestive heart failure include dyspnea on exertion, paroxysmal nocturnal dyspnea (PND), nighttime cough, and ankle swelling. Patients with PND may sit up on the side of the bed for a moment and then get up to drink a glass of water. Patients with severe heart failure may sleep in a sitting position or slumped against a countertop. On physical examination of the heart there may be an S1 gallop rhythm and the point of maximal impulse (PMI) may be shifted laterally and inferiorly. In addition a diffuse PMI may be present. A murmur of mitral insufficiency may be present due to dilated annulus of the heart. The neck veins, which should be flat with the patient’s chest being elevated 30°, may be distended. On lung examination rales may be present from pulmonary congestion and there may be dullness to percussion from pleural effusions.

Diagnostic testing for patients with heart failure includes an ECG, which may show Q waves of a previous myocardial infarction, elevated QRS amplitude of left ventricular hypertrophy, or low QRS amplitude in some patients with severe myocardial dysfunction. An echocardiogram may show evidence of diastolic dysfunction through measurements of compliance, or may show wall motion abnormalities and reduced ejection fraction.

Management of congestive heart failure is indicated when evidence of decompensation is present. Decompensation is manifested by increased symptoms of dyspnea on exertion or PND, the presence of an S3 gallop rhythm, distended neck veins, or an increase in peripheral edema.22 The decision is then made whether or not to admit the patient to the hospital for treatment or to advanced treatment as an outpatient. This is determined more by the severity of the heart failure than the urgency of the surgery. In either case the management includes starting or increasing diuretic therapy, reducing afterload, and in some cases, increasing contractility of the heart. If a diuretic has not been prescribed, furosemide 20 mg daily for 3 to 4 days should suffice in reducing total body salt and water. If a diuretic has already been prescribed, doubling of the dose is indicated. Rarely a second diuretic such as metolazone would be added to boost the loop diuretic.

Afterload reduction is a key tenet in the treatment of congestive heart failure.23 An ACE inhibitor is first-line treatment for congestive heart failure and would be added or increased in dose during an episode of decompensated congestive heart failure. Typically the systolic blood pressure is lowered to between 90 and 110 mm Hg unless significant hypertension was involved in the decompensation. After appropriate diuretic therapy and ACE inhibition, attention may be turned to systolic contractility. In cases of dilated cardiomyopathy the addition of digoxin can be helpful. Its
applicability in other types of heart failure is questionable. Digoxin therapy should be guided by serum digoxin levels. In addition, treatment of decompensated congestive heart failure should include monitoring of electrolytes. If a patient’s known congestive heart failure is compensated, the patient’s surgical risk is greatly reduced toward normal. If the patient has reasonable functional capacity, for instance is able to walk two blocks or more without shortness of breath, the risk factor of heart failure can be discounted, and the patient can come to surgery. In summary a patient with decompensated heart failure is at high risk for major cardiac events, but this risk can be greatly reduced with appropriate management, including diuretic therapy, afterload reduction, and digoxin therapy when needed. Diastolic decompensation is usually treated acutely with diuretic therapy alone, using afterload reduction and the use of β-blockers if hypertension is present or further treatment is needed. While β-blockers are often used in dilated cardiomyopathy, acute use in the treatment of decompensation is not recommended.

Valvular Heart Disease

Most patients with valvular heart disease who have few symptoms or limitations of activity can safely undergo most elective maxillofacial surgery. Diseased cardiac valves pose two general risks: precipitation of cardiac failure and susceptibility to infective endocarditis. The likelihood of causing failure or worsening preexisting cardiac failure is dependent on the location and severity of valve pathology. Prophylactic antibiotics should be used for all patients with a cardiac valve abnormality with a resultant murmur who undergo maxillofacial procedures in which bleeding occurs (Tables 2-3 and 2-4).

Mitral Stenosis Mitral stenosis is almost always a sequela of childhood rheumatic heart disease, although a definite history can be obtained in only half of such cases. Fortunately the incidence of new cases of this problem has decreased substantially since the use of antibiotics to manage streptococcal infections became common practice. The rheumatic disease process causes valve fibrosis, fusion, and calcification. These changes limit valve motion, thus restricting the flow of blood into the left ventricle. The latency period is usually 15 to 20 years. Once valve obstruction occurs the patient will begin to suffer gradually worsening exertional dyspnea and fatigue due to pulmonary vascular congestion and progressive right heart failure. Left arterial enlargement may lead to the appearance of atrial fibrillation (AF) with possible atrial thrombus formation and systemic arterial embolization.

Examination of the patient with clinically significant mitral stenosis may reveal an early diastolic opening snap followed by a low-pitched murmur and a loud first heart sound. Patients in AF will characteristically have an irregularly irregular pulse. A chest radiograph will reveal an enlarged left atrium, pulmonary vascular enlargement, and in more severe cases right ventricular hypertrophy. An ECG may reveal AF, left atrial enlargement, and right ventricular hypertrophy. Echocardiography is

<table>
<thead>
<tr>
<th>High-Risk Category: Prophylaxis Recommended</th>
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<tbody>
<tr>
<td>Prosthetic cardiac valves</td>
</tr>
<tr>
<td>Previous infectious endocarditis</td>
</tr>
<tr>
<td>Complex cyanotic congenital heart disease</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Moderate-Risk Category: Prophylaxis Recommended</th>
</tr>
</thead>
<tbody>
<tr>
<td>Most other congenital malformations</td>
</tr>
<tr>
<td>Acquired valvular dysfunction</td>
</tr>
<tr>
<td>Hypertropic cardiomyopathy</td>
</tr>
<tr>
<td>Mitral valve prolapse with valvular regurgitation</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Negligible-Risk Category: Prophylaxis NOT Recommended</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary artery bypass graft</td>
</tr>
<tr>
<td>Mitral valve prolapse without regurgitation</td>
</tr>
<tr>
<td>Physiologic, functional, or innocent heart murmur</td>
</tr>
<tr>
<td>Isolated secundum atrial septal defect</td>
</tr>
<tr>
<td>Surgical repair of atrial septal defect; patent ductus arteriosus</td>
</tr>
<tr>
<td>Previous rheumatic fever without valvular dysfunction</td>
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<table>
<thead>
<tr>
<th>Oral Procedures in which Prophylaxis is Recommended</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dental extractions and biopsies</td>
</tr>
<tr>
<td>Periodontal procedures</td>
</tr>
<tr>
<td>Dental implant placement</td>
</tr>
<tr>
<td>Periapical endodontic procedures</td>
</tr>
<tr>
<td>Intraligamentary local anesthetic injections</td>
</tr>
<tr>
<td>Dental prophylaxis when bleeding is expected</td>
</tr>
<tr>
<td>Other procedures causing intraoral bleeding</td>
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<table>
<thead>
<tr>
<th>Oral Procedures in which Prophylaxis is NOT Recommended</th>
</tr>
</thead>
<tbody>
<tr>
<td>Routine local anesthetic injection</td>
</tr>
<tr>
<td>Intracanal endodontic therapy</td>
</tr>
<tr>
<td>Suture removal</td>
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<tr>
<td>Taking impressions</td>
</tr>
</tbody>
</table>
usually the definitive test used to detect and characterize mitral stenosis.

Patients with severe mitral stenosis who require elective surgery may need preoperative mitral valve commissurotomy or valve replacement. AF may be managed by preoperative digitalization or β-sympathetic blockade; pulmonary congestion is treated with diuretic therapy. Patients with a known or suspected atrial thrombus are usually on chronic anticoagulant therapy, which may need temporary alteration. Surgeons should note the compromised cardiac output of patients with mitral stenosis.

Acute pulmonary edema is not uncommon following noncardiac surgery on patients with significant mitral stenosis, particularly if excess fluid replacement was given. An additional problem facing these patients is diminished pulmonary compliance that may require postoperative mechanical ventilation longer than is usually necessary.

Mitral Regurgitation Mitral regurgitation or insufficiency is most commonly the result of damage or dysfunction due to coronary artery disease or from prior rheumatic heart disease. The incompetent valve allows left ventricular enlargements as the heart works and expands to maintain cardiac output. Symptoms of congestive failure appear as regurgitation worsens and the enlarging heart transitions to the decompensation (right) side of the Frank-Starling curve.

Physical examination of the patient with significant mitral regurgitation will reveal an apical point of maximal impact displaced inferolaterally, an apical, high-pitched, holosystolic murmur, and a third heart sound (gallop rhythm). Left ventricular hypertrophy and AF may appear on an ECG. Echocardiography will help define the extent of valve disease and, with a measurement of end-systolic left ventricular dimension, the prognosis can be determined (a dimension of more than 55 mm indicates left ventricular dysfunction). Doppler studies or cardiac angiography can be used to determine the severity of dysfunction.

Patients with failure secondary to initial regurgitation are medically managed with sodium restriction, digoxin, diuretics, and preload- and afterload-reducing vasodilators. Eventually surgical valve repair or replacement may be necessary.

There is little increased risk during maxillofacial surgery for patients with well-controlled mitral regurgitation. The surgeon and anesthesiologist must guard against the pulmonary edema to which these patients are prone. Monitoring of pulmonary capillary wedge pressure will help guide therapy.

Mitral Valve Prolapse

Mitral valve prolapse is a common form of mitral regurgitation, most frequently seen in young women, in which one or both of the mitral valve leaflets prolapse into the left atrium during systole, allowing varying degrees of regurgitation to occur. It is characterized by a midsystolic click followed by a late systolic murmur. Symptoms include palpitations and chronic fatigue, but it can be asymptomatic; echocardiography is diagnostic. The prevalence of mitral valve prolapse in women and the general population has been overestimated, with more recent study showing a prevalence of about 3%, equally distributed among men and women; symptoms have been overemphasized as well. Mitral valve prolapse is usually managed symptomatically, using β-sympathetic antagonists to control palpitations.

As with other causes of mitral regurgitation, with medical management there is little increased risk for anesthesia and surgery. Patients should have ECG monitoring to detect intraoperative dysrhythmias, and those with a murmur should be given antibiotics to prevent infective endocarditis.

Aortic Regurgitation Aortic regurgitation or insufficiency occurs when the
Aortic valve becomes partially incompetent, resulting in a backflow of aortic blood into the left ventricle during diastole. This causes left ventricular volume overload resulting in hypertrophy and increased wall thickness, both of which increase myocardial oxygen requirements.

Patients with clinically significant aortic regurgitation will report unusual awareness of their heartbeat, prominent neck pulsations, and symptoms of pulmonary congestion at rest that resolve during exercise. Examination reveals a widened pulse pressure, a bisferious (bifid) carotid pulse, an inferolaterally displaced and prolonged apical PMI, and diastolic decrescendo murmur at the base. In severe cases there may be a third heart sound and apical low-pitched diastolic (Austin Flint) murmur.

The ECG will reveal left ventricular hypertrophy, and a chest radiograph will show left ventricular and aortic root enlargement. Echocardiography with or without a Doppler is used to diagnose and characterize aortic regurgitation. Patients with significant aortic regurgitation will be treated with vasodilators such as calcium channel blockers or ACE inhibitors. β-Blockers should be avoided since they can prolong diastole, increasing the regurgitant flow. Eventually aortic valve replacement may be necessary. Low-risk patients have a near-normal sized left ventricular cavity, while high-risk patients nearing the time for aortic valve replacement show enlargement of end-systolic left ventricular dimensions, corrected for body surface area.29,30

Typically bradycardia or vasodilation cannot be tolerated; thus measures to prevent these changes should be used. The ECG lead V₅ should be monitored perioperatively for signs of subendocardial ischemia. Pulmonary artery catheterization is useful in the perioperative period for measuring left-sided pressure and cardiac output. Afterload reduction may be helpful in patients with normal left ventricular function by reducing the regurgitant fraction, increasing stroke volume, and decreasing left ventricular end-diastolic volume and pressure. Care must be taken when using afterload reducers to not allow aortic diastolic pressure to drop so low as to compromise coronary perfusion.

**Aortic Stenosis**

Aortic stenosis can involve the valve itself or be supra- or infravalvarular. Valve stenosis is most often due to either a congenitally bicuspid valve (which occurs in about 2% of the population) or an aging-related degeneration of a normal trileaflet valve. In either situation valve fibrosis and calcification occur and cause varying degrees of left ventricular outflow obstruction.

Symptoms classically include exertional angina, syncope, or dyspnea. However, many patients can be asymptomatic until surgical stress unMASKS problems. Physical examination of the patient with significant aortic stenosis will typically reveal a weak pulse, narrow pulse pressure, and a nondisplaced but accentuated and prolonged PMI. A diamond-shaped systolic murmur is heard at the base while a fourth heart sound is heard at the apex. Patients typically have little pulmonary hypertension so that many of the classic noncardiac symptoms and signs of heart failure are not present. But because the left ventricle depends on the end-diastolic boost from the left atrium, the development of AF can be catastrophic and should be suspected in a patient with aortic stenosis who suddenly deteriorates.

An ECG shows left ventricular hypertrophy, while the chest radiograph reveals left ventricular and ascending aortic enlargement and calcification. Echocardiography can be used to define the valvular pathology, and cardiac angiography is used to determine the pressure gradient across the valve and to check the status of the coronary arteries. SeVERely stenotic valves may require surgical replacement.

Patients with mild-to-moderate dysfunction requiring maxillocrinal surgery typically require little modification in surgical or anesthetic management. The aortic valve opening must be narrowed to 75% of its normal size before obstructive signs occur. If aortic and mitral stenoses coexist, the problems due to mitral stenosis will predominate. Perioperative risks in patients with isolated aortic stenosis are highest if the history includes exertional dizziness, syncope, or angina and the presence of coronary artery disease.

The preservation of sinus rhythm is important in these patients. Tachydysrhythmias must be avoided since the atrial “kick” supplies needed left ventricular filling. Supraventricular tachycardias should be treated immediately with direct current cardioversion. Sinus tachycardia may require administration of a β-sympathetic antagonist. Bradycardia is also harmful, and rates below 45 bpm should be increased with atropine. Anesthetics that cause myocardial depression should be used cautiously, if at all, and systemic vascular resistance should be maintained. The ECG lead V₅ should be monitored for signs of ischemia; if detected, coronary obstruction must be differentiated from insufficient coronary filling pressure due to aortic stenosis.

**Prosthetic Heart Valves**

Patients with prosthetic heart valves represent a special situation in which properly functioning valves have essentially normal cardiac function but may have new problems directly related to the artificial valve itself. These patients are susceptible to endocarditis (particularly staphylococcal), red cell destruction by the valve, prosthetic valve obstruction by thrombosis or pannus formation, and paravalvular regurgitation. Serum bilirubin, lactate dehydrogenase, and reticulocytes should be measured to detect occult hemolysis. Patients with mechanical (not bioprosthetic) valves are on chronic anticoagulant therapy that needs perioperative management. Patients with prosthetic valves should be given antibiotics to prevent infective endocarditis.
Congestive Heart Failure

The normal myocardium responds to increased physiologic demands by increasing the frequency of contractions and by dilating through the Frank-Starling mechanism, which increases contractility (the end-diastolic wall tension). Heart failure occurs when the heart’s compensatory mechanisms fail to handle the hemodynamic load, causing blood to back up into the pulmonary vasculature, right heart, and major venous beds such as the portal system.

Failure can be produced in two basic ways. First, the heart can be overwhelmed by excessive loads, such as elevated preload (venous return; eg, by hypervolemia) or increased afterload (resistance to ejection; eg, by elevated total peripheral resistance or aortic stenosis). Second, the heart’s ability to compensate for increased demands can be compromised, such as by myocardial infarction or cardiomyopathy.

Long-term management requires that both excessive preload and afterload be modulated. Preload is lessened by limiting intravascular volume through the use of dietary sodium restriction and diuretics, and by venodilation with drugs such as nitrates. Afterload is reduced through the administration of vasodilators. Cardiac contractility is augmented by digoxin. Angiotensin-converting enzyme inhibitors are another common therapeutic drug for failure. Finally, physiologic demands on the heart are controlled by advising the patient to get adequate rest and avoid strenuous exercise.

A failing heart produces many signs and symptoms that vary according to the severity of the decompensation. Dilation of the heart as it tries to compensate can be detected on a posteroanterior chest radiograph. The chest film will also show increased pulmonary vascular markings that occur as pressure forces fluid into interstitial spaces and alveoli, producing pulmonary edema. The signs of rales and decreased breath sounds in dependent portions of the lungs, and symptoms such as dyspnea at rest or on exertion, paroxysmal nocturnal dyspnea, and orthopnea commonly occur. Failure of the heart to propel blood out of the systemic venous system can produce increased interstitial fluid in the lower legs which is revealed as pitting edema of the feet, ankles, and even shins, increased central venous pressure giving jugular venous distention, and portal hypertension causing hepatomegaly.

When surgery is contemplated for a patient with a history of congestive heart failure, preoperative steps should be taken to optimize the patient’s physical status. The patient should be questioned about the amount of exertion necessary to produce dyspnea and about how many pillows are necessary while sleeping to prevent orthopnea, in order to quantitate the severity of the cardiac disability. Nocturnal cough and restlessness and easy fatigability can be early symptoms of problems. Signs of congestive failure include jugular venous distention, presence of a third heart sound (gallop rhythm), pulsus alternans, basilar rales, and pitting edema. A chest radiograph and ECG should be used to measure heart size, to visualize the lung fields, and to help detect AF. If poorly compensated failure is detected, the risk of postoperative pulmonary edema is raised by 25%.

Patients prone to failure can be improved by increased attention to sodium and water restriction and to their compliance with medications such as diuretics, digoxin, and preload and afterload reducers. Potassium levels should be normalized. Mild preoperative hypokalemia can be managed by oral replacement therapy or intravenous administration at a rate of up to 10 mEq/h in concentrations up to 30 mEq/L. Patients taking digoxin should have serum levels measured. Signs and symptoms of digoxin toxicity such as nausea, diarrhea, anorexia, and new dysrhythmias should prompt postponement of surgery until levels are normalized. Consideration should be given to placement of a central venous line for monitoring perioperative central venous pressure or for placing a Swan-Ganz catheter. An indwelling arterial line can also be useful for monitoring mean arterial pressure and for obtaining samples for blood gas analysis. After intubation the patient’s lung compliance should be monitored closely, because decreased compliance is an early sign of pulmonary edema. Mini-dose heparin and elastic stockings can be used postoperatively to decrease the likelihood of deep vein thrombosis and pulmonary embolization. Passive leg exercises and early ambulation postoperatively also help prevent these problems. An early postoperative chest radiograph can reveal early signs of pulmonary edema, as does an elevation of pulmonary capillary wedge pressures. During recovery the patient’s physical activity and emotional stress should be kept low to reduce unnecessary demands on the heart.

Cardiac Dysrhythmias

Patients with diagnosed or occult cardiac rhythm disturbances present a management challenge to the surgeon and anesthesiologist in the perioperative period. Dysrhythmias can compromise cardiac output leading to myocardial ischemia, cerebral ischemia, congestive failure, or shock. In addition, dysrhythmias can predispose towards the formation of intracardiac thrombi and subsequent systemic embolization.

Patients with significant dysrhythmias may or may not have symptoms. The tendency of dysrhythmias to compromise cardiac function frequently depends on overall cardiac health. For example, an otherwise healthy individual can easily tolerate heart rates at the extremes of the range of 40 to 180, whereas someone with a diseased heart would be less tolerant.

Anesthesia and surgery are capable of unmasking a tendency toward dysrhythmias through vagal stimulation, stress-related release of catecholamines, drug-induced histamine release, dysrhythmogenic drugs such
as inhalational anesthetics, and hypoxia due to inadequate ventilation. Statistically, perioperative dysrhythmias, particularly during intubation, are most common in patients with preexisting dysrhythmias or heart disease, or who are on digoxin medication or undergo surgery and anesthesia for longer than 3 hours. In addition, surgery near the carotid sinus can cause atrioventricular conduction disturbances due to the stimulation of intercostal nerves.

The presence of significant cardiac dysrhythmias can often be detected based on symptoms reported during a medical history, such as intermittent palpitations, unexplained syncopal episodes, and transient ischemic attacks. Determination of pulse rate and rhythm should be obtained during the physical examination. An ECG should be obtained in all patients with either suspected or diagnosed dysrhythmias.

Atrial Dysrhythmias  The most common dysrhythmia is sinus tachycardia with a heart rate of 100 to 180. Such an elevated rate compromises cardiac output by lessening diastolic filling time and increasing myocardial oxygen consumption. Sinus tachycardia can have many etiologies including fever, hypovolemia, anemia, hypoxia, drug use, and hyperthyroidism. Therapy is directed at the underlying cause.32

Paroxysmal Atrial Tachycardia  Paroxysmal atrial tachycardia (PAT) is a frequent dysrhythmia with an atrial rate of 140 to 240 and a lower ventricular response rate. PAT can be due to digoxin toxicity or myocardial ischemia, but is usually due to reentrant pathways between the atria and ventricles.

The rhythm is unstable, reverting back to sinus in almost all cases. Risk of surgical procedures is not elevated with a history of PAT; however, if there have been frequent or recent episodes of PAT, a β-blocker may help prevent tachycardia. Ablation of reentrant pathways via electrophysiology procedures is the treatment of choice and is usually curative.33

Atrial Flutter  Atrial flutter (rate 250–300) commonly appears with a 2:1 block producing a ventricular rate of 125 to 150. Patients in atrial flutter who undergo surgery have a 50% mortality rate. It is therefore incumbent on the surgeon to identify and seek correction of this dysrhythmia preoperatively, with direct-current low-energy (25 to 50 watt-seconds) cardioversion.

Atrial Fibrillation  Atrial fibrillation is the second most common cardiac dysrhythmia. It is commonly asymptomatic but characteristically produces an irregularly irregular pulse rhythm and a fibrillation pattern on ECG. The atrial rate is greater than 350, whereas the ventricular rate varies from 140 to 180 bpm. Etiologies include any cause of left atrial hypertrophy, thyrotoxicosis, and coronary artery disease, and may result from the excessive use of caffeine, cocaine, ethanol, diet pills, or nicotine, even in healthy hearts.

The physiologic compromise produced by AF depends on the ventricular response, myocardial health, and duration of the dysrhythmia. A rapid ventricular response increases perioperative mortality by about 15%. Congestive heart failure or myocardial ischemia can appear abruptly in susceptible patients going into AF. Long-standing AF can allow the formation of an atrial thrombus and subsequent thromboembolic complications.

Preoperative management of patients with a history of AF should include consideration of digitalization that by itself may convert AF to a normal sinus rhythm. Intravenous verapamil can also be used but is less successful in converting AF. Both digoxin and calcium channel antagonists decrease chronotropy, thus helping to slow the ventricular response rate to more physiologic levels. Amiodarone has been shown to have prophylactic value.34 Care should be taken to not allow the ventricular rate to fall below 70. Acute onset of AF is most effectively managed with direct-current cardioversion starting at about 200 watt-seconds. Patients with chronic AF should be on anticoagulants, which must be adjusted perioperatively.

Premature Ventricular Contractions  Premature ventricular contractions (PVCs) can be due to many causes including fever, hypoxia, drugs (including digoxin, amphetamine, and inhalational anesthetics), pulmonary artery catheters, electrolyte disturbances, and myocardial ischemia, or they may be idiopathic. The significance of PVC activity, including more complex ectopic ventricular disturbances such as nonsustained ventricular tachycardia, is controversial. Long-term mortality is not reduced in PVC patients without apparent heart disease, but PVCs postmyocardial infarction or with cardiomyopathy do carry increased risk. This is more a function of underlying cardiomyopathy rather than the dysrhythmia itself.

The discovery of significant PVC activity on a preoperative ECG warrants a complete cardiac evaluation, and identified causes of PVCs should be corrected preoperatively. Development of PVCs or runs of ventricular tachycardia during surgery may signal cardiac ischemia or electrolyte abnormalities, which should be investigated and corrected.35,36 The cause of PVCs should be sought and corrected, but note that lidocaine is no longer used to suppress ectopic activity.

Ventricular Tachycardia  The appearance of three or more PVCs in a row is defined as ventricular tachycardia. It has a variety of etiologies including hypoxia, acidosis, myocardial ischemia, digoxin toxicity, hyper- or hypokalemia, and hypercalcemia. Prompt therapy consists of intravenous lidocaine or low-energy direct-current cardioversion.37,38

Heart Blocks  Atrioventricular blocks take several forms. A P–R interval greater than 20 ms constitutes a first degree atrioventricular block and is of little significance perioperatively in the absence of
other cardiac abnormalities. In second degree block, some atrial impulses are not conducted into the ventricles. The Mobitz type I (Wenkebach) second degree block has a P–R interval that progressively lengthens until a nonconducted P wave occurs and the cycle begins again. Mobitz type I rhythms are usually due to digoxin excess, myocardial ischemia, or degeneration of cardiac conduction tissue. Treatment with atropine is necessary only for excessively slow ventricular rates. Mobitz type II second degree blocks have a constant P–R interval but frequent P waves without a ventricular response. This is a worrisome dysrhythmia and perioperative ventricular pacing should be considered.39

Third degree atrioventricular blocks imply a complete block of atrial impulses into the ventricle. The ventricles therefore beat at their low intrinsic rate of about 45. Therapy usually requires the use of a pacemaker.

Bundle branch blocks present no direct contraindication to anesthesia and surgery but usually signal some underlying cardiac disease. Pacing for bundle branch blocks is necessary only if symptomatic bradycardia or complete heart block occurs.

Patients who have permanent cardiac pacemakers pose little increased risk during surgery and above the underlying cardiac problem. If electrocautery is necessary special care should be taken to ensure that it is properly grounded. A magnet to convert a demand pacemaker to the fixed rate mode should be available in the operating suite.

Surgery in the Patient with Respiratory Problems

General Assessment of Airway and Lungs

Maxillofacial surgery itself has minimal effect on pulmonary function compared with general thoracic or abdominal surgery, except when tissue is being transferred from the thorax to the maxillofacial region. However, maxillofacial surgery does sometimes involve prolonged general anesthesia, and procedures can compromise the upper airways. Therefore, it is important to discover and treat airway and lung abnormalities preoperatively or, when not possible, make necessary compensations in surgical and anesthetic plans.

The medical history should ascertain the following about the status of the ventilatory system: the presence of symptoms such as wheezing, productive cough, and low exercise tolerance; the use of pulmonary medications; cigarette smoking; prior thoracic surgery or trauma; and previously diagnosed pulmonary diseases including asthma, pneumonia, chronic obstructive pulmonary disease (COPD), or tuberculosis. In physical examination, points of significance to the assessment of the respiratory system include a careful inspection of the nasal airways, auscultation of lung fields for abnormal sounds, inspection of mucosa and nail beds for signs of cyanosis or clubbing, and measurement of the respiratory rate.40,41

A plain chest radiograph is useful for detecting diffuse or localized parenchymal disease, pulmonary edema, hyperinflation, and consolidations such as pneumonia or neoplasms. However, the yield from routine preoperative chest radiographs is low in patients without a history or examination suggestive of pulmonary disease.

Some pulmonary function testing can be performed at bedside, such as the breath-holding test. The breath-holding test involves having a patient make a maximum inspiration and then hold the breath for as long as possible. Inability to hold one’s breath for at least 15 seconds is indicative of significant pulmonary problems. Spirometry is another useful bedside test for assessing pulmonary function although a delay in surgery is usually unwarranted. Surgeons should request formal pulmonary function testing (PFT) for all patients in whom lung disease is suspected. PFTs help gauge respiratory reserve and measure the potential response to measures taken to improve lung function.42,43

Measurement of arterial blood gases (ABGs) is frequently a part of pulmonary function testing. ABG determination serves both as a baseline for intra- and postoperative measurements, and helps assess the status of pulmonary gas exchange. A low partial pressure of oxygen (PaO₂) may be due to hypoventilation, diffusion impairment, shunting, or a ventilation-perfusion inequality, the last being the most common cause. An elevated partial pressure of carbon dioxide (PaCO₂) is a sign of hypoventilation either due to an inadequate respiratory rate or depth, or to a ventilation-perfusion inequality. Intraoperative capnography and intra- and postoperative oximetry have reduced the need for frequent ABG sampling. Oximetry is also beneficial during the first few hours after maxillofacial surgery, when respiratory insufficiency is most likely to occur.44

Asthma

Asthma is characterized by episodes of wheezing, cough, and production of mucous plugs. It is more common in children, although some adults will have new or relapsed asthma later in life. Chronic uncontrolled asthma can lead to COPD, and asthma complicated by cigarette smoking can lead to COPD as well. Questions regarding history of asthma, frequent or nocturnal coughing, shortness of breath, dyspnea on exertion, and production of mucous plugs are helpful in diagnosing asthma. Physical examination may show wheezing, particularly with forced expiration.45,46

Well-controlled asthma does not pose a significant perioperative risk. Patients with well-controlled asthma should have a dose of albuterol by inhaler or nebulization prior to general anesthesia to prevent intraoperative bronchospasm or laryngospasm.47,48

The patient with a recent history of problematic asthma is at significant risk
when having general anesthesia and surgery. The bronchospasm that characterizes asthma can develop precipitously and compromise ventilation, even with positive pressure, and may be difficult to reverse in time to prevent complications. As with most conditions of this nature, recognition and prevention are the best management strategies.

The airway narrowing in asthma is due to smooth muscle contraction, edema in airway walls, or mucous plugging of airways. Whereas bronchospasm is rapidly reversible with muscle relaxants, edema and plugging are not.

The likelihood of an asthmatic episode occurring during surgery can be judged by a few pieces of historic information. The frequency, severity, duration, and response to therapy of recent asthma attacks will help gauge how well an individual’s asthma is controlled and therefore the safety of proceeding with surgical plans.

When questioning a patient with asthma, key factors are the frequency and nature of attacks, current medication use, last use of steroids, and an indication of the severity of asthma. A history of multiple emergency room visits for asthma, hospitalization for asthma, history of mechanical ventilation for asthma, and steroid dependency are indicators of severe asthma (Table 2-5).

For many years aminophylline-like treatment was the mainstay of asthma and COPD treatment. Several medications have replaced aminophylline and theophylline treatment. For acute treatment albuterol by inhaler or nebulized administration is used. The usual dose is 1 to 2 actuations of a metered-dose inhaler or a nebulization treatment every 4 to 6 hours as needed, although hospitalized patients may receive dosing more frequently. In addition, oral or parenteral steroid treatment is used more liberally than in past years. Patients who are wheezing and are to undergo surgical treatment are usually given steroids to reduce wheezing and the chance of anesthesia-induced laryngospasm and bronchospasm. Steroids are then rapidly tapered and discontinued over 3 to 7 days postoperatively.59

Maintenance therapy in asthma has also broadened to include inhaled steroids, long-acting β-agonists, antileukotriene drugs, and theophylline.50–52 Inhaled steroids using metered-dose inhalers or dry-powder inhalation devices are given on a regular dosing schedule and are not absorbed, preventing systemic complications of steroid use.

Prolonged corticosteroid use carries its own risks as is discussed later in this chapter. The surgeon should confer with the physician managing a patient’s asthma to ensure that the patient has recently been evaluated and that the steroid regimen provides the least amount of drug that is still effective. If possible the patient may benefit from a switch to inhaled corticosteroid use through metered-dose inhalers that may help minimize systemic effects.

Intra- and postoperatively asthmatic patients should be monitored for the appearance of increased airway resistance, wheezing, pulsus paradoxus, tachycardia, fever, hypoxemia, hypercapnia, and acidosis. Atelectasis is common in asthmatics and causes an increased risk of bacterial pneumonia, which is why thorough pulmonary examinations must be given at frequent intervals during recovery.59

**Chronic Obstructive Pulmonary Disease**

Chronic obstructive pulmonary disease (COPD) is an all-encompassing term for lung diseases characterized by loss of lung tissue and its surface area. It includes chronic bronchitis, emphysema, and other conditions, but these distinctions are rather vague and do not result in differing management. Alveolar loss from destruction in COPD results in less surface area to exchange gases and in lower smooth muscle tone of the bronchioles. Emphysematous blebs may replace normal lung tissue. Middle- and large-sized bronchi have lost their cilia and muscle tone, and exude excess mucus, causing pooling of secretions and reduced clearance of dust, smoke, and bacteria. Symptoms and signs of COPD include chronic cough, sputum production, shortness of breath, decreased exercise tolerance, wheezing, and increased anteroposterior thoracic diameter. Patients with advanced disease may purse their lips to increase intrathoracic pressure during exhalation, thus holding open airways that would otherwise close prematurely.53

A chest radiograph may show hyperlucency, kyphosis, and depressed and flattened diaphragms. Pulmonary function tests show a reduced forced expiratory volume in the first second of exhalation (FEV1) and a reduced forced vital capacity/FEV1 ratio. FEV1 is compared to age, gender, and racial norms, and an FEV1 of less than 80% of predicted normal is abnormal, with readings of less than 60 indicating severe obstructive disease. Arterial blood gases may show a loss of oxygenation and elevated carbon dioxide, due to reduced gas exchange and an alteration in the usual respiratory drive. As the term implies, bronchospasm in COPD may be less responsive to bronchodilators than in asthma.

Surgery and anesthesia for patients with significant COPD usually brings few intraoperative risks due to the lung disease itself. However, the likelihood of postoperative pulmonary complications is high in COPD patients. Therefore, proper preoperative identification and preparation are important.

Preparing COPD patients for surgery usually involves reversing pathology able to be altered medically. Hydration to

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<th>Questions for Asthma Patients</th>
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<td>Frequency and nature of attacks</td>
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mobilize mucus secretions, inhaled β-agonists by metered-dose inhaler or nebulization, and inhaled ipratropium are used to optimize preoperative therapy. Oral or parenteral steroids are used if wheezing is detected prior to surgery.

Production of mucopurulent sputum may indicate the need for preoperative antibiotics to help improve COPD symptoms. Amoxicillin, trimethoprim/sulfa combinations, or erythromycin are used most commonly and are given in 7- to 10-day courses.54

Cigarette smoking is the most common cause of COPD and further exacerbates symptoms if continued after irreversible lung pathology occurs. Reversible problems that smoking causes include the release of nicotine, production of carbon monoxide, mucus hypersecretion, impaired ciliary function, and impaired local lung immunity. Preoperative cessation of smoking for 24 hours allows a significant decline in plasma carboxyhemoglobin and nicotine levels, but the rate of pulmonary complications due to smoking takes weeks to fall after smoking is stopped. In the case of coronary artery bypass grafting, the percentage of postoperative pulmonary complications in former smokers does not begin to approach the rate seen in nonsmokers until after at least 8 weeks of abstinence from smoking.55

Other preoperative measures that can prevent postoperative problems in patients with COPD include good nutrition and correction of hypokalemia to improve respiratory muscle strength and familiarization of the patient with incentive spirometry. Preoperative teaching in the use of incentive spirometry, cough/deep breathing exercises, and early ambulation help the patient prepare for recovery before the pain and recovery period from anesthesia occur.

There are several anesthetic considerations for patients with COPD. Volatile anesthetics provide bronchodilatory effects and thus are useful. Nitrous oxide, on the other hand, may cause problems due to its accumulation in bullae potentiating rupture and production of pneumothorax. The respiratory depressive effects of narcotics makes their use in COPD patients hazardous, especially if it is likely that their effects will outlast the duration of needed anesthesia.

The techniques of controlled ventilation must be altered in patients with obstructive airway disease. Ventilatory rates need to be slow enough (typically 6 to 10 per minute) to allow sufficient exhalation time and to compensate for slower diffusion of gases across membranes. Care should be taken to avoid high pressures to lessen the potential of ruptured bullae. Generally COPD patients do best with large tidal volumes at slow rates and do not need positive end-expiratory pressure.56

**Surgery in the Patient with Renal and Urinary Tract Disease**

The kidneys play several roles in helping maintain physiologic normalcy; they are therefore important for continuing or regaining homeostasis during and after surgery and anesthesia. The renal system is necessary to support the processes of fluid, electrolyte, and acid-base balance, drug metabolism and elimination, blood pressure control through the renin-angiotensin system, red blood cell production through erythropoietin production, and vitamin D hydroxylation.

There are several diseases that can affect one or more aspects of kidney function. However, for the maxillofacial surgeon a better gauge of the degree to which the patient’s ability to tolerate anesthesia and surgery is compromised is the adequacy of renal function. The glomerular filtration rate (GFR), normally 100 to 125 mL/min per 1.73 m² of body surface area in an adult, is the single most useful measure of renal health. The GFR is measured clinically by determining the clearance of endogenous creatinine. The body’s serum creatinine (SC) load is highly dependent on muscle mass, and the clearance of creatinine from the serum depends on the number of functioning glomeruli, a number that gradually decreases with age. Also, SC varies inversely with creatinine clearance (CCR). Thus, an estimation of the CCR in males involves obtaining the level of SC and then multiplying its reciprocal by factors that are correct for muscle mass and age.

\[
CCR = \frac{(140 - \text{Age in yr}) \times \text{Weight in kg}}{(\text{SC}) \times 1.73 \times \text{Height in cm}}
\]

For females, the above result is multiplied by 0.85. Although much less accurate, measurement of SC (normal is < 1.5 mg/dL) can be used to help gauge renal function. Although measurement of blood urea nitrogen is used commonly to test renal health, it is a crude measure and may be misleading, especially in patients with poor nutrition or who have been bleeding into the intestinal tract.

Serum electrolyte abnormalities can signal significant renal disease. Poor renal function will often result in decreased secretion of potassium causing hyperkalemia or a concentrating defect leading to urinary sodium wasting and hydrogen ion retention with resultant hyperchloremic metabolic acidosis. Other indications of renal problems include proteinuria, pyuria, and hematuria, all detectable on routine urinalysis.

**Chronic Renal Insufficiency**

The risks of anesthesia and surgery in the patient with known renal insufficiency vary according to the severity of renal compromise. Patients with mild to moderate renal insufficiency (GFR of 25–50 mL/min) usually tolerate the perioperative period well if properly managed. When renal function is severely impaired (GFR of 10–25 mL/min) or frank failure is present (GFR < 10 mL/min), complications of renal origin are much more likely. Patients with severe renal insufficiency have a 60% increase in perioperative morbidity and a 2 to 4% increased mortality compared to healthy patients.57

Extrarenal problems can be produced by renal insufficiency. Normochromic or
normocytic anemia frequently occurs due to several factors, including decreased erythropoietin, decreased red cell survival time, and bone marrow depression. In addition, uremia can also cause decreased platelet aggregating ability and depressed platelet factor 3 release.\(^5\)

Pericardial inflammation or effusion is commonly associated with chronic uremia or hemodialysis, as is myocardial dysfunction. End-stage renal disease is almost always complicated by systemic hypertension. Patients with renal insufficiency have impairment of their immune systems with heightened susceptibility to bacterial, viral, and fungal infections. The cause seems to be faulty neutrophil and lymphocyte production and function. Many of the other problems caused by renal dysfunction affect the gastrointestinal tract. Symptoms of nausea, vomiting, diarrhea, and anorexia frequently accompany uremia. Acute stomatitis and salivary adenitis can occur, as can pancreatitis. The stomach and intestine linings may undergo inflammatory changes. Hepatitis C is present in about 19% of dialysis patients.\(^5\)

Excessive water retention is most easily managed by fluid restriction, which usually helps improve the hypo-osmolar state, and sodium and hydrogen ion balance. Hyperkalemia before elective surgery can be managed with dietary potassium restriction and potassium-wasting diuretics. More acute potassium control may necessitate the use of cation-exchange resins, strategies to drive potassium intracellularly, or dialysis. Hypertension and fluid retention may necessitate diuretic use preoperatively. In cases of renal failure, hemodialysis is recommended to reverse fluid, electrolyte, and acid-base problems, as well as extrarenal disorders such as uremic immunodepression. Dialysis should be performed no more than 24 hours preoperatively. Platelet counts are helpful to identify heparin-induced thrombocytopenia.\(^6\) The lower heparin requirements in newer dialysis techniques prevent many of the residual anticoagulation problems of the past. However, surgeons should remember the capability of heparin to induce thrombocytopenia. Preoperative chest radiographs and an ECG can be used to detect myocardial dysfunction or peri- cardial problems due to uremia or chronic fluid overload. Plans should include the use of prophylactic antibiotics even for minimally invasive procedures.\(^6\)

Intraoperative management of the patient with severe renal insufficiency should include careful cardiac monitoring for dysrhythmias and fluid overload. Intravenous fluids should be administered in quantities only sufficient to replace insensible fluid and blood losses, and be free of potassium. If a hemodialysis vascular access (shunt) is in place, it should be protected from trauma. Intraoperative hemostasis should be especially meticulous if the patient will be dialyzed immediately after surgery.\(^6\)

After surgery, steps should be taken to maintain proper fluid and electrolyte balance, particularly until dialysis can be done. Most surgeons delay postoperative hemodialysis for at least 2 to 3 days to lessen the chance of wound bleeding during heparinization. However, patients with oral or nasal procedures commonly swallow a significant amount of blood, which increases the blood’s nitrogen load and may prompt earlier dialysis than would otherwise be necessary. Extended nasogastric suctioning may help prevent blood swallowing when the likelihood of swallowing large amounts is high.

A significant problem that the anesthesiologist and surgeon face when managing a patient with renal insufficiency is drug elimination and the toxic effects of some drugs on the kidney. Drugs commonly used during maxillofacial surgery that need to be avoided or used with care in the patient with renal compromise include cephalosporins, penicillin, and sulfa antibiotics, nonsteroidal anti-inflammatory drugs, nondepolarizing muscle relaxants, and enflurane. Many references are available that list drugs and dosing modifications needed in renal failure patients.

**Hypertension**

Essential hypertension is one of the most common disorders of adults, so it is not surprising that a large percentage of adult patients who require surgery have hypertension. With more people aware of the hazards of untreated hypertension, many patients seeking the type of care offered by specialty surgeons have had their hypertensive status evaluated and a management regimen prescribed.

Two basic problems can arise in the hypertensive patient requiring anesthesia and surgery. The first is that untreated chronic hypertension can damage many organ systems, particularly the heart, kidneys, and brain. The damaged organs may be less able to tolerate demands placed on them during the perioperative period. The second problem is that for many hypertensive patients, the medications prescribed for controlling hypertension may dull some of the natural responses the body uses to counteract anesthetic and surgical challenges.\(^6\)

Statistically there is no increase in the incidence of adverse effects from untreated hypertension as long as the diastolic pressure is less than 110 mm Hg and no concurrent medical problems exist. When conferring with a patient the surgeon can usually gain an idea of the likelihood of hypertensive organ damage by attempting to learn of the patient’s compliance with antihypertensive regimens. The patient’s physician can often supply this information. Target organ damage can also be detected by various physical and laboratory examinations. Cardiac damage usually manifests initially with left ventricular hypertrophy (LVH). This causes a prolonged and displaced point of maximal impact of the heart apex on palpation. In addition LVH shows on ECG, chest radiographs, and echocardiograms. With time,
signs and symptoms of congestive heart failure arise predisposing the heart to dysrhythmias, ischemia, and the appearance of pulmonary edema.⁶⁷,⁶⁸

The renal damage caused by chronic high blood pressure usually consists of nephrosclerosis. This may be detectable by routine urinalysis, on which proteinuria, hematuria, or pyuria is seen. Renal damage may also cause serum creatinine levels to rise.

Cerebral damage due to hypertension usually manifests later in life with an increased incidence of stroke. In addition the cerebral vascular system’s ability to autoregulate is impaired so that a greater perfusion pressure must be maintained than would otherwise be necessary. Some clinicians also believe chronic hypertension promotes the progress of carotid atherosclerosis and therefore recommend that the surgeon auscultate for carotid bruits.

Many of the vascular changes that occur because of chronic hypertension can easily be seen in the one site where the small vessels are visible; that is, the fundus of the eye. Hemorrhages and exudates seen on fundoscopic examination typically indicate similar changes in other vascular beds.⁶⁹

There is a variety of treatment options available for hypertensive patients, including diuretics, ACE inhibitors, angiotensin receptor blockers (ARBs), β-blockers, calcium channel blockers, selective α₁-blockers, and central α-blockers. The surgeon should be familiar with these drugs and their side effects and risks in surgery.⁷⁰–⁷²

Diuretics can cause hypokalemia and hyponatremia, necessitating screening of electrolytes prior to surgery. ACE inhibitors and, less likely, ARBs can cause hyperkalemia and decreased renal perfusion. β-Blockers reduce heart rate and contractility, although beneficial effects of decreased myocardial demand and preservation of normal sinus rhythm generally outweigh perioperative risks of use. Calcium channel blockers may cause bradycardia but are usually well tolerated. Selective α-blockers may cause first-dose hypotension, but are also usually well tolerated. Central α-blockers may cause drowsiness, depression, and dry mouth.⁷³,⁷⁴

For the patient with poorly controlled hypertension (systolic pressure over 200 mm Hg, diastolic pressure over 110 mm Hg), the surgeon should defer elective surgery until better control is obtained and any end-organ damage is detected; appropriate compensations should be made in the treatment plan. Acute treatment of hypertension can include clonidine given in 0.1 mg increments, or intravenous antihypertensives such as enalaprilat, labetalol, or nicardipine infusion. Sublingual nifedipine should not be used.

Patients whose blood pressure is well controlled preoperatively usually exhibit large swings in their blood pressure during and after surgery. Hypotension usually responds to fluid administration. Hypertension can usually be tolerated if it does not reach severe levels. Excessive increases in blood pressure can be managed with short courses of additional antihypertensive medications until anesthetic drugs or surgery-related stresses have stopped, allowing patients to return to their preoperative status.⁷⁵

Surgery in the Patient with Endocrine Disorders

Diabetes Mellitus

The impact of diabetes mellitus on the anesthetic and surgical management of a patient is highly dependent on the type, severity, and degree of control of the diabetes. Type 1 (insulin-dependent) diabetes mellitus is due to impaired production by or an insufficient mass of pancreatic islet β-cells. Type 2 (non-insulin-dependent) diabetes mellitus occurs due to an altered number and affinity of peripheral insulin receptors. Total insulin production may also be depressed but might be elevated.⁷⁶

The usual daily production of insulin by a lean adult is 33 U; approximately 3 to 5 U are needed for each meal while the basal insulin requirement is about 1 U/h. The ketosis-prone diabetic patient produces less than 10% of the average daily insulin requirement, but the typical type 2 diabetic patient produces an average of 15 U/24 h.

Type 1 diabetes presents the more significant challenge to the well-being of a surgical patient. Patients are usually lean and have had this disease since their youth. Those with long-standing type 1 diabetes cannot go without their insulin for more than 48 hours without diabetic ketoacidosis (DKA) occurring. Hormones that increase during periods of physiologic stress, including cortisol, catecholamines, and glucagon, act to counter the effects of insulin, producing a stress-induced glucose intolerance, even in many healthy nondiabetic patients. This is why type 1 patients who depend on exogenous administration of their insulin commonly have increased insulin requirements from preoperative emotional stress, intraoperative anesthetic stress, and postoperative wound, physiologic, and emotional stress. Studies have shown that elevated blood glucose not only impairs wound healing, but can also depress leukocyte and pancreatic β-cell function. These are reasons, in addition to prevention of DKA, for appropriate insulin supplementation during and after surgery.⁴³,⁷⁷

Type 1 patients, in contrast to type 2 diabetics, have a high rate of systemic problems. Peripheral neuropathies are common, predisposing these individuals to chronic lower leg and foot lesions, which should be detected and noted preoperatively and prevented perioperatively. Long-standing diabetics are also at increased risk for coronary artery disease and may suffer silent (painless) ischemic episodes due to myocardial ischemia.⁷⁸ Insulin-dependent diabetics, particularly those with poor control, handle infections poorly. Therefore, vigilance should be
especially high for breaks in aseptic techniques and consideration given to the use of prophylactic antibiotics. Type 1 patients also have enhanced platelet stickiness that may promote unwanted clotting in surgical flaps. The formation of glycosylated hemoglobin AIC interferes with oxygen release into tissues.79

A rational approach to management of diabetes assists in maintaining glycemic control perioperatively. Care should be given to avoid hypoglycemia at any time during surgery, and to prevent severe hyperglycemia as well. The general range of adequate control is between 120 and 200 mg/dL. This would involve decreasing the usual morning insulin by one-half to allow plasma glucose to rise during the surgery, but providing enough basal insulin to prevent DKA.80,81

If a patient is to have relatively short-duration ambulatory surgery and is required to consume nothing by mouth the morning of surgery, only half the usual morning dose of insulin should be given at the time when intravenous access is gained. Surgery should be early in the morning and intravenous glucose should be given intraoperatively. During surgery the clinician should watch for signs of hypoglycemia such as tachycardia and diaphoresis. The patient should then be encouraged to consume some calorie source by mouth within 3 hours after surgery is completed. Portable glucose monitoring is useful for intra- and postoperative serum glucose monitoring.82

Patients requiring more major surgery and longer duration general anesthesia are usually best managed in a setting in which an anesthesiologist can monitor blood glucose levels in the operating room and administer insulin on an as-needed basis. The morning insulin should be withheld until intravenous glucose is available; then one-half to three-quarters of the usual dose can be administered and supplemented intraoperatively by the anesthesiologist.83,84

When patients are unlikely to enteraly receive their usual caloric supply postoperatively, their insulin should be given based on periodic (every 6 h) plasma glucose sampling. Insulin doses should be gauged to keep the plasma glucose at 150 to 250 mg/dL until normal dietary habits and activity levels return. The patient’s primary care physician can help guide dietary decisions.

Type 2 patients usually have fewer systemic abnormalities due to diabetes and are less likely to suffer perioperative complications. But when major surgery and general anesthesia are performed, these patients usually become hyperglycemic. Not uncommonly patients who are well managed on diet and oral hypoglycemics will need temporary insulin supplementation in the intra- and postoperative periods. As in type 1 patients, blood glucose should be kept at 150 to 250 mg/dL, with insulin supplementation based on periodic sampling.85

**Thyroid Disorders**

The need for normal levels of thyroid hormones to maintain the function of many of the body’s physiologic functions makes proper thyroid gland function important to the surgeon. The gland is composed of follicles, each of which is a lumen filled with thyroglobulin, which is produced by a single layer of epithelial cells lining the follicle. Thyroid hormones, thyroxine (T3) and triiodothyronine (T4), are produced and stored in the gland in a ratio of 10 to 15:1 (T3:T4) and are released on stimulation by thyroid-stimulating hormone, an anterior pituitary hormone. Between the follicles parafollicular cells exist which secrete calcitonin, whose function is to help lower serum calcium by blocking its release from bone.

The majority of T3 and T4 released from the gland are bound to various carrier proteins. Most circulating T3 is produced by conversion from T4 in the liver and kidney. T3 is much more potent than T4, but only the unbound form of either hormone is active, and in the case of T3 an inactive form called reverse T3 (rT3) can be formed. In normal states 35% of T4 is converted to T3 and 40% to rT3. However, in times of physical illness or emotional stress, or if certain drugs (such as corticosteroids) are used, a higher percentage of T3 conversion to rT3 can occur.

The most common laboratory tests of thyroid function are (1) measurements of total thyroid hormone (T) levels by radioimmunoassay (normal is 5,012 pg/dL), in which high values indicate hyperthyroidism and low values indicate hypothyroidism; and (2) T3 resin uptake, in which unoccupied thyroid hormone binding sites on thyroid-binding globulin are measured. High values of T3 resin uptake are associated with hypothyroidism, whereas low values are consistent with hyperthyroidism.86

**Hyperthyroidism** Symptoms of hyperthyroidism include weight loss, palpitations, and restlessness. Exophthalmos occurs in more severe cases owing to increased amounts of retro-orbital fat. Once diagnosed, therapy usually begins with antithyroid drugs such as propylthiouracil or methimazole. β-adrenergic antagonists can be used to control symptoms until thyroid hormone levels decrease. Autoimmune thyrotoxicosis can be allowed time to resolve spontaneously, or treatment with radioactive iodine can ablate the gland. Total thyroidectomy is seldom indicated, except for adenomas or malignancy.87,88

Surgery in the face of hyperthyroidism carries high risks of cardiac dysrhythmias or failure, and the potential for causing a thyroid crisis. Therefore, elective surgery should be deferred until thyroid hormone levels are properly managed. If emergency surgery is necessary on a patient with poorly controlled hyperthyroidism, β-sympathetic antagonists can be used to help control the effects of thyroid
hormones on the heart while intravenous sodium iodide (1 g) can be administered to help block hormone release from the thyroid gland. The β-antagonist should be continued postoperatively until the administered antihyperthyroid drugs have taken effect. Palpation of the thyroid gland should be gentle in patients with known hyperthyroidism to avoid increasing hormone release, and infections should be aggressively managed because they too may precipitate a thyroid crisis.89–91

Hypothyroidism The hypothyroid patient presents a lesser surgical and anesthetic risk when compared with the hyperthyroid patient. The insufficiency of thyroid hormones causes cardiac depression, respiratory depression with weakening of the muscles of respiration, hyponatremia, constipation, neurologic problems with memory loss and depression, and several other metabolic problems. Signs of hypothyroidism include weight gain, periorbital edema, bradycardia, slowed deep tendon reflexes, generalized muscle weakness, and hair loss.

The potential surgical problems in a patient with untreated hypothyroidism include intra- or postoperative heart failure, hypotension, ileus, mental confusion, and delayed wound healing. Therefore, thyroid replacement therapy is advisable prior to elective surgery. In an emergency the surgeon must remain alert to potential problems due to the hypothyroidism and compensate for them if they occur.92

Adrenal Gland Disorders

The adrenal gland, responsible for the production of a variety of hormones including cortisol, aldosterone, and androgens, plays a central role in regulating many metabolic processes. The gland usually comes to the attention of surgeons because of abnormalities in cortisol production. The average daily secretion of cortisol in the adult is 15 to 17 mg (range 8–28 mg). Secretion follows a diurnal pattern, peaking at about 3:00 or 4:00 am, and falling to low levels at about 8:00 or 9:00 pm. Release of cortisol is regulated by adrenocorticotropic hormone (ACTH) secreted by the pituitary, with ACTH release normally increased in time of physiologic stress. It is not unusual for plasma cortisol levels to remain elevated for up to 19 days after major surgery.

Excessive release of cortisol from the adrenal cortex (Cushing’s disease) is rare. These patients show truncal obesity, hypertension, thin skin that heals poorly, and glucose intolerance. These problems can also be seen in patients on long-term therapeutic corticosteroids for problems such as inflammatory joint or bowel disease. Increased surgical risks faced by patients with hypercortisolism include delayed wound healing and a tendency for infections. Delay of elective surgery is warranted until excessive cortisol levels are under control. If surgery cannot wait, techniques designed to compensate for poor wound healing such as better vascularized flaps and the use of prophylactic antibiotics will be helpful.

Adrenal insufficiency is more commonly seen due to exogenous therapeutic steroid administration than to primary adrenal glandular disease. Exogenous corticosteroids will inhibit ACTH release. Current concepts of steroid supplementation for surgery hold that brief periods of steroid use, low-dose steroid use, and alternate-day steroid use do not suppress the hypothalamic-pituitary axis. Thus, if steroids have been used for less than 3 consecutive weeks within the past year, the dose of chronic steroids is 5 mg of prednisone or less, or if alternate-day steroid administration is used, no supplemental (stress-dose) steroids are needed.93 Once adrenal suppression has occurred, a patient is at great risk for problems during major surgery due to their inability to mount a significant cortisol response to the stress. This may precipitate an adrenal crisis, signaled by the onset of lethargy, tachycardia, flank or abdominal pain, vomiting, fever, restlessness, delirium, hypotension, or coma. Because mineralocorticoid production is not controlled by ACTH, its levels remain normal.

Prevention of problems remains the focus of management of patients prone to adrenal insufficiency. For those patients requiring higher doses of steroids, it is prudent to use stress-dose steroids perioperatively. A typical dose is hydrocortisone 100 mg intravenously on call to the operating room, followed by 50 mg every 8 hours for 48 hours postoperatively. The usual dose of oral steroids or its equivalent intravenous dose can then be resumed. Note that more minor procedures usually do not require steroid supplementation.94,95

Surgery in the Patient with Hepatogastrointestinal Disorders

Liver Disease

Surgeons are well aware of the liver’s vital roles in processing nutrients, synthesizing protein, and metabolizing drugs. Fortunately the liver has a tremendous reserve capacity for maintaining function in the face of even severe hepatic pathology.

Protein synthesis is one of the principal liver activities. Of proteins produced, the ones of particular concern to surgeons and anesthesiologists are albumin and several of the clotting factors. Hepatic production of albumin is in the range of 10 to 15 g daily. Albumin helps maintain the oncotic force necessary to restrict excessive loss of intravascular fluid into the interstitium. Albumin also has a large number of reactive sites and can therefore reversibly bind to most drugs. If albumin production slows sufficiently that serum levels fall below 2.5 g/dL, then edema, ascites, and an elevation in the free-to-bound ratio of administered drugs can result.

The vitamin K–dependent coagulation factors II, VII, IX, and X are made in the liver. A significant fall in their levels can be seen with either severe hepatocellular dis-
ease or with impaired vitamin K absorption due to biliary problems.

The liver is responsible for the proper function of several enzyme systems that help to limit drug actions. Plasma cholinesterase is produced by the liver; by breaking ester linkages it inactivates drugs such as succinylcholine and ester-type local anesthetics. The hepatic microsomal enzyme system converts lipid soluble drugs into more water soluble ones that can be excreted by the kidney. Agents such as some benzodiazipines, lidocaine, meperidine, morphine, and alfentanil depend on this system for elimination.

The most common insults to the liver that affect the performance of maxillofacial surgery are ethanol and infectious hepatitis. In the first case many liver functions can be compromised, whereas in the second case, not only is proper liver function jeopardized, the surgeon must also help prevent the spread of the infection to others.96

Other important consequences of liver disease include impaired glycogen storage and gluconeogenesis; hypersplenism due to obstructed portal blood flow, causing thrombocytopenia; and poor handling of large gastrointestinal nitrogen loads such as swallowed blood, which alters the level of consciousness in patients with severe liver dysfunction.

Significant liver problems cause a large number of signs and symptoms so that detection is usually straightforward. Laboratory tests of liver function tend to be nonspecific indicators of tissue damage but are commonly used to evaluate patients with suspected liver disease. Serum aspartate transaminase levels rise because of damage to either liver, heart, kidney, or skeletal muscles. Changes in serum alanine aminotransferase (ALT) levels, on the other hand, are more specific for hepatocellular disease. Lactate dehydrogenase is commonly measured but is another nonspecific indicator of tissue damage, although its isoenzyme-5 fraction is believed to be more specific for liver damage. Elevations in serum alkaline phosphatase indicate obstructed bile ducts. Measurement of serum albumin helps gauge the severity of liver disease, with levels of less than 2.5 g/dL being significant; however, malnutrition can also cause hypoalbuminemia. Severe liver disease is indicated by a prolonged prothrombin time (PT) and a decreased platelet count. Suspicion of an infectious cause of hepatic disease mandates the use of immunologic tests for signals of viral disease. Hepatitis A, typically due to fecal contamination of food and water, is evidenced by hepatitis A antibodies. Acute hepatitis B, transmitted parenterally or venereally, will stimulate production of surface and core antigen antibodies; the chronic form is revealed by the presence of only surface antigen antibodies. Non-A, non-B hepatitis, caused by several different viruses and usually transmitted by infected blood products, causes elevated ALT but no hepatitis A or B antibodies. Finally, hepatitis C (δ-agent), seen most commonly in illicit drug users and multiply transfused patients, causes the appearance of δ-agent antibodies and in its acute form coexists with hepatitis B.97–99

Maxillofacial surgery in the patient with mild to moderate liver disease usually presents few problems because of hepatic reserve. Borderline severe cases require special perioperative attention to prevent complications or a deterioration of liver function. Liver function tests, especially serum ALAT measurement, are useful. A PT and platelet count are necessary to detect a potential coagulopathy. Intravenous vitamin K (5 to 10 mg over 3 to 5 min) can be administered if a deficiency is suspected and will shorten an abnormal PT in 4 to 12 hours. Fresh frozen plasma can be used temporarily to make up for a vitamin K deficiency until the parenterally administered vitamin is effective.

Because patients with severe liver disease have problems with improper gluconeogenesis, the surgeon should closely monitor serum glucose levels. Patients likely to handle nitrogen poorly, particularly those with a history of hepatic encephalopathy, should be placed on dietary protein restriction. If it is likely that blood will be swallowed, the patient may need measures to reduce nitrogen absorption in the intestines, such as administration of nonabsorbable antibiotics or the use of a cathartic such as lactulose; consciousness should be closely monitored.

Drugs used for anesthesia and analgesia may need to be modified in the patient with hepatic disease. Drugs to avoid in patients with severe liver disease include all nonsteroidal anti-inflammatory drugs, tetracyclines, pentazocine, and atenolol. Drugs for which dosages need to be reduced include diazepam, chlordiazepoxide, meperidine, morphine, propoxyphene, theophylline, lidocaine, verapamil, and most β-sympathetic antagonists. Most anesthetics are generally safe to use in patients with hepatic disease, although some feel halothane, fentanyl, and nitrous oxide should be avoided because of their potential for causing liver toxicity.

**Peptic Ulcer Disease**

Peptic ulcers and gastritis are two of the most common afflictions of adults, but they are usually easily controlled with H2 receptor antagonists, which reduce acid secretion, or sucralfate that forms a protective coat over lesions shielding them from the effects of pepsin and acid. Although many patients still use antacids, side effects such as diarrhea (in magnesium-based antacids), constipation (in aluminum-based antacids), and sodium overload make them less desirable.

Signs of active gastrointestinal bleeding include unexplained anemia and a guaiac-positive stools, but the process is usually diagnosed based on the presence of epigastric pain temporarily relieved by food or antacids. Endoscopy is used to confirm clinical suspicions.
Before maxillofacial surgery can be performed in patients with a history of gastritis or peptic ulcer disease or predisposed to these problems due to prolonged physiologic stress, the surgeon must ensure that the patient’s gastrointestinal problem is being addressed properly. The clinician should verify that the patient is compliant with either their H2 receptor antagonist regimen (cimetidine, 800 mg hs; ranitidine, 150 mg bid; or famotidine, 40 mg hs) or with sucralfate (1 g qid). When the patient is unable to take oral medication, cimetidine (300 mg q8h), ranitidine (50 mg q8h), or famotidine (20 mg q12h) can be given intravenously or intra-muscularly.

Patients with a predisposition to gastritis or peptic ulcer disease should not be given non-steroidal anti-inflammatory drugs (NSAIDs). The use of corticosteroids in these patients is controversial. There is no strong scientific evidence that corticosteroids can cause peptic ulcers in most patients, but many clinicians avoid their use in these patients.

**Surgery in the Patient with Disorders of Connective Tissue and Joints**

**Rheumatoid Arthritis**

Rheumatoid arthritis (RA) is a chronic disease causing not only polyarthritis but also problems in serosal surfaces, blood vessels, muscle, skin, and bone marrow. Maxillofacial surgery in patients with RA requires careful evaluation to discover the extent of the patient’s abnormalities and to attempt to have those problems under reasonable control. Classic signs and symptoms of RA include morning stiffness of involved joints, symmetric involvement of proximal hand joints, subcutaneous (rheumatoid) nodules over bony prominences or extensor surfaces, elevated serum rheumatoid factor, and marked bony erosions visible on radiographs.

Nonarticular problems seen with RA include pericarditis, pleuritis, pneumonitis, myopathies, vasculitis, bone marrow depression, and skin ulcers.

Rheumatoid arthritis patients are treated with five classes of drugs: analgesics (NSAIDs), glucocorticoids, slow-acting antirheumatic drugs (SAARDs), or disease-modifying antirheumatic drugs (DMARDs), and anticytokines. Analgesics include acetaminophen, tramadol, and narcotics. NSAIDs range from over-the-counter ibuprofen to newer selective cyclooxygenase-2 (COX-2) inhibitors such as celecoxib, rofecoxib, and valdecoxib. NSAIDs relieve pain and reduce inflammation but do not alter the course of rheumatoid arthritis. COX-2 inhibitors do not have any inherent benefit over older NSAIDs other than less gastrointestinal toxicity. Glucocorticoids effectively suppress inflammation, often at low doses, but carry their own substantial risks. SAARDs and DMARDs include hydroxychloroquine, sulfasalazine, methotrexate, and leflunomide. Methotrexate is now considered to be first-line treatment for active rheumatoid arthritis. Penicillamine, azathioprine, cyclosporine, and gold salts are seldom used. Anticytokines include etanercept, infliximab, adalimumab, and anakinra. These drug classes are often used in combination to control inflammation and slow the progression of the disease.100–103

Patients with RA who require endotracheal intubation should be evaluated preoperatively for their ability to extend at the neck, open their mandible, and move their cricoarytenoid joints.

An early symptom of neck involvement in RA is neck pain with radiation to the occiput. Preoperative cervical spine films should be considered to evaluate for subluxation of the cervical spine.104 The surgeon needs to remain more vigilant than usual to prevent long periods of overextension or flexion of involved joints. Patients with Raynaud’s phenomenon need their fingers and toes kept warm intraoperatively. Patients with Sjögren’s syndrome will require special care to prevent eye desiccation. The skin of RA patients is commonly thin and easily damaged, so additional padding of pressure points is indicated. Preoperative PT and partial thromboplastin time (PTT) measurement will help detect circulating anticoagulants due to the RA. Early postoperative ambulation, heat treatments, and possibly physical therapy of affected joints will help prevent prolonged stiffness.

**Other Connective Tissue Disorders**

The patient coming to surgery may have other connective tissue disorders such as systemic lupus erythematosus (SLE), psoriatic arthritis, ankylosing spondylitis, dermatomyositis, and scleroderma, which have similar perioperative concerns.

Preoperative assessment of patients with SLE and other connective tissue disorders should include a thorough history and physical examination, a urinalysis, electrolyte panel including blood urea nitrogen and creatinine, a complete blood count, and a PT and PTT. Blood typing or screening should be done in advance of surgery to evaluate for blood compatibility. A chest radiograph and ECG are indicated for evidence of pleural or pericardial disease.105

Patients who have taken glucocorticoid therapy should be screened for use of stress-dose steroids, as noted above. Consider stopping NSAID therapy, if possible, to allow return of platelet function. The time needed for this varies from 7 to 10 days for aspirin to 1 day for ibuprofen. Generally NSAIDs other than aspirin should be stopped 3 to 4 days preoperatively, and acetaminophen or narcotics can be used to control pain during this time. There is no evidence that stopping SAARDs or DMARDs prior to surgery conveys any benefit. Anticytokines can limit immune response in severe infections, and in maxillofacial surgery these drugs should be discontinued.
1 week before surgery and resumed 2 weeks postoperatively.

Sjogren’s syndrome patients should have artificial tears or lubricating gel placed in the eyes during anesthesia. Pilocarpine, if used, should be held to avoid confusion over anesthetic complications of bronchospasm, bradycardia, and tremor.

Patients with ankylosing spondylitis have similar spine concerns as RA patients. Scleroderma patients may have limited mandibular movement as a consequence of their disease, causing difficulty with endotracheal intubation. SLE patients may have low platelets, which is generally well tolerated without excessive bleeding. For counts less than 50,000, intravenous immunoglobulin may be used to improve the platelet count. SLE patients may also have evidence of the lupus anticoagulant, manifest by an elevated PTT. The lupus anticoagulant, also referred to as antiphospholipid antibodies, can produce thromboembolism. Patients may be treated with aspirin if antibodies are present and there have been no previous thromboembolic events, or may be fully anticoagulated, requiring adjustment perioperatively.

**Surgery in the Patient with Neurologic and Neuromuscular Disorders**

**Seizure Disorders**

Seizures are typically recurrent transient paroxysms of hyperactive brain function, which can appear as impaired consciousness, involuntary movement, autonomic disturbance, or psychic experiences. They can result from known causes such as fever, ethanol withdrawal, hypoglycemia, hypoxia, or brain damage, or be idiopathic. Most investigators feel the fundamental site of pathology is in the cerebral cortex, which can be detected on an electroencephalogram (EEG).

The reconstructive maxillofacial surgeon is likely to encounter patients who suffer seizures secondary to head trauma (Chronic recurrent seizures occur in 30% of patients with cerebral hematomas, 15% of those with depressed skull fractures, and 5% of patients hospitalized with closed head injuries). Chronic posthead trauma seizures usually do not occur until 6 to 12 months from the time of injury.

Patients providing a past history of any form of seizure disorder (except perhaps febrile seizures in childhood) should be under the care of or evaluated by a neurologist before undergoing major elective surgery. Patients with well-documented seizures and who are under good control can safely have general anesthesia and surgery. Control is usually obtained by the use of antiseizure medications such as dilantin, phenobarbital, valproic acid, carbamazepine, ethosuximide, and clonazepate. Most of these drugs can cause sedation, which can be additive with anesthetic drugs. Side effects of carbamazepine and dilantin include nausea, dizziness, diplopia, and rarely bone marrow depression. Valproic acid can inhibit liver enzymes, potentially causing oversedation with barbiturates.

Newer drugs include lamotrigine, gabapentin, tiagibine, and topiramate. Most of these drugs can cause sedation, which can be additive with anesthetic drugs. Other side effects vary with each drug.

When evaluating a patient with a seizure disorder for surgery, the clinician should learn of the frequency, type, duration, and sequela of seizures to gauge the degree to which control of the seizures has been obtained. Serum drug levels of these agents can be obtained to help check compliance and predict the appearance of seizures, if subtherapeutic, or possible toxic reactions.

**Cerebrovascular Disease**

Patients with a history of cerebrovascular accidents, such as transient ischemic attacks (TIAs) or strokes, requiring maxillofacial surgery need evaluation by their primary physician before surgery. In most cases little can be done preoperatively to diminish the risk of a stroke during surgery. A careful neurologic examination should be performed preoperatively to document residual damage, and again postoperatively to detect evidence of intraoperative problems.

Two situations in which preoperative improvement may be possible are in the patient with either poorly controlled hypertension or severe carotid stenosis. Essential hypertension is a known risk factor for the development of a stroke; therefore, institution of successful antihypertensive therapy before elective surgery is recommended. The preoperative management of patients with carotid lesions is controversial. Part of the problem is that the finding of a carotid bruit by itself does not correlate with the degree or even presence of carotid stenosis. Thus, angiography is necessary if stenosis is suspected, to document the severity of the process. The question is whether to perform a carotid endarterectomy only if a TIA occurs or if carotid artery occlusion is greater than 70%.

Patients with a history of stroke or TIA frequently harbor coronary artery disease as well. A thorough assessment of the risk for coronary disease is indicated, as noted in the above section.

Patients with a history of cerebrovascular disease are often placed on inhibitors of platelet aggregation such as aspirin or dipyridamole. Most physicians will permit these drugs to be stopped at least 1 week preoperatively to prevent bleeding problems perioperatively. Stroke patients may also have trouble clearing secretions or controlling saliva.

**Malignant Hyperthermia**

Malignant hyperthermia is the leading cause of unexpected anesthetic deaths in North America. It is a rare genetic disorder that manifests following treatment with anesthetic agents, most commonly succinylcholine and halothane. The onset of malignant hyperthermia is usually within.
A predisposition to malignant hyperthermia should be suspected in patients with the following characteristics:

- Unusual muscle hypertrophy
- Ptosis, ophthalmoplegia, strabismus
- Pectus deformities or kyphoscoliosis
- Limb girdle weakness
- Hip dislocation, dislocated patella, malaligned feet
- Known central core myopathy
- Young males with previously described appearance
- Any history of myopathy of unknown etiology

Patients with a known or suspected tendency should be considered for local or regional anesthetic techniques. If general anesthesia is necessary a technique that uses nitrous oxide, barbiturates, benzodiazepines, narcotics, or neuroleptic drugs is advisable. Nondepolarizing muscle relaxants should be used if necessary. Drugs such as succinylcholine, amide local anesthetics, ketamine, and volatile anesthetics should be avoided. Premedication with dantrolene (1 mg/kg) orally the day before surgery or as an intravenous bolus the day of surgery is appropriate when malignant hyperthermia is a high probability. In addition a set protocol for its management, should it occur, should be in place before starting anesthesia for patients at risk for malignant hyperthermia.\textsuperscript{111,112}

\textbf{Spinal Cord Disorders}

Paraplegia due to spinal cord damage can cause a number of problems of which the surgeon needs to be cognizant. Abnormal bladder emptying predisposes these patients to urinary tract infections and chronic pyelonephritis. Paraplegia affecting the diaphragm can lead to pneumonia, and inability to exercise the lower extremities and pelvic region setting up a situation in which thromboembolism to the lungs is common. Inability to move can also cause the development of decubitus ulcers. Renal and adrenal functions are often impaired due to amyloidosis, and anemia of chronic disease is frequent in paraplegics.

Maxillofacial surgery for these individuals can be accomplished safely with good patient preparation. Preoperative checks of pulmonary and renal function will reveal patients at high risk for perioperative complications. The sputum and urine should be checked for evidence of infection and blood count obtained to discover if anemia is present. Special care needs to be taken to properly position and pad vulnerable parts of the body during and after surgery. Minidose heparin will help prevent pulmonary embolism, as will keeping the legs elevated during surgery and providing proper physical therapy after surgery. Physical therapy is also necessary to the upper extremities to prevent contractures. Continuous urinary catheterization is needed during surgery, returning to the intermittent bladder catheterization regimen (in place preoperatively) as soon as possible after surgery.

\section*{Surgery in the Patient with a Psychiatric Disorder}

\textbf{Affective Disorders}

Affective disorders such as depression are common problems in modern society. Patients with this disorder need special care during any surgical treatment.

Major depression is characterized by a depressed mood and an inability to enjoy life. Symptoms include sleep disturbance such as early morning wakening, appetite disturbance, fatigue, decreased libido, low self-esteem, and a feeling of hopelessness. Many patients are able to mask or deny their symptoms when under no undue stress, but facing a surgical procedure will usually uncover hidden symptoms of depression.

In addition to the emotional problems that patients with depression incur in the perioperative period, problematic drug interactions can occur between anesthetic agents and many of the agents used to control depression. Selective serotonin reuptake inhibitors are in widespread use for depression, anxiety, and panic disorder, and are well tolerated perioperatively. Tricyclic antidepressants are in common use
for depression, chronic pain, and sleep disorders. They can carry unwanted anticholinergic and hypotensive side effects, which should be remembered when anesthesia is given. An additional problem with tricyclic antidepressants is their tendency to cause increased conduction delays in patients with preexisting heart blocks.

Monoamine oxidase inhibitors (MAOIs) are also used to manage depressive symptoms. They also have anticholinergic and orthostatic hypotensive effects. Drugs with sympathomimetic action should be avoided in patients on MAOIs.

Lithium carbonate is used for patients with bipolar (manic-depressive) disorders. It induces the characteristic ECG changes of inverted and flattened T waves. It can also produce sinus node dysfunction and ventricular irritability. Serum levels should be checked preoperatively in these patients.

Benzodiazepines used for depression pose little risk for safe anesthesia as long as the anesthesiologist is aware of their use. Abrupt discontinuation should be avoided to prevent the appearance of a withdrawal phenomenon.

Conditions such as anorexia nervosa and bulimia should be addressed prior to major surgical procedures due to the impairment to nutritional health and electrolyte balance they produce.113

**Psychotic Disorders**

Psychotic disorders are characterized by delusions and hallucinations. Psychotic patients are usually easily recognized by the results of a comprehensive mental status examination. Antipsychotic drugs such as phenothiazines, thioxanthenes, butyrophenones, and indalones control many of the symptoms of psychosis and cause little increased risk of problems with anesthesia. They do have the tendency to cause sedation and extrapyramidal symptoms in many patients. Introduction of atypical antipsychotic medications has resulted in a large number of patients being converted to these drugs, including respiradone, olanzapine, quetiapine, ziprasidone, and aripiprazole. These medications have many drug-drug interactions, and consultation with a drug reference manual or pharmacist would be prudent to avoid such complications.

Surgery in psychotic patients carries no increased risk of complications as long as the disorder is well controlled.

Acute psychosis, combativeness, and agitation can be disruptive as well as unsafe for the patient and medical staff. After ruling out serious medical complications such as hypoxia, drug or alcohol withdrawal, serious infection, and myocardial infarction, administration of lorazepam 1 to 2 mg PO or IV, or haloperidol 1 to 2 mg PO, IM, or IV, can be used for control of symptoms acutely. Haloperidol also comes in a flavorless liquid formula.

**Substance Abuse**

**Alcoholism** Patients who regularly consume large amounts of ethanol must be allowed to withdraw from the effects of the alcohol before they undergo elective surgery and anesthesia. Failure to follow this strategy risks the appearance of minor alcohol withdrawal syndrome, with its compensatory neuronal excitability and catecholamine release, or the severe syndrome delirium tremens (DT) with hallucinosis, hyperpyrexia, hypertension, and life-threatening cardiac dysrhythmias and seizures.114,115

The following four questions have a high sensitivity and specificity for detecting alcoholism.116

- Have you ever felt the need to cut down on drinking?
- Have you ever felt annoyed by criticism of your drinking?
- Have you ever had guilty feelings about your drinking?
- Have you ever taken a morning “eye opener”?

Previous history of DT and drinking a morning “eye opener” denote a high risk of alcohol withdrawal.

Two strategies are available for the alcoholic patient coming to surgery: continuation of alcohol perioperatively, or avoidance of alcohol with vigilance for withdrawal syndromes. While it seems counterintuitive to continue alcohol use in a hospital or postoperative setting, this strategy can prevent withdrawal; most patients will resume drinking as soon as they can anyway. For patients newly abstaining, those with a prior history of DT may be given scheduled benzodiazepines, such as lorazepam 1 to 2 mg every 8 hours, but most patients should be observed for evidence of DT and treated based on symptoms. Early symptoms include restlessness and tremulousness, followed by agitation, combativeness, fever, and seizures. Symptoms should be treated as soon as they emerge, with oxazepam 15 to 30 mg PO every 6 to 8 hours as needed, or lorazepam 1 to 2 mg PO, IV, or IM every 6 to 8 hours as needed.

Most of the anesthetic hazards in the sober alcoholic patient are due to ethanol-induced hepatic changes (see “Liver Disease”). Chronic ethanol use increases anesthetic requirements for halothane and isoflurane. Clearance of benzodiazepines is also increased, so that larger doses may be necessary in alcoholic patients. Patients with ethanol-induced liver disease are prone to hypoglycemia and need frequent serum glucose determinations during and after surgery.

**Opioid and Illicit Drug Abusers** If surgery is urgently necessary in opioid-dependent patients, it is usually prudent for the surgeon to avoid precipitating the withdrawal syndrome by substituting methadone (2.5 mg equals 10 mg of morphine) for the abused opioid. Usually 20 to 40 mg of methadone is needed daily, administered orally or intramuscularly in 4 to 6 divided doses. Clonidine has also
been found useful for helping prevent symptoms of opioid withdrawal.117

Hypotension is a common problem in opioid abusers during the perioperative period. They also are likely to have difficult veins in which to gain access, necessitating placement of central lines. Intravenous illicit drug abusers also have a high incidence of hepatitis B and C and human immunodeficiency virus positivity.

Cocaine use potentiates problems such as coronary vasospasm, myocardial ischemia/infarction, and dysrhythmias. The rapid metabolism of cocaine in a patient’s system prior to presenting for surgery makes it unlikely that acutely intoxicated patients will be placed under sedation or general anesthesia.118,119

Surgery in the Special Patient

**Obese Patients**

Obesity is a common affliction in modern society due to a combination of poor dietary habits and general lack of physical activity. The excessive weight in an obese individual is due to an overabundance of adipose tissue. Morbid obesity is defined as when a patient is 100% over ideal body weight due to fat accumulation. Calculation of the body mass index (BMI) assists in the diagnosis of obesity, with a BMI of 30 kg/m² and above defining obesity.

\[
\text{BMI} = \frac{\text{Body weight in kg}}{\text{Square of stature (height in m)}}
\]

Obesity by itself does not increase surgical mortality until it becomes severe, but then the risk rises exponentially. The ponderal index has been used to quantitate the increased risk faced by obese individuals. The index is calculated by dividing an individual’s height in inches by the cube root of their weight in pounds. A result greater than 12.5 correlates highly with a significantly heightened risk of complications in the perioperative period. The *Chase method* is another means of gauging risk in obese individuals in which surgical risk is determined by the ratio of weight versus height.

Pulmonary problems are the most frequent complications in the perioperative period in obese patients. These include pulmonary embolism, bronchospasm, atelectasis, and pneumonia. Obesity creates a form of restrictive lung disease, especially when these patients are supine, due to excessive weight on the thorax and abdomen that restricts full inspiration.

Before elective surgery in obese patients a careful history and physical examination are necessary to determine how the obesity may affect anesthesia and to detect a concurrent disease. Specific questions about a history of daytime somnolence and snoring are needed to find if a patient’s airway is easily compromised. Past history of lung disease, heart problems, thrombophlebitis, or pulmonary embolism should also be elicited. Obese patients should also be asked about any previous problems in the establishment of venous access. The usefulness of physical examination of the chest and abdomen is commonly limited in obese patients. Therefore, ancillary examination techniques such as PFTs, ECG, and plain chest radiography are usually warranted.

Because obese patients have heightened risks of pulmonary problems, those who smoke should be helped to quit, hopefully for as long as possible before surgery. A reasonable program of weight reduction should also be recommended. Many patients may benefit from a consultation about potential gastrointestinal surgery for weight control.

When planning surgery the possibility of regional anesthesia should be considered. Deep sedation should be avoided if the airway is likely to be difficult to maintain. If general anesthesia is selected as the method of pain and anxiety control, the patient can be given preoperative instruc-

**Geriatric Patients**

Although many clinicians are concerned that there will be medical complications when treating elderly patients, studies sub-
stinate the fact that most elective surgery is safe in healthy geriatric patients. However, geriatric patients with chronic diseases such as COPD, diabetes, and coronary artery disease are certainly susceptible to the same problems as younger individuals with these same processes. Therefore, when older patients have chronic diseases, preoperative preparation should include efforts to minimize the detrimental effect of the disease process on the patient’s physiology.\textsuperscript{121}

Even though elderly patients can appear frail and sick, a large percentage are actually well. Conversely the appearance of health can be deceiving, because all older individuals experience various changes in physiologic function that can affect their response to the stress of an operation.

Statistically the most common complications that follow major surgery in the elderly are pulmonary embolism, myocardial infarction, pneumonia, and congestive heart failure. The surgeon should be especially vigilant for a past history or perioperative signs of these problems. Furthermore, although geriatric patients usually are able to withstand the initial physiologic stresses of surgery, if a complication occurs, they have less reserve to aid with recovery.

The heart undergoes age-related changes that decrease the maximal heart rate \((220 – \text{age in yr})\). Cardiac output falls (about 1% each year after age 20 yr) because of increased afterload and decreased elasticity of arteries secondary to atherosclerosis. This decreased elasticity also causes any small increase in blood volume to result in sharp increases in blood pressure. Total circulation time at age 20 years is 48 seconds; this rises to 65 seconds at age 70 years. The cardiovascular system also loses much of its responsiveness to catecholamines with age, so that postural hypotension is common. Maximum coronary flow capacity in the elderly is about 65% of that in teenagers.

Pulmonary function also falls as people get older. Loss of lung elasticity and increased stiffness of the chest wall predispose lungs to atelectasis and ventilation-perfusion imbalances, as does the increased residual volume in older lungs. Whereas the \(\text{PaO}_2\) on room air at age 30 years averages about 94 mm Hg, it normally falls to about 74 mm Hg above age 60 years. Vital capacity and expiratory flow rate begin to fall when individuals reach age 30. Muscle weakness prevents forceful coughing, and degeneration of bronchial epithelium leads to less efficient lung cleansing. All of these changes help to account for the relatively high incidence of pulmonary complications following surgery in older patients.

Renal function decreases 20 to 30% between the ages of 30 and 80 years because of natural loss of glomeruli and fibrosis of interstitial tissue. Creatinine clearance falls, but because lean body mass also decreases there is usually no change in measured serum creatinine. An approximation of expected age-related changes in renal function can be gained by the following equation:

\[
\text{Creatinine clearance (mL/min)} = 133 – (0.84 \times \text{Age})
\]

This formula can be used to judge dosages of drugs dependent on renal clearance. Geriatric patients also suffer a loss of renal concentrating and diluting abilities as tubules become less responsive to antidiuretic hormone. For that reason they can easily have intravascular volume disturbances and electrolyte abnormalities. Thirst perception also becomes a problem and thirst cannot be relied on to help gauge fluid requirements in these patients. Prostatic hypertrophy occurs in 80% of men with age, causing urinary problems that are commonly worsened by general anesthesia.\textsuperscript{122,123}

The loss of muscle mass and plasma volume with age may affect drug actions and necessitate changes in drug doses. Older white females are also predisposed to loss of bone strength owing to osteoporosis; extra care should be taken when transporting these patients to and from the operating table. Thinning of skin in older patients also makes them more susceptible to pressure damage, heightening the need for proper intraoperative padding.

Geriatric patients tend to mount poor fever responses to pyrogens. Therefore, other signs of problems such as malaise or altered states of consciousness may need to be used to detect infections. The hearing and visual problems of older patients predispose them to states of confusion owning to sensory deprivation; providing appropriate sensory stimulation helps prevent this problem. A decrease in gastrointestinal motility leads to frequent constipation, and aging often causes impaired glucose tolerance.

Evaluation of elderly patients before elective maxillofacial surgery should begin with a careful medical history. Old records and consultation with the patient’s primary care provider are usually excellent sources of needed information. During the physical examination specific note should be made of the patient’s state of hydration, signs of age-related problems such as carotid or aortic stenosis, and any pulmonary and mental status problems. An ECG and chest radiograph are useful for detecting occult problems and provide a baseline for later comparisons.\textsuperscript{124,125}

Intraoperatively the patient should be kept from excessive loss of heat and over- or underhydration. Postoperatively, the clinician should be alert to possible respiratory depression due to narcotics and signs of myocardial damage such as sudden dyspnea or worsening of congestive heart failure.

Drug modifications in the elderly include reducing benzodiazepine dosages by at least 50%, recognizing the dysrhythmogenic potential of atropine, and being aware that narcotics such as morphine and meperidine have prolonged duration of action, and that water-soluble drugs will have a heightened pharmacologic effect.
while lipid-soluble drugs such as barbiturates will have a long elimination time.123

**Pediatric Patients**

The surgical challenges in pediatric patients are usually due to their small size. However, it is hazardous to consider children as just small adults when considering their anesthetic needs for surgery.

The physiology of pediatric patients is what makes them differ from adults in their response to drugs and anesthesia. Newborns and infants are obligate mouth breathers. Children have relatively small nasal airways, large tongues, small mandibles, short necks, and an abundance of pharyngeal lymphoid tissue; all of these serve to create an airway that is easily compromised. Dead space in children is about 2 mL/kg, and tidal volume is about three times the dead space.

The heart of infants has a fixed stroke volume, so that cardiac output is entirely dependent on heart rate. Blood volume in relation to body weight is high in infants, but ratio decreases with age. Fluid requirements of children vary with weight as shown in Table 2-6. Normal urine output also varies (Table 2-7). Children have relatively large surface areas that can quickly allow excessive heat loss if they are left uncovered in an operating room.

Dosing of drugs to children is usually best decided based on the manufacturer’s recommendations.126

**Pregnant and Lactating Patients**

Pregnancy and lactation are relative contraindications to elective maxillofacial surgery because of the negative effects on the mother and developing child of various drugs, irradiation from imaging studies, and psychological stress associated with surgery.127–129

If surgery cannot be deferred, the patient’s obstetrician should be consulted for guidance with respect to safe drugs to use (Table 2-8). When feasible the surgery should be conducted under local anesthesia. Steps to minimize anxiety are also appropriate. During later stages of pregnancy, patients cannot tolerate long procedures without being allowed to empty their bladder. In addition, pressure from the uterus compromises venous return to the heart by placing pressure on the vena cava when patients are in a supine position.130 Therefore, allowing patients to assume a more left lateral position is necessary. A late-term pregnant patient’s blood pressure or urinary protein must be carefully monitored to detect any early sign of preeclampsia.131,132

Lactating patients need to avoid the use of drugs capable of passing into breast milk and potentially harming the infant (Table 2-9).

**References**

10. Dibra A, Mehelli J, Braun S, et al. Association between C-reactive protein level and subsequent cardiac events among patients with

<table>
<thead>
<tr>
<th>Weight (kg)</th>
<th>Fluid Requirements</th>
</tr>
</thead>
<tbody>
<tr>
<td>1–10</td>
<td>100 mL/kg daily</td>
</tr>
<tr>
<td>10–20</td>
<td>1,000 mL + 50 mL/kg</td>
</tr>
<tr>
<td></td>
<td>over 10 kg daily</td>
</tr>
<tr>
<td>20</td>
<td>1,500 mL + 20 mL/kg</td>
</tr>
<tr>
<td></td>
<td>over 20 kg daily</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Urine Output</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–2</td>
<td>3 mL/kg/h</td>
</tr>
<tr>
<td>2–5</td>
<td>2 mL/kg/h</td>
</tr>
<tr>
<td>5 to adult</td>
<td>1 mL/kg/h</td>
</tr>
</tbody>
</table>


30. Rehder K, Sessler AD, Marsh HM. General
73. Martin DE, Kommerer WS. The hypertensive
Perioperative Considerations

Noah A. Sandler, DMD, MD

Many factors need to be considered when evaluating a patient prior to oral and maxillofacial procedures. Whether a surgery is being performed in an office or operating room, the practitioner must acknowledge the impact of the surgery and the stress the perioperative period potentially entails. In addition, the pathophysiology of concomitant medical ailments that may modify therapy needs to be considered. Preoperative assessment, intraoperative monitoring, and postoperative care need to be modified based on individual patient requirements. The following discussion does not attempt to answer all questions regarding perioperative patient care. Common clinical scenarios and disease processes are presented. Despite our best efforts to prevent problems through assessment and monitoring, problems or emergencies can arise; therefore, this chapter also addresses patient monitoring and emergency management of common clinical situations.

Cardiac Assessment

Since the 1970s risk assessment has been performed in an attempt to identify individuals who may encounter a significant cardiac event (i.e., myocardial infarction [MI] or death) in the perioperative period. In their often-referenced article, Goldman and colleagues identified nine independent factors associated with increased perioperative cardiac risk (Table 3-1). These were assigned a point system based on their relative contribution to cardiac risk. The more points, the higher the risk of significant morbidity or mortality, primarily in the immediate postoperative period (Table 3-2).

Since 1980 the American College of Cardiology in association with the American Heart Association (ACC/AHA) has produced guidelines for the management of cardiovascular disease. In 1996 a committee was developed to assess guidelines in the perioperative evaluation for noncardiac surgery. Expanding on the factors identified by Goldman and colleagues, patient daily function and surgical risk were also considered.

Recent evidence based on 4,315 patients over the age of 50 years undergoing elective noncardiac procedures suggests six major risk factors exist. These are included in a revised cardiac risk index: high-risk type of surgery, history of ischemic heart disease, congestive heart failure, cerebrovascular disease, preoperative treatment with insulin, and preoperative serum creatinine > 2.0 mg/dL. Based on these findings as well as support from similar studies and recent technologic advances in coronary testing and therapies, the ACC/AHA

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Point Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Third heart sound or jugular venous distention</td>
<td>11</td>
</tr>
<tr>
<td>Recent myocardial infarction</td>
<td>10</td>
</tr>
<tr>
<td>Rhythm other than sinus or premature atrial contractions on last echocardiogram</td>
<td>7</td>
</tr>
<tr>
<td>&gt; 5 premature ventricular contractions per minute at any time</td>
<td>7</td>
</tr>
<tr>
<td>Intraperitoneal, intrathoracic, or aortic operation</td>
<td>3</td>
</tr>
<tr>
<td>Age &gt; 70 yr</td>
<td>5</td>
</tr>
<tr>
<td>Important aortic stenosis</td>
<td>3</td>
</tr>
<tr>
<td>Emergent operation</td>
<td>4</td>
</tr>
<tr>
<td>Poor general medical condition</td>
<td>3</td>
</tr>
<tr>
<td>Partial pressure of oxygen &lt; 60 or of carbon dioxide &gt; 50 mm Hg</td>
<td></td>
</tr>
<tr>
<td>K &lt; 30 mEq/L</td>
<td></td>
</tr>
<tr>
<td>Creatinine &gt; 3 mg/dL or blood urea nitrogen &gt; 50 mg/dL</td>
<td></td>
</tr>
<tr>
<td>Chronic liver disease</td>
<td></td>
</tr>
<tr>
<td>Bedridden from noncardiac causes</td>
<td></td>
</tr>
</tbody>
</table>

*As determined in Goldman L et al.1 Adapted with permission from Goldman L et al.1
practice guidelines were updated in 2002. As part of these guidelines, consideration is given to cardiac testing for individuals determined to be at risk for a perioperative event. The following factors are assessed:

- Is the surgery urgent? If delay of the surgery may be detrimental, cardiac assessment may need to be performed at a later time.
- Has the patient undergone coronary revascularization in the past 5 years or percutaneous coronary intervention from 6 months to 5 years previously? If the patient has remained free from symptoms of ischemia, the risk of perioperative cardiac death or MI is extremely low.
- Has the patient undergone a coronary evaluation in the past 2 years? If invasive or noninvasive testing was negative and the person has remained symptom free, no further perioperative testing is indicated.
- Does the individual have an unstable cardiac condition or major clinical predictor of risk? These include acute (within 7 d) or recent (7–30 d) MI, unstable or severe angina, decompensated heart failure, significant arrhythmias, and severe valve disease. These conditions warrant delay of the procedure when possible, and usually coronary angiography is performed.
- Are there intermediate clinical predictors of risk? These include angina pectoris, prior MI as indicated by history or electrocardiography, compensated or prior heart failure, preoperative creatinine > 2 mg/dL (ie, renal insufficiency), and diabetes mellitus (DM), particularly insulin-dependent DM. In addition to these risks, the functional capacity of the individual is determined. This is recorded in metabolic equivalents (METs), where 1 MET is the oxygen consumption of a 70 kg 40-year-old man at rest. Functional capacity is classified as excellent (> 10 METs), good (7–10 METs), moderate (4–7 METs), poor (< 4 METs) (Table 3-3).
- What are the specific risks of the surgery? Considerations include the type of surgery (eg, vascular surgery is high risk) and hemodynamic changes that occur with certain surgeries (eg, significant bleeding or hypotension). Most oral and maxillofacial surgery procedures are considered to be of intermediate risk.

In general, patients with no major and few intermediate predictors of clinical risk and moderate or excellent functional capacity can undergo oral and maxillofacial surgery procedures with little risk of perioperative death or MI. On the other hand, individuals with poor functional capacity who are to undergo higher-risk surgery (eg, head and neck cancer resection) are often considered for further non-invasive testing (eg, stress test, echocardiography). This approach has been demonstrated in recent studies to be efficacious and cost-effective.

Since most oral and maxillofacial surgical procedures are considered to be intermediate risk, the primary cardiac risk factor is the existence of one or more of the major clinical predictors of risk (ie, recent MI, unstable or severe angina, decompensated heart failure, significant dysrhythmias, and severe valve disease). The primary method of initial identification of these factors is a history taking and physical examination. Patients with identifiable risks warrant deferment of surgery with a referral for consideration for a thorough cardiac evaluation.

### Myocardial Ischemia/Angina

The stress of elective surgery begins well before the incision is made. Activation of the hypothalamic-pituitary-adrenal axis is initiated by just scheduling the procedure and persists through the surgical period until at least a week after the surgery. Concomitant with the release of cortisol is stimulation of the adrenal medulla and the activation of the sympathetic nervous system with catecholamine release. These responses may have served an evolutionary purpose and/or aid in aspects of healing; however, they can be detrimental in a debilitated patient with poor reserve. Surgery, itself, necessitates myocardial work. Patients with atherosclerosis and coronary artery disease with narrowing of

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### Table 3-2 Assessment of Morbidity and Mortality Based on Cardiac Risk Factors

<table>
<thead>
<tr>
<th>Class</th>
<th>Point Total</th>
<th>No or Minor Complications* (n = 943)(%)</th>
<th>Life-Threatening Complications† (n = 39)(%)</th>
<th>Cardiac Deaths (n = 19)(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I (n = 537)</td>
<td>0–5</td>
<td>532 (99)</td>
<td>4 (0.7)</td>
<td>1 (0)</td>
</tr>
<tr>
<td>II (n = 316)</td>
<td>6–12</td>
<td>295 (93)</td>
<td>16 (5)</td>
<td>5 (2)</td>
</tr>
<tr>
<td>III (n = 130)</td>
<td>13–25</td>
<td>112 (86)</td>
<td>15 (11)</td>
<td>3 (2)</td>
</tr>
<tr>
<td>IV (n = 18)</td>
<td>&gt; 26</td>
<td>4 (22)</td>
<td>4 (22)</td>
<td>10 (56)</td>
</tr>
</tbody>
</table>

*As determined in Goldman L et al.†Documented intraoperative or postoperative myocardial infarction, pulmonary edema, or ventricular tachycardia.

Adapted with permission from Goldman L et al.

---

### Table 3-3 Metabolic Equivalents for Common Activities

<table>
<thead>
<tr>
<th>Functional Capacity</th>
<th>Metabolic Equivalents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Take care of yourself</td>
<td>1</td>
</tr>
<tr>
<td>Walk a block or two</td>
<td>1</td>
</tr>
<tr>
<td>Climb a flight of stairs</td>
<td>4</td>
</tr>
<tr>
<td>Heavy work</td>
<td>1</td>
</tr>
<tr>
<td>Moderate recreation</td>
<td>&gt;10</td>
</tr>
<tr>
<td>Strenuous sports</td>
<td></td>
</tr>
</tbody>
</table>
the coronary vessels may be unable to meet this increased demand. Myocardial ischemia within 48 hours of surgery results in a ninefold increase in the risk of unstable angina (defined as angina at rest or increasing angina symptoms) and/or MI.

Myocardial work is primarily determined by four factors related to myocardial oxygen demand: heart rate, preload, afterload, and contractility. **Preload** represents all factors that contribute to passive ventricular wall stress (tension) at the end of diastole. It is approximately equal to end-diastolic volume or pressure (ie, the volume of blood left in the heart after diastole). Preload is generally a reflection of the volume status of a patient. It is measured via the central venous pressure or the pulmonary capillary wedge pressure. Additionally, the left ventricular end-diastolic volume determines the cardiac output according to Starling’s law. Clinically, this means increasing preconstriction muscle fiber length by increasing left ventricular end-diastolic volume through volume administration leads to an increase in the force of contraction. **Afterload**, in turn, represents all of the factors that contribute to total ventricular wall stress (tension) during systole. The primary determinants of afterload are the total peripheral resistance against which the heart muscle must pump and changes in intrathoracic pressure. Afterload is indirectly measured through blood pressure and mean arterial pressure. **Contractility** is the ability of the heart muscle to shorten itself in the face of appropriate stimuli. Of these factors, heart rate and afterload are the major contributors to cardiac work and myocardial oxygen consumption. Elevated heart rate is also potentially harmful in that it decreases the time that oxygen and nutrients can be delivered to the myocardial cells (diastolic perfusion time). This is the basis for the goal of maintaining the blood pressure and pulse within 10% of the preoperative value during anesthesia.

Patients with coronary artery disease often have a history of hypertension. Blood pressure is measured using the proper cuff size with patients quiet and comfortable (with back support, if seated) for at least 5 minutes prior to measurement. **Hypertension** is defined as two elevated blood pressure readings separated by at least 2 minutes of ≥ 140/90 mm Hg on two or more separate visits. Healthy patients with persistent elevated pressures ≥ 160/100 mm Hg and those considered to be at high risk (diabetics or patients with clinical cardiovascular disease) should be considered for antihypertensive therapy.

Preoperatively, elevated blood pressure should be managed by deferring treatment for elective procedures. Intraoperative or postoperative hypertension rarely requires treatment. Hypertensive crisis or emergency is a sudden increase in systolic and diastolic blood pressure associated with end-organ damage of the central nervous system, heart, or kidneys. Headache, altered level of consciousness, and less severe manifestations of central nervous system dysfunction are classic findings in hypertensive encephalopathy. Advanced retinopathy with arteriolar changes, hemorrhages, and exudates as well as papilledema are seen on funduscopic examination. Angina, acute MI, or signs of heart failure can be present in hypertensive crisis. Renal failure with oliguria and/or hematuria is present with damage to the kidneys. Less than 1% of patients with a diagnosis of hypertension experience a crisis. In the United States the incidence is higher among African Americans and the elderly. The majority have previously been diagnosed with hypertension and many have been prescribed antihypertensive therapy but with poor control. The incidence of postoperative hypertensive crisis varies depending on the population studied and has been reported in 4 to 35% of patients. Reduction of blood pressure in a hypertensive crisis should be performed with intravenous blood pressure monitoring.

The term **hypertensive urgency** is characterized by severely elevated blood pressure without acute end-organ damage. Postoperative hypertension has been defined arbitrarily as systolic blood pressure > 190 mm Hg and/or diastolic blood pressure ≥ 100 mm Hg. It should be appreciated that most patients with severely elevated blood pressure (diastolic > 110 mm Hg) have no acute end-organ damage. The elevated blood pressure should be treated in a controlled fashion in an intensive care unit. The use of sublingual nifedipine is strongly discouraged as this may result in a precipitous fall in blood pressure. Similarly, intravenous hydralazine may result in severe uncontrolled hypotension. Rapid and uncontrolled reduction of blood pressure may result in cerebral, myocardial, and renal ischemia or infarction. Table 3-4 describes commonly recommended medications and dosages should it be determined that reduction of blood pressure is necessary.

**Recent Myocardial Infarction**

It is important to attempt to avoid the stress of surgery if the patient is experiencing acute ischemia or has a history of recent infarction. Traditionally a 6-month interval between the initial incidence of MI and elective noncardiac surgery has been advocated to avoid stress and the risk of re-infarction. However, recently the importance of this time interval has been called into question. The use of thrombolytics, angioplasty, and risk stratification after an acute MI has been the impetus for this change. Although some patients may continue to have myocardium at risk with subsequent ischemic episodes, others may have critical stenosis converted to widely patent vessels. The AHA/ACC Task Force on Perioperative Evaluation of the Noncardiac Surgery has advocated that the group at highest risk is those who have had an MI within 6 weeks;
Part 1: Principles of Medicine, Surgery, and Anesthesia

After this period risk stratification is based on the presentation of the disease (ie, those with persistent symptoms consistent with active ischemia remain at the highest risk level).12

During severe ischemic episodes the release of intracellular potassium from injured cells may result in partial repolarization of the surviving cardiac cells, particularly along the infarct border. These cells may then initiate areas of ectopia, potentially leading to arrhythmias, especially with concurrent sympathetic stimulation, electrolyte abnormalities, and ventricular hypertrophy. β-Blockers, nitroglycerin, and amiodarone as well as high vagal tone can be protective in this circumstance. In addition, intra-aortic balloon pumps, ventricular assist devices, coronary angioplasty, and revascularization may be indicated.

**Acute Episode of Chest Pain Suggestive of Myocardial Ischemia/Infarction**

Immediate intervention includes the assessment of vital signs and the administration of oxygen and nitroglycerin tablets or spray at 0.4 mg/dose (to be repeated in 5 min intervals for three doses or until the pain is eliminated). If the pain is persistent, intravenous morphine (2–5 mg q5min or until pain relief is achieved) and aspirin 325 mg should be given. The local Emergency Medical Service should be contacted early as the protocol calls for the performance of an early 12-lead echocardiography (preferably by Emergency Medical Service personnel) and screening of the patient for an antifibrinolytic or reperfusion (ie, an angioplasty with stent placement or coronary artery bypass graft) procedure.13

**Decompensated Congestive Heart Failure**

A history of worsening shortness of breath (dyspnea), difficult ventilation when assuming the supine position (orthopnea), or gasping for oxygen when assuming the supine position when asleep (paroxysmal nocturnal dyspnea) should alert the practitioner to the possibility of acute congestive heart failure. Signs of cardiac failure include raised jugular venous pressure, added heart sounds (S3 [the presence of a third heart sound], in particular), pulmonary crackles (indicating pulmonary edema), hepatomegaly, and peripheral edema. The presence of any of these signs or symptoms warrants a complete cardiac evaluation prior to initiating any elective procedure.1,3

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| Table 3-4 Common Antihypertensive Agents Used to Actively Lower Blood Pressure in Hypertensive Crisis |
|---------------------------------|----------------|------------------|------------------|
| **Drug** | **Mechanism** | **Dosage** | **Comments** |
| Clonidine | Central α2-agonist | 0.1 mg PO q20min | Useful in hypertensive urgency; gradually decreases BP |
| Diazoxide | Smooth muscle relaxant | 1–3 mg/kg IV, maximum single dose of 150 mg | Causes rapid BP decrease |
| Enalaprilat | Angiotensin converting enzyme inhibitor | 1.25 mg over 5 min q6h | Blocks angiotensin II |
| Esmolol | β1-selective blocker | 0.5 mg/kg followed by infusion of 25–300 µg/kg/min | Rapid onset (60 s), short duration (10–20 min) |
| Fenoldopam | Dopamine agonist | Initial dose 0.1 µg/kg/min titrate; maximum 1.6 µg/kg/min | Short acting, increases renal perfusion |
| Labetalol | α- and β-blocker (α:β = 1:7) | Loading dose of 20 mg followed by 20–80 mg dose at 10 min intervals or 1–2 mg/min infusion | Avoid larger bolus doses; can cause hypotension |
| Nicardipine | Ca channel blocker | 5 mg/h increasing 2.5 mg/h q5min (maximum 15 mg/h) | Useful for cardiac and cerebral ischemia; dose independent of weight |
| Nitroprusside | Arterial/venous dilatation | Infusion; usually < 2 µg/kg/min | Rapidly decreases BP; risk of cyanide toxicity |
| Phentolamine | α-blocker | IV 1–5 mg boluses | Can cause tachyarrhythmias, angina |
| Trimethaphan | Nondepolarizing ganglionic block | IV infusion 0.5–1 mg/min; maximum 15 mg/min | Adrenergic block is therapeutic effect; cholinergic block of side effects |

BP = blood pressure.
**Arrhythmias**

The normal pattern of electric transmission of the heart starts with the initiation of the impulse in the sinoatrial (SA) node, spreading through the atria with a convergence of the impulse at the atroventricular (AV) node. There is a delay of conduction through the AV node, accounting for the P–R interval on the echocardiogram (ECG; 100 ms). This interval is prolonged by parasympathetic (vagal) stimulation and shortened by sympathetic activity. Activation of the ventricles starts on the left side of the interventricular septum, crossing over to the right at the midpoint of the septum. The impulse spreads through the Purkinje system to the apex. The wave of depolarization then moves along the walls of the ventricles from the endocardium to the epicardium to reach the AV groove.

Perioperative cardiac arrhythmias are caused by abnormalities of cardiac impulse formation, impulse conduction, or a combination of both. There is a higher incidence of arrhythmias in the perioperative setting, and anesthetic agents are known to alter cardiac impulse generation and conduction. Perioperative catecholamines owing to exogenous administration or endogenous release in the presence of ischemia set the stage for new arrhythmia during this period.14,15

Volatile agents directly decrease SA and AV node automaticity, but increasing extracellular calcium can antagonize this phenomenon. A common occurrence with the use of volatile agents is isorhythmic AV dissociation, in which the AV node generates the pacemaker at a modestly higher rate than the SA node. This is a result of direct depression of the SA node by the volatile agent and some stimulation of the AV node by sympathetic activity. Serious hemodynamic consequences are not usually seen in healthy individuals but are a concern with ventricular noncompliance such as ventricular hypertrophy as a result of atherosclerosis of the aorta or peripheral vessels. Inhalation agents in general are not otherwise arrhythmogenic, but arrhythmias can be produced in the presence of triggering agents and clinical situations that generate a high catecholamine state. This includes light anesthesia levels (with hypertension and tachycardia), hypoxemia, hypercarbia, and the use of exogenous epinephrine or aminophylline (the latter of which indirectly causes the release of endogenous catecholamines). The arrhythmogenic dose in micrograms per kilogram of epinephrine administered by inhalation with various inhaled agents are 2.1 with halothane, 3.7 with halothane and lidocaine, 6.7 with isoflurane, and 10.9 with enflurane.16

Paroxysmal supraventricular tachycardias (PSVTs) arise from the SA or AV node, atrium, or an accessory AV connection. They are common arrhythmias that are usually seen in cardiac surgical patients (20–40%) but can develop in patients undergoing noncardiac surgery (usually major vascular, cancer, or orthopedic procedures). The onset and termination of these rhythms are usually abrupt, with rates between 120 and 300 beats per minute (bpm). The ECG typically identifies the area of origin of the ectopic conduction with a positive P wave being present in SA-node reentry PSVTs, absent or inverted P waves in AV-node origin PSVTs, and altered P wave morphology in intra-atrial reentry PSVTs.

The most common PSVT is atrial fibrillation (> 90% of SVTs in the postoperative period). It can occur as the result of cardiac disease, such as mitral valve disease, congestive heart failure, coronary artery disease, or pericarditis. It can also be the result of systemic processes such as thyrotoxicosis, pulmonary embolus, chronic obstructive pulmonary disease (COPD), alcohol or caffeine excess, or electrolyte disturbances. Changes seen on the ECG are most evident in lead II as an irregular rhythm.

Untreated PSVT can result in ventricular rates that exceed 120 to 200 bpm, which can cause significant hemodynamic instability. If uncontrolled ventricular rates occur acutely in the perioperative period, prompt treatment is necessary. Rate control is achieved with verapamil (a calcium channel blocker noted for decreasing conduction at the AV node), digoxin, or esmolol (a β1-selective blocker). If patients do not convert to sinus rhythm with these agents, electrocardioversion with prior anticoagulation is attempted.

It is interesting to note that a recently performed meta-analysis has demonstrated that β-blockers reduce the incidence of postoperative atrial fibrillation, whereas digoxin and verapamil have no effect. If a PSVT is detected upon routine monitoring, patients should be referred for further evaluation. Acute evaluation is required if the individual is symptomatic and/or the rate is poorly controlled. A complete discussion of the causes and treatment protocols of PSVTs is beyond the scope of this chapter. The reader is hereby referred to the most recent advanced cardiac life support protocols released by the American Heart Association.13

Abnormal conduction pathways can present as an irregular rhythm. Wolff-Parkinson-White syndrome is a condition in which such a pathway connects the atria to the ventricles, bypassing the AV junction through the bundles of Kent. As a result of impulses traveling through this accessory pathway, the electrocardiogram demonstrates a shortened P–R interval (< 0.12 s), a wide QRS complex (> 0.10 s), and a characteristic slurring of the upstroke of the R wave (called a delta wave) (Figure 3-1). This extra or accessory electric pathway is present in approximately 1.5 per 1,000 people. It runs in families in < 1% of cases. In the majority of individuals, it is completely silent and is only detected on a routine ECG. In a small proportion of patients, the extra electric pathway generates an electric circuit that produces a very rapid heart rate. Most patients tolerate this well, but some experience very troublesome palpitations,
light-headedness, and blackouts. A very small minority of patients may die suddenly from ventricular fibrillation. The ideal treatment in patients with symptoms is to destroy the extra electric pathway using radiofrequency ablation. Younger patients (< 25 yr) are most at risk of sudden death and require further tests to assess their possibility of developing life-threatening electric disturbances. This is best done with an exercise test under the supervision of a cardiologist. The abrupt disappearance of the delta wave on the ECG as the heart rate increases is a good sign, obviating the need for further investigation. If this does not happen, further electrophysiologic testing is recommended.\(^\text{17}\)

Ventricular arrhythmias can be classified as benign, potentially malignant, and malignant. Benign ventricular ectopy (ie, premature ventricular contraction) occurs in a normal heart with or without a previous history of arrhythmias, is asymptomatic, and generally does not warrant treatment unless hemodynamic perturbations are noted. Nonspecific cardiac challenges such as hypoxemia, hypercarbia, acidemia, sympathetic surge, drug effects, and electrolyte disturbances should be investigated and treated as necessary. A recently completed study demonstrated a 6.3% incidence of premature ventricular beats, but only 0.62% suffered severe adverse outcomes, which, according to the author, may have been related more to the aggressive treatment employed in these cases. More than six premature ventricular contractions per minute, especially if they are multifocal, are considered to be ventricular tachycardia and should be treated accordingly.\(^\text{16}\) Ventricular tachycardia with a pulse is treated using cardioversion or antiarrhythmia medication in a controlled monitored setting. Pulseless ventricular tachycardia is managed in the same manner as ventricular fibrillation, as described below. After assessing an unconscious victim for responsiveness, breathing, and a pulse, the airway should be opened, two rescue breaths given, and cardiopulmonary resuscitation initiated until a defibrillator is obtained. The rhythm should be assessed, and if ventricular tachycardia without a pulse or ventricular fibrillation is detected, progressive electric shocks should be administered at 200 J, 200 to 300 J, and 360 J using a conventional defibrillator or an automatic external defibrillator. Less energy is needed for a biphasic defibrillator (eg, 120 J, 150 J, and 200 J). If the rhythm is persistent, epinephrine in 1 mg doses every 3 to 5 minutes or vasopressin as a single 40-unit dose should be administered. Defibrillation at maximum dose (360 J or the biphasic equivalent) should be repeated after the catecholamine (epinephrine or vasopressin dose). If unsuccessful, doses of amiodarone, lidocaine, procainamide, or magnesium may be attempted followed by defibrillation at a maximal dose. For the most part, these drugs have only preventive roles in case of recurrence of the arrhythmia.\(^\text{13,17}\)

**Automatic Implantable Cardioverter Defibrillators and Pacemakers**

The first automatic implantable cardioverter defibrillator (AICD) was placed in 1980 and became commercially available in 1986. In recent years the use of AICDs has become widespread and has significantly reduced cardiac death in this susceptible population from 40 to 60% to < 2 to 3% over a 3-year postimplantation period. They are primarily used in cases of ventricular ectopy or spontaneous/recur rent episodes of ventricular tachycardia/fibrillation despite drug therapies. For the practitioner treating an individual with an AICD, it is important to realize that basic and advanced cardiac resuscitation should proceed as if the individual does not have the device. The shock delivered by the appliance may be discernible but does not pose any risk to the caregiver. The proper functioning of the device should be checked after resuscitation. In addition, the use of magnetic resonance imaging (MRI) is contraindicated when the device is in place. Since electrocautery can cause the device to administer an inappropriate shock, the device should be inactivated prior to using any electrosurgical equipment.

Presently there are over 1,500 types of pacemakers working in over two million individuals. In general, they are used for bradycardia and to prevent resultant low-cardiac output states. Modern devices adapt the rate to the metabolic needs of the patient. Sensors of oxygen saturation, right ventricular pressure, central venous blood temperature, and body movements help to adapt the rate. No pacemaker beats are observed if the intrinsic rate is greater than the threshold of the pacemaker. If the pacemaker is functioning, there should be a pacemaker spike on the down slope of the R wave, ST segment, or T wave with a QRS complex following in a one-to-one relationship. Pacemaker failure in the perioperative period can occur as a result of hypothermia, hyperkalemia, hyperventilation, or acute ischemia. Some pacemaker generators can be affected by electrocautery. It is advisable to use bipolar cautery with the lowest possible current and to avoid using cautery within 13 cm of the

![FIGURE 3-1](image_url) **Demonstration of the delta wave in Wolff-Parkinson-White syndrome.**
Electrolytes and Acid-Base Disturbances

With any arrhythmia, coexisting acid base and electrolyte disturbances should be identified and corrected. Part of the perioperative assessment of hypoxia is the maintenance of acid-base balance. Normal pH of arterial blood is 7.4 and is maintained to within 0.05 (ie, the normal pH range of the blood is 7.35 to 7.45). The main buffering of acids occurs through the lungs (through the conversion of carbonic acid [H₂CO₃] to CO₂ and H₂O) and the kidney (through the base bicarbonate [NaHCO₃]).

Respiratory acidosis occurs when the lungs are not exhaling CO₂ adequately. This can occur with emphysema or respiratory depressive states such as oversedation, respiratory insufficiency, and arrest. Conversely, respiratory alkalosis occurs when too much CO₂ is expelled as in hyperventilation, neurogenic disorders, and salicylate toxicity (which, interestingly, is accompanied by metabolic acidosis).

Metabolic acidosis is caused by a deficit of the base bicarbonate. Normally there is an H₂CO₃-to-NaHCO₃ ratio of 1:20. H⁺ is excreted in the urine, and bicarbonate is reabsorbed into the renal tubules to maintain this ratio. With the presence of excess acid, the bicarbonate combines with this source of H⁺, is excreted, and is therefore no longer available for its usual buffering role. This results in an upset of the 1:20 ratio and acidosis. Lactic acid from muscle activity or anaerobic conditions, diabetic ketoacidosis, renal failure, or exogenous sources such as methanol, ethanol, or paraldehyde can all serve as the alternative acid source. A method to determine whether metabolic acidosis is present is to calculate an anion gap (if information on electrolytes is available):

\[
\text{Anion gap} = \text{Na}^+ - (|\text{Cl}^-| + |\text{HCO}_3^-|)
\]

A normal range is 10 to 14 mEq/L.

Metabolic alkalosis is caused by a relative increase in bicarbonate. Only rarely is this caused by the exogenous administration of bicarbonate since the kidney normally excretes excess bicarbonate in an individual who is well hydrated and has good kidney function. More commonly this condition occurs owing to electrolyte disturbances such as occur as a result of vomiting, nasogastric suctioning, or diuretic use. Primarily this can occur through shifts in intracellular potassium.

Hypokalemia increases the excitability and automaticity of cardiac muscle, increasing the possibility of arrhythmias. Hypomagnesemia can potentiate this effect by decreasing the extrusion of intracellular calcium, which is also arrhythmogenic in cardiac conduction cells. Assessment of electrolytes and their correction is therefore warranted in acid-base perturbations.

**Examples of Acid-Base Analysis**

1. Note the pH value: pH < 7.35 = acidosis; pH > 7.45 = alkalosis.

2. Note the value of partial pressure of carbon dioxide in arterial blood (PaCO₂ value).
   - If it is the same sign as the pH, the condition is metabolic in nature. If it is the opposite in sign, the condition is respiratory. Therefore, pH < 7.35 and PaCO₂ < 40 mm Hg indicate metabolic acidosis; pH < 7.35 and PaCO₂ > 40 mm Hg signify respiratory acidosis.
   - This represents a method of analysis that is easy to remember. The basis involves the underlying cause of each condition. Respiratory acidosis is primarily caused by an elevation of CO₂, causing a compensatory elevation of carbonic acid in the lung with a resultant decreased pH. Metabolic acidosis is caused by the addition of an acid source to the normal acid-base buffering system. This acid source lowers the pH. One of the methods of buffering this acid is the carbonic acid system in the lung. Respiration rate and depth increase in an attempt to eliminate the additional CO₂ produced, lowering the CO₂. Ultimately, however, this system cannot eliminate all of the additional acid and maintain the normal acid-base ratio.

3. Confirm the acid-base relationship through analysis of the bicarbonate level (assuming normal kidney compensations are present).
   - In respiratory acidosis the kidney should retain bicarbonate and reestablish the normal 1:20 acid-to-base ratio (ie, the bicarbonate level should remain at its normal value of 24 mEq/L). In metabolic acidosis there is usually a bicarbonate deficit (ie, bicarbonate level < 24 mEq/L).

**Case Example 1**

A 54-year-old man is referred for lethargy. A review of systems reveals polydypsia, polyphagia, and polyuria. His laboratory results are as follows: arterial chemistries reveal Na = 130 mEq/L, Cl⁻ = 94 mEq/L, K = 4.5 mEq/L, and glucose = 600 mg/dL.

In this example, the pH is < 7.35; therefore, it is a case of acidosis. The PaCO₂ is < 40 mm Hg; therefore, the process is metabolic acidosis. The bicarbonate level (12 mEq/L) confirms a relative bicarbonate deficiency consistent with metabolic acidosis. An anion gap analysis is as follows:
Na\(^+\) – ([Cl\(^-\)] + [HCO_3\(^-\)])
130 – (12 + 94) = 31.5

This reveals the presence of an anion gap metabolic acidosis, consistent with diabetic ketoacidosis based on the clinical presentation and elevated glucose level (600 mg/dL).

Case Example 2 A 75-year-old woman was recently started on furosemide to treat pedal edema. She describes a loss of energy and a light-headed sensation when arising from a seated position. Her arterial blood gases indicate a pH of 7.53, PaCO\(_2\) of 52 mm Hg, and HCO\(_3\(^-\)\) of 32 mEq/L. Serum chemistries show the following levels: Na = 129 mEq/L, Cl\(^-\) = 90 mEq/L, K = 3.0 mEq/L, and glucose = 120 mg/dL.

In this case, the pH (7.53) and PaCO\(_2\) (52 mm Hg) reveal the presence of an alkalotic state. This is confirmed by the bicarbonate level (32 mEq/L). Metabolic alkalosis is often caused by secondary volume depletion with resultant electrolyte shifts. The loss of intracellular potassium can cause the shift of protons (H\(^+\)) into the cell to maintain neutrality.

Renal Insufficiency
It is interesting to note that an elevated creatinine is presently included as a factor in risk assessment for surgery.\(^2\) Acute renal failure is primarily a result of intraoperative renal hypoperfusion. It is usually seen in cardiopulmonary bypass procedures and thoracoabdominal and abdominal aortic aneurysm repairs, where its incidence is reported to be as high as 15%, 25%, and 5.4%, respectively.\(^{19-21}\) In addition to surgical type, preoperative renal insufficiency is the single consistent predictor of postoperative renal failure.\(^{19}\) Additional insults that may further predispose a patient to perioperative kidney failure are the presence of an already ischemic state caused by renal artery stenosis, volume depletion, and diabetes, or a recent acute ischemic event caused by hemorhage or exposure to radiocontrast agents. Many other conditions can predispose the kidneys to ischemic injury, including sepsis, cirrhosis, jaundice, hepatorenal syndrome, congestive heart failure, shock, malignant hypertension, preeclampsia, sickle cell anemia, collagen vascular diseases, and multiple myeloma. Many drugs also potentiate the risk of ischemic renal injury through alterations in intrarenal hemodynamics, including angiotensin-converting enzyme inhibitors, nonsteroidal anti-inflammatory drugs, cyclosporine, tacrolimus, and amphotericin B.\(^{19,22}\)

The most susceptible area to ischemic injury is the tubular cells of the thick ascending loop of Henle and a portion of the proximal convoluted tubules located in the renal medulla (Figure 3-2). The cells in this region are rich in mitochondria and are responsible primarily for chloride ion absorption. A combination of low blood flow (compared with that in the renal cortex) and high metabolic demand accounts for this susceptibility. Initially there is a loss of urine-concentrating ability as the normal medullary gradient dissipates, followed by a decline in urine output as tubules become obstructed and denuded.

Traditionally, the management of acute renal failure has been the maintenance of urine output through the use of intravenous hydration and diuretics such as furosemide and mannitol in addition to low-dose dopamine to maintain renal perfusion. Recently this practice has come into question since increasing renal blood flow elevates the oxygen demand at the medulla and may lead to further injury.\(^{19}\) Present research is directed at regulating renal vasoactive substances discovered in animal models including prostaglandins (especially prostaglandin E\(_2\)), angiotensin II, nitric oxide, endothelin, and adenosine.\(^{23}\)

Since volume depletion and hypotension are risk factors for the development of acute renal failure, preoperative testing of blood urea nitrogen and creatinine should be conducted in patients with a known history of renal insufficiency or a disease mechanism (eg, diabetes mellitus) in which kidney damage may be present and significant volume loss or hypotension may occur. In addition, the use of intraoperative invasive monitoring (ie, central venous pressure or pulmonary capillary wedge pressure) may be warranted in these cases.\(^{19}\)

Pulmonary Assessment

Asthma
Asthma is a disease characterized by an episodic variable airflow obstruction with increased airway reactivity. Recently the importance of submucosal inflammation and its control in managing asthma has been stressed. Bronchoconstriction in asthmatics is triggered by a stimulus such as an antigen, exercise, or exposure to cold. The trigger elicits an acute inflammatory cascade, characterized by degranulation of mast cells and activation of eosinophils and macrophages in the airway. Released leukotrienes, histamines, and bradykinins increase vascular permeability and resultant edema. The airways fill with mucus and inflammatory cells, and smooth muscles contract as a response to released mediators and an increased cholinergic tone.\(^{24,25}\)

Heightened airway responsiveness can increase the likelihood or severity of bronchospasm under anesthesia. Aspects of the patient’s history that may indicate the potential for problems to arise include frequent nocturnal awakenings from bronchospasm, increased necessity for inhaler use, recent hospitalizations or emergency department visits, a change in the amount or quality of secretions, or a recent viral illness or cold symptoms. Spirometry is helpful in the initial diagnosis and chronic management of reactive airway disease. Its routine use adds little information to the preoperative assessment that cannot be ascertained by the recent history and physical examination. Repeat assessments over time can be helpful, however, as subtle changes in flow rates can be detected by spirometry before they
become symptomatic; this allows preventive treatment to be initiated.

The most common parameters that are assessed over time are the forced expiratory volume generated in the first second of exhalation and the peak expiratory flow rate (Figure 3-3). These parameters can be measured with inexpensive handheld devices. A 20% variation in peak expiratory flow rates is normal. Rates that fall to 50 to 80% below normal are considered a moderate exacerbation. Flow rates < 50% of baseline are considered severe and require prompt medical attention.

The term reactive airway disease is considered by some individuals to be synonymous with asthma. However, airway reactivity is also increased owing to allergic rhinitis, bronchitis, emphysema, and respiratory viral infections. Bronchospasm is a physical sign of acute increased airway resistance. It is associated with tachypnea, wheezing, air trapping, and worsened gas exchange. Under anesthesia wheezing and bronchospasm can occur with or without a prior history of reactive airway disease. Most wheezing is self-limited and requires no intervention, but it can indicate the initiation of a more severe bronchospasm. Patients with symptoms of bronchospasm preoperatively should have elective procedures postponed.

Whereas asthmatics have chronic hyperactivity of the airways, patients with upper respiratory tract infections (URIs) have acute airway reactivity that can last up to 6 weeks after recovery from the initial infection. Airway hyperreactivity in URIs is neurally mediated with an increase in vagal-mediated bronchoconstriction. Children with a concomitant URI are especially susceptible to bronchospasm. These children are two to seven times more likely to have adverse events in the perioperative period, and there is an increased risk of postoperative desaturation in these patients. The risks are highest in those patients undergoing endotracheal intubation (in whom there is an 11-fold increase in perioperative respiratory complications). Definitive criteria for canceling a surgery to be performed under sedation or general anesthesia have not been established, and the decision is often subjective. Suggested criteria for cancellation include the necessity of endotracheal intubation, parental observation that the child is acutely ill the day of surgery, the presence of nasal congestion and cough, concomitant exposure to passive smoke, and active sputum production. Most surgeons agree that the planned surgery, if elective, should be postponed until after the acute symptoms have resolved and have not recurred for a 3-week period after the initial evaluation.

**Treatment of a Reactive Airway**

Inhaled short-acting β₂-adrenergic agonists are the drug of first choice for the treatment
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of acute bronchospasm. \( \beta_2 \)-Agonists directly relax smooth muscle, aid in the stabilization of mast cells, and inhibit the release of acetylcholine from postganglionic cholinergic nerves. The inhibition of the cholinergic response is important because bronchospasm during a surgery is often mediated by a vagal response. Doses should be limited by side effects rather than by an arbitrary number of inhalations. In general, intubated patients require twice the drug dose since the delivery of drug through the endotracheal tube is inefficient. If the patient is unconscious, the \( \beta_2 \)-agonist can be delivered subcutaneously or intravenously, usually as epinephrine. Intravenous epinephrine can be used safely in low doses, but dysrhythmias and other undesirable effects may occur in older individuals.

Glucocorticoids are useful in asthma in patients who have not adequately responded to \( \beta_2 \)-agonists. Their reported benefits include reduction of inflammation, histamine, and arachidonic acid metabolites. Anticholinergic drugs such as ipratropium cause bronchodilation directly and blunt bronchoconstriction from cholinergic-mediated triggers. Both steroids and anticholinergic agents enhance the activity of \( \beta_2 \)-agonists but are not indicated for acute exacerbations of bronchospasm.

In the past theophylline was frequently recommended for acute exacerbations of bronchospasm; however, this has been encouraged less in recent years since its potency as a bronchodilator is less than the \( \beta_2 \)-agonists and it frequently produces toxicity and undesirable side effects including dysrhythmias. In some patients with chronic asthma or COPD, theophylline can decrease the severity and frequency of attacks and decrease steroid requirements. Its mechanism of action also has been questioned recently. Although it does increase concentrations of cyclic nucleotides (ie, cyclic adenosine monophosphate) in airway smooth muscle and inflammatory cells by inhibiting the phosphodiesterase isozyme, it also has been demonstrated in dogs to produce bronchodilation by increasing the release of endogenous catecholamines. (Halothane appears to block this effect.) The drug also acts as an adenosine-receptor antagonist, which may help to mediate its effects on ventilation and mediator release.\(^{28}\) Cromolyn sodium reduces the degranulation of mast cells, inhibiting the release of histamine and leukotrienes. As such, it is useful as a prophylaxis against acute attacks in patients with asthma. It has no beneficial value in the management of acute bronchoconstriction. The latest approach in reactive airway management is to block the conversion of arachidonic acid to leukotrienes. Similar to other measures that are directed at reduction of the inflammatory response, these medications prevent acute exacerbations of asthma or bronchospasm but are not appropriate for acute attacks. An example of a leukotriene inhibitor is montelukast sodium, which specifically blocks the leukotriene D\(_4\) receptor.

Respiratory arrest in the perioperative period is commonly caused by airway obstruction, laryngospasm, or a foreign body in the airway. A further differential diagnosis and treatment algorithm is provided in Figure 3-4.\(^{29,30}\)

**Perioperative Effects of Tobacco Smoking**

Cigarette smoke contains over 3,000 constituents, some of which are toxic or tumorigenic. Carbon monoxide, produced as an end product of burning tobacco, has
a 200 times greater affinity than oxygen for the hemoglobin (Hb) molecule. Carboxyhemoglobin, which can be as high as 15%, predisposes a patient to perioperative hypoxia. Pulse oximetry fails to recognize the presence of carboxyhemoglobin (COHb) as distinct from oxyhemoglobin. Therefore, a patient with 10% COHb may display a saturation of 100% when, in fact, the actual saturation may be closer to 90%. In addition, carboxyhemoglobin has the effect of shifting the oxygen dissociation curve to the left (ie, less oxygen is delivered to tissues; Figure 3-5). The relative hypoxia detected by the body (more specifically, the kidneys) results in an increased release of erythropoietin with a resultant thrombocytosis. In addition, carbon monoxide has a direct effect on the myocardium with increased automaticity and a lower threshold for ventricular fibrillation.31

The pulse oximeter functions by positioning a pulsating arterial bed between a two-wavelength light-emitting diode and a detector (photodiode). One wavelength is 660 nm (red), and the other is 940 nm (infrared). Oxygenated hemoglobin absorbs more of the 940 nm wavelength than does reduced hemoglobin, which, in turn, absorbs more of the 660 nm wavelength. The percent saturation reading (SpO2%) is determined from the ratio of oxygenated hemoglobin to the total hemoglobin. A common difficulty in determining SpO2 occurs secondary to changes in the strength of the arterial pulse or patient movement, resulting in either no signal or artificially low readings. Causes of these errors include hypothermia, hypotension, the use of vasopressors, electrocautery, artificial or opaque nail finishes, and additional monitors such as an automatic blood pressure cuff or arterial line on the same arm. The effects of other potential sources of error in SpO2 measurement are given in Table 3-5.32

Nicotine as a vasoconstrictor can have a significant effect on the cardiovascular system. Similar to other vasoconstrictors, increases in heart rate, blood pressure, and peripheral vascular resistance are seen secondary to the activation of the sympathetic nervous system and the release of catecholamines from the adrenal medulla. This effect persists for 30 minutes after smoking a cigarette. Coronary artery vascular resistance is similarly affected, potentially leading to further limited blood flow in areas predisposed to ischemia. Nicotine can also lower the threshold for ventricular fibrillation. Carbon monoxide and nicotine have a relatively short half-life (carbon monoxide t1/2 = 4 h; nicotine t1/2 = 30–60 min). With regard to potential cardiac complications, there is a direct benefit of abstinence from smoking for 12 to 24 hours.
Unfortunately, detrimental effects on ciliary function and mucus overproduction by respiratory mucosa as a response to tobacco can last for months after smoking cessation. Additional detrimental effects include increased bronchial reactivity, macrophage dysfunction, and changes in pulmonary surfactant. Assuming a smoker has not had long-term deleterious effects related to COPD, these changes require 6 to 8 weeks for complete reversal. Postoperative pulmonary complications including atelectasis, pneumonia, and bronchospasm are much more likely to occur in individuals who smoke.

Interestingly, increased pulmonary complications have been demonstrated when a patient ceases smoking < 8 weeks prior to a planned surgery. Therefore, recommendations to the smoking patient should include at least a 12- to 24-hour smoking “fast” or, more desirably, a cessation of smoking for 8 weeks or more. Patients should be counseled that cessation for periods < 8 weeks may actually predispose the individual to increased pulmonary complications.

In recent studies the effects of second-hand or passive smoke have been analyzed. The risks of chronic bronchitis, asthma, and wheezing were all higher in patients exposed to involuntary tobacco exposure, especially in the workplace with a daily exposure of > 8 h/d. The exposure levels in the workplace have been estimated to be higher than at home, and the time spent at work is usually longer. It is prudent to determine secondhand smoke exposure in the perioperative management of the surgical patient.

### Obesity

The difference between normality and obesity is arbitrary, but an individual with increased fat tissue to such an extent that physical and mental health are affected and life expectancy is reduced should be considered obese. Body mass index (BMI) is widely used in clinical and epidemiologic studies. It is the ratio of body weight (in kilograms) to height (in meters squared). A patient with a BMI of < 25 kg/m² is considered normal. A patient with a BMI of 25 to 30 kg/m² is overweight but at relatively low risk for serious medical complications; one with a BMI of > 30 kg/m² is obese with a higher risk of morbidity and mortality. Morbidly obese individuals have an increased risk of death from cardiorespiratory and cerebrovascular disorders, diabetes mellitus, and certain forms of cancer in addition to many other diseases. These risks are proportional to the duration of obesity. Weight loss reduces the risks but only over time; weight reduction immediately prior to surgery has not been shown to reduce perioperative risk.

Approximately 5% of obese individuals have obstructive sleep apnea (OSA), which is characterized by episodes of apnea or hypopnea during sleep. Obstructive apnea is characterized by apnea despite a continuous respiratory effort against a closed airway. Central apnea is characterized by the loss of ventilatory effort. Many patients diagnosed with OSA can have periods of central apnea during sleep as well. Apnea is typically defined as 10 seconds or more of total cessation of airflow. Hypopnea is defined as a reduction in airflow (typically 30–50%) or a reduction sufficient to lead to a 4% decrease in arterial oxygen saturation. The number of apneic or hypopneic episodes believed to be significant is five or more per hour. The exact number is arbitrary, as are the definitions of apnea and hypopnea used by various sleep laboratories. Often individuals with OSA are noted to have nocturnal snoring and daytime hypersomnolence. OSA can lead to hypercapnia, systemic and pulmonary hypertension, and cardiac arrhythmias.

In the perioperative period, episodes of OSA are most frequent during rapid eye movement sleep, the extent of which is relatively low in the initial postoperative period but in excess on the third to fifth postoperative nights. Caution should therefore be exercised any time anesthetic agents are used in a patient with a history of OSA. In addition, the continued use of medical therapies including continuous positive airway pressure should be stressed in the perioperative period.

Morbid obesity is characterized by reductions in functional residual capacity (the volume remaining in the lungs after a normal quiet expiration), expiratory reserve volume (the volume of air that can forcefully be expired after a normal resting expiration), and total lung capacity. These changes have been attributed to mass loading and splitting of the diaphragm (Figure 3-6). Anesthesia compounds these problems and impairs the ability of the obese to tolerate periods of apnea.
Ventilation and Capnography

Capnography is defined as the measurement and display of exhaled carbon dioxide. Increases in end-tidal CO₂ combined with decreases in the respiratory rate of the individual have been demonstrated to be an effective way to detect hypoventilation and respiratory depression. Pulse oximetry, in contrast, indirectly measures oxygenation (partial pressure of oxygen in arterial blood). Based on the oxygen-hemoglobin dissociation curve (see Figure 3-5), there can be a significant decline in oxygen saturation that can go undetected by the pulse oximeter. Capnography, by detecting hypoventilation, may be used to prevent hypoxia; upon noting hypoventilation, the practitioner can take measures to improve patient ventilation. Proponents of capnography for non intubated sedation advocate its use over other forms of ventilatory monitors that can experience interference from operatory noise, clothing, or surgical drapes. These methods include observation of chest wall movements, plethysmography, auscultation of breath sounds (precardial stethoscope), or palpation or movement of the reservoir bag.

Opponents to the use of capnography for nonintubated sedation cite sampling errors, particularly in individuals who are mouth breathing when nasal sampling is being used.38–40

Endocrine Assessment

Diabetes Mellitus

Perioperative care of the diabetic patient depends on identification and assessment of the current status of end-organ disease. Long-standing diabetics frequently have compromise in one or more organ system. Commonly associated diseases include atherosclerosis, coronary artery disease, hypertension, cardiomyopathy, cerebrovascular disease, peripheral vascular disease, peripheral and autonomic neuropathy, and/or renal insufficiency. Preoperative evaluation should focus on these concerns, and events of prior surgeries should be reviewed. For more complex procedures, laboratory values that may be reviewed include blood glucose, blood urea nitrogen, creatinine, urinalysis (for glucose, ketones, and proteins), and glycosylated hemoglobin (Hb A₁c) levels. Hb A₁c levels reflect the adequacy of glucose control during the previous 1 to 3 months. Levels in nondiabetics range from 5 to 7% of hemoglobin. Levels in diabetics with poor long-term glucose control exceed 8%.

With more procedures being performed on an outpatient basis and the length of hospital stays being shortened dramatically, perioperative management of the diabetic patient has become more complicated. Many factors are present that determine the glycemic response, including insulin secretion, insulin sensitivity, overall metabolism, and nutritional intake in addition to the stress and length of the procedure. Surgical stress and some general anesthetic agents, themselves, are associated with increases in the counter-regulatory hormones epinephrine, norepinephrine, glucagon, growth hormone, and cortisol. The effect of these hormones is to elevate insulin resistance, which increases hepatic glucose production and decreases peripheral glucose use. Patients receiving pharmacologic therapy to control their diabetes may also be susceptible to hypoglycemia, especially when fasting preoperatively. Although hypoglycemia can cause significant morbidity, marked hyperglycemia should also be avoided since it can lead to dehydration and electrolyte disturbances and impaired wound healing and predispose to infection or diabetic ketoacidosis in the patient with type 1 DM. This is not to say that patients with historically poor control of their disease should be rapidly normalized presurgically; little evidence supports this approach. In general, the goal for glucose control during surgery should be between 150 and 200 mg/dL. The more unstable the diabetes, the more frequently this level should be assessed in the perioperative period.

As in all patients, underlying cardiac, pulmonary, renal, and electrolyte disturbances and anemia should be evaluated. Assessment should include a focus on the microvascular (ie, renal insufficiency,
retinopathy), macrovascular (including atherosclerosis, coronary artery disease, hypertension), and neuropathic signs related to poor diabetes control. Medication use and insulin regimen should be recorded. Management of the patient should be coordinated with the individual who manages the patient’s daily protocol. The following are recommended guidelines in the management of patients with diabetes who require a period of nothing by mouth prior to their planned procedure.

Type 2 DM Controlled by Diet Only
Measurement of blood glucose should be considered prior to the procedure, after the procedure, and intraoperatively for longer surgeries. Hyperglycemia is treated with short-acting insulin (regular or lispro), usually administered subcutaneously. It is prudent to remind patients prior to discharge of the signs and symptoms of hyperglycemia (discussed below) and to reinforce guidelines for contacting their physician.

Type 2 DM Treated with Oral Hypoglycemic Agents
Oral hypoglycemic agents are generally administered the day prior to surgery and withheld the day of surgery. If patients manifest marked hyperglycemia, supplemental insulin may be indicated; the surgery may be performed if electrolyte levels are acceptable. Table 3-6 provides information on common oral hypoglycemic agents.

Types 1 and 2 DM Treated with Insulin
For individuals who take long-acting insulin (ie, extended zinc suspension or glargine; Table 3-7), a switch to an intermediate-acting type is initiated a day or two prior to surgery. The regulation of intermediate insulin is then adjusted based on the likelihood of the patient eating lunch. If the likelihood of oral intake at lunch time is high, two-thirds of the normal intermediate dose is given on the morning of the procedure. If the patient is treated with a twice-daily dose of insulin, then one-half of the total morning dose of insulin (including short-acting) should be administered in the morning as intermediate insulin. If the likelihood of consuming lunch is low, one-half of the total morning dose of insulin (including short-acting) should be administered as intermediate-acting insulin for the patient treated with a single insulin dose and one-third for those on a twice-daily regimen. For the patient taking multiple doses of short-acting insulin, one-third of the pre-meal dose of short-acting insulin is administered. Patients treated with continuous insulin infusion therapy (with an insulin pump) are treated with their usual basal infusion rate.

Individual modifications of insulin therapy may be required, and it is advisable to discuss the management with the patient’s physician. Procedures scheduled later in the day can be more complex to manage, and intravenous glucose infusion and/or supplemental short-acting insulin may be necessary. Long complex operative procedures may require intravenous insulin regimens. Table 3-7 reviews the common types of insulin and their onset, peak activity, and duration.

Hypoglycemia and Hyperglycemia: Identification and Management
Direct neurologic symptoms and an adrenergic response characterize the manifestations of hypoglycemia. Neuroglycopenia generally begins with confusion, irritability, fatigue, headache, and somnolence. Prolonged severe hypoglycemia can cause seizures and even focal neurologic deficits, coma, and death. Therefore, any new neurologic symptom in the postoperative period should be investigated for hypoglycemia because prolonged deficit of glucose can result in irreversible neurologic deficits. The adrenergic symptoms include anxiety, restlessness, diaphoresis, tachycardia, hypertension, arrhythmias, and angina owing to catecholamine release in response to hypoglycemia. Recognition of perioperative hypoglycemia can be difficult initially because presenting symptoms can be altered or absent as a result of the effects of anesthetic agents, analgesics, and sympatholytic agents. In addition, diabetics with autonomic neuropathy have blunting of the adrenergic response associated with hypoglycemia.

Hypoglycemia is defined as glucose < 50 mg/dL in adults and < 40 mg/dL in

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Example(s)</th>
<th>Mechanism</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>α-Glucosidase inhibitors</td>
<td>Acarbose, miglitol</td>
<td>Inhibit intestinal brush border oligo- and disaccharidases</td>
<td>No efficacy until patient is eating</td>
</tr>
<tr>
<td>Biguanides</td>
<td>Metformin</td>
<td>Sensitize target tissue (muscle, fat) to insulin action</td>
<td>May potentiate the risk of developing lactic acidosis perioperatively</td>
</tr>
<tr>
<td>Thiazolidinediones</td>
<td>Pioglitazone, rosiglitazone, troglitazone</td>
<td>Improve peripheral glucose uptake</td>
<td>No increased incidence of lactic acidosis</td>
</tr>
<tr>
<td>Sulfonylureas</td>
<td>Glipizide, chlorpropamide</td>
<td>Stimulate insulin secretion</td>
<td>Higher potential for developing perioperative hypoglycemia</td>
</tr>
</tbody>
</table>
children. Its treatment is a glucose source if oral intake is possible; however, to avoid the risk of aspiration and delay in absorption, 50 mL of 50% (25 g) of glucose should be administered intravenously. Each milliliter of D50 raises the blood glucose approximately 2 mg/dL. Glucagon (1–2 mg), diazoxide, and octreotide have been used but are typically reserved for sulfonyl urea–induced hypoglycemia.

Perioperatively many regulatory hormones that oppose insulin action are released. Catecholamines, glucocorticoids, growth hormone, and glucagon can cause plasma glucose levels of > 180 mg/dL, exceeding the capacity of the kidney and resulting in glycosuria. Glucose-induced diuresis can occur, resulting in dehydration or the formation of ketone bodies, which, in turn, results in diabetic ketoacidosis. Treatment includes the use of intravenous insulin and appropriate rehydration. One unit of regular insulin typically lowers the glucose 25 to 30 mg/dL in a 70 kg individual. Subcuticular injection should be avoided in the perioperative period owing to unpredictable cutaneous blood flow.

### Adrenal Assessment

Adrenal insufficiency is classified as either primary, owing to disease of the adrenal glands themselves, or secondary, owing to decreased adrenocorticotropic hormone (ACTH) because of pituitary or hypothalamic disorders. Primary adrenal insufficiency is also known as Addison’s disease and is thought to be the result of an autoimmune process. Other causes of primary adrenal insufficiency include chronic granulomatous disease including tuberculosis.

Secondary adrenal insufficiency is most commonly seen in patients on chronic glucocorticoid therapy. Patients on steroid therapy may have ACTH suppression a full year after steroid therapy. Symptoms include fatigue, weakness, anorexia, nausea and vomiting, and weight loss. Only in primary adrenal insufficiency is ACTH elevated, indirectly resulting in increased skin pigmentation, especially in skinfolds. In primary adrenal insufficiency, aldosterone levels are low, resulting in dehydration with hyponatremia and hyperkalemia since the role of aldosterone in the kidney is resorption of sodium (and water) and excretion of potassium. In secondary adrenal insufficiency, there are often other endocrine abnormalities present.

In individuals with an intact hypothalamic-pituitary-adrenal axis undergoing a stressful event such as a surgical procedure, the adrenal glands increase their baseline secretion of cortisol. Increasing cortisol helps maintain hemodynamic stability in the face of stress. Patients with long-term exogenous steroid use have a blunted response to surgical stress compared to that of normal controls, with resultant lower cortisol levels.

Adrenal crisis is usually seen in patients with adrenal suppression and is precipitated by a stressor, typically surgery, trauma, or sepsis. Patients may experience intractable nausea and vomiting, abdominal pain, fever, lethargy, and coma. Hypotension and a narrow pulse pressure (the difference between systolic and diastolic pressure) are evident as shock ensues. Based on these potential risks and anecdotal reports published, supraphysiologic corticosteroid regimens have been recommended for patients on exogenous steroids.

Recent evidence suggests that patients on long-term steroids who receive no perioperative coverage suffer a 1 to 2% risk of incurring a hypotensive crisis. Studies support maintaining patients on their daily steroid dosage throughout the perioperative period or providing smaller steroid dosages rather than the supraphysiologic dosages once routinely recommended. An exception to this practice is the critically ill patient, in whom supraphysiologic dosages are often administered. An example of a suggested steroid regimen based on the degree of stress is provided in Table 3-8.

### Thyroid Assessment

Hyperthyroidism primarily affects women, with a female-to-male ratio of approximately 8:1. Common causes of hyperthyroidism include Graves’ disease (a toxic diffuse goiter secondary to an autoimmune reaction caused by stimulatory antibodies to the thyroid-stimulating hormone receptor), toxic nodular goiter, exogenous thyroid hormone (iatrogenic), and iodine administration. The effects of excess thyroid hormone include tachycardia, atrial fibrillation, premature ventricular contractions, worsening of angina pectoris, and high-output cardiac failure.

### Table 3-7  Onset, Peak, and Duration of Common Insulin Preparations

<table>
<thead>
<tr>
<th>Type of Insulin</th>
<th>Example</th>
<th>Onset</th>
<th>Peak</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rapid-acting</td>
<td>Lispro</td>
<td>5–15 min</td>
<td>30–75 min</td>
<td>2–4 h</td>
</tr>
<tr>
<td>Short-acting</td>
<td>Aspart</td>
<td>5–15 min</td>
<td>1–2 h</td>
<td>3–6 h</td>
</tr>
<tr>
<td>Intermediate-acting</td>
<td>Regular</td>
<td>30–45 min</td>
<td>2–3 h</td>
<td>4–8 h</td>
</tr>
<tr>
<td>Intermittent-acting</td>
<td>NPH</td>
<td>2–4 h</td>
<td>4–8 h</td>
<td>10–16 h</td>
</tr>
<tr>
<td>Long-acting</td>
<td>Zinc suspension</td>
<td>2–4 h</td>
<td>4–8 h</td>
<td>10–16 h</td>
</tr>
<tr>
<td>Prolonged intermediate-acting</td>
<td>Extended zinc</td>
<td>3–5 h</td>
<td>8–12 h</td>
<td>18–20 h</td>
</tr>
<tr>
<td>suspension</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Premixed combination insulin</td>
<td>70/30 or 50/50</td>
<td>30–60 min</td>
<td>Early–late</td>
<td>About 18 h</td>
</tr>
<tr>
<td>(NPH/regular)</td>
<td></td>
<td></td>
<td>peak: 2–12 h</td>
<td></td>
</tr>
</tbody>
</table>
Failure to recognize that a patient has uncontrolled hyperthyroidism can result in a thyroid storm, which can manifest either during the procedure or in the postoperative period. It is characterized by marked tachycardia, hyperthermia, weakness, and an altered level of consciousness. Untreated, the result can be congestive heart failure and/or cardiovascular collapse. Treatment includes airway and ventilatory support with increased minute ventilation to control excessive CO₂ production. Body temperature should be aggressively managed with cool intravenous fluids, cooling blankets, and decreased ambient temperature. β-Blocker administration should be started immediately to interrupt the adrenergic response. Traditionally, a nonselective β-blocker, propranolol, has been used. More recently the use of esmolol, a shorter-acting β₁-selective blocker has been advocated. Patients with COPD, asthma, and congestive heart failure are more likely to tolerate therapy with a β₁-selective agent. Hemodynamic monitoring and the correction of fluid and electrolyte imbalances should be performed. The differential diagnosis of a thyroid storm includes malignant hyperthermia (MH; see below), neuroleptic malignant syndrome, and pheochromocytoma.

Women are ten times more likely to develop hypothyroidism than are men. The most common cause is iatrogenic, secondary to surgical resection or radioactive ablation of the thyroid gland. Hashimoto’s thyroiditis, an autoimmune disorder characterized by the presence of antimicrobial antibodies, is the most common noniatrogenic cause of hypothyroidism. Hypothyroidism is usually insidious in onset and often goes unrecognized despite multisystem effects. The most common signs and symptoms include lethargy, constipation, cold intolerance, weight gain, and anorexia. Although severe hypothyroidism can result in increased morbidity and mortality, most experts agree that mild to moderate hypothyroidism poses no increased surgical risk. Elective surgery should be postponed in hypothyroid patients until adequate replacement therapy is administered. Usually this can be accomplished by oral thyroxine supplementation. Two weeks are required before the patient has symptomatic improvement. Triiodothyronine, which is the active hormone, can be administered for a more acute response, but it usually takes more than 2 weeks until the thyroid-stimulating hormone, the marker for adequate thyroid function, normalizes.⁴²

Malignant Hyperthermia
MH is a rare autosomal dominant trait in which individuals inherit hypersensitivity to specific trigger agents that cause the rapid accumulation of calcium into the sarcoplasmic reticulum of skeletal muscle. This causes sudden hypermetabolic reactions, leading to hyperthermia and massive rhabdomyolysis. Trigger agents include potent volatile anesthetic agents and succinylcholine (a depolarizing muscle relaxant). Halothane has traditionally been described as a causative agent and forms the basis of the diagnostic test to confirm MH. However, all volatile agents, including sevoflurane according to recent reports, can induce MH.⁴⁵

The reaction that typically occurs is abrupt and severe, requiring immediate attention. Elevation of end-tidal CO₂ is an early sign, prior to temperature elevation. The main treatment is dantrolene, a non-specific muscle relaxant. Its mechanism is likely the blockade of the release of calcium from the sarcoplasmic reticulum. In an acute episode of MH, a supply of at least 36 vials of dantrolene should be available for immediate use; this corresponds to a maximum dose of 10 mg/kg in a 70 kg adult. In an acute attack dantrolene is administered repeatedly in 2 to 3 mg/kg doses every 5 to 10 minutes. Each vial needs to be reconstituted with 60 mL of

<table>
<thead>
<tr>
<th>Surgical Stress</th>
<th>Steroid (Hydrocortisone) Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>25 mg on day of surgery</td>
</tr>
<tr>
<td>Moderate</td>
<td>50–75 mg on day of surgery, 1 or 2 d taper</td>
</tr>
<tr>
<td>Major</td>
<td>100–150 mg on day of surgery, 1 or 2 d taper</td>
</tr>
</tbody>
</table>

Adapted from Salem M et al.⁴₄
sterile water. Although the use of dantrolene has reduced the mortality risk from 50% prior to its use, there still is approximately a 10% mortality rate.

There is an estimated occurrence of MH in 1 of 15,000 children and 1 of 50,000 adults. Those at risk for an attack include survivors of an MH reaction and individuals with muscular dystrophy. The clinical sign of masster muscle spasm during anesthesia with halothane or succinylcholine may also indicate a susceptibility to MH. The in vitro caffeine halothane contracture test is used to evaluate individuals susceptible to developing MH when exposed to triggering agents. Diagnostic tests based on deoxyribonucleic acid are currently available for MH-susceptible individuals. In addition to trigger agents, phenothiazines (such as prochlorperazine) should be avoided since there is a possible association between MH and neuroleptic malignant syndrome (NMS). NMS is a rare, occasionally lethal, idiosyncratic complication associated with neuroleptic antipsychotic drugs. NMS is characterized by high temperature and muscle rigidity. Anxiety and agents with sympathomimetic activity, especially α-agonists, have been demonstrated to aggravate MH experimentally. Agents that some authors have recommended to be avoided owing to sympathetic effects include ketamine and atropine. The use of dantrolene prophylaxis in MH patients is uncommon in view of the low likelihood (0–0.62%) of an MH reaction when a trigger-free anesthetic regimen is used. Dantrolene is associated with a high frequency of muscle weakness and postoperative nausea. In the past, outpatient surgery was discouraged. It is now recommended that careful postoperative monitoring be continued for at least 4 hours. However, most oral and maxillofacial surgeons likely avoid performing outpatient sedation for someone with a personal or family history of malignant hyperthermia owing to the factors described.45,46

References
Preoperative Patient Assessment

Joel M. Weaver, DDS, PhD

The primary purpose of preoperative patient assessment is to provide sufficient information to the surgical and anesthetic team members to permit them to formulate the most appropriate surgical and anesthetic plans. The same process should be used for both office and hospitalized patients, including trauma victims; medically, mentally, or physically compromised patients; and healthy patients having elective surgery with either local anesthesia alone, conscious sedation, deep sedation, or general anesthesia. Depending on the variables discovered in the assessment, modifications to the usual surgical and anesthetic regimens may be necessary to improve the chances of attaining a satisfactory outcome.

The components of the preoperative assessment are (1) a review of the previous medical records if available, including all medical, surgical, and medication information; (2) a personal interview with the patient or knowledgeable guardian to obtain additional past medical and surgical histories; (3) a focused physical and psychological examination of the patient, with emphasis on the cardiovascular and respiratory systems and the adequacy of the airway in regard to the potential for difficulty in attaining and maintaining its patency during deep sedation or general anesthesia; (4) a review of results of the medical tests and referral for consultation if needed; (5) a determination of the patient’s perioperative risk; and (6) a thorough explanation of the various treatment options in discussion with the patient or guardian to assist with their treatment decisions and to obtain their informed consent.

Information such as current medications, drug allergies, the likelihood of pregnancy, family history of malignant hyperthermia, a significant medical or surgical history, and, if the procedure is scheduled at the time of evaluation, an assessment of fluid or food ingestion may influence the surgeon’s choice on how to proceed.

A review of the previous medical records can provide a wealth of information that the patient may not know or be able to relate during their interview. For example, if there is previous documentation of a “difficult airway” whereby an anesthesiologist had significant difficulty with mask ventilation and needed multiple attempts to intubate a severely retrognathic patient, an oral surgeon might not choose to administer deep sedation or light general anesthesia to that patient in the office. Better alternatives might include light conscious sedation in the office with only those drugs for which pharmacologic antagonists exist, or possibly an awake fiberoptic intubation in the office, surgicenter, or hospital prior to the induction of general anesthesia. For patients who are poor historians, previous medical records may be the sole source of information concerning previous surgeries and medical problems. Unfortunately, timely access to previous medical records may be difficult or impossible.

Usually, information concerning the patient’s past medical, surgical, and anesthetic history can be gathered by a personal or telephone interview. Although completion of a health questionnaire or medical history form by the patient may be a starting point for the interview, it alone does not meet the important goal of establishing a personal dialogue with the patient to ensure that this information is as complete and accurate as possible. The true value of the medical history form is to alert the interviewer as to which areas need further explanation. For example, a positive indication of asthma by the patient on a health screening questionnaire is relatively worthless information by itself; it must be followed up with further questioning concerning the frequency of attacks, its precipitating factors, successful measures for treatment, the most recent attack, and the degree of severity of symptoms, including previous emergency room treatments for severe asthmatic episodes, hospital admissions, or even endotracheal intubation in the intensive care unit for status asthmaticus. Only after appropriate questioning has been completed for each positive item on the past medical history form can the patient’s past medical, surgical, and anesthetic history be considered adequate.
Obviously, the additional information gleaned from the patient must be written on the form for review at the time of the procedure as well as for proper medicolegal documentation.

Once the information is gathered, the surgeon should categorize the surgical patient according to the American Society of Anesthesiologists (ASA) Classification of Physical Status (Table 4-1), even if only local anesthesia is to be used. ASA PS-1 patients would be expected to have a lower risk of perioperative complications than ASA PS-4 patients. Despite a lack of absolute precision in accurately classifying the perioperative risk for all patients, this index is, nevertheless, commonly used to help identify certain risk factors so that modifications in the treatment plan can be accomplished. For instance, ambulatory general anesthesia in a dental office for ASA PS-1 and many ASA PS-2 patients is considered safe and cost effective, whereas ASA PS-4 patients would only receive local anesthesia and perhaps light levels of anxiolysis in an office setting.

**Assessment of Cardiovascular Disease**

**Cardiac Disease**

Cardiac disease can be subdivided into ischemic and nonischemic disease. Ischemic disease includes atherosclerotic heart disease, angina pectoris, and previous myocardial infarction. Nonischemic disease includes a wide variety of etiologies, such as vascular (polyarteritis nodosa), congenital (tetralogy of Fallot), infectious (bacterial endocarditis), inflammatory/autoimmune (scleroderma), traumatic (cardiac contusion), toxic (alcoholic cardiomyopathy), pulmonary (cor pulmonale), metabolic (obesity), neoplastic (carcinoid), and endocrine (hyperthyroidism).

In a landmark article, Goldman and colleagues developed a multifactorial index to assess cardiac risk associated with a variety of noncardiac procedures such as orthopedic and general surgery. This prospective study followed 1,001 patients older than 40 years at Massachusetts General Hospital until discharge and recorded all complications. Various potential risk factors for cardiac complications were correlated with actual complications, and a risk index based on a points system was subsequently formed. Of the 537 Class I patients, with 0 to 5 points, only 0.7% had life-threatening complications and 0.2% experienced cardiac death. Patients with 6 to 12 points were placed into Class II, whereas those with 13 to 25 points comprised Class III. Class IV patients, with 26 or more points, had a 22% incidence of life-threatening complications and 56% experienced cardiac death. Of all these factors, a previous history of congestive heart disease was the most predictive of complications, followed by a myocardial infarction within the previous 6 months.

Detsky and colleagues modified the Goldman Index by including unstable angina and remote myocardial infarction as additional risk factors for perioperative cardiac complications in vascular surgery patients. They simplified the scoring system of Goldman and colleagues into three classes, improving predictive accuracy. Table 4-2 represents Goldman and colleagues’ and Detsky and colleagues’ factors for perioperative cardiac risk.

Although anesthetic and surgical care have markedly improved in the last 25 years and risks may be less in some areas, Kenchaiah and colleagues recently reported that in both men and women who are obese, the risk of heart failure was doubled. With the increasingly high prevalence of obesity in the United States, this risk factor, among others, will prove more important in determining the risk of poor outcomes in the future.

Ischemic Heart Disease  **Angina Pectoris and Coronary Artery Disease**  Angina pectoris is typically a substernal chest pain or pressure that may radiate to either arm, the neck, or the mandible that is initiated by exercise, mental stress, pain, or other factors that produce increased myocardial oxygen demand in the presence of reduced oxygen delivery to the myocardium. It is most often caused by coronary artery disease, although other precipitating factors include severe anemia, hypotension, vasoconstrictor overdose, and coronary artery spasm. Angina pectoris may be classified as stable, unstable, or variant.

Unfortunately, the symptoms of angina pectoris may be confused with mitral valve prolapse, esophageal reflux, esophageal spasm, peptic ulcer disease, biliary disease, hyperventilation, musculoskeletal disease, and pulmonary disease. The diagnosis of angina pectoris is therefore not necessarily easy for the clinician to establish.

Stable angina pectoris is diagnosed when there is minimal change over 2 months regarding precipitating factors, frequency, intensity, duration, and treatments for successful termination of the attacks. Unstable angina pectoris relates to

### Table 4-1  American Society of Anesthesiologists Physical Status Classification

<table>
<thead>
<tr>
<th>Classification</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>PS-1</td>
<td>Normal healthy patient</td>
</tr>
<tr>
<td>PS-2</td>
<td>Patient with mild systemic disease</td>
</tr>
<tr>
<td>PS-3</td>
<td>Patient with severe systemic disease</td>
</tr>
<tr>
<td>PS-4</td>
<td>Patient with severe systemic disease and a constant life threat</td>
</tr>
<tr>
<td>PS-5</td>
<td>Moribund patient who is not expected to survive without the operation</td>
</tr>
<tr>
<td>PS-6</td>
<td>Declared brain-dead donor patient for organ harvest</td>
</tr>
</tbody>
</table>

recent changes in some or all the above factors. Thus, unstable angina is defined by chest pain encountered during less than the usual exercise, or that lasts longer, is more intense, more frequent, or requires more than normal measures to terminate it. Unstable angina is also termed preinfarction angina since it may be the harbinger of an impending myocardial infarction. Variant angina, also known as Prinzmetal’s angina, may occur in patients who have no detectable coronary artery disease but in whom coronary vasospasm occurs periodically, even at rest or with ordinary exercise. Cardiac dysrhythmias are frequently present during such spasms. These patients are frequently prescribed calcium channel antagonists prophylactically.

Patients who elicit a history of angina pectoris must be thoroughly interviewed to permit the practitioner to properly place them into the appropriate category. Patients who are judged to have reasonable cardiac reserve and are considered stable are certainly good candidates for relatively simple office procedures while being carefully monitored. Light to moderate levels of conscious sedation may prove beneficial in preventing an angina attack, particularly in the anxious patient, by reducing the stress of the procedure and decreasing myocardial oxygen demand. Using profound local anesthesia with no more than 40 µg of epinephrine has been recommended by Malamed for medically compromised dental patients. These patients should be told to take their usual prophylactic medications such as β1-adrenergic antagonists perioperatively, and to bring their nitroglycerin sublingual tablets or spray on the day of surgery to abort an attack if it were to occur.

Common risk factors for coronary artery disease include advanced age, diabetes mellitus, hypertension, peripheral vascular disease, hypercholesterolemia, obesity, cigarette smoking, sedentary lifestyle, and family history of coronary artery disease. According to Tarhan and colleagues, the perioperative risk of an acute myocardial infarction in patients without a history of myocardial infarction is 0.13%. Numerous retrospective studies involving large groups of patients indicate that the risk of a second myocardial infarction in the perioperative period seems to stabilize at approximately 6% after 6 months from the initial infarction. However, the 6% re-infarction rate is considerably higher than the 0.13% incidence of perioperative infarction for the same procedures in patients without previous myocardial infarction.

**Congestive Heart Disease** Multiple studies indicate that the presence of congestive failure is the single most important risk factor for perioperative cardiac morbidity independent of the presence of dysrhythmias, cardiomyopathy, valvular disease, or coronary artery disease. Appropriate strategies for perioperative management include optimization with careful attention to fluid management and maximizing therapies such as inotropes, diuretics, vasodilators, and antidysrhythmics.

The New York Heart Association (NYHA) functional classification of patients with heart disease (Table 4-3) is useful in categorizing patients who have
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Mitral stenosis is usually...
Mitral stenosis without regurgitation causes left atrial enlargement and ultimately congestive heart failure. Critical mitral stenosis is usually defined as an area < 1 cm². Because the atrial outflow is reduced, tachycardia reduces the flow into the left ventricle, which increases pulmonary congestion and decreases cardiac output. Thus, heart rate must remain reasonably normal, and the atrial “kick” associated with sinus rhythm may be necessary for maintaining cardiovascular stability.

**Mitral Insufficiency** Mitral insufficiency is frequently associated with mitral stenosis as the result of rheumatic heart disease. It produces a holosystolic blowing murmur heard best at the apex. It is often tolerated until the patient begins to develop signs and symptoms of congestive heart failure. Mitral insufficiency is associated with risk factors such as congestive heart failure or recent myocardial infarction. As in aortic insufficiency, attention must be given to preventing excessive fluid administration and to maintaining forward blood flow with moderate increases in heart rate and vasodilation.

**Mitral Valve Prolapse** Mitral valve prolapse, or Barlow’s syndrome, is associated with a bulging or prolapse of the mitral valve leaflets into the left atrium during systole. Typically, it produces a nonejection click cardiac murmur, often called “click-murmur syndrome,” heard best at the cardiac apex and may be associated with a regurgitant murmur. The diagnosis is normally confirmed with echocardiography. Although not a benign condition, it is less likely to be problematic than many of the above valvular diseases. It is often associated with a history of chest pain, anxiety attacks, dizziness, supraventricular tachycardia, and palpitations. These patients are at risk of paroxysmal tachydysrhythmias and sudden death. Occasionally, mitral valve prolapse is associated with significant regurgitation and endocarditis. Appropriate care includes measures to prevent significant positive inotropic and chronotropic responses to stress by adequate control of anxiety and pain, judicious use of β-adrenergic agonists such as epinephrine, and careful monitoring of cardiovascular parameters during surgery.

**Cardiomyopathy** Cardiomyopathy may result from a variety of causes not related to valvular or coronary disease, such as systemic disease, infection, or drug and alcohol abuse. The degree of cardiac impairment can be estimated by invasive or noninvasive measurement of the cardiac ejection fraction (percent EF); this is the percentage of left ventricular blood volume ejected into the aorta during each contraction. The normal value is approximately 70% and should increase with exercise or stress, whereas an EF of 30% is usually associated with decreased exercise tolerance. Patients with an EF of 15% or less have significant physiologic impairment and may be candidates for cardiac transplantation.

There are three classes of cardiomyopathy: dilated, nondilated, and hypertrophic. The typical findings associated with dilated cardiomyopathy include a marked increase in left ventricular end-diastolic volume. The perioperative implications of dilated cardiomyopathy include optimization of function including careful fluid management and maximizing therapies such as inotropes, diuretics, vasodilators, and antidysrhythmics, as in the management of congestive heart failure.

Patients with nondilated cardiomyopathy, also known as restrictive cardiomyopathy, present with rigid ventricles that impair diastolic filling, although the contractile function may remain somewhat intact. Right ventricular failure and elevated venous pressures are common. Dysrhythmias are a common cause of death in these patients; therefore, careful monitoring of cardiovascular parameters is essential to facilitate rapid recognition, diagnosis, and treatment of life-threatening dysrhythmias during any surgical procedure.

Hypertrophic cardiomyopathy, also known as idiopathic hypertrophic subaortic stenosis (IHSS), is usually an inherited autosomal dominant characteristic, although it can also be a result of longstanding hypertension. The intraventricular septum may be greatly thickened in asymmetric septal hypertrophy, or the hypertrophy may be concentric. Depending on the area of hypertrophy, left ventricular outflow obstruction may occur during systole. Furthermore, the septal leaflet of the mitral valve may not function properly owing to the hypertrophy of the septum, and mitral regurgitation may result. Fatal ventricular dysrhythmias may result in sudden death even in apparently healthy teenagers with undiagnosed hypertrophic cardiomyopathy. Ischemia within the hypertrophic segment may also result in myocardial infarction. Preparation for surgery would include careful monitoring of vital signs and minimization of those factors associated with increases in cardiac inotropy and rate, such as hypotension, vasodilation, β-adrenergic drugs, pain, and anxiety. Preoperative β-blockade, adequate hydration, and local anesthetics without epinephrine, unless absolutely necessary, are the usual components of good operative planning.

**Hypertension**

Hypertension is a very common disease. Although it can occur secondarily as a result of a definable cause such as hyperthyroidism or pheochromocytoma, it is most often a multifactorial primary disease of poorly understood origin, termed essential hypertension. In their seventh report, the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure recently revised their definition of hypertension.
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from previous reports, recognizing that early detection and treatment of prehypertension and hypertension is important and ultimately reduces risk. Important key messages in the joint committee’s latest report are as follows: (1) for patients < 50 years of age, systolic blood pressure > 140 mm Hg is a more important risk factor for cardiovascular disease than is diastolic pressure elevation; (2) beginning with a pressure of 115/75 mm Hg, the risk of cardiovascular disease doubles with every incremental increase of 20/10 mm Hg; and (3) a systolic pressure of between 120 and 139 mm Hg or a diastolic pressure of between 80 and 89 mm Hg is prehypertension, and lifestyle modifications are recommended to prevent cardiovascular disease (Table 4-4).11

Major risk factors for hypertension include smoking, dyslipidemia, diabetes mellitus, age > 60 years, gender (men and postmenopausal women), and family history of cardiovascular disease in women > 65 and men > 55 years. If untreated, it commonly causes coronary artery disease, cardiomegaly, congestive heart failure, and end-organ damage to vital tissues such as the heart, kidneys, retina, and brain. Elevated systolic blood pressure in the elderly appears to be a better predictor than elevated diastolic blood pressure of terminal end-organ damage, such as coronary artery/cardiovascular disease, stroke, renal failure, postoperative myocardial ischemia, and overall death.11,12

Because the increased peripheral vascular resistance produces a contracted intravascular volume, hypertensive patients are highly susceptible to the vasodilator effects of sedative and anesthetic agents that may result in a relative or absolute severe hypertensive episode.

Prolonged excessive hypotension in a patient with significant peripheral vascular disease who needs a relatively high pressure to perfuse vital organs may be more detrimental during surgery than permitting a modest degree of hypertension to continue. For patients planning for elective surgery who are found to be significantly hypertensive at the preoperative assessment, it is best to postpone the procedure until their physician can optimize their pressure and volume status. It is recommended that surgery be delayed, if possible, for poorly controlled hypertensive patients with blood pressure above the mild to moderate range (> 180/110 mm Hg).11,13

Acute treatment of hypertension at the time of elective surgery may produce blood pressure numbers that initially make the practitioner more comfortable before starting anesthesia and the procedure, but the less-than-optimized patient is much more likely to have significant labile hypertensive and/or hypotensive episodes during the course, and this may increase their risk of morbidity or mortality. As a general rule, patients with hypertension should take all of their normal antihypertensive medications with blood pressure numbers that initially make the practitioner more comfortable before starting anesthesia and the procedure, but the less-than-optimized patient is much more likely to have significant labile hypertensive and/or hypotensive episodes during the course, and this may increase their risk of morbidity or mortality. As a general rule, patients with hypertension should take all of their normal antihypertensive medications with blood pressure

Table 4-4 Classification of Hypertension

<table>
<thead>
<tr>
<th>Systolic BP</th>
<th>Diastolic BP</th>
<th>Category</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 120</td>
<td>&lt; 80</td>
<td>Normal</td>
</tr>
<tr>
<td>120–139</td>
<td>80–89</td>
<td>Prehypertension</td>
</tr>
<tr>
<td>140–159</td>
<td>90–99</td>
<td>Stage 1 hypertension (mild)</td>
</tr>
<tr>
<td>&gt; 160</td>
<td>&gt; 100</td>
<td>Stage 2 hypertension (moderate)</td>
</tr>
</tbody>
</table>

Adapted from the seventh report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure.11

BP = blood pressure.
Table 4-5  Duke Activity Status Index

<table>
<thead>
<tr>
<th>Activity</th>
<th>METs</th>
<th>Functional Capacity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Walk in house</td>
<td>1.75</td>
<td>Poor</td>
</tr>
<tr>
<td>Personal care (dress, bath, toilet)</td>
<td>2.75</td>
<td>Poor</td>
</tr>
<tr>
<td>Walk 1–2 blocks</td>
<td>2.75</td>
<td>Poor</td>
</tr>
<tr>
<td>Light work: dusting, washing dishes</td>
<td>2.7</td>
<td>Poor</td>
</tr>
<tr>
<td>Moderate work: vacuuming</td>
<td>3.5</td>
<td>Poor</td>
</tr>
<tr>
<td>Yard work: raking, mowing</td>
<td>4.5</td>
<td>Moderate</td>
</tr>
<tr>
<td>Sexual relations</td>
<td>5.25</td>
<td>Moderate</td>
</tr>
<tr>
<td>Climb stairs</td>
<td>5.5</td>
<td>Moderate</td>
</tr>
<tr>
<td>Golf, bowling</td>
<td>6</td>
<td>Moderate</td>
</tr>
<tr>
<td>Swim, basketball, ski</td>
<td>7.5</td>
<td>Excellent</td>
</tr>
<tr>
<td>Run</td>
<td>8</td>
<td>Excellent</td>
</tr>
</tbody>
</table>

Adapted from Hlatky MA et al14; Hollenberg SM.15
MET = metabolic equivalent; 1 MET = 3.5 mL/kg/min oxygen use.

infarction and the risk stratification for various noncardiac surgical procedures.13 Using these guidelines, the oral and maxillofacial surgeon can estimate the cardiac risks associated with the surgical procedure and decide whether the patient’s medical condition warrants further cardiac consultation. For instance, according to the algorithm in Figure 4-1, a cardiac patient with intermediate predictors of cardiac risk (mild angina or controlled congestive heart failure) with good exercise tolerance (equal to or greater than 4 METs) who is scheduled for a low-risk surgery (tooth extraction or tori removal) should not need an extensive cardiac work-up. However, that same patient scheduled for hemimandibulectomy, partial pharyngectomy, laryngectomy, or radical neck dissection with flap reconstruction that would entail large fluid shifts while under anesthesia for many hours (high surgical risk) and who has poor exercise tolerance (< 4 METs) should receive cardiac testing prior to surgery. Likewise, a patient with minor predictors of cardiac risk (advanced age or previous stroke) scheduled for the above high-risk surgery would not need cardiac consultation if his or her exercise tolerance was good but should be referred if the exercise tolerance was poor.

Although the guidelines in Figure 4-1 do not specifically define the surgical risk category of the most common oral surgical procedures, the surgeon should attempt to compare the severity of their proposed surgery with that of the examples provided. Perhaps a Le Fort III fracture would be similar in risk to an intermediate-risk acetabular fracture, whereas a dental implant would be considered a low-risk superficial procedure.

Assessment of Pulmonary Disease

Patients with pulmonary disease must be carefully assessed preoperatively because even healthy patients may develop pulmonary complications as a direct result of surgery and anesthesia. Pulmonary disease can be classified as either restrictive or obstructive. Restrictive disease may be the result of, for instance, severe scoliosis or morbid obesity and results in a decrease in all measured lung volumes. Obstructive disease is usually the result of smoking or asthma and may be characterized by marked increases in residual volume and functional residual capacity. A thorough past medical history and physical examination related to the pulmonary system prior to sedation or general anesthesia is mandatory. Unfortu-

nately, many patients, particularly smokers, are not aware that they have significant pulmonary compromise until it is very advanced.

As first reported by Morton in 1944, smoking is a risk factor for postoperative pulmonary complications, even among smokers without signs or symptoms of chronic obstructive pulmonary disease.16,17 The risk declines from 33 to 14.5% after only 8 weeks following cessation of smoking, whereas those who stop smoking for < 8 weeks have a higher risk of complications than do current smokers.18

The assessment should start with questions regarding dyspnea on exertion and functional level of physical activity that can be accomplished, such as how many flights of stairs can be managed without rest. Patients with mild or only occasional symptoms usually need no further investigation, whereas those with frequent or severe symptoms may need further evaluation and management prior to surgery. Although physical limitations may also be indicative of cardiovascular disease or pulmonary disease, they often present simultaneously because smoking is a major risk factor for cardiovascular disease.

Physical examination of the patient with obstructive pulmonary disease may reveal an increased anteroposterior diameter of the chest, a depressed diaphragm, a hyperresonant thorax on percussion, and wheezing, particularly during expiration. The chest radiograph may demonstrate hyperinflated lungs. The forced expiratory volume in 1 second (FEV₁) is usually < 80% of the vital capacity. Obstructive disease may be reversible, as in bronchial asthma, or it might have a reversible component. Common irreversible diseases include emphysema, chronic bronchitis, and bronchiectasis. However, antibiotics and bronchodilator therapy may reverse at least some of the components of acute symptoms of chronic bronchitis.
Asthma
Bronchial asthma is a common pulmonary condition that must be respected for its potential to cause life-threatening complications during surgery and anesthesia. In addition to taking a careful history with regard to asthmatic triggers, frequency, severity, emergency room visits, and hospitalizations, one can also assess the potential for an acute event by noting the number of different asthma medications required to control symptoms and the frequency and efficacy of their use. Wheezing from asthma immediately prior to the induction of anesthesia and surgery

FIGURE 4-1  Preoperative cardiac assessment algorithm for surgical risk of cardiac death/nonfatal myocardial infarction. Major predictors of risk: unstable angina, decompensated heart failure, significant dysrhythmias, and severe valvular disease; intermediate predictors of risk: mild angina, prior myocardial infarction, controlled heart failure, diabetes, and renal insufficiency; minor predictors of risk: advanced age, abnormal electrocardiogram, nonsinus rhythm, poor functional capacity, prior stroke, and uncontrolled hypertension. High-risk surgeries: emergent major surgery, major vascular surgery, and prolonged cases/major blood loss/fluid shifts; intermediate-risk surgeries: carotid endarterectomy, head and neck, intraperitoneal, intrathoracic, orthopedic, and prostate; low-risk surgeries: endoscopic, superficial, cataract, and breast. METs = metabolic equivalents; OR = operating room. Adapted from Eagle K et al.13
Chronic bronchitis, characterized by a chronic excess of mucus in the bronchi, is due to enlarged mucous glands that reduce the luminal diameter of the airways and increase resistance to airflow. Chronic bacterial infections are common and produce inflammation and fibrosis that further contribute to increased resistance. Patients with chronic bronchitis develop hypoxemia and carbon dioxide retention relatively early in the course of the disease compared with emphysema patients. Cor pulmonale, manifested by hepatopulmonary reflux and peripheral edema, also develops comparatively early and results in the patient being termed a “blue bloater.” The preoperative evaluation and management of chronic bronchitis is similar to that for emphysema.

**Emphysema**

Emphysema is characterized by irreversible enlargement of the alveolar air ducts and by destruction of the walls of these air spaces. The loss of elasticity of these structures permits collapse of the airways during exhalation, resulting in increased airway resistance. To keep their airways from collapsing, patients with severe emphysema can be observed to purse their lips during exhalation to attain positive end-expiratory pressure in their airways. The chest radiograph typically demonstrates low flat diaphragms and extremely hyperlucent lung fields, consistent with gas trapping and loss of lung parenchyma.

Preoperative management may decrease the incidence of postoperative pulmonary complications. Those with suspected significant obstructive disease may be candidates for preoperative pulmonary function testing and analysis of arterial blood gases. Many emphysema patients, commonly known as “pink puffers,” have reasonably normal arterial blood gases as they are able to increase their minute ventilation and cardiac output to compensate for increased airway resistance. With increasing pulmonary artery pressures above a mean of 20 mm Hg, cor pulmonale develops as the right ventricle begins to fail, resulting in hypoxemia, venous congestion, and systemic edema.

Measurement of the ratio of FEV$_1$ to forced vital capacity (FVC) may help to discern the severity of the disease and predict the chance for respiratory failure if the ratio is $< 50\%$. Carbon dioxide retention typically occurs when the FEV$_1$/FVC ratio is $< 35\%$.

**Bronchiectasis**

Bronchiectasis occurs when there is an abnormal enlargement of the bronchi that are frequently filled with purulent sputum and highly vascularized granulation tissue. There is risk of significant hemoptysis and an increased risk of pulmonary edema, pulmonary hypertension, and cor pulmonale.

**Summary**

The surgeon must complete a careful and thorough past medical history and physical examination to assess the risk of pulmonary disease. Recognition of poor exercise tolerance, clubbing of the fingertips, chronic cough and dyspnea with minimal exertion, decreased breath sounds, wheezes, rhonchi, and excessive expiratory effort are ominous signs of significant pulmonary disease that may warrant further evaluation and treatment prior to surgery and anesthesia.

Many patients with severe pulmonary disease require continual administration of supplemental oxygen via a nasal cannula at home. This should be continued during dental treatment. In the event of a medical emergency such as chest pain, giving of 100% oxygen by face mask and monitoring of the respiratory rate are highly recommended for all such patients. Should the respiratory rate of a patient who is a chronic carbon dioxide retainer decrease because of loss of respiratory drive caused by the additional oxygen, the practitioner may simply need to remind the conscious patient to breathe, or manually ventilate the unconscious patient with positive pressure oxygen. Only if a severely compromised pulmonary patient is left unmonitored while breathing 100% oxygen by face mask would there be danger of oxygen causing hypoventilation in the dental office that is properly equipped with airway adjuncts needed for artificial ventilation.

**Assessment of the Airway**

Assessment of the airway is one of the most important facets of the preanesthesia evaluation process because the inability to maintain a patent airway and provide adequate ventilation and oxygenation is frequently responsible for anesthesia-related morbidity and mortality. In a closed claims study by the American Society of Anesthesiologists, Caplan and colleagues reported that 34% of 1,541 liability claims were for adverse respiratory events. This was the largest source of adverse outcomes in their study. Of these cases approximately 75% were related to either inadequate ventilation (38%), esophageal intubation (18%), and difficult intubation (17%). Although the current universal use of the pulse oximeter and end-tidal carbon dioxide monitoring have undoubtedly decreased some of these events, at least some of the difficult intubations could have been situations in which the anesthesiologist could neither intubate nor mask ventilate an apneic patient. Thus, the oral and maxillofacial surgeon must carefully assess the potential for this type of catastrophic failure to maintain the airway during any sedation or anesthesia administered in the office or other surgical venue and be prepared to properly manage that circumstance should it occur despite careful assessment and planning to avoid it.
The American Society of Anesthesiologists has developed and updated an algorithm for management of the difficult airway.\textsuperscript{22} As seen in Figure 4-2, these guidelines enable anesthesiologists, nurse anesthetists, dentist anesthesiologists, and oral and maxillofacial surgeons to have a detailed series of plans and alternatives to facilitate the management of the difficult airway. This reduces the likelihood of adverse outcomes such as death, brain death, myocardial injury, and airway trauma. These guidelines recommend that a careful airway history and examination be conducted prior to the induction of anesthesia to detect medical, surgical, and anesthetic factors including previous anesthetic records, if available, that may identify the difficult airway.

Congenital and acquired diseases or conditions, for instance, may alter the airway anatomy to such an extent that attaining and maintaining a patent airway during anesthesia may be difficult or impossible. Congenital conditions such as Pierre Robin, Treacher Collins, Goldenhar’s, Klippel-Feil, and Down syndromes are associated with abnormalities such as restricted movement of the neck and mandible, micrognathia, maxillary and mandibular hypoplasia, and macroglossia. Examples of acquired conditions include obesity, oropharyngeal space infections, epiglottitis, tonsillitis, rheumatoid arthritis, tumors, temporomandibular joint disorders, head and neck cancer surgery, and oropharyngeal radiation therapy.

A careful physical examination of the airway must be accomplished. Anatomic characteristics associated with difficult intubation include a short large-diameter neck, retrognathia with obtuse mandibular angles, protruding maxillary incisors, decreased mobility of the temporomandibular joint, and a high-arched palate.

Although there is no airway rating system that can accurately predict a difficult airway with high sensitivity and specificity, the modified Mallampati classification is widely used.\textsuperscript{23} The hypothesis of Mallampati and colleagues is that the base of the tongue in certain individuals is disproportionately large, which makes direct laryngoscopy difficult. The tongue base is therefore compared with other anatomic features that it may obscure. To perform this test correctly, the patient should be sitting or standing upright and asked to open their mouth as widely as possible without phonating. In Class I patients the uvula, faucial pillars, and soft palate are visible. In Class II patients only the faucial pillars and soft palate are visible, whereas in the Class III patients, only the soft palate is observed. Class I patients are expected to have normal airways, whereas patients in Class II are somewhat more likely to be difficult to intubate. Intubation in Class III patients is even more likely to be difficult.

Samsoon and Young later added a fourth category to the original Mallampati classification.\textsuperscript{24} Their fourth class included visualization of the hard palate but not the soft palate or other structures. Class IV patients have the highest risk for a difficult intubation (Figure 4-3).

Although difficult intubation does not always coincide with difficult mask ventilation, one must recognize that patients in modified Mallampati Classes III and IV pose an increased risk of loss of a patent airway during nonintubated deep sedation or general anesthesia. When compounded with other risk factors such as mandibular retrognathia, obesity, or postradiation therapy, the practitioner may elect to administer only light conscious sedation with drugs that are pharmacologically reversible or to secure the airway via awake fiberoptic intubation prior to induction of general anesthesia.

In certain instances additional evaluation of the airway may be prudent. For example, fiberoptic pharyngoscopy, soft tissue radiography, computerized tomography, and magnetic resonance imaging may be helpful in identifying the extent of airway compromise and tracheal deviation associated with severe dentofacial and neck infections. Patients with a severe infection and significant trismus, orthopnea, dysphagia, drooling, and dyspnea may easily lose the patency of their tenuous airway with even modest doses of sedative, anxiolytic, or opioid analgesic medications given prior to attempted fiberoptic intubation. Preparations for an immediate surgical airway must be made well in advance.

Assessment of Endocrine Disease

Any of the major endocrine disorders can impact the course of anesthesia and surgery and should be considered in the preoperative assessment.

Adrenal Gland

A lack of adrenal cortical activity, as in Addison’s disease, may decrease the production of cortisol and aldosterone and alter cardiovascular stability. Patients who take supplemental glucocorticosteroids may have a suppression of adrenocorticotropic hormone from their pituitary gland and may need preoperative supplementation of cortisol. An overproduction of epinephrine and norepinephrine in the adrenal medulla from a pheochromocytoma may create a hypertensive-tachycardiac crisis intraoperatively.

Thyroid Gland

Hypothyroidism Hypothyroidism has many potential causes and is usually determined by an assessment of levels of thyroid stimulating hormone (TSH), triiodothyronine (T\textsubscript{3}), and thyroxin (T\textsubscript{4}). Patients who complain of fatigue and intolerance to cold and who are hypotensive may suffer from myxedema. Theoretically, myxedematous patients may be more susceptible to the depressant effects of anesthetics and less responsive to adrenergic vasopressors and cardiac inotropes. However, a retrospective study demonstrated no significant difference
1. Assess the likelihood and clinical impact of basic management problems:
   A. Difficult Ventilation
   B. Difficult Intubation
   C. Difficulty with Patient Cooperation or Consent
   D. Difficult Tracheostomy

2. Actively pursue opportunities to deliver supplemental oxygen throughout the process of difficult airway management.

3. Consider the relative merits and feasibility of basic management choices:
   A. Awake Intubation
   B. Non-Invasive Technique for Initial Approach to Intubation
   C. Preservation of Spontaneous Ventilation

4. Develop primary and alternative strategies:
   - Confirm ventilation, tracheal intubation, or LMA placement with exhaled CO2
     a. Other options include (but are not limited to): surgery utilizing face mask or LMA anesthesia, local anesthesia infiltration or regional nerve blockade. Pursuit of these options usually implies that mask ventilation will not be problematic. Therefore, these options may be of limited value if this step in the algorithm has been reached via the Emergency Pathway.
     b. Invasive airway access includes surgical or percutaneous tracheostomy or cricothyrotomy.
     c. Alternative non-invasive approaches to difficult intubation include (but are not limited to): rigid bronchoscope, esophageal-tracheal combitube ventilation, or transtracheal jet ventilation.
     d. Consider re-preparation of the patient for awake intubation or canceling surgery.
     e. Options for emergency non-invasive airway ventilation include (but are not limited to): rigid bronchoscope, esophageal-tracheal combitube ventilation, or transtracheal jet ventilation.

* Confirm ventilation, tracheal intubation, or LMA placement with exhaled CO2

**FIGURE 4-2** Algorithm for management of a difficult airway. LMA = laryngeal mask airway. Reproduced with permission from the American Society of Anesthesiologists.22
in hemodynamic instability, imbalance in fluid and electrolytes, necessity for vasopressors, myocardial infarction, sepsis, bleeding, extubation time, or time to discharge compared with matched controls.\textsuperscript{25} The conclusion of the study was that mild hypothyroidism is not a contraindication for surgery. However, severe myxedema can lead to coma, cardiovascular collapse, and heart failure and necessitates a postponement of surgery until it can be corrected.\textsuperscript{26}

**Hyperthyroidism** Graves’ disease is the most common type of primary hyperthyroidism. Symptoms include hyperexcitability, weight loss, hypertension, and tachycardia. Thyroid storm during anesthesia can resemble malignant hyperthermia. Propylthiouracil or methimazole is frequently prescribed to reduce thyroxin secretion prior to surgery, and $\beta$-adrenergic antagonists are used to stabilize the adrenergic activity prior to and during surgery.

**Goiter** Enlargement of the thyroid gland may adversely influence the patency of the airway. Substernal goiter may be difficult to recognize on physical examination without a chest radiograph, but it may produce symptoms of dyspnea and dysphagia. Large superficial goiters may increase the difficulty of endotracheal intubation.

**Pituitary Gland**

The pituitary gland has a wide influence on many glands and organs. Increased production of hypothyseal pituitary tropic hormones can produce secondary hyperthyroidism (TSH), secondary Cushing’s syndrome (adrenocorticotropic hormone), and acromegaly (growth hormone).

Acromegaly predisposes the patient to cardiomyopathy, dysrhythmias, and sudden death.\textsuperscript{27,28} The excessive growth hormone increases the production of insulin-like growth factor I (IGF-I) by the liver and other tissues. Excessive levels of IGF-I can produce headaches, profuse sweating, joint disorders, soft tissue swelling, and overgrowth of the hands, feet, mandible, and viscera. The patient with acromegaly may therefore present with a difficult airway, particularly for endotracheal intubation.

**Diabetes Mellitus**

Diabetes mellitus is a common disease with far-reaching implications, primarily owing to the microangiopathy-related impairment of normal blood flow and subsequent end-organ damage. Patients diagnosed with insulin-dependent diabetes at a young age are less commonly seen than those diagnosed with non–insulin-dependent diabetes later in life, who are generally able to control it with oral hypoglycemic agents. Insulin-dependent diabetics generally have more severe signs and symptoms related to their diabetes and have increased potential to suffer the consequences for a longer period of time than non–insulin-dependent diabetics. Preoperative evaluation of all diabetics includes an assessment of the degree of blood glucose control and a search for evidence of end-organ damage. As the degree of end-organ damage progresses, the likelihood of perioperative complications, often cardiovascular in nature, increases.

Blood sugar is usually measured several times a day when insulin therapy is needed. Although blood sugar concentrations can vary widely throughout the day, a measurement at the preoperative assessment appointment can give the practitioner an idea of the degree of control that the patient might have at that time. The practitioner may also discern that the patient is in optimal control by measuring the glycosylated fraction of adult hemoglobin (HbA\textsubscript{1c}) for a long-term picture of overall control. Hemoglobin A\textsubscript{1c} binds with glucose to form HbA\textsubscript{1c}, which is a relatively stable complex that provides more of an average blood glucose level over a period of 1.5 to 2 months. Thus, taken together, these two measurements provide the practitioner with information on both short- and long-term control.

A well-controlled diabetic is expected to have fewer perioperative complications including reduced incidences of wound infection and diabetic ketoacidosis. Although long-term tight control should reduce end-organ damage, tight control in the immediate perioperative period may predispose the patient to hypoglycemia, which can result in central nervous system damage.

A rational approach to properly managing diabetic patients is based on knowledge of the type of diabetes present, the degree of its control by the patient, the stress associated with the surgical procedure, and the likelihood of the patient quickly resuming a normal diet and hypoglycemia medication postoperatively.\textsuperscript{29} Short-term control by the sliding scale may be best in the perioperative period for many patients; however, others may do well administering their insulin after surgery in
their usual manner, as if surgery never happened. An individualized approach to diabetic management is essential.

End-organ damage from diabetes may result in problems that directly affect surgery and anesthesia. Renal failure may be the result of diabetic nephropathy, which may alter fluid and electrolyte balance and drug elimination. The lack of erythropoietin production by the kidney may result in significant anemia. Diabetic sensory neuropathy may permit myocardial ischemia and silent myocardial infarction to go unrecognized by the patient and is an independent predictor of perioperative cardiac morbidity. Diabetic autonomic neuropathy may also increase the risk of aspiration of gastric contents during deep sedation or general anesthesia by delaying gastric emptying. In addition, it may cause unpredictable cardiovascular responses to anesthetic drugs and to other cardiovascular-active drugs.

Metabolic acidosis with hyperglycemia > 300 mg/dL in the diabetic defines ketoacidosis. Insulin-resistance owing to trauma, surgery, or infection may be a contributing factor. The conversion of fatty acids to acetoacetic acid, β-hydroxybutyrate, and acetone in the absence of insulin produces metabolic acidosis and the fruity smell on the breath that may be recognized during the preoperative assessment. Extraglomerular potassium increases as it leaves the cells, and this results in intracellular depletion of potassium in the presence of hyperkalemia. Significant hypovolemia results from the osmotic diuretic effect of glucose in the urine. All of these deviations must be corrected with insulin, fluid, and electrolytes before proceeding with all but the most urgent surgery.

Assessment of Liver Disease
Preoperative assessment for liver disease is particularly important for those individuals with cirrhosis or acute hepatitis because morbidity and mortality rates with these diseases are markedly increased.

When the practitioner suspects liver disease during the perioperative assessment, several screening tests are available. Acute or chronic hepatocellular damage is indicated with elevations of aspartate aminotransferase (AST) and alanine aminotransferase (ALT). Acute damage can produce very high enzyme elevations, whereas chronic damage may produce only mildly elevated levels. ALT is more specific to hepatocytes.

Unconjugated bilirubin from normal red cell destruction may increase in the presence of severe liver disease if the hepatocytes cannot conjugate it with glucuronide. Elevated serum bilirubin is responsible for the yellow jaundiced appearance.

Serum albumin and nearly all of the clotting factors such as prothrombin are produced in the liver. Severe liver disease can decrease the synthesis of many important proteins, as reflected in decreased serum albumin levels. Additionally, because many anesthetic drugs are normally highly bound to albumin, reduced serum albumin levels over a period of many weeks may permit unusually high levels of free drug to exist in the plasma, which could produce a markedly enhanced effect from a relatively small dose. Reduced prothrombin levels would be reflected in an increased prothrombin time (PT) and International Normalized Ratio (INR) and serve as additional markers for the severity of hepatic disease. Because significant liver disease influences so many bodily functions, only necessary simple procedures under local anesthesia and perhaps nitrous oxide–oxygen conscious sedation should be attempted in an office setting for those patients with significant hepatic compromise.

Assessment of Renal Disease
Renal disease has a great impact on perioperative morbidity and mortality. The mortality rate associated with acute renal failure ranges from 42 to 88%. Levy and colleagues demonstrated that acute renal failure is an independent risk factor for mortality, regardless of other risk factors. He also noted that because the mortality of contrast medium–associated acute renal failure is above 30%, elective surgery should be postponed if possible until renal function returns to baseline in these patients. Although newer less toxic contrast agents are now available, acute renal failure can still occur.

As previously discussed, renal failure is often a consequence of diabetes and long-standing hypertension. It can be responsible for congestive heart failure, fluid and electrolyte imbalance, anemia, hypertension, and azotemia. When renal disease is suspected from the history and physical examination, several tests can be completed to assess its presence and the degree of impairment. Because urea and creatinine are excreted by glomerular filtration and their blood levels are therefore inversely proportional to the glomerular filtration rate, blood urea nitrogen and serum creatinine levels are commonly obtained to initially assess renal function. Creatinine serum levels are normally in the range of 0.6 to 1.5 mg/dL. Approximately a 50% loss in kidney function is indicated by a creatinine level > 2.0, whereas a 75% loss of function would be indicated by a creatinine level > 4.8. Creatinine levels > 10.0 are consistent with end-stage renal disease (ESRD).

Patients with ESRD who depend on hemodialysis often present for perioperative assessment in either a hypervolemic or hypovolemic state, depending on whether they need dialysis soon or have just completed it. Chronic hyperkalemia and anemia are commonly seen. Patients on hemodialysis are usually treated on the day after dialysis, when they have somewhat stabilized their physiology and when the effects of their dialysis-associated heparin are no longer present. Many of these patients are quite sensitive to small doses of sedatives and anxiolytics; therefore, slow careful intravenous titration of
Assessment of Bleeding Disorders

A careful history regarding bleeding problems is essential prior to surgery. Excessive bleeding may result from a variety of causes. For instance, drugs such as acetylsalicylic acid and other nonselective nonsteroidal anti-inflammatory analgesics may inhibit platelet function. Liver disease may decrease the production of clotting factors. A family history of bleeding may be the result of autosomal dominant transmission of von Willebrand’s disease to males and females, whereas hemophilia A and B are both inherited as sex-linked recessive traits. These patients may be taking various factors to bring their levels to the normal range or may have had a history of intravenous desmopressin administration to acutely elevate levels of factor VIII and von Willebrand’s factor prior to surgery. A decreased ristocetin cofactor activity is the most sensitive and specific screening test for von Willebrand’s disease because large multimers of von Willebrand’s factor are important in ristocetin-induced platelet aggregation.

To help uncover previously unrecognized bleeding disorders prior to major dental surgery, Holtzman and colleagues recommend preoperative laboratory assessment of hemostasis prior to orthognathic surgery. However, there are a large number of studies that generally concur that routine hemostatic testing of asymptomatic patients does not significantly alter treatment and is not cost-effective for the low yield.

Wahl reviewed more than 950 patients continuously receiving anticoagulants who underwent more than 2,400 dental surgical procedures, and only 12 (< 1.3%) required more than local measures to control bleeding. Conversely, of the 526 patients who stopped their anticoagulant therapy, 5 suffered serious embolic complications and 4 of the 5 died. Wahl recommends that most dental surgery patients should remain at therapeutic levels of their anticoagulant during the perioperative period.

When a bleeding disorder is suspected, the usual screening tests include the PT or INR to test the activity of the extrinsic and final common pathways and the activated partial thromboplastin time to test the intrinsic and final common pathways. Platelet counts may be important when thrombocytopenia is suspected and bleeding time is prolonged.

Assessment of Neurologic and Neuromuscular Disorders

The oral and maxillofacial surgeon may encounter a variety of patients with neurologic disorders. Neurologic examination may reveal important findings that may alter treatment planning. For instance, head-injured trauma patients are classified according to the Glasgow Coma Scale (Table 4-6).

Protection of the airway without increasing the chances of worsening any existing neurologic impairment is of prime importance in severely traumatized patients. The preoperative assessment of some of these patients may be, by necessity, quite limited during resuscitative procedures. Nevertheless, it is absolutely necessary to accomplish to whatever degree is possible.

Neuromuscular disorders such as Parkinson’s disease or multiple sclerosis may increase the risks of ventilatory insufficiency during spontaneous breathing and aspiration during sedation or anesthesia when the airway is relatively unprotected. Duchenne’s muscular dystrophy may be a risk factor for development of malignant hyperthermia or neuroleptic malignant syndrome in response to various anesthetic drugs.

Epilepsy

Epilepsy is a common neurologic disorder that requires careful assessment. Patients with a history of seizure should maintain their antiseizure therapy during the perioperative period. The practitioner should be aware of the frequency and duration of the seizures, including the most recent one, and what to expect should a seizure occur. Despite maximal doses of multiple medications, some patients remain poorly controlled, and the surgeon must then determine the most appropriate venue for surgical treatment, while considering that the risks of pulmonary aspiration and respiratory insufficiency during seizure episodes are increased.

Transient Ischemic Attack and Stroke

Patients with a history of transient ischemic attacks (TIAs) or stroke should be evaluated in the same manner as those with angina pectoris and myocardial infarction. Those who are deemed to have unstable TIAs or who have had a stroke within the previous 6 months are managed

<table>
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<tr>
<th>Table 4-6 Glasgow Coma Scale</th>
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<tr>
<td><strong>Action</strong></td>
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<tr>
<td>Eye opening</td>
</tr>
<tr>
<td>Spontaneously</td>
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<tr>
<td>To speech</td>
</tr>
<tr>
<td>To pain</td>
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<tr>
<td>Motor response</td>
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<tr>
<td>Obeys</td>
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<tr>
<td>Localizes pain</td>
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<tr>
<td>Withdraws from pain</td>
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<tr>
<td>Flexion to pain</td>
</tr>
<tr>
<td>Extension to pain</td>
</tr>
<tr>
<td>None</td>
</tr>
<tr>
<td>Verbal response</td>
</tr>
<tr>
<td>Oriented</td>
</tr>
<tr>
<td>Confused</td>
</tr>
<tr>
<td>Inappropriate</td>
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<tr>
<td>Incomprehensible</td>
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<td>None</td>
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Adapted from Teasdale G, Jennett B. Patient’s score determines category of neurologic impairment: 15 = normal; 13 or 14 = mild injury; 9–12 = moderate injury; 3–8 = severe injury.
similarly to those with unstable angina and recent myocardial infarction, respectively. The hypercoagulable state associated with the stress of surgery is more likely to manifest itself in patients with preexisting disease in coronary and cerebral arteries.

Preoperative Screening Tests for Asymptomatic Patients

With the advent of high-tech automated equipment in the past several decades that can quickly complete a large number of preoperative screening tests, practitioners who wished to gather as much information as possible about their patient to optimize care and reduce poor outcomes began to order “universal testing,” even for apparently healthy asymptomatic patients, in a futile attempt to “leave no stone unturned.” Unfortunately, the indiscriminate ordering of multiple laboratory tests has many drawbacks and usually does not uncover diseases that normally should be discovered by other means such as a thorough history and physical examination. For instance, Rabkin and Horne identified 165 patients who had been diagnosed as having “new electrocardiographic changes.”42 However, of that number, 163 were identified as having changes consistent with their history and physical examination, so these changes were not unexpected. Of the 2 patients whose new electrocardiographic changes were not consistent with the basic information recorded in their chart, 1 patient was found to be in atrial fibrillation, which should most likely have been discovered by palpation of an irregular pulse during the examination process. The other patient had no physical examination performed. Thus, this study indicated that a thorough history and physical examination should be the key to determining whether the practitioner should look for new electrocardiographic changes.

Domoto and colleagues performed 19 screening tests in 70 asymptomatic elderly patients whose mean age was > 80 years.43 Most abnormalities were minimally outside normal ranges, and only 0.1% of the patients had a resulting change in treatment. Most importantly, no patient received an important benefit from the tests. Likewise, Dzankic found that the prevalence of abnormal preoperative electrolyte values and thrombocytopenia was small and had a low predictive value in elderly surgical patients.44 Although more prevalent, abnormal hemoglobin, creatinine, and glucose values were also not predictive of postoperative adverse outcomes. Thus, the routine preoperative testing in geriatric patients for hemoglobin, creatinine, glucose, and electrolytes on the basis of age alone may not be indicated. Selective laboratory testing, as indicated by history and physical examination, determines a patient’s comorbidities and surgical risk.

Narr and colleagues studied 3,000 ASA PS-1 and PS-2 patients who received elective surgery and found no benefit from the tests.45 Archer and colleagues completed a meta-analysis of over 14,000 patients and concluded that the practice of obtaining routine preoperative chest radiographs should be abandoned.46

It is important to understand that the “normal values” of various tests are often set around a normal distribution that would include values of perhaps 95% of a healthy population. However, some healthy individuals may fall above or below the normal range yet still be without disease. When one considers the variable selectivity of individual tests, it is not unreasonable to expect that from a large battery of tests, at least one may reveal a falsely positive result. Such a result may prompt the clinician to seek additional information from more invasive tests, which may result in a severe complication. Therefore, indiscriminate testing can actually do more damage than the potential harm of some unrecognized disease that it is designed to discover. Additionally, in an era of cost containment, testing asymptomatic patients in hopes of improving outcomes is generally not cost-effective for the resulting low yield.47–50 Although many patients with significant diseases such as diabetes and coronary artery disease, as well as women of child-bearing age who are not sure of their pregnancy status, need certain laboratory testing preoperatively, routine testing of healthy asymptomatic patients with no complicating factors is unwarranted.51–53

A carefully taken medical history and a thorough physical examination remain the most important aspects of optimal patient care when supplemented by specific tests that are indicated by this information.

Summary

Having obtained and evaluated all of the appropriate information from the above sources, the oral and maxillofacial surgeon must, in the end, judge whether the benefit-to-risk ratio of completing a procedure for a particular patient, using a particular sedative/anesthesia technique in a specific venue (office, ambulatory surgical center, or hospital), is acceptable. For some medically, physically, or mentally complex patients, an alternative surgical procedure, surgeon, anesthesia provider, anesthesia technique, and/or venue may be deemed more appropriate than for those same variables with the healthy patient. Sound professional judgment of the surgeon is the hallmark of successful oral surgical practice, and a complete preoperative assessment of each patient provides an opportunity to influence that judgment for a safe and successful operation. The oft-mentioned statement “never treat a stranger” is indeed profound.

References

47. Roven MF, Kaplan EB, Schreider BD, et al. The relative roles of the history and physical examination, and laboratory testing in...


Intravenous 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 intravenous 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 at least 20% of surgical procedures are performed with office-based sedation or general anesthesia.

While it is neither possible nor the intention of the authors to present the full scope of anesthetic medications including emergency medications in this chapter, we will 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. 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 into or out of the cell, including chloride, potassium, sodium, and calcium. 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 towards 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 activate GABA receptors, 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. Nondepolarizing neuromuscular blockers are competitive antagonists for the acetylcholine 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, as they do not compete for the same binding site. Many pesticides are an example 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. Commonly identified by the route of administration, such as per oral (PO), intravenous (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 prior to entrance into the central circulation. This process potentially reduces the plasma concentration of drug that reaches the effector site, such as the CNS. Since 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 intravenous and inhalation administration provide direct entry into the central circulation, reaching peak plasma concentration very quickly following drug administration. Inhalation pharmacokinetics will be discussed in the following section “Inhalation Anesthetics.”

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. Following 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. Since 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. Following initial intravenous 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 (approximately 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. The degree to which the drug binds to plasma proteins such as albumin and α1-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 as the majority of the additional administered agent will be free drug that is able to cross the blood-brain barrier. Careful titration of intravenous
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 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 the 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, including 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 additional medications is possible. Induction is isof orm specific; a coadministered drug will only be affected by enzyme induction 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 simetidine for treatment of gastric ulcers may experience prolonged residual CNS effects from diazepam, as simetidine inhibits the hepatic enzymes that normally metabolize diazepam. Various tables have been published which 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 (eg, 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 (eg, 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 intravenous 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. Figure 5-1 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 programmed 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.

**Benzodiazepines**

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

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

Characteristics shared by benzodiazepines include sedation, anxiolysis, anterograde amnesia, muscle-relaxing properties, and anticonvulsant activity. Indeed, any intravenous 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 prior to the procedure and as a sleep adjunct the night before surgery. In clinical practice they are also used for conscious 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. Additionally, even smaller 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 are then excreted by the kidney in urine.

Side effects of 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. Intravenous injection can be painful, although injecting into a larger vein or pre-administration of lidocaine or an opioid can reduce discomfort. Intramuscular injection is painful and absorption can be unpredictable.

Diazepam is still used for intravenous conscious 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 compared to midazolam. However, the much longer elimination time of diazepam may contribute to lingering sedative effects. Diazepam can also be given orally (5 to 10 mg) for preoperative anxiolysis and mild 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 also 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, due 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 solution, rather than propylene glycol, resulting in less pain on intravenous and intramuscular injection. It is 2 to 3 times as potent at 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 diazepam after bolus intravenous administration.

Midazolam is currently more popular than diazepam for intravenous sedation for short oral surgical procedures. For conscious sedation 0.05 to 0.15 mg/kg IV 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 intramuscular injection of 0.5 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 to 1 mg/kg (maximum 15 mg), usually mixed into a flavored syrup or in a commercially available premixed product; this route may be better accepted by pediatric patients. 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 only available in an oral formulation as 0.125 mg and 0.25 mg tablets. This sleep adjunct can be used off-label for anxiolysis and 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 with clinically effective sedation lasting from 30 to 90 minutes.

**Flumazenil**

Flumazenil is a highly specific competitive antagonist for the benzodiazepine receptor and is used as a reversal agent for benzodiazepine agonists. It will reverse benzodiazepine sedation, excessive disinhibition, and the additive ventilatory depression related to benzodiazepines when combined with opioids. Flumazenil is given 0.2 mg IV 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 since 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 mild sedation or euphoria. It is important to note that narcotic 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 barbiturates to provide analgesia and augment the desired level of anesthesia.

While the term opiate refers to any drug derived from opium, opioid medications include all substances, natural and synthetic, which bind to the opioid receptors. Common opioid medications are shown in Figure 5-3. 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. Agonist activity at these receptors either modifies or decreases neuronal transmission of pain signals. Several subtypes of opioid receptors (eg, μ, κ, δ) with differential effects have been identified. The μ and κ receptors are predominately responsible for analgesia, and most clinically used opioids are agonists for the μ receptor. A subset of opioids, termed agonist-antagonist opioids, are agonists at κ receptors and 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 μ receptor 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 (CO₂) levels in the brainstem respiratory centers. Decreased respiratory rate and arterial hypoxemia may result without supplemental oxygen (O₂) and appropriate monitoring (eg, pulse oximetry). Opioids are often titrated incrementally to balance the analgesic effect against respiratory depression.

Bradydardia 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 (eg, 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 has a slow onset. Peak effect following IV administration occurs in 15 to 30 minutes and the analgesic effect lasts approximately 4 hours. Because of its slow onset and longer duration of activity, it is commonly used in anesthesia for postoperative pain management rather than intravenous 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.

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 intravenous 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, which is half the potency of meperidine. When mixed with monoamine oxidase inhibitors, meperidine administration may produce a dangerous excitatory hyperthermic reaction. With repeated dosing, particularly in renally compromised patients, accumulation of normeperidine may lead to seizures. 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 to 20 min). With
such characteristics fentanyl is a frequent choice for intravenous conscious sedation for short office-based procedures. It is typically given in 25 to 50 µg increments towards a total dose of approximately 1 to 2 µg/kg. It is also given during induction of general anesthesia, both for 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 rigidity and glottic rigidity, making ventilation impossible; there are reports that even lower doses (eg, 100 µg) can trigger this centrally mediated effect. Fentanyl-associated chest wall rigidity is treated with either naloxone 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 following 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 towards the end of the procedure.

Remifentanil is used in a total intravenous infusion anesthetic technique to maintain anesthesia during dental 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, and 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 cause 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 intravenous 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 US Drug Enforcement Agency Schedule II controlled substances, nalbuphine is not currently a scheduled controlled substance and does not require state and federal documentation of use.

**Naloxone**

Naloxone is a pure opioid antagonist that is active at all opioid receptor subtypes. It will reverse both the ventilatory depressive and 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 (eg, chronic pain management, illicit opioid users, methadone therapy for opioid abuse), naloxone must be used with caution as the antagonist effect may precipitate acute opioid withdrawal and acute congestive heart failure may result.

The initial dose is 0.4 to 2 mg IV for acute reversal. Naloxone can also be titrated in 0.04 mg increments when gradual adjustment of mild 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.

**Barbiturates**

Barbiturates are sedative-hypnotic medications that have long been employed as induction agents of general anesthesia. Barbiturates produce sedation, loss of consciousness, and amnesia. These drugs do not provide analgesia and may actually 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 intravenous barbiturate can also suppress acute seizure activity.

Barbiturates are derivatives of barbituric acid (Figure 5-4). The characteristics of the individual barbiturate are determined by the side chains attached to the barbiturate ring (Figure 5-5). 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 intravenous 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 as 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 intravenous 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 prior to 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.

**Thiopental**

Thiopental is an ultrashort-acting barbiturate that is commonly used at 3 to 5 mg/kg IV to induce loss of consciousness for general anesthesia prior to endotracheal intubation. It is associated with a longer recovery than methohexital due to its decreased plasma clearance and is generally not used as a continuous infusion to maintain anesthesia due to significant storage in multiple drug compartments. A 2.5% solution of thiopental is less...
expensive than other induction agents, but when rapid recovery is desired during outpatient anesthesia, other agents such as methohexital and propofol have proven more popular. Thiopental can release histamine, which is a concern in asthmatic patients.

**Methohexital**

Methohexital is an ultrashort-acting barbiturate that is commonly employed for outpatient oral surgical procedures, primarily for its more rapid recovery compared to thiopental and its lower cost compared to propofol. As an oxybarbiturate, methohexital is less lipid soluble than thiopental but is associated with a more rapid awakening because of its increased hepatic clearance. Psychomotor function returns more quickly with methohexital than thiopental, allowing for earlier discharge following an outpatient procedure.

Methohexital is reconstituted into a 1% solution and given at 1.5 to 2 mg/kg IV 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 following methohexital anesthesia. Methohexital exhibits clinically insignificant histamine release.

**Propofol**

Propofol has become one of the most popular sedative-hypnotic drugs used for ambulatory surgery. Propofol, 2,6-diisopropylphenol (Figure 5-6), 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 following administration.

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 IV produces unconsciousness within 30 seconds. In the intermittent bolus technique frequently used for deep sedation in oral surgery, small increments of propofol (10 to 30 mg) are periodically administered after a baseline conscious 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 intravenous infusion. The dosages for conscious 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 conscious sedation to general anesthesia, highlights the lower margin of safety of this drug, especially if the intended level of sedation is conscious sedation. US Food and Drug Administration labeling prohibits use of propofol by those involved in the conduct of the surgical or diagnostic procedure.}

**Pentobarbital**

Pentobarbital is an intravenous 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 the ultrashort-acting agents.

**Nonbarbiturate Induction Agents**

Other medications are available for sedation and induction of general anesthesia. These include propofol, etomidate, and ketamine, all of which can produce unconsciousness but with several differing characteristics from barbiturate medications.

**FIGURE 5-6 Chemical structure of propofol.**

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normally be associated with such a drop in blood pressure. Hypotension may therefore be very significant following bolus administration of propofol, particularly in the 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 to 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. All these features make propofol an attractive choice for outpatient procedures where decreased time to discharge is desirable.

Even with an available generic formulation the higher cost compared to barbiturates is still apparent. The increased cost can overshadow the advantages of using propofol infusions, especially if the surgical time is long (> 2 h), or if quick discharge is not required.

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 pre-administration 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, as the egg protein contained in the suspension is lecithin, whereas most egg allergies consist of a reaction to egg albumin. The original proprietary agent, Diprivan, uses ethylenediaminetetraacetic acid as an antibacterial agent, whereas the generic version contains a sulfite. 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. Both drug suspensions 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, as sepsis is a possibility.

**Etomidate**

Like midazolam, etomidate contains an imidazole structure (Figure 5-7). 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 IV. 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 because it is more expensive than other induction agents.

Spontaneous respiration may be maintained. Respiratory depression is less pronounced with etomidate compared to 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 and recovery that is faster than with thiopental but slower than with methohexital or propofol.

**Ketamine**

Ketamine is a phencyclidine derivative (Figure 5-8) that induces a state of “dissociative anesthesia.” This is characterized as a “dissociation” between the thalamocortical and limbic systems, producing a cataleptic state during which the patient may appear awake but does not respond to commands. 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, which involve the excitatory neurotransmitter glutamate. 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.

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 IV 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 will 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 postoperative nausea and vomiting. 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 a primary indication for ketamine is intramuscular 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 intramuscular 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 is 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 an IV deep-sedation technique. Conscious sedation is first achieved with a benzodiazepine, followed by subanesthetic doses of 10 to 30 mg of ketamine until a state that is similar to deep sedation is achieved. Although ketamine is quite analgesic some surgeons also add an opioid in the baseline sedation. Alternatively, if a standard deep-sedation technique with methohexital has been applied (see “Methohexital,” above) and large doses of the barbiturate become necessary to achieve adequate sedation, or unwanted patient movement persists despite high methohexital doses, the addition of small boluses of ketamine can often enhance the quality of sedation.

“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 doses less than 2 mg/kg IV. It may be attenuated with prior or concurrent administration of benzodiazepines, which should be routine when intravenous sedation techniques are used.

Inhalation Anesthetics

Inhalation anesthetics include nitrous oxide (N₂O) as well as the potent volatile halogenated agents, such as halothane, isoflurane, sevoflurane, and desflurane. N₂O alone is commonly used in dental offices for anxiolysis and mild 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.

The pharmacokinetics of these anesthetic agents differ from those of intravenous 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. Except for halothane most of 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. Cardiac output influences the rate of uptake from the alveoli. 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

FIGURE 5-8 Chemical structure of ketamine.
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 5-1) 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 blood stream and then into the alveoli. Gases with lower blood:gas coefficients will likewise “offload” from the blood stream into alveoli more quickly and can translate into a faster offset of action.

Unlike intravenous medications these inhaled drugs are not administered in doses of mg/kg. The equivalent of the effective dose (ED50) 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 5-1. 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 N2O 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 35-year-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 N2O 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. The exception is halothane, which in addition to directly depressing myocardial contractility, blocks the baroreceptor reflex. This resulting decrease in cardiac output can lead to a precipitous drop in systemic blood pressure with higher doses of halothane.

Halothane also has the highest association with cardiac dysrhythmias. Halothane induction commonly suppresses sinoatrial node activity, leading to the development of functional rhythms. It also sensitizes the myocardium to catecholamine-related

<table>
<thead>
<tr>
<th>Molecular weight</th>
<th>Nitrous Oxide</th>
<th>Isoflurane</th>
<th>Enflurane</th>
<th>Halothane</th>
<th>Desflurane</th>
<th>Sevoflurane</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitrous Oxide</td>
<td>44</td>
<td>184.5</td>
<td>184.5</td>
<td>197.4</td>
<td>168</td>
<td>218</td>
</tr>
<tr>
<td>Isoflurane</td>
<td>Gas</td>
<td>238</td>
<td>172</td>
<td>243</td>
<td>664</td>
<td>160</td>
</tr>
<tr>
<td>Enflurane</td>
<td>105</td>
<td>1.2</td>
<td>1.6</td>
<td>0.77</td>
<td>6.0</td>
<td>2.0</td>
</tr>
<tr>
<td>Halothane</td>
<td>0</td>
<td>0.2</td>
<td>2.4</td>
<td>20</td>
<td>0.02</td>
<td>3</td>
</tr>
<tr>
<td>Desflurane</td>
<td>0</td>
<td>0.2</td>
<td>2.4</td>
<td>20</td>
<td>0.02</td>
<td>3</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>0</td>
<td>0.2</td>
<td>2.4</td>
<td>20</td>
<td>0.02</td>
<td>3</td>
</tr>
</tbody>
</table>

MAC = minimum alveolar concentration.
ventricular dysrhythmias (Figure 5-9), particularly under conditions of hypercarbia.\textsuperscript{15} Isoflurane, sevoflurane, and desflurane are not significantly associated with an increased incidence of epinephrine-associated dysrhythmias. Epinephrine contained in local anesthetic solutions should be limited to a maximum dose of 1 to 2 µg/kg during halothane anesthesia whereas up to 3 to 4.5 µg/kg is considered safe with the other three agents. Under halothane anesthesia, administration of 1.0 to 1.5 mg/kg cardiac lidocaine IV immediately prior to intubation reduces the incidence of ventricular dysrhythmias during this stimulating period when endogenous epinephrine release may occur. Hypoxia and hypercarbia also lower the threshold for dysrhythmias and should be especially avoided with halothane anesthesia. Treatment of the presenting dysrhythmia should be managed as required, including hyperventilation, deepening of anesthetic level and, if indicated, discontinuation of halothane with administration of an alternative anesthetic agent.

At usual doses N\textsubscript{2}O 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 breaths, but the faster respiratory rate does not fully compensate for the smaller tidal volumes. Therefore, minute ventilation is reduced and arterial CO\textsubscript{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 0.1 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. Hepatotoxicity is more of a concern with halothane administration. 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 should be at least 2 L/min to minimize compound A accumulation in the CO\textsubscript{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\textsubscript{2}O, nondepolarizing neuromuscular blockers, opioids, benzodiazepines, and other intravenous anesthetic agents do not trigger MH. Exposure to these medications causes an abnormal receptor in skeletal muscle cells to release excessive intracellular calcium, leading to uncontrolled muscle contractions. As a result of CO\textsubscript{2} production increases quickly and exhaled CO\textsubscript{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\textsubscript{2} given, preferably through a different circuit and machine. Dantrolene at 2.5 to 10 mg/kg IV 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 following the episode. Reemergence of the reaction is common, requiring re-administration 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\textsubscript{2}O is commonly administered in dental offices for anxiolysis and mild sedation. It is a colorless and odorless gas, available in blue cylinders. In the dental setting it is commonly administered with a nasal hood and appropriate scavenger system. Concentration ratios of N\textsubscript{2}O:O\textsubscript{2} range up to 70:30 on most N\textsubscript{2}O and anesthesia machines. High levels of N\textsubscript{2}O:O\textsubscript{2} alone can produce sedation and significant analgesia. Unexpected respiratory depression or airway obstruction can occur when N\textsubscript{2}O is added to other sedative agents.

N\textsubscript{2}O in O\textsubscript{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\textsubscript{2}O 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\textsubscript{2}O sedation, 3 to 5 minutes of 100% O\textsubscript{2} is administered to prevent diffusion hypoxia; if room air O\textsubscript{2} is given instead, the rapidly exiting N\textsubscript{2}O can
dilute the O₂ concentration in the alveoli to hypoxic levels during recovery.

With a low blood:gas solubility coefficient of 0.47, N₂O has a very quick onset and recovery. While N₂O 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 is often 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 side effects. N₂O is also inexpensive and can reduce the total cost of administered drugs.

There are a few contraindications for the use of N₂O. It can enter closed spaces faster than nitrogen can exit, leading to distention of the closed space. In oral surgical practice the implication of this property is to avoid N₂O use in patients with current otitis media and sinus infections and with emphysema (blebs). Other contraindications of N₂O use include current respiratory disease and a history of severe postoperative nausea.

Several precautions should be exercised when using N₂O. 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 N₂O sedation. Because low levels of N₂O 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 N₂O 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 halothane, isoflurane, sevoflurane, and desflurane. As seen in Figure 5-10, all are derivatives of ether except for halothane. 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.

**Halothane** Halothane has a sweet non-pungent odor that does not irritate the airway mucosa to the extent of isoflurane and desflurane, and is therefore useful for inhalation induction of general anesthesia. Halothane is very potent, with a MAC value at 0.75 but a relatively high blood:gas solubility of 2.54. Therefore, halothane will have a slow onset of inhalation induction unless high doses are used. Recovery from anesthesia will be slower than with other agents with lower solubility coefficients.

Halothane is the oldest and most inexpensive of currently available potent gases but presents with the most deleterious side effects. As noted above, halothane is associated with significant cardiovascular changes and dysrhythmias. These should be monitored closely during induction and epinephrine administration, such as with local anesthesia, when dysrhythmias are more commonly encountered. Unlike the other agents, at least 15% of the halothane molecules are metabolized by the liver, and hepatotoxicity is more significant with halothane, especially after repeated and prolonged administration. Halothane hepatitis is very rare but can result in hepatic necrosis and death. Of all the halogenated agents it also appears to be the most potent trigger for MH.

**Isoflurane** Isoflurane is more pungent than halothane and is not a good choice for inhalation induction. It has an intermediate potency (MAC 1.2) and blood:gas partition coefficient (1.46). This agent is a common choice for maintenance of anesthesia, as recovery time is in the intermediate range and shorter than halothane. Isoflurane is also much more cost-effective for longer periods of anesthesia compared to two other popular agents, sevoflurane.
and desflurane; its cost per bottle is significantly lower and the total amount used is less due to the lower MAC.

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 following a short anesthetic (< 1 h) is more rapid than either isoflurane or halothane due 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 to isoflurane. The recovery time is also not significantly improved compared to isoflurane, as 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 amount of inorganic fluorine released during sevoflurane use has not been associated with renal damage. Sevoflurane and CO₂ 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 is not usually the agent of choice for patients with renal disease. Even in healthy patients many practitioners recommend limiting sevoflurane use to less than 2 hours and maintaining 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 as its vapor pressure is close to atmospheric pressure. It also possesses only fluorine substitutions which, 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 N₂O. This confers a quick onset and offset, and recovery can be very rapid following a short anesthetic with desflurane. Like sevoflurane, desflurane is more expensive than the other gases, and considering its higher MAC value (6.0), much more of the gas will be used per minute, resulting in a significantly higher cost if desflurane is used for a longer procedure.

**Perioperative Analgesic Medications**

Opioid medications, which have been discussed previously, are the classic 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 like morphine or hydromorphone is commonly administered by the practitioner prior to the end of the procedure, in anticipation of postoperative pain. During the initial phase of postoperative care these medications may be given either by the nursing staff or patient, administered via computer-aided patient-controlled analgesia pumps.

Another option is ketorolac tromethamine, currently the only available intravenous NSAID medication in the United States. This agent can provide effective analgesia for many dentoalveolar procedures at IV and IM doses of 30 to 60 mg or 0.5 to 1.0 mg/kg. Onset time is 10 to 15 minutes, with an analgesic duration of approximately 6 hours. Ketaorolac 30 mg IM is the analgesic equivalent of 10 mg of parenteral morphine and does not produce opioid-related respiratory depression, nausea, or sedation. 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 (gastrointestinal ulcers, inflammatory bowel disease, blood dyscrasias, liver failure, etc.) 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, and those taking angiotensin-converting enzyme inhibitors or angiotensin II receptor antagonists, may require renal perfusion to maintain adequate 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 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 intravenously NSAID combinations can provide effective analgesia for many dentoalveolar procedures at IV and IM doses of 30 to 60 mg or 0.5 to 1.0 mg/kg. Onset time is 10 to 15 minutes, with an analgesic duration of approximately 6 hours. Ketaorolac 30 mg IM is the analgesic equivalent of 10 mg of parenteral morphine and does not produce opioid-related respiratory depression, nausea, or sedation. 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 (gastrointestinal ulcers, inflammatory bowel disease, blood dyscrasias, liver failure, etc.) 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, and those taking angiotensin-converting enzyme inhibitors or angiotensin II receptor antagonists, may require renal perfusion to maintain adequate renal perfusion, and NSAID administration can result in acute fluid retention. This drug is also associated with a higher cost than other analgesic medications.

**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 twentieth 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 s) and shortest duration (5–10 min) of the neuromuscular-blocking agents and is typically used to treat laryngospasm not relieved with positive pressure (20 to 40 mg, or 0.1 to 0.2 mg/kg). It is also given to facilitate tracheal intubation (1 to 1.5 mg/kg IV) 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 sinus 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. SCh is therefore contraindicated in these patients, along with patients in renal failure. SCh is a trigger for MH (see section on malignant hyperthermia). Its use should also be avoided in patients with pseudocholinesterase abnormalities, as 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 administration. The chemical structures of these drugs fall into two classes: benzylisoquinolines and aminosteroids. Characteristics of currently available nondepolarizing muscle relaxants are outlined in Table 5-2.

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 mivacurium has the shortest. 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 twitches produced by stimulation of specific muscles, such as adductor pollicis and orbicularis orbi. Paralysis may need to be reversed by an anticholinesterase to ensure adequate recovery of airway and respiratory muscle function prior to 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.

Most of the nondepolarizing agents are metabolized by the liver and excreted by the kidney. Three of these are less dependent on hepatic or renal function. Mivacurium, like SCh, is metabolized by pseudocholinesterase and is affected by its deficiency. 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. 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 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.

### Table 5-2  Common Neuromuscular-Blocking Medications and Their Properties

<table>
<thead>
<tr>
<th>Time to Intubate (min)</th>
<th>Metabolism and Elimination</th>
<th>Histamine Release</th>
<th>Vagolyis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Succinylcholine</td>
<td>1</td>
<td>1</td>
<td>5–10</td>
</tr>
</tbody>
</table>

The two most commonly used acetylcholinesterase medications are listed. Acetylcholinesterase and anticholinergic medications are given in recommended combinations according to similar 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.

### Table 5-3  Reversal Doses of Acetylcholinesterase and Anticholinergic Medications

<table>
<thead>
<tr>
<th>Cholinesterase</th>
<th>Cholinesterase Dose (mg/kg)</th>
<th>Anticholinergic</th>
<th>Anticholinergic Dose (mg/mg of cholinesterase)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neostigmine</td>
<td>0.4–0.8</td>
<td>Glycopyrrolate</td>
<td>0.2</td>
</tr>
<tr>
<td>Edrophonium</td>
<td>0.5–1.0</td>
<td>Atropine</td>
<td>0.014</td>
</tr>
</tbody>
</table>

Clinical uses in anesthesia of atropine, glycopyrrolate, and scopolamine are defined by their varied effect at the muscarinic receptor sites of different organs (Table 5-4). 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

Postoperative nausea and vomiting (PONV) is one of the most common complaints following 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 is not uncommon. Swallowed blood and secretions stimulate the gag reflex and are potent gastric irritants. Drugs used during sedation and anesthesia, such as N₂O, 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 5-11) 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 (eg, prochlorperazine), and butyrophenones (eg, droperidol). They effectively reduce PONV but are associated with adverse effects such as sedation and extrapyramidal reactions. 5-HT₃ antagonists including ondansetron and dolasetron are expensive, but produce less sedation and other adverse effects than the dopamine antagonists. Antihistamines such as promethazine (which also possesses a phenothiazine structure) and diphendygryamine can cause significant sedation. Anticholinergic medications (eg, scopolamine) are rarely used for PONV, although the antihistamines promethazine and diphendygryamine also possess anticholinergic effects.

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 IV 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 history of PONV. Avoidance of known nausea triggering agents such as N₂O, ketamine, and longer-acting opioid medications may also reduce PONV.

| **Table 5-4 Varied Effects of Anticholinergic Medications** |
|-----------------|-----------------|-----------------|-----------------|-----------------|
| **Anticholinergic Medication Characteristics** |
| Tachycardia | Bronchodilation | Sedation | Antisialagogue |
| Atropine | ### | ## | ≠ | ### |
| Glycopyrrolate | ## | ## | ≠ | ## |
| Scopolamine | ≠ | ≠ | ### | ### |

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

FIGURE 5-11  Diagrammatic representation of the chemoreceptor trigger zone (CTZ). Adapted from Watcha MF and White PF.¹⁸ 5-HT₃ = 5-hydroxytryptamine (serotonin); N₂O = nitrous oxide.
References

Pediatric Sedation

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Jeffrey B. Dembo, DDS, MS
Kevin J. Butterfield, DDS, MD

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 and Physiologic Considerations

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.\(^1\)\(^2\) Such effects can be exacerbated by a decreased minute ventilation and airway tone secondary to sedative medication used during the anesthetic administration.

There are anatomic differences unique to the pediatric upper airway that 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. Since 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 compliance 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 since 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.\(^3\) Additionally, 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
Exchange of gas takes place within the alveoli. Closing volume, which is 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 than in the adult. 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.4-6

Functional residual capacity (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.7 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 to approximately 2:1 in an adult.7 FRC decreases further in the sedated patient. The FRC provides a pulmonary oxygen reserve.8 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.9-11 One model comparing the child to 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.12

Endotracheal Intubation There are also anatomic differences between the pediatric and 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 post extubation. Uncuffed tubes are used by most anesthesiologists for patients less than 8 to 10 years of age.13 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 calculating the appropriate size of endotracheal tube ([age (yr) +16]/4) and the appropriate length of endotracheal insertion ([age (yr)/2 + 12]).14 However, 28% of the time the initially selected uncuffed endotracheal tube does not provide an adequate seal, and re-intubation may be necessary.15 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 cm H2O, 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 cm H2O 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 further 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.16

Certain congenital anomalies are well recognized for their altered anatomy. Some of the most commonly encountered disorders are Crouzon 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 anomaly (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.

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

Cardiac Output Perfusion is dependent on cardiac output and peripheral resis-
Neural Innervation The myocardium is innervated by both the sympathetic and 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 compared with the 2- to 3-year-old. 17

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 since 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.

Preoperative Evaluation of the Patient
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 (eg, cardiovascular status), while performing an appropriate physical examination dictated largely by the patient’s medical history.

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 < 6 years of age frequently cannot comprehend the need for or benefits of the surgical procedure. Children > 6 years old or those who have better-developed social skills (eg, acquired from daycare programs) may be more capable of understanding the situation and expressing their concerns. 18 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. 19

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. 20 Clear, simple, and succinct explanations appropriate for the age of the child may minimize adverse behavior.

Preoperative Fasting
The risk of pulmonary aspiration of gastric contents in the pediatric patient during anesthesia is reported to be up to 10 incidents per 10,000 cases. 21-23 Morbidity secondary to aspiration includes obstruction from particulate material as well as aspiration pneumonitis that is dependent on both the quantity and

<table>
<thead>
<tr>
<th>Function</th>
<th>Age (yr)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2–6</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>100 (80–120)</td>
</tr>
<tr>
<td>Systemic arterial pressure (mm Hg)</td>
<td>75–115/50–75</td>
</tr>
<tr>
<td>Cardiac output (mL/kg/min)</td>
<td>150–170</td>
</tr>
</tbody>
</table>

Table 6-1 Means and Ranges of Normal Cardiovascular Function
acidity of the aspirate. Establishing parameters that minimize the risk of particulate gastric contents as well as decrease the quantity and acidity of residual gastric fluids can decrease the incidence of this morbidity.

Gastric emptying of solids is variable. A 6- to 8-hour fast from solids is recommended to allow gastric emptying and minimize the risk of particulate aspiration. 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 pediatric patients up to 2 hours prior to surgery does not significantly increase the quantity of gastric volume or gastric acidity.24-28 Guidelines have thus been established for healthy pediatric patients that allow unlimited amounts of clear liquids to be consumed up to 2 to 3 hours prior to surgery. This recommendation avoids the need for an extended fast, which has the potential to make the patient irritable and uncomfortable and to increase the incidence of hypotension secondary to dehydration. However, in most cases it still may be simplest to state that the child should have nothing by mouth (NPO) after midnight and to schedule the procedure as the first case in the morning. Children who are scheduled in the afternoon may have a light breakfast at least 6 hours prior to the surgery.

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 he or she 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 H2-antagonist such as cimetidine 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. Glycopyrrolate also reduces the acidity and volume of gastric contents.29 Atropine, alternatively, decreases the tone of the lower esophageal sphincter and predisposes to gastroesophageal reflux of stomach contents.

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.30 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 cuff.31-34 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.35-42 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.43 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 since the incidence of adverse respiratory events is less in a patient anesthetized with a face mask or laryngeal mask airway 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 colleagues demonstrated that a patient with an active or recent URI requires approximately 30% less apneic time to desaturate than does a healthy patient.44

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.\(^{45}\) Considering the above, a delay of 2 weeks is probably acceptable before performing a short office-based minor dentoalveolar procedure in which the patient is not intubated.

**Cardiovascular Evaluation**

The child who presents for surgery with a previously undiagnosed cardiac murmur poses a diagnostic challenge. Innocent murmurs are heard in up to 50% of normal pediatric patients at some point during childhood. The cause of these murmurs is usually turbulent blood flow through any of the great vessels. Features that commonly identify innocent murmurs include those that are crescendo-decrescendo and of short duration and low intensity, and those that occur early in systole. All diastolic murmurs are pathologic. The patient's history may also suggest signs and symptoms of cardiac pathology. These may include limited exercise tolerance, pale color, frequent respiratory problems, hypoxemia, palpitations, or dysrhythmias. A murmur in an asymptomatic child is frequently not pathologic, and no special anesthetic considerations are required. However, if there is uncertainty regarding the significance of a murmur, a consultation with a cardiologist is recommended. For patients with congenital heart disease, prophylaxis against bacterial endocarditis is necessary.

**Pregnancy Testing in the Adolescent Patient**

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%.\(^{46}\) 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.\(^{47}\) However, many patients in this age group may not provide an accurate history, especially in the presence of their family. This is not an acceptable rationale for routine testing. 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.

**Sedative Techniques**

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 nontreating 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 prior to 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.\(^{48}\)

**Routes of Administration**

Sedative medication may be administered by many routes, including oral, intranasal, transmucosal, rectal, intramuscular, inhalational, and intravenous.\(^{49}\) 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 (eg, 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-gauge or 22-gauge angiocatheter. The intraosseous needle is recommended primarily for children < 6 years of age because they still have red bone marrow (Figure 6-1). 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-stilleted 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 inhalational induction is accomplished by administering oxygen or a mixture of oxygen (minimum concentration of 30%) and nitrous oxide using a full face mask. 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 agent and hold his or her 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 (eg, extraction of a deciduous tooth), once general anesthesia is achieved, the face mask can be removed, the procedure performed, the face mask reapplied, and the patient allowed to awaken breathing 100% oxygen. Some clinicians advocate maintaining the general anesthesia by con-
tinuing 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 face mask 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 face mask. 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 (eg, scented lip gloss) selected by the child to the face mask 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 face mask. 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 face mask. 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 age 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. Although frequently used as a sole sedative agent by many surgeons, an oral sedative agent can be used as a premedicant prior to establishing intravenous access or inducing general anesthesia by a different route (eg, 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 felt 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 central nervous system (CNS) through the cribriform plate. Medication may be absorbed through the cribiform 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 felt 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 IV, IM, 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.\(^{57,58}\)

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.\(^{59}\) 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 both in placing or removing the catheter may result in a laceration of the mucosa and cause a greater absorption of drug.

**Pharmacologic Agents**

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. There are numerous agents that 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 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 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 dilatation. 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 intravenous 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 signifi-
cant 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. 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. 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 (eg, benzodiazepines, propofol).

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

Ketamine can be administered IV, IM, 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 as the drug has a rapid onset, within 3 to 5 minutes. Dosing recommendations up to 10 mg/kg IM 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 IM should provide effective sedation. 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. 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 the above 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. Coadministra-

Pediatric Sedation
Midazolam is a water-soluble oral medication. Bioavailability is approximately 81%. 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 IV 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.

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 IV, IM, 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 nasal pharynx and is swallowed. In a study that compared oral to intranasal administration of midazolam, children were found to be less tolerant of the intranasal administration.

Oral midazolam is probably the most widely used premedicant in children. The recommended dose of midazolam is 0.5 to 1.0 mg/kg to a maximum of 20 mg. Midazolam is a water-soluble oral medication. Bioavailability is approximately 81%. 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 IV 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.

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zolam 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 at which 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 more sustained midazolam plasma level.97,98

Higher doses of oral midazolam (0.75 to 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 paradoxic 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 5, “Pharmacology of Outpatient Anesthesia Medications.” 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, IM, and IV. 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 prior to parental separation. Rectal administration, however, can be distressing, as discussed above. 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 IV. 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 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 to 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 US Food and Drug Administration (FDA) for use in children > 3 years of age 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 unproven, clinicians should be aware of the risk for this reaction in children and limit the dose and duration of propofol therapy accordingly.
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.\textsuperscript{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 coadministration 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.\textsuperscript{116–118}

Although nitrous oxide lacks sufficient potency to solely induce general anesthesia, halothane, sevoflurane, desflurane, and isoflurane have sufficient potency to induce and maintain general anesthesia (Table 6-2). 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 (eg, coughing, breath holding, laryngospasm), whereas desflurane and isoflurane tend to irritate the airway if used for mask induction.\textsuperscript{119–121}

The blood and tissue solubility of an inhalational agent is 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.

Since 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 ($\text{PaCO}_2$). 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.\textsuperscript{122} Not all respiratory effects are detrimental. All inhalational agents are beneficial in that they produce bronchial dilatation and are advantageous in the management of the patient with chylothoracic 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).\textsuperscript{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.\textsuperscript{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

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Table 6-2  Inhalational Anesthetic Agents

<table>
<thead>
<tr>
<th>Agent</th>
<th>Blood Solubility</th>
<th>Maximum Acceptable Concentration (%: 1–12 yr</th>
<th>Adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitrous oxide</td>
<td>0.47</td>
<td>—</td>
<td>105.00</td>
</tr>
<tr>
<td>Halothane</td>
<td>2.40</td>
<td>0.87</td>
<td>0.76</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>0.69</td>
<td>2.5</td>
<td>1.70</td>
</tr>
<tr>
<td>Desflurane</td>
<td>0.42</td>
<td>7.98–8.72</td>
<td>7.30</td>
</tr>
<tr>
<td>Isoflurane</td>
<td>1.40</td>
<td>1.60</td>
<td>1.20</td>
</tr>
</tbody>
</table>

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 for halothane 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 (eg, increasing an agent by 0.5–1% after a few breaths) or a high initial concentration of an agent (eg, 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. 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). 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 < 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. In one study, in which children were subject to a 4-minute anesthesia, time to eye opening was 102 seconds with halothane and 167 seconds with sevoflurane.

The last factor that needs to be considered both in comparing sevoflurane and halothane 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. The incidence, which may be as high as 1 in 6,000 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. 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 > 50 µmol. Renal injury has also been associated with the formation of compound A, which is a product of the reaction between sevoflurane and CO₂ 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 h), 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. 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 antihistaminic 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. The difference in bioavailability results from the amount of drug that is swallowed and the amount of drug that is 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 to 3 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.

**Perioperative Complications**

**Laryngospasm**

Intraoral surgery in the anesthetized non-intubated patient renders the patient susceptible to airway obstruction and airway irritation. Such irritation can result in a
laryngospasm, which is the apposition of the supraglottic folds, the false vocal cords, and the 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 1,000 patients in the total population and 17.4 per 1,000 in patients <9 years of age.39

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, then 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 (eg, tracheal tug, paradoxic respiration) should be treated aggressively. The first maneuver is to apply gentle continuous positive airway pressure with 100% O2 by face mask. 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 the child. 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 that is 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 the child is hypoxemic, atropine 0.02 mg/kg should precede the administration of the succinylcholine to prevent a bradycardia secondary to the muscarinic effect of succinylcholine. If intravenous access is not available, succinylcholine may be administered intralingually or IM (succinylcholine 4 mg/kg).141

There are several potential complications 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 CO2 is the most sensitive sign of malignant hyperthermia.142,143

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.144,145

Alternative neuromuscular agents have been developed that can provide rapid onsets 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.

**Cricothyrotomy**

Three approaches to emergency surgical opening of the airway are mentioned in the literature: emergency tracheotomy, emergency cricothyrotomy, and emergency transtracheal ventilation.146 In the experience of most, emergency tracheotomy cannot be performed rapidly enough in dire situations. Likewise, transtracheal jet ventilation is extremely hazardous in children because barotrauma may occur owing to the restricted egress of ventilatory gas. Therefore, when endotracheal intubation cannot be accomplished, the most rapid method for oxygenating the patient in an emergency situation is cricothyrotomy.147
Nausea and Vomiting

Postoperative 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. PONV occurs in 6 to 42% of all pediatric surgical patients. 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 pediatric patients.148

Anesthetic drug selection can have an effect on the incidence of PONV. Preoperative midazolam has been associated with reduced PONV in children.149 Sub-sedative doses of propofol also provide antiemetic effects. This contrasts with methohexital, which is associated with a higher incidence of PONV than is propofol in adults. Studies are lacking comparing the incidence of PONV of these two agents in a pediatric population. 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.140 As discussed above, ketamine is an excellent agent for pediatric sedation. An unfortunate adverse effect associated with ketamine is a reported incidence of PONV that is as high as 50%. Nitrous oxide also has emetic effects. However, concentrations < 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 children undergoing surgery is not justifiable as the majority do not experience PONV or have, at most, one or two episodes. The agents used are the same as those used to manage PONV in the adult. The following discussion identifies points significant to the management of PONV in the pediatric patient.

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 between phenothiazines, with little sedation produced by perphenazine compared with the other phenothiazines.150

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. 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.151,152

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 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.153 Transdermal scopolamine has been used successfully to reduce PONV in children receiving morphine but is associated with a significant increase in sedation and dry mouth.154 Other potential side effects include dysphoria, confusion, disorientation, hallucinations, and visual disturbances.

Serotonin Receptor Antagonists Serotonin antagonists were discovered serendipitously when compounds struc-
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. Psychomotor and respiratory function were unaffected by ondansetron. Prophylactic ondansetron 0.05 to 0.15 mg/kg IV or orally reduced the incidence of PONV in children after a variety of surgical procedures.

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 up to 1 mg/kg IV (maximum dose 25 mg) was effective in reducing postoperative vomiting in children after tonsillectomy. However, low-dose dexamethasone 0.15 mg/kg IV was not as effective as perphenazine 70 µg/kg IV in preventing emesis after tonsillectomy in children. This class of drugs is better used in combination with another antiemetic than as the sole agent to prevent PONV.

**Special Considerations**

Oral and maxillofacial surgeons treat a diverse group of patients. Simplistically, the pediatric patient differs from the adult patient anatomically, physiologically, and behaviorally. Beyond these differences the pediatric population is a diverse group within itself. Oral and maxillofacial surgeons are involved with the management of patients with craniofacial syndromes as well as other physical or mental impairments. The craniofacial syndromes may result in anatomic and physiologic alterations as well as mental disabilities. Potential airway abnormalities include macroglossia, micrognathia, choanal atresia, limited mouth opening, kyphoscoliosis, or cervical spine abnormalities. These abnormalities may make the patient more susceptible to upper airway obstruction and compromise spontaneous ventilation, oxygenation, mask ventilation, or laryngoscopy and intubation. Many of these patients may have significant cardiovascular disease associated with their syndrome. Mental impairment may also be associated with several congenital syndromes. Alternatively, 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. Lastly, substance abuse among children and teens has reached epidemic proportions.

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. It 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 US high-school population without a diagnosed medical condition is reported to abuse this drug. 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. Chronic use of the medication may decrease anesthetic requirements.

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 IM 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.\textsuperscript{162} 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 impacts 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.\textsuperscript{163,164} 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 often times necessitate the use of psychiatric medications; antipsychotics are the most prevalently prescribed medications in this group.\textsuperscript{165} 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 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.\textsuperscript{169} 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 that are 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.\textsuperscript{167} Mental impairment is most common in patients with spastic cerebral palsy. It is important to recognize that > 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 increase 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.\textsuperscript{167} 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.\textsuperscript{168}

Down Syndrome

Down syndrome, or trisomy 21, is a common chromosomal disorder occurring at a rate of 1.5 per 1,000 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 temporomandibular joint 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 prior to 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 to 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, although milder, to 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.169

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 < 35% of normal are at increased risk. Muscle weakness also contributes to obtund laryngeal reflexes and an inability to clear tracheobronchial secretions. Patients are at increased risk for aspiration secondary to the obtund 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. Additionally, patients are susceptible to an unexplained late respiratory depression. Ambulatory surgery may be unadvisable but at a minimum requires prolonged observation prior to discharge.170

**Substance Abuse**

Substance abuse amongst 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 prior to the survey interview. This estimate represents 7.1% of the population ages 12 years old or older. Among youths ages 12 to 17 years, approximately 10% 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 (approximately 3%), cocaine (approximately 0.5%), hallucinogens (approximately 1%), and inhalants (approximately 1%). An adequate history taking prior to 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.
Alcohol  Alcohol is the most commonly used and abused substance among teenagers. Most alcohol use by US 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 pre-adult abuser. Nonetheless, laboratory examinations may reveal elevation of γ-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 > 1. A mean corpuscular volume > 100 is strong confirmatory evidence of alcoholism.

Aspiration risk is significantly increased in the chronic alcoholic as 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 negate 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 paralysis 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 γ-aminobutyric acid (GABA)-ergic stimulation.

Amphetamine  Amphetamine, a racemic mixture of β-phenylisopropylamine, is an indirect sympathomimetic drug. It is a powerful CNS stimulant with peripheral α and β 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. Chronic amphetamine use is associated with a markedly diminished anesthetic requirement. 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 sniffed (intranasal), injected (intravenous), or smoked (inhaled). Its administration provides an intense euphoria. Cocaine use amongst 12- to 17-year-olds in the United States is approximately 0.8%,174

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. 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”  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.

In 2001 an estimated 8.1 million (3.6%) of Americans ages 12 or older had tried ecstasy at least once in their lifetime. The principle constituent of ecstasy (MDMA) can produce robust deleterious effects on serotonergic functioning in animals, including serotonin depletion and the degeneration of serotonergic nerve terminals. 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, methylenedioxymethamphetamine, 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. 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** Approximately 1% of 16-year-olds in the United States used lysergic acid diethylamide (LSD) in 2001. 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 > 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.

**Marijuana** Marijuana is the most commonly used nonalcohol illicit drug for people < 18 years old. In 2001 it was used by 76% of current illicit drug users. 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 hyperreactivity. Cases of laryngospasms within 36 hours of its use have been reported. A β2-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. Myocardial depression can occur, and the threshold for sympathomimetic-induced dysrhythmias is lowered.

**PCP** PCP is a dissociative anesthetic that originally was synthesized for intravenous use. Because of its postoperative emergence reactions (ie, 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
Maintaining normotension and avoiding potential for tachycardia, tachyarrhythmia, similar to its congener, ketamine, with the systemic management.

PCP's effects, are the standard for anesthetics, which may exacerbate sympatomimetics, which may exacerbate PCP's effects, are the standard for anesthetic management.

Summary

Ambulatory anesthesia in the pediatric 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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Part 2

DENTOALVEOLAR SURGERY
The management of impacted teeth is a basic component of most oral and maxillofacial surgery practices. Although the majority of impacted teeth are third molars, any other tooth may be impacted. The usual care for impacted third molars is removal; however, the care for impacted teeth other than third molars may include exposure (with or without attachment of an orthodontic bracket), uprighting, transplantation, or removal. These teeth often pose challenges in treatment planning and surgical care. This chapter includes information on incidence, etiology, evaluation, and surgical treatment options.

Incidence

The incidence of impacted permanent teeth has been addressed in several studies. Grover and Lorton examined 5,000 army recruits and found a high frequency of impacted teeth (Figure 7-1). Although maxillary and mandibular third molars were the teeth most commonly impacted, 212 teeth excluding third and fourth molars were impacted. This study identified the maxillary canine as the tooth most likely to be impacted following maxillary and mandibular third molars. Impactions of every permanent tooth were identified except the mandibular incisors and first molars. Thilander and Myrberg examined more than 6,000 Swedish school children and found a 5.4% prevalence of impacted teeth excluding third molars. In an evaluation of 3,874 full-mouth radiographs, Dachi and Howell found the incidence of impacted canines in the maxilla to be 0.92% and of other non–third molar teeth to be 0.38%. This study also identified maxillary canines as the most commonly impacted teeth after maxillary and mandibular third molars. In a study of middle-aged and older Swedish women, Grondahl found approximately 25 non–third molar impacted teeth in 1,418 women evaluated. Again, the canine tooth was the most frequent non–third molar impaction identified, followed by premolars and second molars. This study examined an older population than did most of the other studies and had a lower incidence of non–third molar impacted teeth. Presumably symptomatic teeth and those with pathologic findings were removed at earlier ages in this population. These studies are all similar in identifying the maxillary canine as the tooth most likely to be impacted following third molars. The next most likely teeth to be impacted are mandibular bicusps, followed by maxillary bicusps and second molars. Impactions of first molars and incisors are relatively uncommon (Figure 7-2). Although impaction of permanent teeth is a relatively common finding, the lack of eruption of a primary tooth is apparently quite rare. When it occurs it is almost always a mandibular molar. Submerged teeth are common in the primary dentition but generally reflect teeth that erupted into a normal position and later became ankylosed and secondarily submerged. Bianchi and Roccuzzo have identified 10 cases in the literature of the past 20 years that appear to illustrate primary impaction of deciduous teeth. A recent review suggests that primary tooth impaction is usually associated with defects in the development and eruption of the permanent successor, suggesting the need for long-term follow-up.
Etiology

The definition of an impacted tooth is “a tooth that can not, or will not, erupt into its normal functioning positions, and is, therefore, pathologic and requires treatment.”\(^8\) Causes of impacted permanent teeth include systemic and local factors. Impaction of teeth in the hereditary syndrome of cleidocranial dysplasia (Figure 7-3) is more properly termed primary retention.\(^5\) Endocrine deficiencies (hypothyroidism and hypopituitarism), febrile diseases, Down syndrome, and irradiation are other systemic factors that may influence impaction of permanent teeth.\(^9,10\) In all of these systemic conditions, multiple teeth are generally involved. 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.\(^5,11,12\)

Because the maxillary canine is relatively commonly impacted, it has been studied to identify the causes of this tooth impaction. Jacoby separates labially unerupted maxillary canines from palatally impacted canines in his evaluation of the cause of failure of eruption of these teeth.\(^11\) 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.\(^11\) In a review of impacted maxillary canines, Bishara stated 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.\(^9\)

Impacted second molars have been studied to determine the cause of these impactions. Although maxillary second permanent molars are infrequently impacted, in a study of these impactions, Ranta found that the third molar was generally positioned occlusally and palatally in relation to the second molar, acting as an obstruction (Figure 7-4).\(^13\) In a similar study Levy and Regan identified the most probable cause of impaction of developing second molars as malposition of the tooth germs of the maxillary third molars.\(^10\) A typical finding was deformation of the mesial surfaces of the crowns and roots of the third molars. Raghoebar and colleagues stated that impaction of first molars is often diagnosed as ectopic eruption, whereas impaction of second molars is usually associated with arch-length deficiency.\(^5\)

Clinical problems have been identified associated with impacted permanent teeth. Failure of teeth to erupt into their normal position in the arch may result in problems that include malocclusion, loss of arch length, migration or loss of neighboring teeth, periodontal disease, root resorption of adjacent teeth, resorption (internal or external) of the impacted
tooth, dentigerous cysts or odontogenic tumors, and pericoronitis.5,9

Evaluation
Clinical 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 unerupted position of the tooth.

Radiographic assessment of the impacted teeth is important in the preparation for surgical or orthodontic treatment. Most techniques for localization of an impacted tooth have been studied primarily with maxillary canines. These techniques, however, can be generalized to other teeth in the oral cavity. Ericson and Kurok have studied the radiographic appearance of ectopically erupting maxillary canines and have found that a palpable canine generally erupts in a relatively normal position.14 Most canines can be evaluated with accuracy from conventional periapical films. Axial or panoramic films were less useful.14 When polytomograms were used, root resorption was diagnosed with greater accuracy. This study indicated that the optimal age for evaluating an ectopically positioned canine was 10 to 13 years, depending on individual development.15 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.16 However, the higher cost and radiation dose of CT limits its use to impacted teeth in unusual positions or in proximity to vital structures.

Standard radiographic techniques may be used to localize the unerupted teeth. These include the tube shift method, buccal object rule, and periapical occlusal method.17 The tube shift method uses two periapical radiographs, shifting the tube horizontally between exposures. If the unerupted tooth moves in the same direction in which the tube is 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.17 The buccal object rule uses two radiographs taken with different vertical angulations of the x-ray beam. An object located on the buccal side moves inferiorly with the beam directed inferiorly, whereas an object located in a lingual or palatal position moves superiorly. 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.17

Panoramic films can be used to assess maxillary canine position (Figure 7-5).18 This technique uses the property that an object closer to the tube (palatal) is relatively magnified, and is most accurate when the tooth is close to the alveolar crest. A study comparing magnification from a panoramic radiograph with a vertical parallax from occlusal and panoramic films showed a slight superiority for the vertical parallax method. Both methods were better at localizing palatal cuspids than labial cuspids.19

Surgical Treatment
Treatment of impacted permanent teeth must be based on clinical and radiographic evaluation as well as a determination of future risks. Clearly, teeth that are symptomatic, have caused infection in the surrounding tissues, or have radiographic evidence of development of changes (cyst formation, resorption of adjacent teeth, or root resorption of the impacted teeth) require surgical treatment. Treatment of the asymptomatic tooth must take into account many factors, including age, specific prevalence of pathologic conditions, severity of potential pathology associated with impacted teeth, progression of untreated conditions, frequency and severity of potential complications of treatment, potential patient discomfort and inconvenience associated with either treatment or nontreatment, and economic consequences of treatment.4 Methods of treatment of impacted permanent teeth include orthodontic assistance through surgical exposure with or without attachment of the tooth, surgical uprighting, transplantation, and surgical removal.

Exposure
Surgical exposure is a procedure that allows natural eruption of impacted teeth.9,20 Ohman and Ohman studied 542 impacted teeth in 389 patients.20 In this study the crowns of the teeth were surgically exposed with removal of tissues in the direction most appropriate for crown movement. The wounds were packed until they were totally epithelialized. 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 < age 19 years.20

In a study of impacted premolars, Thilander and Thilander showed that surgical exposure alone resulted in eruption, provided that space was present in the arch.21 However, mesially tipped premolars had a poor prognosis and required orthodontic
guidance. Laskin and Peskin 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.22

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 adequate space within the dental arch for the impacted tooth, and anchorage. Many appliances have been advocated, including polycarbonate crowns and pins inserted into the structure of the tooth. Both of these techniques are used rarely because of the problems of availability of bonded orthodontic brackets/buttons.

Wires placed around the cervical line of the tooth have been a common method of orthodontic guidance; however, this technique has been regarded as relatively invasive. A clinical report in 1981 identified external resorption as a possible sequela of the wide exposure at the cementoenamel junction (CEJ) that is necessary for placement of a cervical wire.23 This complication was studied by Kohavi and colleagues in 1984 in 23 patients who had surgical exposure and attachment of a cervical wire to the tooth.24 The teeth were separated into two groups; one had "light exposure" for placement of a band not exposing the CEJ, and the second had "heavy exposure" involving the removal of bone, complete removal of the follicular sac, and full exposure of the CEJ. This study showed significantly more damaging effects of the heavy exposure technique, and the authors recommended avoiding exposure of the neck of the tooth for placement of a cervical wire.24

Although the use of attachments such as rare earth magnets has been advised for the movement of teeth, the most common method is the placement of a bonded orthodontic bracket.25 This can usually be done with a conservative exposure of the tooth, removing only enough soft tissue and bone to place the bonded bracket, and avoiding exposure of the CEJ.9

Studies have compared simple exposure with packing to maintain a gingival path for eruption, with exposure and bonding of a bracket. Iramaneerat and colleagues found that there was no difference in total orthodontic treatment time for the two techniques.26 Pearson and colleagues found that bracketing was more costly and more likely to require reoperation.27 Nonetheless, placing a bracket is the more popular technique, perhaps owing to orthodontist preference and patient comfort.

For the most common type of non–third molar impaction, the maxillary palatal cuspid, the typical surgical exposure involves reflection of the full-thickness palatal flap, conservative exposure of the tooth, and bonding of a bracket to its palatal surface (Figure 7-6). If the tooth is near the free edge of the flap, soft tissue may be removed to leave the 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. 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.28

Management of the cuspid that is impacted on the labial side follows the same general principles as for the palatally impacted cuspid. A position in the arch must be established by preliminary orthodontic treatment prior to cuspid exposure. An additional important factor for the labially impacted cuspid is preservation of attached mucosa adjacent to the cervical line of this tooth. Generally the most appropriate technique is to begin with a full-thickness mucoperiosteal flap to identify the position of the impacted tooth. The crown of the tooth is conservatively uncovered, and a bonded bracket is attached; then vertical releasing incisions are made to provide a broadly based flap that is superiorly repositioned to cover the CEJ of the tooth. The bonded bracket helps to support the attached gingiva in this apical relationship (Figure 7-7). As the
Management of Impacted Teeth Other than Third Molars

That avoiding treatment of unerupted or submerged teeth may result in occlusal and periodontal problems for adjacent and opposing teeth. An important factor in the treatment of impacted molars is removal of the third molars that prevent the second molars’ normal eruption (Figure 7-8). Ranta stated that it is typical for impacted second molars to erupt normally when the offending third molar is removed. 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. Vig also recommends routine removal of the third molar when a second molar is impacted.

Consequently, surgical repositioning of impacted mandibular second molar teeth and occasionally first molars is the usual treatment of choice. When impaction of a second molar is identified, consideration should be given to correcting the impaction before the roots are fully formed. 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. The technique for second molar uprighting begins with the removal of the third molar (Figure 7-9). 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 CEJ of the second molar. After adequate distal space is obtained, the second molar may be gently lifted superiorly and posteriorly to clear the height of contact of the adjacent first molar.

Most second molars are relatively stable after being lifted past the height of contour of the first molar. Usually it is not necessary to fix the tooth into position (see Figure 7-9B). An extremely important part of this surgical procedure

Uprighting

Surgical uprighting of teeth has been applied most commonly to impacted molars. Reynolds identifies several reasons for uprighting lower molar teeth, including providing occlusion with opposing teeth and proximal contacts with adjacent teeth, minimizing the risk of caries and periodontal disease, and assisting in orthodontic treatment. Palczyny adds...
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is ensuring that there are no occlusal forces on the repositioned second molar. This generally does not require equilibration on the opposing tooth, but an occlusal adjustment can be performed if necessary. Antibiotics are prescribed following this procedure.

An endodontic evaluation should be performed 3 weeks following the uprighting of the tooth. When a tooth with fully developed roots is repositioned, endodontic treatment, if indicated, should be undertaken approximately 6 to 8 weeks following the surgery. Radiographs should be taken at 6-month intervals for 2 years to evaluate the postoperative course (see Figure 7-9C).31

Transplantation

Transplantation of teeth has been advocated as an alternative to other methods of treatment of impacted teeth. It may be appropriate for the adult patient who cannot undergo conventional orthodontic movement of a canine or premolar. Sagne and Thilander studied 47 patients with 56 canines that were surgically transplanted.33 The advocated technique is a careful wide exposure of the impacted tooth. The tooth is then moved into its position 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 following surgery. 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 7-10).33

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. Impacted canines should be approached from the surface of the maxilla with which they are most closely associated. Labially impacted canines are frequently removed with an elevator technique, but palatal canines 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.

Impacted maxillary bicuspids may be removed much like canines. Mandibular bicuspids 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 bicuspid 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 bicuspid to be pushed through to the lingual. Removal of impacted molars is similar to removal of impacted third molars.

Summary

Impacted teeth other than third molars are relatively common findings. Much can be done to preserve these teeth and allow their functional positioning within the dental arch. Surgical exposure with or without orthodontic guidance, surgical uprighting, and transplantation of teeth are valuable techniques that can be mastered by oral and maxillofacial surgeons. Although some studies have indicated that routine removal of impacted teeth is not necessary, removal is indicated in many different situations.
References
Impacted Teeth

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Removal of impacted teeth is one of the most common surgical procedures performed by oral and maxillofacial surgeons, and most surgeons cite third molar removal as the operation most likely to humble them. Extensive training, skill, and experience are necessary to perform this procedure 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 is no less problematic. In many situations this decision is made based on clinical experience and professional judgment; in others the decision is clear cut based on available scientific data. Contemporary medical and dental practices demand evidence-based decision-making, and the surgeon is called on more and more frequently to justify surgical procedures, including the removal of third molars.

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

Development of the Mandibular Third Molar

The mandibular third molar is the most commonly impacted tooth. It also presents the greatest surgical challenge and invites the greatest controversy when indications for removal are considered. When the surgeon is determining whether a specific third molar will become impacted and whether it should be removed, he or she needs to have a clear understanding of the development and movement of the third molar between the ages of 7 and 25 years.

A number of longitudinal studies have clearly defined the development and eruption pattern of the third molar.4–7 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. By age 24 years 95% of all third molars that will erupt have completed their eruption.

The change in orientation of the occlusal surface from a straight anterior inclination 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.

Most third molars do not follow this typical 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 claims that there may be differential root growth between the mesial and distal roots, which causes the tooth to either remain mesially inclined or rotate to a vertical position depending on the amount of root development.7,8 In their studies they have found that underdevelopment of the mesial root results in a mesioangular impaction. Overdevelopment of the same root results in over-rotation of the third molar into a distoangular

†Deceased.
impaction. Overdevelopment of the distal root, commonly with a mesial curve, is responsible for severe mesioangular or horizontal impaction. The Belfast Group has noted 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 over-rotation from mesioangular to distoangular. These over-rotations 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 there is inadequate bony length, there is a higher proportion of impacted teeth. In general, patients with impacted teeth almost invariably have larger-sized teeth than do those without impactions. Even when the tooth-bone relationship is favorable, a lower third molar that is positioned lateral to the normal position almost always fails to erupt. This may also be the result of the dense bone present in the external oblique ridge.

A final factor that seems to be associated with an increased incidence of tooth impaction is retarded maturation of the third molar. 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 in a 20-year-old, an impacted third molar with partially developed roots is less likely to erupt than a similarly positioned tooth with fully developed roots.

### 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 the factors that influence eruption potential.

It is important to remember that eruption of lower third molars is complete at the average age of 20 years but that it can occur up to age 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. It is fairly well established that the position of retained third molars does not change substantially after age 25 years, although there is some evidence of continued movement as late as the fourth decade. Many patients are evaluated for third molar removal in their late teens, and the surgeon must therefore attempt to discern the probable outcome of the eruption process based on more than tooth position alone.

Numerous studies have evaluated the influence of various factors on the eruption potential of a lower third molar. Two factors consistently emerge as most prognostic: angulation of the third molar and space available for its emergence. By age 18 to 20 years, lower third molars that are horizontal or strongly mesioangular have much less eruption potential than do those that are 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 > 1 or if the tooth orientation diverges substantially from vertical, the tooth is unlikely ever to erupt fully.

### Indications for Removal 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.

#### Pericoronitis Prevention or Treatment

When a third molar, usually the mandibular third molar, partially erupts through the oral mucosa, the potential for the establishment of a mild to moderate inflammatory response similar to gingivitis and periodontitis exists. In certain situations the patient may actually experience a severe infection, which may require vigorous medical and surgical treatment. The bacteria that are most commonly associated with pericoronitis are *Peptostreptococcus*, *Fusobacterium*, and *Bacteroides* (*Porphyromonas*). Initial treatment of pericoronitis is usually aimed at debridement 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.
Prevention of recurrent pericoronitis is usually achieved by removal of the involved mandibular third molar. Although operculectomy has been recommended for management of this problem, the soft tissue redundancy usually recurs owing to the relationship between the anterior border of the ramus and the fully or partially erupted mandibular third molar. Pericoronitis can occur whenever the involved tooth is partially exposed through the mucosa, but it occurs most commonly around mandibular third molars that have soft or hard tissue lying over the posterior aspect of the crown.23

Approximately 25 to 30% of impacted mandibular third molars are extracted because of pericoronitis or recurrent pericoronitis.14,24–27 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.

**Prevention of Dental Disease**

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.14,24–27 As with pericoronitis, the presence of caries and eventual pulpal necrosis are responsible for an increasing percentage of extractions with age.

The presence of the partially impacted third molar and the patient’s inability to clean the area thoroughly may result in early advanced periodontal disease. This is the primary reason for removal of approximately 5% of impacted third molars.14,24–27 Even young patients in otherwise good general periodontal health have a significant increase in periodontal pocketing, attachment loss, pathogen activity, and inflammatory markers at the distal of the second molar and around the third molar.28–30 In patients whose dental health is poor and who have partially erupted third molars, the periodontal condition around the second molar and partially erupted third molar can become extremely severe at an early 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 below.

**Crowding of Mandibular Incisors** Perhaps one of the most controversial issues regarding mandibular third molars has been the issue of their influence on anterior crowding of mandibular incisor teeth, especially after orthodontic therapy. A variety of studies have been reported that support both sides of the controversy. Many of these studies have been reviews of small numbers of patients or of anecdotal information.31,32 More recent literature includes longitudinal reviews of orthodontically treated patients in larger numbers,33,34 and the preponderance of evidence now suggests that impacted third molars are not a significant cause of post-orthodontic anterior crowding. In fact, anterior incisor crowding is associated with deficient arch length rather than the mere 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. On the other hand, following maxillary down-fracture a deeply impacted upper third molar is often easily approached superiorly 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.35 The general incidence of neoplastic change around impacted molars has been estimated to be about 3%.36,37 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.14,24–27 These pathologic entities are usually seen in patients under age 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%. 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 and the mandibular third molar displaces significantly into the roots of the second molar, 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 more than 40 years old 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 often need to be removed and the dental prosthesis either altered or refabricated.

Each situation must be viewed individually, and the risks and benefits of removing the impacted tooth must be given careful 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, martial arts, and some so-called noncontact sports such as basketball, 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. Additionally, 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. 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.

When a patient presents with this type of complaint, the surgeon must make sure that all other sources of pain are ruled out before suggesting surgical removal of the third molar. In addition, the patient must be informed that removal of the third molar may not relieve the pain completely.

Summary

The preceding discussion has dealt with the indications for removal of symptomatic impacted third molars. Most clinicians agree that if a patient presents with one or more of the above pathologic problems or symptoms, the involved teeth should be removed. It is much less clear what should be done prophylactically with teeth that are impacted before they cause 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.

Contradictions for Removal 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.

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. Although some clinicians report that removal of the tooth bud of the developing third molar at age 8 or 9 years can be accomplished with minimal surgical morbidity, the general 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.
relativelyatraumatically 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.

As a patient becomes older there is decreased healing response, which may result in a greater bony defect postoperatively than was present because of the impacted tooth. Additionally, 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. As a patient ages, the response to surgical insult is tolerated less easily 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 following third molar extraction is much higher in the patient over age 40 years compared with patients under age 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 over age 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 younger compromised patient but also the younger compromised patient, the surgeon occasionally needs to remove symptomatic as well as asymptomatic third molars. The compromised medical status becomes 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 (eg, 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. When fully developed, totally bone-impacted third molars are present around the inferior alveolar nerve; it may be best to leave that impacted tooth in place and not risk permanent anesthesia of the inferior alveolar nerve. In such situations the potential risk of development of pathologic problems would be relatively small, and, therefore, the advantage of removal of such a tooth would not outweigh the potential risks. Surgical extraction of impacted third molars can result in significant bony defects that may not heal adequately in older patients and, in fact, may result in the loss of adjacent teeth rather than the improvement or preservation of periodontal health. This also would be viewed as a contraindication to removal of the impacted tooth.

**Surgery and Perioperative Care**

**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 importantly, 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.45

Other factors have been implicated in making the extraction process more difficult. Roots can be either conical and fused 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 is one that must be learned on a theoretic basis and then performed repeatedly to gain adequate experience. There is more variety in presentation of the surgical situation of impacted third molars than in any other dental surgical procedure. Therefore, extensive experience is required to master their removal. A variety of textbooks are available that describe in detail the technique for removal of the different types of impactions.46,47

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 debrided 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, which 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 8-1A 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 (Figure 8-1C 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, and this may be bothersome during the early portion of the surgery.

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. High-resolution magnetic resonance imaging has demonstrated that the lingual nerve may be intimately associated with the lingual cortical plate in the third molar region in 25% of cases and be above the lingual crest in 10%.48 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 8-2A 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 (Figure 8-2C and D), although it
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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 low-torque air-
driven handpiece, although a few surgeons
still choose to use a chisel for bone
removal. The most recent advance is the
relatively high-speed high-torque electric
drill, which has some significant advan-
tages in reducing the time required for
bone removal and tooth sectioning. It is
essential that the handpiece exhaust the air
pressure away from the surgical site to pre-
vent tissue emphysema or air embolism,
and that the handpiece can be sterilized
completely, usually in a steam autoclave.

Bone on the occlusal, buccal, and
cautiously on the 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 8-3). 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.

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 impact-
ed tooth is divided is dependent on the
angulation of the impaction. Tooth sec-
tioning is performed either with a bur or
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 eleva-
tor 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 usu-
ally 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 deliv-
ered, and the remainder of the tooth is
removed with 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

FIGURE 8-1 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 overly-
ing 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. Adapted from Peterson
LJ. Principles of management of impacted teeth. In: Peterson LJ, Ellis E III, Hupp JR, Tucker MR, edi-
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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 8-5).

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 8-6). 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 8-7). 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.

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 8-8).

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

![Figure 8-2](image-url)  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 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. Adapted from Peterson LJ, Ellis E III, Hupp JR, Tucker MR, editors. Contemporary oral and maxillofacial surgery. 4th ed. St Louis: CV Mosby; 2003.

![Figure 8-3](image-url)  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. Adapted from Peterson LJ, Ellis E III, Hupp JR, Tucker MR, editors. Contemporary oral and maxillofacial surgery. 4th ed. St Louis: CV Mosby; 2003.
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the dental follicle. Finally, the socket and wound should be thoroughly irrigated with saline or sterile water (30 to 50 mL is optimal).49 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.

**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

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**FIGURE 8-4 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. Adapted from Peterson LJ, Ellis E III, Hupp JR, Tucker MR, editors. Contemporary oral and maxillofacial surgery. 4th ed. St Louis: CV Mosby; 2003.

**FIGURE 8-5 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. Adapted from Peterson LJ, Ellis E III, Hupp JR, Tucker MR, editors. Contemporary oral and maxillofacial surgery. 4th ed. St Louis: CV Mosby; 2003.
exact incidence of postoperative infection is unknown. In the usual sense of the word, infection probably is a rare occurrence following 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. 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 following third molar surgery.

A more subtle type of wound healing problem that occurs after the surgical removal of the impacted mandibular third molar.
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.53–60 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 are 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 use of perioperative systemic antibiotic administration does not seem to be valid.

**Use of Perioperative Steroids**

Just as the oral and maxillofacial surgeon desires to minimize the incidence of infection following 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 following surgery. The two most widely used steroids are dexamethasone and methylprednisolone. Both of these are almost pure glucocorticoids, with little mineralocorticoid effect. Additionally, these two appear to have the least depressing effect on leukocyte chemotaxis. Common dosages of dexamethasone are 4 to 12 mg IV at the time of surgery. Additional oral dosages of 4 to 8 mg bid on the day of surgery and for two days afterward result in the maximum relief of swelling, trismus, and pain. Methylprednisolone is most commonly given 125 mg IV at the time of surgery followed by significantly lower doses, usually 40 mg PO tid or qid, later on the day of surgery and for two 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.61–65

**Expected Postoperative Course**

Surgical removal of impacted third molars is associated with a moderate incidence of complications, around 10%.66,67 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.68–70 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.12 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. 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). A large multicenter prospective study, the Third Molar Project, has produced detailed data on this issue.71,72

The performing surgeon must be intimately familiar with this information if he or she 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 following surgery. The most effective way to achieve hemostasis following 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 (eg, 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. 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 following 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. 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; 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 analgesics 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 correlate 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.

**Complications of Impaction Surgery**

**Infection**

An uncommon postsurgical complication related to the removal of impacted third molars is infection. The incidence of infection following the removal of third molars is very low, ranging from 1.7 to 2.7%. Infection after removal of mandibular third molars is almost always a minor complication. About 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.
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. 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 following 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%. 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. 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. 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%. Copious irrigation of the surgical site with large volumes of saline is also effective in reducing dry socket. Topical placement of small amounts of antibiotics such as tetracycline or lincomycin may also decrease the incidence of alveolar osteitis.

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.

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 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 or alveolar and lingual nerves following third molar surgery is about 3%. 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.

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. 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.\textsuperscript{93} 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 textbook.

**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 return 2 to 4 weeks later to remove it. If the tooth is asymptomatic, is not causing any restriction in jaw movement, and is not causing 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 made 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.\textsuperscript{94}

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 following extraction in patients over age 40 years.

**Periodontal Healing after Third Molar Surgery**

Two of the important reasons for removing impacted third molars is to preserve periodontal health or, in some situations, to treat a periodontitis that already exists.\textsuperscript{23} 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 contraindicted 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,\textsuperscript{95–97} although some studies have indicated a net gain in bone level after surgery.\textsuperscript{98} 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 following the 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.\textsuperscript{99–101} 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.\textsuperscript{95,102,103} 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.\textsuperscript{97} The long-term healing in this group continues for up to 4 years after surgery, with continuing reduction in probable pocket depths.\textsuperscript{100} However, long-term follow-up of older patients clearly demonstrates that this long-term healing does not occur.\textsuperscript{98,100} 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 following third molar extraction.

In summary, periodontal healing following 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. 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 periodontal healing after surgery as possible. 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 age 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 over the past three decades. 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 may be expected to appear in the scientific literature for the next several years.

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 most attempts at very early prediction of impaction and removal of tooth buds at age 8 or 9 years have now been generally abandoned, it is reasonable that by age 17 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 17 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.

References


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 esthetically.

In the late 1970s Brånemark and colleagues 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 > 30% of patients older than 65 years are completely edentulous. 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. Since 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 twenty-first 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 esthetics.

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. As a result, there is proportionally a qualitative and quantitative loss of tissue, resulting in adverse skeletal relationships in essentially all spatial dimensions (Figure 9-1).

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
Part 2: Dentoalveolar Surgery

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 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 the above effects, a goal-oriented approach to treatment is the most appropriate. The overall objectives include the following: (1) to eliminate pre-existent or recurrent pathology; (2) to rehabilitate infected or inflamed tissue; (3) to reestablish maxillomandibular relationships in all spatial dimensions; (4) to preserve or restore alveolar ridge dimensions (height, width, shape, and consistency) conducive to prosthetic restoration; (5) to achieve keratinized tissue coverage over all load-bearing areas; (6) to relieve bony and soft tissue undercuts; (7) to establish proper vestibular depth and repositioning of attachments to allow for prosthetic flange extension if necessary; (8) to establish proper notching of the posterior maxilla and palatal vault proportions; (9) to prevent or manage pathologic fracture of the atrophic mandible; (10) to prepare the alveolar ridge by onlay grafting, corticocancellous augmentation, sinus lift, or distraction osteogenesis for subsequent implant placement; and (11) to satisfy facial esthetics, 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

![Diagram A](image1)

![Diagram B](image2)

![Diagram C](image3)

![Diagram D](image4)

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.

Prior to 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 above. 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 prior beginning bone reconstruction.2 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 anterior-posterior and transverse deficient maxilla opposing a wide mandible that is excessive in its anterior-posterior dimension.

Residual ridge form has been described and classified by Cawood and Howell3 (Figures 9-2 and 9-3) 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 work-up of the patient’s predilection for metabolic disease, including serum calcium, phosphate, albumin, alkaline phosphatase, and calcitonin levels.4 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.

**Hard and Soft Tissue Examination**

A problem-oriented physical examination should include evaluation of the maxillary-mandibular 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.

The soft tissue evaluation should involve careful visualization, palpation, and functional examination of the overlying soft tissue and associated muscle attachments (Figure 9-4). 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 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 where adequate alveolar ridge height and width is imperative for implant placement (Figure 9-5). 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. Caliberation 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.

**FIGURE 9-2**  
*Maxillary horizontal measurements (A). Classification of resorption of maxillary alveolar ridge: anterior (B) and posterior (C). Adapted from Cawood JI, Howell RA.*
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 9-6).9

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 9-7). 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 9-8).

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 esthetic and surgical considerations; bridge the gap between the model surgery and the operation; and allow cooperation between the surgeon, laboratory technician, periodontist, prosthodontist, and orthodontist, which results in a cost-effective prosthetic reconstruction with improved esthetic results (Figure 9-9). 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 proven superior in providing increased patient function, confidence, and esthetics. 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 the reconstructive surgeon can plan treatment for his or her 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. Since acceptable prosthetic reconstruction can be achieved with a variety of treatments that vary in complexity, invasiveness, time to completion, simplicity of maintenance, functional attributes, esthetic 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.

**Principles of Bone Regeneration**

There are many approaches 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

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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 esthetic 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 or the mandibular condyle. 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 peristeum. Because bone matrix is calcified, it must be vascularized to grow, regenerate, or be sustained. In
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 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 colleagues 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 9-10. 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 > 5 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.

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. This concept states that bone, itself, does not regulate the rate of bone growth. Instead, it is the functional soft tissue matrix related to bone that 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.

In 1989 Ilizarov 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. On the other hand, 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 BMP in regenerate.

Bone Grafts

Bone graft principles are discussed in Chapters 12, “Bone Grafting Strategies for Vertical Alveolar Augmentation,” 39, “Bony Reconstruction of the Jaws,” 40, “Microvascular Free Tissue Transfer,” and 43, “Reconstruction of the Alveolar Cleft.” 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. Since 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.

Cancellous grafts have the greatest concentration of osteogenic cells, and the particulate form of these grafts has the greatest cell survival owing to better diffusion and rapid revascularization. These grafts must completely undergo a two-phase mechanism of graft healing. Osteoblasts that survive transplantation proliferate and 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. 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 endostem, 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; since 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. Since 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, serologic tests be performed, and all specimens be sterilized and verified by culture prior to 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 only retain 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 γ-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.

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 colleagues 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 above 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. 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 US Food and Drug Administration (FDA) approval for spine fusion surgery in 2002. To date, US human clinical trials related to maxillofacial reconstruction include complete 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 after completion of a sinus lift dose-response study. A 20-center study of pivotal sinus lifts is near completion; its dual end points include the evaluation of bone regeneration at end point one and the evaluation of 2-year loaded implant data at end point two. To date, a time frame for submitting this data for FDA approval has not been established.
At our center 9 patients were enrolled in the pivotal study, with 21 evaluated sinus lifts sites. All study sites were confirmed before treatment by CT scan to have 5 mm or less of natural bone. Six months after graft placement, comparative CT scans were obtained from all study sites and the presence of graft and graft dimensions were assessed. All sites had enough bone for placement of implants at least 4 mm in diameter and 12 mm high. Trephine- procured biopsy specimens obtained at the time of implant placement were used to verify the presence of homogeneous vascular osteocyte-rich bone with a normal trabecular and marrow-space architecture. At our center all 21 implants have remained functionally loaded for at least 36 months. These results are preliminary and may not reflect the findings of all centers. 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. Our results to date show that this technique has 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 characteristics of bone desirable for implant placement (Figure 9-11).

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 de-emphasized. 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 alveolaroplasty 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 tissue regeneration. In cases where 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 (ie, 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. 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

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**FIGURE 9-11**  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 < 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 (×100 original magnification; hematoxylin and eosin stain). B, BMP was placed in an identical site to that shown in Figure A (×75 original magnification; 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 (×128 original magnification; hematoxylin and eosin stain).
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. 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 as it promoted giant cell rather than osteoclastic resorption. 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.

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. 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.

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. 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 curedtted 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 maturati

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 over-compression and over-reduction 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 over-reduction. For large bony defects, rotary instrument recontouring is preferred. Normal saline irrigation is used to keep bony temperatures < 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. 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-lock-stitch 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 protruberance. Vertical releasing incisions may be necessary if adequate exposure cannot be obtained since 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 protruberance is complete and visualization confirms that no irregularities or undercuts exist, exposure 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 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 elliptic fashion from edges of thecrestal incision to allow a tension-free passive closure (Figure 9-12). 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 his or her 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. 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 over-reduce the palate and expose the floor of the nose. Final contouring may be done with an egg-shaped recontouring bur (Figure 9-13). 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.

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 as 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 9-14). 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 as 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 with particular attention to hemostasis is completed. 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, 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. 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 debridement, loop electrocautery as described by Guernsey, and laser ablation with a carbon dioxide laser. 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 9-15 and 9-16). 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 anatomic frenum and assists with the excision. An elliptic 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 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 periosseum. 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 the tongue’s 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 9-17). 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 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 9-18). 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 9-19). Closure of the incision 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 postoperatively. 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 postoperatively.

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**Figure 9-17**

A, Lingual frenum attachment encroaching 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.
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. 

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.

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. A denture with a soft reline is extended to the desired depth (Figure 9-20). 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 9-21).

These procedures provide satisfactory results provided that adequate mandibular height exists preoperatively. A minimum of 15 mm is acceptable for the above 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 as well as Obwegeser and MacIntosh, both labial and lingual extension procedures can be performed to effectively lower the floor of the mouth (Figure 9-23). 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.

Today, with the incorporation of endosteal implants and the fabrication of implant-borne prostheses, lingual and buccalabial flange extensions to stabilize mandibular prostheses are not necessary. Consequently, attention is directed toward preservation or preparation of the alveolus for implants rather than extension of the fixed tissue attachments. As a result, these procedures are rarely used today.

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 esthetics 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 prior to 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), some form of augmentation is indicated. Onlay, interpositional, or 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 9-24 and 9-25). A labial 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, 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), 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. In a similar approach to that described above, 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

FIGURE 9-22 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 amount of vestibular depth extension compared with the old vestibular depth, which is marked by the previous denture flange. A, B adapted from and C–E reproduced 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 (PA): JB Lippincott Company; 1992. p. 1121.
sources of retention and stabilization of the graft and alveolus at the time of augmentation. Implant success ranges from > 90% initially and falls to 75% and 50%, respectively, at 3 and 5 years postoperatively. 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) posteriorly, resulting in an increased interarch space. Because this method involves a Le Fort I osteotomy, true skeletal discrepancies between the maxilla and 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 9-26). Although early studies entertained the simultaneous placement of dental implants at the time of augmentation, 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. A relapse of 1 to 2 mm has been demonstrated in interpositional grafts using the Le Fort I technique with rigid fixation. 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 infracture 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 9-27). 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 where the alveolus meets these requirements and therefore elect to place implants approximately 6 months later. Block and Kent 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%. 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 work-up 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 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 Chapter 57, "Maxillary Orthognathic Surgery," 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 esthetic restorations (Figure 9-28). 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 Chapter 56, "Principles of Mandibular Orthognathic Surgery" and Chapter 57, "Maxillary Orthognathic Surgery." 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). 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.\textsuperscript{53–57}

**Inferior Border Augmentation** Inferior border augmentation has been demonstrated using autogenous rib or composite cadaveric mandibles combined with autogenous cancellous bone (Figure 9-29).\textsuperscript{58–60} 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 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 sub-mandibular 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, but holes are drilled throughout the specimen to facilitate vascularization. Autogenous bone is then obtained from the ileum, morselized, and placed in the cadaver 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 < 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.61 Peterson and Slade as well as Harle described the visor osteotomy in the late 1970s (Figure 9-30).62,63 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 and then Stoelinga and Tideman described a horizontal osteotomy with interpositional grafts to augment mandibular height, with repositioning of the inferior alveolar neurovascular bundle (Figures 9-31 and 9-32).64,65 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.

**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 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 involves 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 work-up 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. Additionally, 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.

There are both intraosseous and extraosseous devices that have been designed for alveolar distraction. The Lead R System device designed by Chin 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 where the vertical dimension of occlusion is compromised. The rod is also visible anteriorly, which may be an esthetic 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 above are the DISSIS distraction implant and the Veriplant. The Lead device mentioned above 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 9-33).

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/d 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 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.

**References**

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**FIGURE 9-33** 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.
Part 2: Dentoalveolar Surgery

Osseointegration

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History of Dental Implants

Replacement of lost dentition has been traced to ancient Egyptian and South American civilizations.¹ In ancient Egyptian writings implanted animal and carved ivory teeth were the oldest examples of primitive implantology. In eighteenth and nineteenth century England and colonial America, poor individuals sold their teeth for extraction and transplantation to wealthy recipients.² The clinical outcomes of these transplanted dentitions were either ankylosis or root resorption. Continued research prolonged allotransplant survival but did not appreciably improve predictability.

In 1809 Maggiolo placed an immediate single-stage gold implant in a fresh extraction site with the coronal aspect of the fixture protruding just above the gingiva.³ Postoperative complications included severe pain and gingival inflammation. Since then various implant materials were used ranging from roughened lead roots holding a platinum post to tubes of gold and iridium.³–⁶ Adams in 1937 patented a submergible threaded cylindrical implant with a ball head screwed to the root for retention for an overdenture in a fashion similar to that done today.⁷

Due to inadequate alveolar bone height in certain sites of the jaws, subperiosteal implants were developed. In 1943 Dahl placed a metal structure on the maxillary alveolar crest with four projecting posts.⁹ Multiple variations to this initial design were fabricated but these devices often resulted in wound dehiscence. Blade implants were introduced by Linkow and by Roberts and Roberts.¹⁰,¹¹ There were numerous configurations with broad applications, and the implants became the most widely used device in implantology in the United States and abroad (Figure 10-1).

A two-staged threaded titanium root-form implant was first presented in North America by Brånemark in 1978.¹² He showed that titanium oculars, placed in the femurs of rabbits, osseointegrated in the femurs of rabbits after a period of healing. Two-staged titanium implants were first placed in patients in 1965 and studies showed prolonged survival, free-standing function, bone maintenance, and significant improvement in benefit-to-risk ratio over all previous implants.¹³ This breakthrough has revolutionized maxillofacial reconstruction. Subsequently, various implant designs have been manufactured and research in implantology has grown exponentially. The frontiers of implantology are rapidly being advanced and esthetics continue to be an integral part of this progress.

Implant Materials and Surface

Implant materials have undergone a number of different modifications and developments over the past 40 years. Commercially pure titanium has excellent biocompatibility and mechanical properties. When titanium is exposed to air, a 2 to 10 nm thick oxide layer is formed immediately on its surface.¹⁴ This layer is bioinert. However, strength issues with pure titanium have led manufacturers to use a titanium alloy to enhance strength of the implant. Most abutments are made of titanium alloy. The use of alloy significantly increases strength, which can be an issue with small-diameter and internal connections. Titanium alloy (Ti-6Al-4V) is becoming the metal of choice for endosseous dental implants.
Several attempts have been made to improve implant anchorage in bone by modifying the surface characteristics of titanium implants (Figure 10-2). In order to enhance the bone connection to the implant, a thin coating of hydroxylapatite (HA) has been plasma-sprayed onto a roughened and prepared titanium implant. HA coatings usually range from 50 to 70 µm and are applied to the implant surface with plasma-spray technology. A pressurized hydrothermal postplasma-spray increases the crystalline HA content from 77 to 96%, with an amorphous content of 4%. This coating offers an improved bone adhesion as shown in several studies.

Because of the success in orthopedics with roughened titanium surfaces for endosteal appliances, dental implant manufacturers have modified the titanium surface either by adding titanium to the surface through plasma-spray technology or by reduction procedures involving etching and blasting the surface. The titanium plasma-sprayed surface was the first rough titanium surface introduced into implant dentistry. The titanium plasma-sprayed (TPS) surface process is characterized by high-velocity molten drops of metal being sprayed onto the implant body to a thickness of 10 to 40 µm. Its original intent was to obtain a greater surface area for bone attachment. TPS implants demonstrated satisfying long-term results in fully and partially edentulous patients.

Roughened titanium surfaces can also be produced by reduction techniques such as sand- or grit-blasting, titanium oxide blasting, acid etching, or combinations of these techniques. In 2000 Cordioli and colleagues reported mean bone-to-implant contact values at 5 weeks of 72.4% for the acid-etched surface, 56.8% for TPS, 54.8% for grit-blasted, and 48.6% for machined surface implants. Reduced healing times have been documented which are believed to result in the need for less time from implantation to loading and better results in poorer-quality bone.

Despite the success with machined smooth titanium implants, the use of a roughened surface has been substituted by all manufacturers and clinicians as the current surface of choice. With rare exceptions most endosseous implants have a roughened surface texture.

Surgical Protocol Generic for All Implants

Placement without Trauma to the Soft and Hard Tissues

Heat generation during rotary cutting is one of the important factors influencing the development of osseointegration. It is widely accepted that heat increases in proportion to drill speed, and that by extension,
high-speed drilling causes physiologic damage to bone. In 1983 Eriksson and Albrektsson demonstrated the occurrence of irreversible histologic damage in the rabbit tibia when heat exposure at a temperature of 47°C was longer than 1 minute. An even greater injury occurred after heating the bone to 53°C for 1 minute, and heating to temperatures of 60°C or more resulted in permanent cessation of blood flow and obvious necrosis that showed no sign of repair over follow-up period of 100 days.

Minimal heat during implant site preparation has been recommended to achieve optimal healing conditions. Although the relationship between speed and heat generation is still under debate, the consensus has been to recommend speeds of less than 2,000 rpm with copious irrigation for preparation of implant sites. In 1986 Eriksson and Adell showed that the Brånemark drilling system had a mean maximum temperature of 30.3°C during drilling, with a maximum temperature of 33.8°C. The duration of maximum temperature never exceeded 5 seconds.

Watanabe and colleagues measured heat distribution to the surrounding bone with three different implant drill systems, in 1992. Generation of heat in the presence or absence of irrigation when drilling with spiral or spade-type drills was observed in the pig rib via thermography. The maximum temperature generated without irrigation was significantly greater than with irrigation for each drill. The heat generated continuously spread to the surrounding bone even after the bur or drill was removed from the bone, and the original temperature returned in about 60 seconds. The spiral drill required the longest time to generate heat, with gradual increase of temperature. The round bur and cannon or spade drill could finish cutting in a short time, with rapid generation of heat. Maximum temperature without irrigation was higher than with irrigation for any drill. With irrigation at proper speed, minimal heat was generated. When cortical bone was prepared using the spiral drill, irrigation decreased the maximum temperature by 10°C or more. It is recommended by all manufacturers that the bur be moved up and down while preparing the implant site, to allow accessibility of irrigation to the cutting edges of the bur, neutralizing heat generation and removing bone debris.

**Time for Integration**

Historically a nonloading healing period of machined-surfaced dental implants has been 4 to 6 months for the mandible and 6 months for the maxilla. The 4- to 6-month recommendations were made to prevent the development of a fibrous encapsulation of the implant fixtures that occurs with premature loading. These early recommendations for implant surgical protocol were developed based on clinical observations and not necessarily based on an understanding of the biologic principles of implant integration. The original Brånemark protocol has been greatly modified due to the advances in implant microtopographic surfaces and design. In recent years histologic and experimental studies have shown that specifically designed microtopographic implant surfaces can result in increased bone-to-implant contact at earlier healing times than obtained with machined-surface implants. Over the years histologic and clinical studies investigating early and immediate implant loading revealed that implants can be placed into function earlier than previously recommended. In 1998 Lazzara and colleagues evaluated the efficacy of loading Osseotite dental implants at 2 months to determine the effect of early loading on implant performance and survival. The cumulative implant survival rate was 98.5% at 12.6 months. The cumulative postloading implant survival rate was 99.8% at 10.5 months. Testori and colleagues investigated the clinical outcome of 2 months of loaded Osseotite implants placed in the posterior jaws, with a follow-up period of 3 years. The overall cumulative implant survival rate after functional loading was 97.7% in the mandible and 98.4% in the maxilla. Cooper and colleagues investigated the early loaded implants in clinical function without risking the result of osseointegration. They demonstrated a 96.2% implant survival rate with loaded unsplinted maxillary anterior single-tooth implants 3 weeks after one-stage surgical placement. The majority of the tapered threaded implants were placed in type 3 bone with a minimal length of 11 mm. The mean change in marginal bone level was 0.4 mm with a mean gain in papilla length of 0.61 mm at 12 months. In a recent report unsplinted implants placed by a single-stage procedure were successful when loaded by a mandibular overdenture prosthesis. Further developments in implant surfaces will greatly reduce integration time (Figure 10-3).

**Key Reasons for Failure**

Endosseous dental implants have been used successfully throughout the past few decades. Unfortunately implants are not always successful. Improper implant placement can result in a framework design that compromises esthetics and distribution of force on implants. Endosseous implants distribute occlusal load best in an axial direction, but if the occlusal load is in a lateral direction, many damaging stresses, including shear stresses, are generated directly at the crest of bone. Lazzara proposed that off-angle implant positioning requiring over 25° of angle correction will cause an implant to fail. Overheating bone during placement will result in a fibrous tissue against the implant surface rather than the bone. Placing implants into bone of poor quality without consideration to the mechanical forces of loading can result in early or late failure. Lack of bone contact at the time of placement is also a factor leading to lack of integration or marginal
integration. The presence of infection when placing an implant can lead to suboptimal healing and eventual lack of integration, infection within a week of placement, or lack of bone formation that results in early failure after loading.

Keratinized gingiva has been shown to promote soft tissue health around teeth. However, around dental implants, the presence of keratinized gingiva may or may not be important for preservation of crestal bone. Krekeler and colleagues suggested that there is a strong correlation of keratinized gingiva with implant failure and the absence of an adequate band of keratinized mucosa surrounding the abutment. This suggested relationship was based on the ability of the keratinized mucosa to withstand bacterial insult and ingestion, which can lead to periimplantitis.

Clinical trials with HA-coated implants indicate that the presence of keratinized gingiva is important for long-term success of endosseous implants. There was a significant relationship between implant survival and crestal bone level maintenance with posterior mandible implants in the presence of a 1 to 2 mm thick band of attached keratinized gingiva. The early Brånemark reports indicate that crestal bone levels were not affected by the presence of keratinized gingiva in the anterior mandible, although the presence of transient gingivitis was increased in patients without the protective effect of keratinized gingiva. Thus, keratinized gingiva is important for overall periimplant health. Procedures to create and preserve keratinized gingiva are recommended when placing and exposing implants. When placing a one-stage implant, incision design should result in keratinized gingiva labial to the implant.

The most important factors for implant success, identified by Block and Kent in 1990, are surgery without compromise in technique, placing implants into sound bone, avoiding thin bone or implant dehiscence at the time of implant placement, established balance restoration, and ensuring appropriate follow-up hygiene care. Implants placed into thin ridges or that had dehiscence of their surface did not uniformly gain bone attachment levels during the healing period. Labial bone implant defects should be grafted with particulate hydroxylapatite. In the posterior maxilla, vertical bone loss seems to be due to excessive cantilever-type forces placed on the implants. The use of sinus grafting is recommended to provide adequate bone support in the atrophic posterior maxilla. The presence of keratinized gingiva strongly correlated with bone maintenance in the posterior mandible. Consequently, implant surgical techniques should preserve all keratinized gingiva. Most patients who receive implants for dental restorations have lost teeth due to caries or periodontal disease. Patients need to maintain meticulous oral hygiene. If pocket probing greater than 3 mm around the implant occurs, additional antibacterial solution application or pocket elimination is recommended for hygiene purposes.

Wound Healing

Bone healing is a physiologic cascade of events in which complex regenerative processes restore original skeletal structure and function. Bone is generated by two separate mechanisms: endochondral and membranous bone formation. Endochondral bone formation occurs at the epiphyseal plates in long bones and condylar head of the mandible and accounts for growth in length. It entails the laying down of a preformed cartilaginous template, which is gradually resorbed and replaced by bone. Membranous bone formation or primary bone healing requires differentiation of mesenchymal cells into osteoblasts, which produces osteoid. The osteoid is then mineralized to form bone. This type of bone formation occurs in the calvaria, most facial bones, the clavicle, and the mandible. Osseointegration belongs to the category of primary bone healing. The word osseointegration was defined as “a direct structural and functional connection
between ordered, living bone and the surface of a load carrying implant.\textsuperscript{24}

Wound healing consists of three fundamental phases: inflammation, proliferation, and maturation. The induction of bone formation at surgical interfaces reflects a major alteration in cellular environment. These crucial events involve an inflammatory phase, a proliferative phase, and a maturation phase.

**Phase One: Inflammatory Phase**

Bone healing around implants results in a well-defined progression of tissue responses that are designed to remove tissue debris, to reestablish vascular supply and produce a new skeletal matrix. Platelet contact with implant surfaces causes liberation of intracellular granules that, when released, are involved in the early events associated with tissue injury.\textsuperscript{33} Release of adenosine diphosphate, serotonin, prostaglandins, and thromboxane A\textsubscript{2} promotes platelet aggregation, resulting in a hematic plug. Platelets continue to degranulate during the formation of the hematic plug and release constituents that increase vascular permeability (serotonin, kinins, and prostaglandins) and contribute to the inflammatory response accompanying tissue injury.\textsuperscript{33}

Acute wound healing consists of a cellular inflammatory response dominated mainly by neutrophils. Migration of the neutrophils to the site of injury generally peaks during the first 3 to 4 days following surgery.\textsuperscript{34} These cells are attracted to the local area by chemotactic stimuli and then migrate from the intravascular space to the interstitial space by diapedesis. The role of these cells is primarily phagocytosis and digestion of debris and damaged tissue. Digestion of tissue is feasible via the release of digestive enzymes such as collagenase, elastase, and cathepsin.\textsuperscript{34} By the fifth day macrophages predominate and remain until the reparative sequence is completed.\textsuperscript{32} These cells are derived from circulating monocytes that originate from the bone marrow via monoblast differentiation. Macrophages can be activated by products of activated lymphocytes and the complement system. Macrophages have the ability to ingest inflammatory debris by phagocytosis and to digest such particles by releasing hydrolytic enzymes.\textsuperscript{32}

**Phase Two: Proliferative Phase**

Microvascular ingrowth from the adjacent bony tissues during this phase is called neovascularization.\textsuperscript{35} Cellular differentiation, proliferation, and activation result in the production of an immature connective tissue matrix that is later remodeled. The local inflammatory cells (fibroblasts, osteoblasts, and progenitor cells) proliferate within the wound and begin to lay down collagen.\textsuperscript{36} This combination of collagen and a rich capillary network forms granulation tissue with a low oxygen tension. This hypoxic state, combined with certain cytokines such as basic fibroblast growth factor (bFGF) and platelet-derived growth factor, is responsible for stimulating angiogenesis. bFGF seems to activate hydrolytic enzymes, such as stromelysin, collagenase, and plasminogen, which help to dissolve the basement membranes of local blood vessels.\textsuperscript{32} Reestablishment of local microcirculation improves tissue oxygen tension and provides essential nutrients necessary for connective tissue regeneration.

Local mesenchymal cells begin to differentiate into fibroblasts, osteoblasts, and chondroblasts in response to local hypoxia and cytokines released from platelets, macrophages, and other cellular elements.\textsuperscript{32} These cells begin to lay down an extracellular matrix composed of collagen, glycosaminoglycans, glycoproteins, and glycolipids. The initial fibrous tissue and ground substance that are laid down eventually form into a fibrocartilaginous callus. The initial bone laid down is randomly arranged (woven type) bone.\textsuperscript{36} Woven bone formation clearly dominates wound healing at this point for the first 4 to 6 weeks after surgery.

**Phase Three: Maturation Phase**

After the establishment of a well-vascularized immature connective tissue, osteogenesis continues by the recruitment, proliferation, and differentiation of osteoblastic cells.\textsuperscript{32} Differentiated osteoblasts secrete a collagenous matrix and contribute to its mineralization. Osteoid-type bone within a vascularized connective tissue matrix becomes deposited at dental implant surgical interfaces.\textsuperscript{16} Eventually this matrix envelopes the osteoblastic cells and is subsequently mineralized. This cell-rich and unorganized bone is called woven bone. Loading of the dental implant stimulates the transformation of woven bone to lamellar bone.\textsuperscript{16} Lamellar bone is an organized bone displaying a haversian architecture. Bone remodeling occurs around an implant in response to loading forces transmitted through the implant to the surrounding bone. The lamellae around the implant are remodeled according to the exposed load, which with passage of time, shows a characteristic pattern of well-organized concentric lamellae with formation of osteons in the traditional manner.\textsuperscript{16}

Under normal circumstances healing of implants is usually associated with a reduction in the height of alveolar marginal bone. Approximately 0.5 to 1.5 mm of vertical bone loss occurs during the first year after implant insertion.\textsuperscript{35} The rapid initial bone loss is attributed to the generalized healing response resulting from the inevitable surgical trauma, such as peristeal elevation, removal of marginal bone, and bone damage caused by drilling.

**Options for the Edentulous Mandible**

Options for patients with an edentulous mandible include a conventional denture, a tissue-borne implant-supported prosthesis, or an implant-supported prosthesis (Figure 10-4).
Part 2: Dentoalveolar Surgery

Physical Examination of the Edentulous Patient

The depth of the vestibule and the mentalis muscle attachments are noted to determine the necessity of a vestibuloplasty. The width of keratinized gingiva on the alveolar crest and the distance from the alveolar crest to the junction of the attached and unattached mucosa are noted. Identification of the mental foramen by digital palpation is useful to determine subsequent implant location. In a relaxed vertical position of the jaws, the relationship of the anterior mandible to the maxilla is observed to determine the benefits of positioning the implants to correct or mask a Class II or Class III skeletal jaw relationship. Alveolar ridge palpation will determine the slopes of the labial and lingual cortices and the alveolar height. The location of the genial tubercles should also be noted.

Radiologic Examination of the Edentulous Patient

Radiologic evaluation of the patient prior to placing implants is focused on the determination of vertical height and the slopes of the cortices in relation to the opposite arch. A panoramic radiograph is the baseline radiograph used to evaluate the implant patient. The lateral cephalogram is useful to demonstrate the slopes of the cortices of the anterior mandible and the skeletal ridge relationships of the mandible to the maxilla, and to provide a simple and inexpensive radiographic assessment of anterior alveolar height. Additional radiographic techniques include the use of complex motion tomography or reformatted computed tomography (CT) scans. CT has a less than 0.5 mm error when reformatted cross-sectional images are examined. As clinical experience increases most surgeons agree that there is less need for these more expensive radiographic techniques for preparation of placing implants. CT scans are becoming popular in combination with models of the bone for accurate treatment planning and the fabrication of final prostheses prior to the actual surgical procedure.

Incision Design Considerations

Based on the location of the muscle attachments and the height of the mandible, the surgeon makes the decision regarding which incision to use to expose the bone and subsequently place implants into the edentulous mandible. If the attachment of the mentalis muscle is 3 mm or more labial to the location of the attached gingiva on the alveolar crest, a crestal incision can be used. If the mentalis muscle is in close proximity to the alveolar crest, resulting in mobile unattached gingiva directly against the implant abutment, a “lipswitch” vestibuloplasty is performed to inferiorly reposition the muscle attachments.

**Two Implants**

In general, when placing two implants for an overdenture, one should take into consideration the potential need for additional implants at a later time. Some patients enjoy the overdenture prosthesis but may complain of food getting caught under the denture, mobility of the prosthesis when speaking, swallowing, or chewing, and a desire to eliminate changing clips, O rings, or locator-type attachments. These patients may then desire the retention of a fixed or fixed-removable prosthesis. For these patients three additional implants may be placed to result in a total of five implants in the anterior mandible, which is sufficient to support an implant-borne prosthesis. Taking this into consideration when placing two implants into the anterior mandible, locating the implants 20 mm apart, each 10 mm from the midline of the mandible, allows for later implant placement if needed.

Implant placement at the correct height in relation to the alveolar crest is crucial. If the implant is placed such that the cover screw is superficial to the adjacent bone, a chance of incisional dehiscence or mucosal breakdown may occur. It is advantageous to countersink implants in the anterior mandible sufficiently (1 to 2 mm depending on the type of external or internal connection of the specific implant used) to allow the height of the cover screw to be in a flush relationship with the adjacent alveolar bone. The surgeon should follow the guidelines for the specific implant system being used. For one-stage implants temporary healing abutments are placed as recommended by the manufacturer. Accidental loading from poorly relined dentures can lead to trauma to the implants and eventual loss. Thus it is prudent to excessively relieve and use appropriate soft liners for the transitional denture during the healing period.

The anterior mandible may have a dense cortical plate with an abundant marrow space, or it may have very minimal marrow with an abundance of cortical bone. The smaller the mandible, the more cortical bone and less cancellous bone is available. When encountering very dense bone it is important to periodically clean the drill bits to keep the cutting surfaces clean of debris during the preparation of the implant site. For coated implants a threadformer type of bur is used to create threads in the bone. For self-tapping implants the surgeon may need to use a slightly larger bur than is customarily used in other areas of the mouth. For example, rather than using a 3.0 mm bur prior to self tapping a 3.75 mm implant, a 3.25 mm diameter drill may be necessary to allow for ease of implant insertion into very dense bone.

**Four or More Implants**

Four or more implants are placed when considering an implant-borne prosthesis. Implant-borne prostheses include hybrid screwed-retained, crown-and-bridge type, or fixed/removable with milled bars and retentive devices (see Figure 10-4). The incision design is similar for placement of four or more implants into the anterior mandible. The subperiosteal reflection should be sufficient to expose the lingual and labial cortices and the mental foramen bilaterally. After the periosteal reflection is completed, the surgeon has an excellent view of the operative site, the contours of the bone, and the location of the mental foramen. A caliper is used to mark the alveolar ridge at no less than 5 mm anterior to the mental foramen. This distance is usually the anterior extent of the nerve, as it loops forward in the bone prior to exiting the bone at the mental foramen. A small round bur is used to place a depression in the bone to locate the implant site on one side of the mandible. A similar mark is placed on the opposite side of the mandible, no less than five mm anterior to the mental foramen. The caliper is then set to 7 or 8 mm and the next implant locations are marked in a similar manner anterior to the two distal locations. If a fifth implant is to be used, then a mark is made in the midline of the mandible. By using the caliper, the implant bodies are placed a sufficient distance apart to ensure adequate space for restoration and hygiene. The use of CT-generated models of the mandible can result in surgical templates that can be secured to the jaws with pins or the implants themselves, resulting in precise implant location by preoperative planning. As the planning process matures with CT-generated applications and templates, incisions will be needed less often.

After the implant locations are identified, the first drill in the implant drilling sequence is used. If available a surgical stent is placed in order to correctly locate the implants in relation to the teeth. For Class III mandibles the implants can be angled slightly lingually, for Class II mandibles the implants can be angled slightly anteriorly, and for Class I mandibles the implants are placed vertically in relation to the inferior border of the mandible. Regardless of the angulation of the implants, the crestal location of the implants is the same, with the implants exiting the crest midcrestally without excessive labial or lingual location.

**Augmentation of the Atrophic Mandible**

If the patient is in satisfactory health for a bone graft harvest procedure, the indication for bone augmentation of the anterior mandible is a patient with less than 6 mm of bone height. Patients with greater than 6 mm of bone height can do well with implants without bone augmentation. Most clinicians will use iliac crest corticocancellous blocks to augment the height in an atrophic mandible. The procedure can be performed through either an intraoral or an extraoral incision, depending on clinician preference (Figure 10-5). The placement of implants at the time of bone graft placement is also
clinician dependent. If implants are placed at the time of bone graft placement, then the patient’s time to restoration is decreased, the graft can be secured to the mandible with threaded implants, and the shorter time to functional loading may prevent graft resorption. The disadvantages of placing implants at the time of bone graft placement include possible partial resorption of the graft and exposed portions of the implants, which is difficult to treat, malposition of the implants due to lack of proper angulation at placement, which can be technically challenging from an extraoral approach, and potential lack of integration secondary to poor graft remodeling. Technically the graft procedures are similar, with the exception of the surgical preparation of the sites for the implants.

Intraoral incisions for placement of blocks of bone can be made either crestally or within the vestibule. The crestal incision places the incision over the bone graft, but it also allows the surgeon to have the best chance to avoid incisal dehiscence secondary to vascular insufficiency. A vestibular incision places the incision away from the bone graft; however, blood supply to the edge of the vestibular incision travels through the dense fibrous tissue over the crest and thus may be prone to breakdown secondary to vascular insufficiency. Both of the intraoral incisions and their subsequent release will result in obliteration of the vestibule, which will require secondary soft tissue grafting. One should note that the mental foramen is often palpable on the alveolar crest, with some portion of the inferior alveolar nerve dehisced from the mandible secondary to resorption of the alveolar crest bone.

The bone grafts are harvested and trimmed as necessary. The goal of the graft should be to restore the mandible to approximately 15 mm of vertical height; however, for a 3 mm mandible, gaining this amount of bone may be excessive. For the extremely small 1 to 5 mm tall mandible, restoring the mandible to 10 to 13 mm is considered a great success. Two or three pieces of corticocancellous bone blocks are trimmed and placed over the superior aspect of the mandible. The edges are smoothed and the grafts are stabilized in position with screws placed through the grafts, engaging the inferior border of the mandible. If implants are placed at the time of graft placement, the clinician must weigh the possibility of partial graft resorption and subsequent implant failure. Implants can be placed 4 months after the graft was performed, and combined with a simultaneous vestibuloplasty.

The disadvantages of using an extraoral approach to graft the atrophic mandible include avoidance of intraoral incision breakdown, avoidance of an intraoral communication with the bone graft and potential infection, maintenance of the vestibular attachments, which may eliminate the need for vestibuloplasty, and ease of reflection of the inferior alveolar nerve from the alveolar crest without incising over the nerve (Figure 10-6). These advantages often are significant and offer the patient the least chance of incisal dehiscence; hence, this approach is the method of choice for these authors. From this approach bone grafts can be placed in either block or particulate form, with implants used as “tent poles” to maintain space over the graft.38

Most clinicians will allow at least 4 months to healing of the iliac crest corticocancellous bone graft prior to placing implants. Iliac crest corticocancellous


grafts heal well but start resorbing after 3 to 4 months, so the surgeon may need to place the implants at 3 months, depending on consolidation and remodeling of the bone graft, which is determined radiographically. If necessary a split-thickness dissection can be made intraorally and a palatal or split-thickness dermis or skin graft can be placed to restore some semblance of vestibule. At the time of vestibuloplasty, rigid fixation screws can be removed and implants placed, engaging the inferior border of the mandible. When simultaneously performing a vestibuloplasty with implant placement, one should countersink the implants below the level of the periosteum so that the graft can lay flush and not be tented up off the host tissue bed by the dome-like prominence of the cover screws of implants.

**Placement of Implants into Atrophic Mandibles without Grafting**

The majority of patients with atrophic mandible with less than 10 mm of bone height and at least 5 to 6 mm of height are not good candidates for bone grafting secondary to health-related issues. For these patients four implants can be placed, with 1 to 2 mm of the implant through the inferior border of the mandible, and 1 to 2 mm supracrestal as necessary. It is important to gently prepare the bone with new sharp drills and pretap these bones since they can be brittle and have minimal blood supply. The implants should be placed to avoid labial protrusion (see Figure 10-6).³⁷

**Options for the Edentulous Maxilla**

Treatment planning for the edentulous maxilla is usually initiated at the restorative dentist’s office. This includes establishment of the patient’s goals of what he/she desires at the completion of implant therapy. Once these goals are established the surgeon is seen and an assessment of bone availability is performed.

A panoramic radiograph and a physical examination are often all that are required to delineate satisfactory bone bulk for the placement of implants into the maxilla. From the panoramic radiograph one can estimate the amount of vertical bone available throughout the entire maxilla. Occasionally a reformatted CT scan is obtained to confirm the presence of bone prior to implant placement. If cross-sectional radiography is planned, using a radiopaque stent at the time of the radiography significantly increases the amount of information gathered. The teeth in the patient’s prosthesis are made radiopaque by using a radiopaque material, typically 20 to 30% barium sulfate combined with clear acrylic so that the teeth are included in the cross-sectional image. This provides information concerning the relationship of the bone to the desired teeth.

Parel’s classification of the edentulous maxilla is useful for conceptualization of the prosthetic plan (personal communication, 1991). The Class I maxilla involves the patient who seems to be missing only the maxillary teeth, but has retained the alveolar bone almost to its original level (Figure 10-7). The Class II maxilla has lost the teeth and some of the alveolar bone, and the Class III maxilla has lost the teeth and most of the alveolar bone to the basal level.

For the Class I patient a fixed restoration, borne by implants, can be fabricated because the patient has adequate alveolar bone for support of the soft tissues and is missing only the teeth. There is usually greater than 10 mm of bone height in both the anterior and posterior maxilla. For a fixed crown-and-bridge restoration, implants need to be placed within the confines of the teeth of the planned restoration. The implants should be
placed to avoid the embrasure regions in order to promote esthetics and oral hygiene. For a fixed crown-and-bridge restoration, the implants should be placed 3 mm apical to the gingival margin of the planned restoration in order to allow the restorative dentist to develop a natural emergence of the crowns from the gingiva. If the Class I patient desires a tissue-borne overdenture on four implants because of financial constraints, then the design of the overdenture bar must be such as to avoid excessive space-occupying designs, since the patient is missing only their teeth, not the alveolus.

The Class II patients rarely can be esthetically managed with a fixed crown-and-bridge prosthesis since they require the labial flange of the maxillary prosthesis to support the nasal-labial soft tissues. In order to distinguish the need for acrylic to support the soft tissues, it is useful to duplicate their maxillary dentures and remove the labial flange, leaving only the teeth. The resultant soft tissue profile with the modified duplicated maxillary denture will easily help the implant team and patient decide on a treatment plan. If the patients look good without the flange of their denture, indicating sufficient nasal-labial support, a fixed crown-and-bridge restoration can be fabricated using pink porcelain or acrylic to decrease apical gaps from lost alveolar bone. In addition the deficiency of alveolar bone necessitates placing the implants more apical than is ideal, resulting in excessively long teeth, teeth with pink acrylic, a removable lip “plumper,” or a hybrid-type prosthesis with space between the prosthesis and the implants.

A fixed crown-and-bridge, fixed/removable (spark erosion or milled prosthesis), or removable overdenture-type prosthesis may be prescribed. The implant-borne fixed and fixed-removable prostheses require at least six, or preferably eight, endosseous implants to adequately support a maxillary implant-borne prosthesis. The exception is the use of the Zygomaticus implant fixtures. These prostheses require posterior maxillary vertical height of bone for implants placed in the first molar region. The removable prosthesis requires two to four implants placed into the anterior maxilla to support a bar that has retentive vertical stress-breaking attachments. Edentulous maxillary prostheses are usually fabricated with cross-arch stabilization of the left and right implants. Cross-arch stabilization significantly increases implant survival long term.

**Placement of Eight Implants into the Anterior Maxilla**

For the patient with adequate anterior vertical bone height, and for whom a treatment plan has been made for anterior implants for overdenture support, four implants can be placed. It is recommended to place at least four implants for a tissue-supported overdenture in the maxilla. Four implants in the anterior maxilla are used to support a rigid bar, often combined with vertical stress-broken attachments placed at the distal aspects. Implants for overdentures are typically placed with their centers slightly palatal to the crest to avoid dehiscence and thin bone over the facial aspect of the implants. The incisive canal should be avoided as a site for implant placement. Specifically, implants for overdentures are place in the canine and premolar locations, dependent on the availability of bone. An implant can be placed in the lateral incisor position if necessary. However, implants placed in the central incisor locations complicate the prosthetic rehabilitation since the presence of the abutments and a bar near the midline may result in excessive palatal bulk in the denture, which may be bothersome to the patient.

**Placement of Eight Implants without a Graft**

If the goals of the patients are to have a denture or prosthesis that will enable them to have a palateless prosthesis and allow them to chew all textured foods without the prosthesis depending on the tissues for support, then a sufficient number of implants is required to resist the forces of mastication. For these patients it is recommended to use six to eight implants for an implant-supported fixed or fixed/removable prosthesis, with an adequate number of implants located posteriorly to support the molars.

Eight implants in the anterior and posterior maxilla are used to support a suprastructure for a totally implant-borne restoration with tissue contact only for speech. If a bar-type structure is planned, the implants should be placed within the confines of the borders of the planned prosthesis, and not labial or outside the borders of the teeth. The implants should be placed to avoid impingement of the teeth in the overdenture and to allow space for the fabrication of the bar. For many of these implant-borne cases, implants are placed from the canine region extending posteriorly, with a minimal number of implants placed into the incisal region. This pattern of placement makes the design of the anterior portion of the prosthesis easier.

The implants for fixed/removable overdentures are typically placed with their centers slightly palatal to the crest in order to avoid dehiscence and thin bone over the facial aspect of the implants. The implants can be positioned from second molar to central incisor; however, most restorative dentists prefer to avoid the central incisor and second molar sites. The second molar site can be used in select cases, but it does make the placement of screws, abutments, and transfer copings difficult. In addition the bars may need the space of the second molar site for attachments, depending on the prosthesis design of the retentive bar.

**Placement of Eight Implants with Sinus Grafts**

Patients who have received a treatment plan or an implant-borne restoration but who
have insufficient vertical bone for the placement of implants in the maxilla posterior to the canines are considered for a combination of sinus grafting and implant placement. The sinus grafts can be performed as one surgery, followed 6 to 12 months later with implant placement, or the sinus graft can be performed and the implants placed at the time of the sinus graft. If the sinus graft is performed prior to implant placement, the surgeon should verify that bone has formed within the graft.

We and our colleagues perform sinus grafting with immediate placement of implants. Currently, the recommended sinus graft material is autogenous bone, harvested from the jaws, tibia, or iliac crest. If necessary the autogenous bone volume can be augmented with demineralized bone in a ratio not to exceed 1:1. Hydroxylapatite-coated implants are used for immediate placement into sinus grafts.

**Single- and Multiple-Unit Restorations**

There are different surgical concerns when placing single- or multunit restorations in the anterior maxilla or other areas where esthetics are less of a concern. Placement of implants into premolar and molar locations can usually be performed with less concerns of papilla and root eminence morphology (Figure 10-8).

**Premolar or Molar Restorations**

Diagnosis and treatment planning will indicate whether there is sufficient space and bone available for implant placement. Periapical radiographs are necessary for single-tooth restorations to confirm that the roots of the adjacent teeth do not impinge in the space that will be used by the implant. If root angulation is a problem, then preoperative orthodontics will need to be performed prior to implant placement, or a fixed bridge can be made rather than placement of an implant.

Careful attention should be directed to the final restorations and the mechanical loading that the restoration and hence implants will feel. Canine guidance or group function is usually present and can affect the position of the implants. Canine discursion is recommended when placing posterior implants for fixed restorations. The ideal single premolar or molar restoration has a balanced occlusion that will result in traumatic forces upon the implant. Single-tooth implants should be placed such that the implant is under the working cusp of the tooth, to avoid excessive cantilever forces. Maximal length implants should be used whenever possible. Short implants in the posterior jaws tend to have less long-term survival than longer implants. The crown-to-root ratio needs to be addressed. Complete treatment planning, which includes knowledge of the final restoration, will increase success and limit complications.

The surgical incision is made slightly palatal to the crest, with vertical releasing incisions flaring into the vestibule in order to keep the base of the flap wider than the crestal incision width. Full-thickness subperiosteal labial and palatal flaps are reflected to expose the crest and to provide visualization of the vertical cortices of bone. The implant should be placed with its axis parallel to the occlusal forces, with the emergence of the implant angling to meet the buccal cusps of the mandibular teeth.

**Multiple Implant–Borne Restorations for the Posterior Maxilla**

Since these restorations commonly involve the distal teeth, assessment of the availability of bone in relation to the sinus is critical. If 10 mm of bone is not available, then a sinus augmentation is indicated. If two long implants can be

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**FIGURE 10-8**

A, This patient required a single implant for replacement of a premolar in the maxilla. A tissue punch was used to access the crestal bone. The implant site was prepared and the implant placed through this circular soft tissue hole. This implant has an internal connection. B, A fixed abutment was placed immediately into the implant and prepared. A provisional crown, not in occlusion, was fabricated. C, This is the final crown. Note the excellent soft tissue reaction to the crown, abutment, implant complex.
placed without the need for a sinus graft, along with sinus elevation of a third site by the use of osteotomes, then 8 mm of bone for the third implant is acceptable. However, the use of osteotomes to elevate the sinus floor by 2 mm is not a procedure that has abundant scientific validation. Therefore the patient must be apprised of the risks and potential failure. When in doubt a sinus elevation is performed. The mechanics of the final restoration need to be taken into consideration when placing multiple implants for a full quadrant restoration.

There are patients who have sufficient vertical bone but are deficient in the width projection of the bone. After maxillary teeth are extracted for a variety of reasons, facial bone resorption can occur, leaving the palatal bone intact, with the alveolus thin and deficient. Placing the implant in the ideal position may result in facial bone dehiscence. For the thin ridge in the posterior maxilla, with sufficient bone height, several surgical options are available. These include the use of particulate bone grafting with membrane coverage, the use of onlay bone grafts harvested from the symphysis or ramus, and ridge expansion using osteotomes or osteotomies.

**Restorative Options for Single-Unit Restorations in the Anterior Maxilla**

Esthetic implant restorations represent a challenge to reproduce normal-appearing restorations with normal-appearing soft tissue profile and integrity. Most implant sites that require esthetics have deficiencies in the ideal bone and overlying soft tissue, and must be enhanced with a variety of surgical techniques. A tooth may be missing because of lack of tooth development, caries, external or internal resorption of teeth following trauma, root canal complications, bone loss from periodontal disease, or recent dentoalveolar trauma. Each of these etiologies has secondary effects on the proposed implant site. It is common to find a deficiency in labial bone with loss of the previous root eminence form of the ridge. In addition, the overlying soft tissue at the level of the alveolar crest may be thin, resulting in a lack of stippling, variations in gingival color, and increased translucency resulting in parts of the implant and abutment showing through the gingiva.

The majority of anterior maxillary single-tooth sites present with inadequate bone and soft tissue, requiring both bone and soft tissue augmentation. The height of the papilla reflects the underlying crestal bone height on the adjacent teeth. Careful assessment of the bone levels on the adjacent teeth enables the surgeon and restorative dentist to inform patients of the realistic expectations of retaining or creating papilla for an esthetic single-tooth restoration.

The presurgical assessment, using the aesthetic tooth wax-up, results in the ability of the surgeon to estimate the height and width of a bone graft, if one is indicated. For severe bone deficiency, which prevents implant stabilization, a bone graft should be placed at least 4 months prior to implant placement, allowing future implant placement in the ideal location horizontally and vertically. When the deficit of the bone is such that the implant can be placed and is mechanically stable, with a portion of its surface exposed through the bone, then a hard tissue particulate graft is placed at the same time as the placement of the implant. The material used for grafting depends on the extent of the implant bone fenestration. Autogenous bone is used for larger fenestrations, with a gradual increase in hydroxyapatite used as the implant bone dehiscence decreases in size.

**Incision Considerations for Esthetic Sites**

When placing an implant in the central incisor location, careful attention to the detail of gaining access to the underlying bone is critical for obtaining a perfect result, without ablation of the papilla or vertical scars from poor incision design and technique. If there is 5 mm from the contact point of the teeth to the crestal bone of the adjacent tooth, then the use of sulcular incisions is indicated. If there are papillae present but the teeth are long, with an excess of 5 mm between the contact point to the crestal bone of the adjacent tooth, then the patient needs to be warned that papillae may not be present after implant placement. When necessary, vertical incisions should be beveled to allow for esthetic scar healing. When the bone anatomy permits, the use of a tissue punch and avoidance of incisions will allow for no scars and no loss of papilla.

Angulation of the implant should result in the axis of the implant being oriented to emerge slightly palatal to the incisive edge of the planned restoration. If placed at or anterior to the incisive edge of the tooth, there may be difficulty in developing the emergence profile of the restoration. If the implant is placed too far labial, with the anterior edge of the implant at the edge of the gingival margin of the planned tooth, then with addition of the abutment and porcelain, the gingival contour will be excessive and gingival recession results. As the platform (ie, diameter of the implant) increases, the clinician must be cautious to ensure that the labial edge of the implant is not excessively labial, or emergence of the crown will be compromised and will result in an obese crown form. Most restorations require more than 1 mm of clearance from the labial surface of the implant to the eventual clinical crown, secondary to development of the emergence profile of the restoration from the subgingival portion of the implant restoration.

The depth of the implant in relation to the planned gingival margin is also critical. If the implant is placed too shallow, with 2 mm or less from the top of the implant to the gingival margin, then several adverse
events can occur. The metal from the implant may be visible through the gingival margin. Because the distance from the top of the implant to the gingival margin is minimal, metal showing through the gingiva is difficult to camouflage. A minimal distance between the gingival margin and the top of the implant may also result in difficulty in adjusting the margins of the abutment, with porcelain extending to the implant itself. It is then difficult to develop a natural appearance since the gingival margin region of the restoration is excessively bulked or round in shape. The use of ceramic abutments may help in these adverse situations. However, proper implant placement is a simple means to avoid these problems.

Immediate Loading and One-Stage Protocol

The evolution of implant-related therapies in the modern era was based on the work of Brånemark and colleagues, who scientifically validated the process of placing an implant into bone, waiting a period of time for bone to heal to the implant, followed by long-term functional loading. During the 1970s and early 1980s a one-stage threaded titanium plasma-coated implant was used for overdenture retention with immediate loading. The “Swiss screw” was placed into the anterior mandible and had excellent long-term success. Other one-stage implant systems were slow to develop, but as they have emerged with data to support a one-stage process (ie, with no need for exposure surgery), the concept of a one-stage endosseous implant therapy has gained credibility. The Strauman system has long-term data indicating that a one-stage unloaded implant system can work in all areas of the mouth, in distinction to the Swiss screw and the Brånemark protocols. Recently, more interest has arisen for placement of implants into the esthetic zone of the maxilla, with either immediate loading or the use of a healing abutment that mimics the natural shape of the tooth. The hypothesis is that by placing a healing abutment with natural contours, the soft tissue response will be enhanced, potentially resulting in a more esthetic final restoration.

Treatment planning for a one-stage or immediately temporized anterior maxillary restoration begins with a list of contraindications. If a tooth is present and needs to be extracted, a one-stage exposed implant placement at the time of extraction will require the following:

- No purulent drainage or exudate from the site
- Excellent gingival tissue quality without excessive granulation tissue
- Lack of periapical, uncontrolled radiolucency
- Adequate bone levels circumferentially without the need for additional soft or hard tissue grafting

The clinician has several options (Table 10-1). At the time of tooth extraction, if there are any of the contraindications present as described above, either a graft can be placed into the extraction socket, or no graft is placed. The decision to avoid a graft is based on the thickness of the labial bone and the prior healing patterns of the patient, if known. However, in our institution, an anterior extraction site without a socket graft is more prone to labial bone resorption and hence less-than-ideal bone is available at the time of implant placement. If a graft is placed into the socket, then after 3 to 6 months, depending on the material placed, the implant can usually be placed in an ideal location.

If there is ideal bone and soft tissue present at the time of extraction, an implant can be placed at the time of extraction. The clinician should decide prior to extraction if a provisional restoration is to be placed at the time of implant placement, or if the implant is to have a healing abutment placed for a one-stage protocol, or submerged for a two-stage protocol.

Preoperative planning for immediate temporization after implant placement involves fabrication of a surgical guide that precisely locates the implant in one position. The surgeon must work closely with the restorative dentist to ensure that the planned placement of the implant will indeed be able to be performed. The restorative dentist should be available during surgery to guide the surgical placement and be able to adapt the temporary restoration after implant placement.

After the implant is placed and the orientation approved by the restorative dentist, the abutment is placed, and removed as necessary so that changes in its height and contours can be accomplished outside

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Adjunctive Treatments</th>
<th>Advantage</th>
<th>Disadvantage</th>
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</thead>
<tbody>
<tr>
<td>Extract tooth</td>
<td>No graft; wait 8 wk</td>
<td>Short time to implant placement</td>
<td>Labial bone loss and need for adjunctive tissue grafts</td>
</tr>
<tr>
<td>Extract tooth</td>
<td>Immediate placement of implant</td>
<td>Less time for overall treatment</td>
<td>Increased chance for infection; may not have ideal bone support upon placement</td>
</tr>
<tr>
<td>Extract tooth</td>
<td>Graft extraction site; wait 4 mo for implant placement</td>
<td>Provides ideal placement site</td>
<td>Extended time for treatment</td>
</tr>
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</table>
of the mouth. The abutment and temporary crown may be prepared on a model prior to surgery in selected cases. The abutment is placed and tightened to the implant and the temporary crown completed. The occlusion should be relieved to avoid loading the implant during the healing period. In some patients who may be prone to loading the implant because of athletics, weight lifting, or their occlusion, an anatomic healing abutment or a custom healing abutment can be placed in order to preserve the morphology of the gingiva, without the presence of a tooth form.

Procedures performed during the integration or healing period are delayed until implant integration has occurred, in order to avoid disturbance of this critical aspect of implant success. Approximately 2 months after the implants have been placed, the patients are seen by the restorative dentist and surgeon to decide, based on the esthetic set-up, whether the implant site requires additional augmentation of the ridge. The goal is to achieve a convex ridge profile and develop the site's shape to allow for the restoration to emerge from the gingiva, similar to a natural tooth. Our experience indicates that 70% of the implant sites that required hard tissue grafts also benefited from subepithelial connective tissue grafts placed 3½ months after implant placement.

Summary
The successful restoration of the patient with dental implants can result in a change in dental function and health, with a happy patient. The basis for the use of dental implants is initiated by the normal sequence of wound healing, the translation of surface engineering to implant design, and evidence-based trials that verify and confirm efficacy of treatment methods.

References


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 periimplant 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 esthetic 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 esthetic 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 prior to 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 periimplant 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 11-1). 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 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 11-1). 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 re-adaptation 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. Additionally, the buccal bevel maximizes the amount of attached tissue reflected with the buccal flap (see Figure 11-1).

As suggested above, 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 esthetic case types (Figures 11-2–11-6).

Surgical Maneuvers for Management of Periimplant 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. There are three distinct soft tissue surgical maneuvers that 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 periimplant 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

![Figure 11-1](image-url)  
**Figure 11-1** 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.

![Figure 11-2](image-url)  
**Figure 11-2** 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.
The width of the gingival tissues 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 anterior-most 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 11-7). The contoured flap is then repositioned apically and secured around the abutments with a suture passing through each interimplant area, and 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, this maneuver facilitates primary closure and circumferential adaptation around the transmucosal implant structures while preserving an adequate band of attached tissue around the 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 11-8). 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...
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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 as 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 11-9).3 This maneuver is especially suited for completely edentulous or posterior partially edentulous implant case types, where 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 Class 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 esthetic 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 esthetic 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:

- 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
• Pre-existing 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 < 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 esthetic importance of the site. In a nonesthetic area the surgeon can use the various surgical maneuvers described above to obtain primary closure and then reevaluate the need for soft tissue grafting based on the health and volume of periimplant attached tissues obtained after initial healing. In contrast, when the total width of attached tissue present is < 3 mm in an esthetic area, soft tissue augmentation is indicated prior to 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 esthetic areas, small-volume soft tissue esthetic ridge defects can be corrected simultaneously with submerged or nonsubmerged implant placement with subepithelial connective tissue grafting, whereas large-volume soft tissue esthetic ridge defects are most predictably reconstructed prior to 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 (eg, 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 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 above. 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 prior to 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. 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 below, 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. When gingival grafting is performed after implant abutment connection or delivery of the final restoration, a horizontal incision is made through the inter-implant 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 11-10A). 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
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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 11-10B. Subsequently, the mucosal flaps are excised and residual elastic or muscular tissue are 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 11-10C, 11-11, and 11-12). The following graft immobilization pressure is 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 prior to implant placement in the severely atrophic maxilla or mandible that is < 10 mm in height and has < 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 11-13–11-15). 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 (ie, open approach). Alternatively, the graft can be secured in a split-thickness pouch prepared at the recipient site (ie, 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 esthetic blending at the recipient site. The subepithelial connective tissue graft can be used during initial implant-site development prior to implant placement or simultaneous with submerged implant placement for the correction of small-volume soft tissue esthetic 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 esthetic ridge defect is present, a series of connective tissue grafts is usually required for reconstruction of these esthetic ridge defects prior to 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 11-16A). 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 then made approximately 2 mm apical to the first incision to complete an ellipse (Figure 11-16B). 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 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 subepithelial planes of dissection. This approach begins with a full-thickness curvilinear incision, as described above. 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 11-17), thereby assisting the surgeon to establish the appropriate subepithelial plane of dissection. The remainder of the surgical procedure is identical to the procedure described above for the dual-incision technique.

The advantage of the dual-incision approach is that it is easier to perform. Since 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 esthetic 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 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 < 4 mm apically 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 11-18). 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
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. 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 clamped suture material 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 11-18) 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 11-19). 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 de-epithelialized with a fresh no. 15C scalpel. This further extends the wound margin, thereby reducing flap retraction and greatly enhancing incision line esthetics. 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.

Next, the cover flap is secured coronally with interrupted sutures passing through the papillae. These sutures should pass through the facial flap and the de-epithelialized 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, it 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 above. 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 vascularized interpositional periosteal connective tissue flap (VIP-CT) flap is an innovative technique that provides for reconstruction of large-volume soft tissue esthetic ridge defects with a single procedure. 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 esthetic 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, a reduction in 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 where previous hard and soft tissue site-development techniques have fallen short. This technique has also proven 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 11-20). It is a predictable means of resubmerging an implant in the anterior area when an unexpected soft tissue dehiscence compromises the final esthetic 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 11-21 and 11-22), as previously discussed.

Of greatest significance, this technique provides the implant surgeon with a proven technique for predictable simultaneous hard and soft tissue esthetic implant-site development at compromised anterior sites with large-volume combination esthetic ridge defects (Figure 11-23). 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

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**FIGURE 11-20** 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 following 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. Reproduced with permission from Sclar A.
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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 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 11-24A).

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 11-24A). 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 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 neurovascular 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 (Figure 11-24B). 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 greatest 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 (Figure 11-24C). 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 above 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 11-25). When used for root or abutment coverage, the basement membrane side of the graft should be oriented toward the exposed root or abutment (Figure 11-26). 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 apico-coronal dimension) and immobilizing a larger AlloDerm graft compared to 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. In these instances the AlloDerm graft is first rehydrated in non-activated anticoagulated PRP solution prior to its immobilization at the recipient site. Subsequently, 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.

Conclusion

This chapter provides the implant surgeon with the basic information necessary for successful management of periimplant 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, as 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.

References

Strategies to increase alveolar vertical dimension fall into six general categories: (1) guided bone graft augmentation, (2) onlay block grafting, (3) interposition alveolar bone graft, (4) alveolar distraction osteogenesis, (5) iliac corticocancellous augmentation bone graft, and (6) the sinus bone graft.

The difficulty in gaining and maintaining alveolar vertical augmentation is well established in the literature, but the various procedures that have been used have been complicated by relapse and resorption.1–3 Augmentations without the placement of implants generally resorb unless a nonresorbable grafting material such as hydroxylapatite is used.4–6

This chapter reviews the indications and contraindications for the above procedures, all of which have found their niche in oral and maxillofacial surgery reconstruction using osseointegrated implants.

Alveolar vertical defects have been classified according to the size of the defect.7 Deficiencies can range from 1 or 2 mm to more than 20 mm in height. In general monocortical grafts or guided bone graft augmentations are useful for smaller augmentations. Interpositional grafts work well for moderate-sized defects, whereas distraction osteogenesis is reserved for more extensive alveolar defects. Large bone mass deficiencies, where there is not enough bone to distract, require iliac bone graft reconstruction, though a vertical gain of 10 mm is difficult to achieve in these settings. Finally, there is the sinus bone graft, which functions as an “endosteal” expansion of alveolar vertical bone mass.

Guided Bone Graft Augmentation

Vertical bone augmentation of deficient alveolar ridges can be obtained with guided bone regeneration techniques. These techniques allow vertical augmentation of up to 10 mm both in the posterior and anterior maxilla and mandible. A barrier membrane is placed and stabilized with tacks or screws in order to protect an autogenous bone graft usually harvested from the retromolar area in the mandible. The membrane is maintained in the site completely covered by the soft tissues for a period of at least 6 months.

The implants can be placed either at the time of bone regeneration or at the membrane removal surgery. Figure 12-1 illustrates a posterior mandible atrophy in which 7 mm of vertical bone height is required. After full thickness flap elevation, a couple of 10 mm long tenting screws have been placed in order to avoid the membrane collapse toward the bone ridge. The cortical bone has been perforated with a round bur (see Figure 12-1A). Autogenous bone chips have been placed and covered with a titanium-reinforced expanded polytetrafluoroethylene (ePTFE) membrane (see Figure 12-1B). After 6 months of uneventful healing, a mucoperiosteal flap has been elevated (see Figure 12-1C), and the membrane has been removed to expose the regenerated bone (see Figure 12-1D). Two Bränemark implants have been placed (see Figure 12-1E). Figure 12-1F and 12-1G show the final porcelain-fused-to-metal prosthesis and the periapical x-ray after 3 years of occlusal loading.

Mandibular Block Autografts for Localized Vertical Ridge Augmentation

Mandibular block autografts have been used extensively for alveolar ridge augmentation with great success and include
Part 2: Dentoalveolar Surgery

The vertically deficient ridge presents the greatest challenge for reconstruction, and success with these grafts can be achieved with defects of up to 6 mm. The posterior maxilla and mandible are the most common areas of the mouth where this type of deficiency occurs. This section focuses on posterior maxillary and mandibular reconstruction in a staged manner prior to implant placement. Implants are placed in a submerged or nonsubmerged mode after appropriate healing time with the block grafts.

Typically, there is loss of alveolar bone height in the posterior maxilla and mandible secondary to periodontal disease and after tooth removal. Tooth loss results in buccal plate compromise and a reduction in alveolar width. This bone resorption process continues in a medial direction until a knife-edged ridge forms. This may then result in a deficiency of alveolar height that would preclude implant placement. The cortical plate may be minimal or absent, further complicating implant placement. Finally, occlusal forces are greater in the posterior than in the anterior area of the mouth, necessitating appropriate surgical and prosthetic treatment planning for long-term implant success.

Treatment planning in these areas must include solutions to reduce stress. A primary plan includes increasing the number of implants. No pontics are used, so one implant per buccal root is the treatment planned for each case. In addition, no cantilevers are allowed. Splinting of all crowns is also indicated for biomechanical force distribution. Occlusal considerations include eliminating lateral interferences during any excursive movements. The final factors involved in decreasing undesirable stress to the implants are interrelated. They include increasing the bone density and maximizing the diameter of implants. These two goals are accomplished with mandibular block grafts. The quality of bone from the ramus buccal shelf is typically type 1, and the symphysis normally exhibits type 2 and occasionally type 1 quality bone. These grafts create areas for the use of larger diameter implants that increase the surface area over which the stresses of occlusal forces are distributed.17,18

There are four key principles that should be followed for mandibular block graft success. First, recipient site preparation must be done to allow access for trabecular bone blood vessels and osteogenic cells, which is critical for predictable bone incorporation. Also, platelet release from

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**Figure 12-1**

A, An edentulous posterior mandible is flapped open, and perforations are made through the cortex in preparation for the bone graft. “Tent pole” bone screws are placed at the desired height, up to 10 mm. B, Reinforced membrane is tacked into place. C, Six months later, the membrane is exposed. D, Bone formation after membrane removal. E, Placement of two dental implants. F, Final restoration. G, Periapical x-ray after 3 years of loading.
damaged blood vessels produces platelet-derived growth factor and transforming growth factor (TGF-β), which accelerate wound healing. Site preparation facilitates intimate adaptation of the graft to its underlying bony bed. Second, two-point fixation of each block is important to prevent microtortion of the graft resulting in incomplete bone incorporation. Low-profile self-tapping screws are recommended. Third, primary closure without tension of the wound site is critical to prevent dehiscence, which is the primary complication of monocortical block grafts. Careful attention to undermining the flap will allow for complete relaxation prior to closure. Prosthesis contact with the ridge is not allowed for the entire duration of healing. Finally, implant placement must follow graft incorporation and should never be done simultaneously. This staging provides predictable bone volume and optimal bone density to be created prior to stage 1 surgery.

The symphysis can provide a range of dense cortical cancellous bone ranging from 4 to 11 mm, in contrast to a typical ramus buccal shelf block graft that is 3 to 4 mm. These grafts can be used for predictable horizontal augmentation of 5 to 7 mm and vertical augmentation of up to and including 6 mm.

**Symphysis Block Graft Harvest**

A sulcular incision design is preferred for the symphysis block graft harvest as opposed to the more conventional vestibular design. This approach can be safely used if the periodontium is healthy and no crowns are present in the anterior dentition. Also, a highly scalloped thin gingival biotype is contraindicated.

The incision begins in the sulcus from second bicuspid to second bicuspid. An oblique releasing incision is made at the mesial buccal line angle of these teeth and continues into the depth of the buccal vestibule. A full thickness mucoperiosteal flap is reflected to the inferior border of the mandible. This allows for good visualization of the entire symphysis, including both mental neurovascular bundles. It also provides easy retraction at the inferior border and results in a relatively dry field. Contrast this with the vestibular approach, which results in more limited access, incomplete visualization of the mental neurovascular bundles, and more difficulty in superior and inferior retraction of the flap margins. Also, there is typically bleeding secondary to the mentalis muscle incision resulting in the need for hemostasis. Finally, wound dehiscence from the sulcular approach is rare. The vestibular incision can result in wound dehiscence and scar band formation.

A 702L tapered fissure bur in a straight handpiece is used to penetrate the symphysis cortex via a series of holes that outline the graft. It is important to not encroach within 5 mm of the apices of the incisor and canine teeth as well as the mental neurovascular bundles. Also, the inferior osteotomy is made no closer than 4 mm from the inferior border. All holes are then connected to a depth of at least the full extent of the bur flutes (7 mm). The graft is then harvested using straight and curved osteotomes or modified bone spreaders. The donor site is packed with gauze soaked in either saline or platelet-poor plasma. Closure of the site is done after graft fixation and includes a particulate graft. This graft is not critical to the esthetic outcome; however, grafting of the donor site to allow for a secondary block harvest can be done.

**Ramus Buccal Shelf Block Graft Harvest**

A full thickness mucoperiosteal incision is made distal to the most posterior tooth in the mandible and continues to the retro-molar pad and ascending ramus. An oblique release incision can be made into the buccinator muscle at the posterior extent of this incision. The incision continues in the buccal sulcus opposite the first bicuspid where an oblique release is made to the depth of the vestibule. A full thickness mucoperiosteal flap is then reflected to the inferior border allowing for visualization of the external oblique ridge, buccal shelf, lateral ramus and body, and mental neurovascular bundle. The flap is further elevated superiorly from the ascending ramus and includes stripping of the temporalis muscle attachment.

There are three complete osteotomies and one bone groove that need to be prepared prior to graft harvest. A superior osteotomy is created with a 702L fissure bur in a straight handpiece. It begins opposite the mandibular second molar and continues posteriorly to the ascending ramus approximately 4 to 5 mm medial to the external oblique ridge. The length of this osteotomy depends on the graft size. The anterior extent of this bone cut can approach the distal aspect of the first molar, depending on the anterior location of the buccal shelf. A modified channel retractor is used for ideal access to the lateral ramus body area to allow for two vertical bone cuts. The osteotomies begin at each end of the superior bone cut and continue inferiorly approximately 12 mm. All osteotomies just barely penetrate cortical bone. Finally, a no. 8 round bur is used to create a groove connecting the inferior aspect of each vertical osteotomy. The graft is then harvested using modified bone spreaders that are malletted along the superior osteotomy. The graft will fracture along the inferior groove and should be carefully harvested as to avoid injury to the inferior alveolar neurovascular bundle. The sharp ledge that is created at the superior extent of the ascending ramus is then smoothed with a large round fissure bur. Gauze moistened with either saline or platelet-poor plasma is then packed into the wound site. Closure of the donor site can be done after graft fixation.
**Case 1**

A healthy 59-year-old white female was referred for implant evaluation. Clinical and radiographic examination revealed a missing right maxillary second bicuspid and all molars (Figure 12-2A). The edentulous space exhibited a deficiency in alveolar height of approximately 4 mm, along with minimal sinus pneumatization precluding the need for sinus grafting (Figure 12-2B and C). The treatment plan included vertical bone augmentation using a right ramus buccal shelf block graft prior to implant placement for a three-unit fixed bridge.

The recipient site was exposed via a full thickness buccal flap reflection (Figure 12-2D). Site preparation included slight decortication and perforation prior to block grafting (Figure 12-2E). A right ramus buccal shelf graft was harvested in the conventional manner (Figure 12-2F–H) and contoured to size (Figure 12-2I and 12-2J). Platelet-rich plasma was then placed on the recipient site prior to block graft fixation (Figure 12-2K and L). Particulate demineralized freeze-dried bone allograft was mortised superior to the graft (Figure 12-2M), and additional platelet-rich plasma was placed over the graft complex (Figure 12-2N). Primary closure without tension was accomplished prior to particulate grafting and administration of platelet-rich plasma. A posterior vertical release incision was also made to allow for advancement of the full thickness flap (Figure 12-2O and P). Five months later the site was reentered revealing excellent block incorporation (Figure 12-2Q). Implants were placed in a nonsubmerged mode because of the excellent type 1 quality bone (Figure 12-2R and S).
Case 2

A healthy 62-year-old white female was referred for implant evaluation. This patient was unhappy with her existing bilateral distal extension partial denture and desired fixed prosthetic work in both edentulous areas (Figure 12-3A and B). Clinical and radiographic examination revealed missing mandibular molars bilaterally (Figure 12-3A–C). Also noted was a vertical deficiency of more than 5 mm in the right posterior mandible and 4 mm in the left posterior edentulous mandible. The treatment plan included vertical ridge augmentation of the right side with a symphysis graft and of the left side with a right ramus buccal shelf block graft.

The right edentulous site was exposed, appropriate crestal decortication and perforation was done, and a symphysis block
graft was fixated to the crest (Figure 12-3D and E). Platelet-rich plasma was applied to the recipient site prior to graft fixation. Five months later both sites were reentered and revealed no evidence of bone resorption (Figure 12-3F and G). The right side revealed vertical augmentation of 5 mm. Three threaded Spline implants were placed in a nonsubmerged mode because of the excellent type 1 quality bone (Figure 12-3H and I). The left edentulous space was augmented 4 mm with a right ramus buccal shelf block graft in the same fashion and three threaded implants were also placed nonsubmerged (Figure 12-3J–L). Both sites were ultimately grafted with epithelial palatal tissue for enhanced keratinized gingiva (Figure 12-3M and N), and three-unit fixed bridgework was fabricated for each site (Figure 12-3O).

Mandibular block autografts for vertical alveolar ridge augmentation are predictable and offer many advantages. These grafts are primarily cortical in nature, exhibit minimal resorption, and tend to incorporate exceptionally well with recipient bone in a relatively short time. They also maintain post-implant placement bone volume and retain their radiographic density to the augmented site. Despite the many advantages block grafts offer for alveolar ridge augmentation, there are complications with posterior mandibular autografts when used for horizontal and vertical augmentation. Morbidity with this grafting protocol is associated with both donor and recipient sites. This includes experience with 434 grafts harvested between August 1991 and December 2002: 208 symphysis grafts and 226 ramus buccal shelf grafts.

Symphysis donor site morbidity includes intraoperative complications such as bleeding; mental nerve injury; soft tissue injury of cheeks, lips, and tongue; block graft fracture; and potential bicortical harvest. Bleeding episodes are intra-bony and can be taken care of with cautery, local anesthesia, and collagen plugs. Injury to the mental neurovascular bundle is avoidable with proper surgical technique, especially in the use of the sulcular approach for bone harvest. Block fracture and bicortical block harvest can also be prevented by following good surgical technique. Pain, swelling, and bruising occur as normal postoperative sequellae and are not excessive in nature. Use of platelet-rich plasma has decreased overall soft tissue morbidity. Infection rate is minimal (< 1%). Neurosensory deficits include altered sensation of the lower lip, chin (temporary 19%; permanent < 1%), and dysesthesia of the anterior mandibular dentition (transient 53%; permanent < 1%). No evidence of dehiscence was seen using the sulcular approach.

The ramus buccal shelf harvest can also result in intraoperative complications including bleeding, nerve injury, soft tissue injury, block fracture, and mandible fracture. Intrabony bleeding and soft tissue bleeding can be handled with cautery. Injury to the inferior alveolar neurovascular bundle and the lingual neurovascular

![Figure 12-3](image-url)

**Figure 12-3**  A, Right posterior edentulous mandible. B, Left posterior edentulous mandible. C, Radiograph indicating bilateral posterior mandibular vertical deficiency. D, Block graft fixation with platelet-rich plasma application. E, Block graft fixation. Note butt joint at anterior recipient donor interface. F, Excellent block graft incorporation at 5 months. (CONTINUED ON NEXT PAGE)
bundle can be avoided with proper soft tissue manipulation and meticulous osteotomy preparation. Block fracture is also an avoidable problem with proper surgical technique. Postoperative morbidity includes trismus (approximately 34%) but is certainly transient and can take up to 2 weeks to resolve. Pain, swelling, and bruising are typically mild to moderate and, again, are minimal with use of platelet-rich plasma. Infection rate is less than 1%. Altered sensation of the lower lip or chin occurs approximately 8% of the time, with less than 1% being permanent. Altered sensation of the lingual nerve has also been reported but has been transient only. No incidence of altered sensation of mandibular dentition has been found. Infection rate is less than 1%.

Recipient site morbidity includes trismus, bleeding, pain, swelling, bruising, infection, neurosensory deficits, bone resorption, dehiscence, and graft failure. Trismus can be expected, as the surgical protocol for reconstruction of the posterior mandible includes manipulation of the posterior mandibular musculature. Incidence is less than 40% and is transient. Bleeding of the recipient bed is intentional secondary to meticulous site preparation (decortication and perforation), but excessive bleeding, although rare, can occur secondary to both intrabony and soft tissue vessel transection. Pain, swelling, and bruising are mild to moderate and are minimized with platelet-rich plasma. Infection rate is less than 1% and is usually secondary to graft exposure. Nerve neurosensory deficits can occur secondary to site preparation and block fixation because normal anatomy is violated. Dehiscence and graft failure (approximately 2.5%) are seen secondary to soft tissue closure with tension or prosthesis contact with the graft site. (Strong recommendation: avoid the use of any type of prosthesis secondary to posterior mandibular block graft reconstruction.) Finally, block graft resorption at

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**FIGURE 12-3 (CONTINUED)**  
Part 2: Dentoalveolar Surgery

Stage 1 surgery is minimal (0 to 1.5 mm) but can be excessive if dehiscence of the graft occurs. In summary, overall morbidity of mandibular block autografts for atrophic posterior mandibular reconstruction is minimal. Most complications are preventable. Those that occur can be handled predictably with minimal adverse effects to the patient.

Interpositional Bone Graft

The interpositional bone graft is placed between a mobilized segmental osteotomy and the basal bone. A typical vertical gain is 4 or 5 mm in the maxilla but 5 to 10 mm in the mandible. The indication for the procedure is an alveolar defect where there is insufficient vertical height for placement of implants such as in the anterior maxilla or in the posterior mandible when a stable vertical augmentation is required, usually over a three- or four-tooth segment.

Figure 12-4A to C illustrates an anterior maxillary defect treated with interpositional grafting. Figure 12-4D shows a posterior mandibular deficiency with 6 mm of bone available above the inferior alveolar nerve. An osteotomy was done (Figure 12-4E) through a vestibular incision to maintain both lingual and crestal blood supply. An interpositional cortical bone graft harvested from the ramus was placed at the osteotomy site, raising the alveolus about 7 mm (Figure 12-4F). The raised segment rotated slightly lingually, but this was compensated for by using a bone plate to establish both the final vertical height and the crestal axis of the osteotomized segment (Figures 12-4G and H).

Alveolar Distraction Osteogenesis

A deficient alveolus can be distracted to improve vertical dimension for implant placement. Sufficient width (5 mm) and vertical height (8 to 10 mm) of a distraction site are needed in order to ensure sufficient (5 × 5 mm) bone mass of the segment to be translated.

Figure 12-5A to G illustrates a case where severe atrophy of both soft and hard tissues left a significant alveolar retrognathia and a vertical defect of at least 10 mm (see Figure 12-5A and B). Using a
vestibular approach, a flared osteotomy was made (see Figure 12-5C). Then a biphase distractor plate was placed in order to gain vertical and horizontal displacement (see Figure 12-5D). Following a vertical distraction of 12 mm (see Figure 12-5E), horizontal movement was achieved by tightening the nut on the horizontally placed screws for a 5 mm horizontal movement. Four months later, implants were placed (see Figure 12-5F). The final restoration was placed an additional 4 months later. A 1-year postrestorative finding is shown in Figure 12-5G to J, indicating a stable bone pattern and reasonable esthetic restoration.

**Iliac Corticocancellous Grafting**

When the jaw is too deficient to do monocortical grafting or osteotomies, bone graft augmentation with iliac corticocancellous graft is needed. Major grafting is usually required when bone mass needs to be expanded in order to gain enough bone for osseointegration.

Figure 12-6A to G shows a patient who had severe maxillary atrophy in which iliac bone graft was combined with sinus augmentation and Le Fort I advancement. Figure 12-6A shows the preoperative finding of severe bone loss including maxillary retrognathia. A 5 mm maxillary advancement with a Le Fort I osteotomy fixated with resorbable bone plates was done. The anterior reconstruction relied on onlay corticocancellous block graft supported by particulate marrow. Graft preservation strategies such as barrier membrane and titanium mesh may be helpful, but in this case a cortical
graft was placed laterally, which minimizes the need for a barrier membrane. Figure 12-6B shows the down-fractured maxilla, where both sinus and nasal membranes are elevated and preserved. The advanced maxilla augmented laterally and vertically around the arch is shown in Figure 12-6C. Figure 12-6D shows the augmentation 6 months after grafting the area is exposed for implants indicating modest shrinkage of the graft, still adequate for implant placement. E, Implant exposure 6 months later (1 year after the initial iliac graft). F, G, The final prosthesis and restoration. H, Implant findings 2 years after placement into iliac graft indicating a stable bone loss pattern to 1st and 2nd screw thread.

Sinus Bone Graft

The sinus bone graft is well established as one of the most stable vertical augmentation procedures in the surgeon’s armamentarium.

Three techniques are used, including:

1. Sinus intrusion osteotomy
2. Lateral approach sinus membrane elevation
3. Alveolar augmentation combined with sinus elevation (shown above)

The sinus intrusion osteotomy can be done on the day of extraction if the wound is clear of soft tissue and infection. In the case shown in Figure 12-7A, the intrusion was done with a bone graft and implant placement 6 weeks after the dental extraction. At this stage epithelial closure of the wound was present, and a residual infection had resolved. A bone graft was taken from the mandible and intruded into the sinus floor using an osteotome. Bone graft was also placed into defects within the extraction socket. Figure 12-7A to C show the sinus grafting and implant procedure. At this stage epithelial closure of the wound was present, and a residual infection had resolved. A bone graft was taken from the mandible and intruded into the sinus floor using an osteotome. Bone graft was also placed into defects within the extraction socket. Figure 12-7D show the final bone graft consolidation 1 year after final restoration.

The lateral sinus graft is done through a Caldwell-Luc approach by elevating the
Bone Grafting Strategies for Vertical Alveolar Augmentation

To preserve a "closed wound" sinus membrane in order to preserve a "closed wound." Bone graft material is packed against the sinus floor, taking care to remove all soft tissue that might be present there. This approach can be used for both simultaneous and delayed implant placement. Barrier membranes are usually not required but benefit over the grafted site if a large "window" is made. Small windows and the use of autogenous bone as graft material generally lead to primary osseous healing of the osteotomy site.

The use of piezoelectric surgery is helpful in avoiding perforation of the membrane. The technique is particularly helpful in areas where a robust thickness of bone is present or when the membrane is extremely thin. The advantage of using this technology is that piezoelectric surgery does not "cut" soft tissue, so sinus membrane perforation is much less likely to occur. Figure 12-8 demonstrates the piezoelectric procedure leading to elevation of the membrane without perforation.

After grafting, the period for consolidation of the bone graft varies with the grafting material used. Allogeneic bone actually slows down the consolidation process. The use of combination grafts including bovine xenograft, algipore, or various other alloplasts all form bone adequate for osseointegration.18

Though bone quality varies considerably as shown by human trephine biopsy results of the various grafting materials, the capabilities of the sinus graft to gain enough bone to form load-bearing osseointegration are remarkable. The 5-year failure rate of implants by almost any grafting technique is less than 20%.19,20

Though grafting material must be osseoconductive, inductivity is not required in order for bone to form. The sinus floor grows bone with blood clot alone. Whatever the technique, bone migrates "endosteally" up the side of the implant. If only a few millimeters of migration occurs, in addition to the residual bone, there is often enough gain to form and maintain osseointegration. Therefore, the principal success of the sinus grafting is not one of implant macro- or microarchitecture or even the type of graft material, be it alloplast, allograft, or autograft, but the intrinsic bone-forming capacity of the sinus floor itself and to a lesser degree the investing sinus membrane.21

In cases of severe atrophy the surgeon must make every effort to use the best available technique and bone graft material.
possible in a highly compromised site. This setting argues for the use of particulate bone marrow harvested from the tibia or ilium and possibly adjuncts such as platelet-rich plasma.

Summary
The difficulty of treating alveolar vertical defects requires the surgeon to be skilled in all of the above modalities. In skilled hands, various approaches can be used in treating the same type of defect.

In most cases defect sites are not strictly vertically deficient. Skill in alveolar width augmentation, or combined treatment, is needed as well. With all of these measures, the ultimate restorative goal is to obtain orthoalveolar form, a concept that now encompasses a broad array of surgical innovation.

References
Severely resorbed edentulous maxillae present very complex problems for the surgeon and restorative dentist. Lack of internal osseous stimulation and nonphysiologic crestal bone loading results in continued resorption of an already atrophic edentulous maxilla. The end result is an inability to use a conventional full denture prosthesis.

In 1999 Dr. Per-Ingvar Brånemark and colleagues introduced the zygoma implant (P-I Brånemark, personal communication, 1999). In their initial study over a 10-year period, 110 implants were placed. Each patient had an additional two to four conventional implants placed in the anterior maxilla, which was restored with cross arch stabilization. Of the zygoma fixtures placed and restored in the initial study, only two were lost in the first year of occlusal loading, and three failed in the subsequent 8 years for a long-term success rate of > 95%.

The availability of the zygoma implant has provided a viable alternative for treatment of patients with extreme resorption of the edentulous maxilla or large pneumatized maxillary sinuses. Before the introduction of this fixture, implant-supported or -retained fixed or removable prostheses in the atrophic maxilla could only be considered after extensive ridge preparation. This preparation usually included major autologous bone grafting, prolonged treatment times, long-term inability to wear any prosthesis, and a higher failure rate for conventional implants placed in large bone grafts.

**Zygoma Implant**

The zygoma implant is an extended-length (30–52.5 mm) machined titanium fixture that is placed through the crestal (slightly palatal) aspect of the resorbed posterior maxilla transantrally into the compact bone of the zygoma. In addition to two to four conventional fixtures in the anterior maxilla, initial stability of this elongated fixture is assured by its contact with four osseous cortices (Figure 13-1):

1. At the ridge crest
2. The sinus floor
3. The roof of the maxillary sinus
4. The superior border of the zygoma

The zygoma implant provides posterior maxillary anchorage when the existing osseous structures do not allow standard implant placement. The alternative in this situation includes bone graft augmentation (sinus lifts and onlay grafts) with their attendant costs, discomfort, prolonged treatment times, and higher complication rates. The zygoma fixture is suggested in the following circumstances:

- When full maxillary edentulism is accompanied by advanced posterior resorption that would otherwise require grafting. At least two and preferably four anterior standard implants are needed in combination with bilateral zygoma implants.
- In partial or incomplete maxillectomy patients when additional implants can be placed in other sites such as the
piriform sinus, orbital rims, palatal shelves, or pterygoid plates to support cross-arch stabilization.

**Indications**

While the zygoma implant is most often used in cases of moderate to severe atrophy, it can be considered a valuable procedure for any patient in need of posterior maxillary implant support with or without significant atrophy. The ability to avoid grafting in many patients, along with the continuous use of an interim maxillary prosthesis also makes the zygoma implant approach appealing as a treatment option.

**Moderate Atrophy**

The majority of patients who present with a medium- to long-term history of denture wear will have a moderate degree of atrophy (Figures 13-2 and 13-3). This category of denture experience constitutes the majority of patients who seek implant therapy to reverse the effects of continuing bone loss and prosthesis instability. Many will be candidates for grafting procedures, such as sinus augmentation or block onlay techniques, as a means of creating additional osseous structure to allow enough implant sites for predictable support. The ability to avoid such grafting is one of the principal benefits of considering the zygoma implant alternative (Figure 13-4).

**Severe Atrophy**

Although most of these patients will essentially be graft candidates, there are some who, because of history or physical circumstances, cannot or will not undergo these procedures. A history of consistent graft failure or a systemic compromise that contraindicates grafting are examples of mitigating factors that may require considering an alternative approach such as use of the zygoma implant (Figure 13-5A–D). Experience to date with these patients is not extensive, but early indications of implant survival are seen as encouraging, even with the most severely compromised maxillae (Figures 13-5E and 13-6).

Prosthesis design for the severely atrophic maxilla with implant support may be influenced by the relative size disparity between the two jaws. Most such atrophy results in an undersized maxilla relative to the corresponding mandible, even in cases where both arches are equally resorbed. Cantilever considerations and implant stress distribution may mandate the use of an overdenture prosthesis rather than a fixed restoration in order to manage occlusal alignment and lateral spacing (Figure 13-7).

**Inadequate Posterior Support**

Occasionally patients will present with adequate bone for anterior or premaxillary implants but have sinus extensions that eliminate the potential for posterior implants without augmentation (Figure 13-8). If such grafting is indicated but countermanded by patient request or health considerations, the zygoma approach can be equally effective.

**Syndrome Patients**

Another less frequent indication for the zygoma approach can present in patients with various anodontias from syndromes such as cleidocranial dysostosis or ectodermal dysplasia. Radiographs may show either impacted and unerupted teeth or missing dentition, resulting in growth patterns of the maxilla that are disrupted and minimized (Figures 13-9 and 13-10). These individuals often present with insufficient bone for adequate numbers of implants and can be difficult to graft because of space or soft tissue limitations. Zygoma implants can be valuable in these instances when combined with conventional fixtures to provide the basis for
The Zygoma Implant

long-term prosthetic support at a relatively early age (Figures 13-11 and 13-12).

Acquired and Congenital Defects

Maxillary defects created by secondary intervention, such as tumor removal or by trauma, can often be treated with zygoma implant therapy to provide retention for an obturating prosthesis (Figures 13-13). Similarly, congenital defects such as an unrepaired adult cleft palate (which are

FIGURE 13-5  A, Severe maxillary atrophy is demonstrated on this survey film. The patient had a history of several failed onlay bone graft procedures. B, At one point, these implants were placed in graft and native bone. All failed, with a resultant destruction of functional support bone. C, Maxillary dimensions from continuous lateral atrophy resulted in a residual anatomy that did not require sinus invasion for implant placement. Even though this is unusual, it did not affect the structural integrity of the implants. D, Implants were placed on either side of the two zygoma fixtures for stability. E, All implants were successfully integrated and were positionally suitable for prosthesis construction.

FIGURE 13-6  A, A definitive restoration has been functioning for over 5 years with no evidence of significant implant challenge. B, Radiographically, the 5-year follow-up shows normal bone response.

FIGURE 13-7  A, An overdenture bar splint was constructed with lateral extensions to keep the retentive elements aligned with the occluding surfaces. B, The undersurface of the overdenture illustrates the mechanical retention provided. C, Frontal view of the finished prosthesis.
increasingly rare owing to early surgical closure) can often be treated with conventional implants in combination with zygoma fixtures to support a removable prosthetic appliance. Situations such as these are rarely the same because of the wide variations in residual soft tissue and bone anatomy, and each case will require careful individual planning to assess the potential for implant placement or zygoma use. For many, however, the ability to use remote bone anchorage with implants around the defect periphery can create excellent supplemental retentive possibilities for these often large and otherwise poorly supported prosthetic devices.

**Immediate Loading**

Literature citations supporting the possibility of immediate loading of maxillary implants increasingly support this concept.6–9 The criteria for attempting this approach are generally the same as for immediate loading anywhere in the oral cavity: adequate initial stability, good bone receptor sites, and initial cross-arch splinting with rigid materials (Figure 13-14A and B). In situations where these criteria can be met, the survival prospects for both conventional and zygoma fixtures appear to be equivalent to the rates attained with the delayed approach. The benefits in patient comfort, convenience, and enhanced function make this a desirable option in appropriately selected cases (Figure 13-14C–F).

**Partial Edentulism**

The original concept of the zygoma implant, used with anterior implants and cross-arch stabilization, would theoretically not have application for posterior maxillary partial edentulism (Figure 13-15A). In practice, however, there is potential for using the zygoma implant through the sinus, with additional fixtures on either side, to support a fixed partial denture (Figure 13-15B–E). This approach has not been thoroughly investigated, and clinical trials do not provide enough longevity to make a definitive statement regarding the efficacy of this technique. Being able to gain strong intermediate support through sinus areas that would otherwise have to be grafted does have enough merit, however, to warrant further investigation.

**Contraindications**

Other than the most obvious contraindications, such as systemic compromise or sinus disease, there are only two specific situations that would complicate the use of the zygoma implant or make it unnecessary. First, where adequate maxillary bone exists for implant placement in numbers

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**FIGURE 13-8** A, This patient initially presented with good bone and five anterior implants, which had not been loaded, opposing an intact restored lower dentition. His physical stature presented the possibility of heavy loading potential to the upper arch, and grafts were recommended posteriorly for additional implant placement. B, The patient refused grafting, so 52 mm zygoma implants were placed bilaterally to provide the necessary support posteriorly.

**FIGURE 13-9** This ectodermal dysplasia patient presents with partial anodontia and associated findings typical of this syndrome.

**FIGURE 13-10** The effects of long-term overdenture use without adequate caries control are evident intraorally.

**FIGURE 13-11** A, The arches were treated with a staged approach, which included mandibular extractions, implant placement, and immediate loading of several fixtures. The maxilla was debrided at the same time, with no implant placement. Tooth bud removal was incomplete. B, Eventual maxillary implant placement after healing included zygoma fixtures bilaterally in lieu of grafting procedures.
and positions to support a prosthetic appliance, the zygoma implant is not needed. The second situation is where there is not enough premaxillary support for at least two stable implants with good potential longevity. Differential diagnosis, in fact, often depends more on the volume and condition of anterior bone than existing posterior anatomy to determine whether some edentulous patients may be candidates for this procedure. In such instances, bone-grafting procedures should be considered preprosthetically, to create an adequate osseous base for effective cross-arch stabilization.

Complications

The most significant complication to zygoma implant therapy is the loss of the implant (Figures 13-16A–C). Our experience to date indicates this is a relatively infrequent occurrence, but the impact on the original treatment plan is significant. Without this support element, posterior anchorage may be severely compromised and cantilever extensions to the first molar region may overstress the remaining components. Correcting the resultant imbalance using a zygoma approach will require a healing period for bone regeneration in the original site and eventual


A
B

FIGURE 13-13  A, Gunshot trauma created significant maxillomandibular discontinuities. B, Reconstructive efforts over several years have resulted in effective osseous restructuring in both arches. C, Traditional anatomic landmarks are difficult to identify, and normal arch contours are significantly disrupted in the repaired maxilla. D, While anchorage in the zygoma was adequate, absence of alveolar bone was noted on one side. The ability to use zygoma implants in this situation was significantly advantageous. E, Maxillomandibular relationships were lateralized as depicted by the mounted casts of each arch. While not ideal, this was still a workable situation. F, Radiographic view of the completed prosthesis. G, Clinical view, in occlusion, of the completed rehabilitation. Lateral jaw relationship discrepancies required a lingual cantilever and crossbite on the lower bridge.
replacement of a second implant. Inter-

im therapy may include the use of a pro-

visional restoration on the remaining

integrated implants but should not

include a cantilever extension on the

affected side (Figure 13-16D). To date,

this rescue approach has proven effective

in the two instances that we have experi-

enced in zygomatic implant failure. Both

have ultimately been restored to com-

plete function using both the original

and rescue zygoma fixtures for posterior

support (Figure 13-16E–G).

Presurgical Assessment: Clinical

Current use of the zygoma implant dic-
tates ultimate restoration with cross-arch
stabilization of the fixtures with addi-
tional implants. Adequate bone must be
available to place and retain at least two

but preferably four anterior maxillary
conventional implant fixtures, which are
joined to the zygoma fixtures with a cast
base. The patient must have pathology-
free maxillary sinuses and have accept-
able soft tissues in the area in which the
implants will be placed. The patient’s

presurgical planning should be completed
before insertion of the implants for both

the maxillary and mandibular arches.

Patients should be physically and med-
ically stable enough to withstand a surgi-
cal procedure approximately 2 hours long
and to tolerate a general anesthetic or
deep intravenous sedation. The patient’s
mandibular range of motion must be
adequate to provide access for placement
of fixtures 30 to 52.5 mm long
transpalatally in the area of the zygo-
matic buttress. The opposing mandibular

Teeth, if present, may limit access to the
site of the zygoma fixture placement. If
using deep sedation, local anesthesia in
the mandibular arch, as well as in the sur-

gical site itself, is advisable.

Presurgical Assessment: Radiographic

Adequate radiographic examination is
needed prior to surgery to identify or rule
out sinus or other pathology and to evalu-
ate the osseous anatomy of both the zygo-
ma and maxilla. The thickness of the
remaining alveolar bone inferior to the
sinus in the second premolar–first molar
region should be sufficient to provide
some support for the long implant near
the abutment connection. The apex of the

sinus just lateral to the orbital floor should
be identified and the quality and quantity
of the bone that will support the apical
end of the zygoma implant evaluated. The
anterior maxillary alveolus should also be
evaluated to determine if enough residual
bone is available to place two to four ante-
or implant. Panoramic, periapical,
cephalometric, and plain tomography or
computerized exposures are all helpful in
this evaluation.

FIGURE 13-14  A, These five anterior and two zygoma implants were loaded immediately with a rein-
forced resin bridge converted from the original denture. B, The cantilever extensions are limited at the
provisional stage, but the reinforced bridge provides a rigid cross-arch effect. This prosthesis was deliv-
ered immediately following surgery. C, Radiographically, all implants appear integrated at
5.5 months. The provisional fixed partial denture has not been removed during that time period.
D, The soft tissue response viewed at removal of the provisional prosthesis shows relatively good epithe-
lial recovery. The deep tissue response in the zygoma regions results from the long-term resin connection
subgingivally. E, The definitive prosthesis was completed approximately 8 months after stage I surgery.
F, Radiographically all implants appear well integrated and functioning normally.
FIGURE 13-15  A, Sinus graft procedures were recommended for this patient, but were declined. As an alternative approach, zygoma implants were considered for the support needed to create fixed partial dentures bilaterally. B, The zygoma fixtures are augmented mesially and distally with conventional implants. A delayed approach to restoration was used. C, The radiographic presentation immediately after stage I surgery. D, The completed right-side fixed partial denture was constructed using porcelain-fused-to-metal technology. E, The occlusal view shows the bilateral restorations, each with a central zygoma implant.

FIGURE 13-16  A, An impression coping has been attached to the zygoma implant at the final impression appointment. B, It was noted that there was rotational instability of this fixture with movement of the coping. C, The implant was removed without resistance. There was no sign of bone adherence to any of the implant surface. D, A provisional restoration was created for interim use while the failure site healed and during the healing period for another zygoma implant. The cantilever extension to the affected side has been reduced to only premolar occlusion. E, Occlusal view of the completed restoration on healthy zygoma implants bilaterally. F, Frontal view of ceramometal restoration. G, Radiographic view. The right side zygoma implant side shows an integrated replacement fixture.
Surgical Protocol

Surgery for zygoma implant placement is best performed using deep intravenous sedation or a general anesthetic. Local anesthesia with vestibular infiltration, second-division nerve blocks, and percutaneous blocks or infiltration lateral and superior to the zygomatic notch just lateral to the orbital rim should be administered. Bilateral inferior alveolar nerve blocks are also helpful if the procedure is performed with sedation because significant retraction of the tongue, lower lip, and mandible are needed to ensure adequate access for the procedure.

A crestal incision, placed slightly to the palatal aspect of the ridge in the first molar–second bicuspid region is made from the right- to left-tuberosity regions with bilateral releasing incisions at the incision ends. A releasing incision at the maxillary midline is also helpful for flap development and retraction. The lateral maxilla is exposed by elevating full-thickness mucoperiosteal flaps sufficient to visualize the zygomatic buttress from ridge crest to the superior surface of the zygoma at the zygomatic notch, just lateral to the orbit. The anterior maxilla is exposed to the piriform rims to avoid tearing the flap during retraction and to allow placement of conventional anterior maxillary implants. The entire lateral surface of the zygomatic buttress is exposed using a palpating finger extraorally at the zygomatic notch to ensure that the dissection is not directed into the orbital floor. During the dissection, the infraorbital nerve should be identified and protected.

A fissure bur, usually a 703 or 702, in a straight surgical handpiece is used to make a “slot” exposure vertically in the lateral wall of the sinus near the height of the zygomatic buttress. The slot should parallel the planned course of the zygoma implant just medial to the lateral sinus wall. The slot should extend from near the sinus floor at the planned site of implant placement superiorly to near the roof of the sinus. Preparation of the slot in the sinus wall allows the surgeon to visualize directly the passage of all drill preparations and implant insertion through the lateral sinus. When preparing the slot, the Schneiderian membrane in the sinus is removed to allow good visualization and to prevent its interference with site preparation and implant insertion. If portions of the membranes are “picked up” by the implant and carried into the implant preparation in the body of the zygoma, they could interfere with osseointegration.

A series of long drills are used for incremental preparation of the implant site. The zygoma implant varies in length from 30 to 52.5 mm (Figures 13-17 and 13-18). The apical two-thirds of the implant is 4 mm in diameter and the alveolar one-third is 5 mm in diameter. The initial drill is a round bur, which is used to start the implant preparation at the second bicuspid–first molar area as near the crest of the residual alveolar ridge as possible—usually slightly to the palatal aspect. The surgeon must preserve enough bone lateral to the site to fully surround the alveolar portion of the implant. The round bur is directed through the sinus floor and through the lateral sinus superiorly following the axis of the lateral wall slot preparation to the top of the sinus where it indents the site of the preparation in the zygoma body. The slot preparation allows direct visualization of the passage of the drill and the subsequent instrumentation and implant insertion (Figures 13-19–13-21). A custom-designed zygoma retractor with a toe-out tip is kept in position over the zygomatic notch throughout the site preparation to provide good visualization and protect the surrounding anatomy. The retractor also has a midline marker that parallels the site preparation and assists in orientation of the drills in the proper direction (see Figure 13-20). Subsequent drills to complete the preparation are, in sequence, long 2.9 mm diameter twist drills, a 2.9 mm to 3.5 mm pilot drill, and a 3.5 mm twist drill. The preparation is carried through the body of the zygoma, through the cortical bone of the sinus roof, and through the cortex at the superior border of the zygoma body at the notch. The soft tissues at the superior portion of the preparation are protected by the zygoma retractor (Figure 13-22). Each fissure bur has incremental markings from 30 to 52.5 mm, which help the surgeon determine the needed implant length.
preparation is complete, final determination of implant length is made using the zygoma implant depth gauge. Lastly, if the residual alveolar bone is substantial, a 4 mm twist drill is used to complete the alveolar portion of the preparation. If the residual alveolar bone is spongy, this step is usually eliminated.

The zygoma implant has an angulated abutment platform. The 45° angulation allows the platform of the implant to emerge in the same plane as that of the conventional implants that will be placed in the anterior maxilla. Premounted implant carriers are already attached to the zygoma implants for handling of the fixture with the handpiece. The implant is inserted with copious irrigation, directly visualizing its passage through the lateral sinus through the slot preparation (Figure 13-23). During insertion, the implant must stay in the same plane as the drills in order to ensure its engagement in the preparation site at the zygoma body. The slot preparation should be extended superiority far enough to allow visualization of the preparation. When site preparation has been adequately performed, the handpiece will stall when the apical portion of the implant engages 2 to 3 mm of dense zygomatic bone. When this occurs, a manual driver is used to complete implant insertion. Proper angulation of the abutment platform is determined by placing a screwdriver in the implant carrier screw head and seating the implant until the screwdriver is perpendicular to the crest of the edentulous ridge. The implant carrier is removed and a cover screw is placed (Figure 13-24).

After placement of the zygoma implants, two to four regular platform Mark III or Mark IV Nobel Biocare implants are placed in the anterior maxilla (Figure 13-25). The flaps are repositioned and sutured. The maxillary denture is relieved, hollowed out at the implant emergence sites, and soft-lined with a tissue conditioner. Prior to closure, implant-level impressions are made. This allows for fabrication of a rigid bar to be placed at second-stage surgery about 6 months later.

The patient’s denture prosthesis is relined as often as is necessary over the 6-month osseointegration period. At second-stage surgery, the cast rigid bar is attached to the implant fixtures, providing immediate cross-arch stabilization. The denture is further hollowed out and relined or a transitional fixed prosthesis is constructed and attached. Four to 6 weeks later, after the soft tissues are healed,
prosthetic procedure

healing phase

the maintenance of the zygoma implant patient is an ongoing process from the completion of stage I surgery through the entire healing phase (Figure 13-26). As noted earlier, the existing or provisional upper denture can be modified for immediate use (Figures 13-27–13-30), giving the patient a continuous esthetic presentation. There will be some significant limitations for functional use, such as changes in retention or chewing capability, but the option of having teeth throughout the entire process is usually far more appealing than the transitional periods of no prosthesis use that accompany many graft procedures.

protective splinting

one of the unique features of these implants is the strength they provide when used with splinting and cross-arch stabilization. When used or loaded independently, however, it is felt that the off-axis load transfer can be detrimental and possibly counterproductive for maintenance of osseointegration.10 immediately following stage II surgery, or exposure of all implants with abutment connections, it is recommended that some protective measures be used to prevent independent stress transfer from the denture base to the implants individually. To this end, the current protocol calls

figure 13-24 zygoma implant fully inserted. note the cover screw on the abutment platform positioned near the crest of the alveolar process. the implant “hugs” the lateral wall of the sinus.

figure 13-25 near ideal positioning of the zygoma implants. a, presurgical panoramic radiograph. b, postsurgical panoramic radiograph. c, posterior-anterior radiograph. d, lateral head radiograph.

figure 13-26 immediately after implant placement cover screws are attached to all of the fixtures used in the maxillary arch, and the tissues are sutured to create a watertight primary closure. this radiograph shows the implant positions immediately after placement.

figure 13-27 the patient’s original denture is hollow ground in the area of the premaxillary ridge crest and distally onto the alveolar ridge and palatal mucosa areas where the two zygoma implants will eventually exit. it is also important to relieve the intaglio surface of the labial flange to prevent unnecessary apical pressure in the vestibular area.
for splinting all of the newly exposed implants with a soldered bar within 24 hours of abutment connection (Figures 13-31 and 13-32). This is accomplished by making an impression immediately after the abutments are delivered and sending it to the dental laboratory for rapid turnaround (Figure 13-33). A gold bar of approximately 2 mm in diameter is bent to contour so that it touches a set of gold cylinders attached to the abutment analogs on the cast (Figure 13-34). With a microwelding device the bar and cylinders can be soldered together and within a short time period a passive protective splint can be fabricated. The bar splint is delivered, usually the next day, and the denture is hollow ground to allow complete seating without bar interference (Figure 13-35). At this time, a complete soft liner can be applied to the upper prosthesis to enhance comfort and retention (Figures 13-36 and 13-37). The bar splint may not be necessary in situations where the patient is not wearing an upper prosthesis, but for all other cases where continuous denture wear is desirable, the bar splint protocol should be used.

**Final Prosthesis Construction**

Final impressions can be made following an adequate healing period, usually 3 to 4 weeks (Figures 13-38–13-40). The procedure for this and ensuing steps is the same as for all
fixed bridge construction on implants. Jaw relation records are obtained using implant-stabilized record bases and wax rims (Figure 13-41). The try-in with teeth follows the trial set-up done in the laboratory, and patient approval of the esthetic presentation is confirmed (Figures 13-42–13-44). Silicone putty indexes are made of the approved wax-up and are used to provide a matrix for creation of a metal bar structure (Figures 13-45 and 13-46). Following a second try-in appointment for evaluation of passive fit and esthetics, the prosthesis is processed with heat polymerizing resin (Figure 13-47). Delivery is
accomplished using appropriate screws and screw torques to provide even and complete seating (Figures 13-48 and 13-49).

The bar structures are generally waxed and cast in precious metals but can also be milled from solid blocks of titanium with excellent passive fit properties (Figures 13-50–13-54). In select situations, such as minimal interocclusal distance or high load forces, it may be beneficial to use a porcelain-fused-to-metal restoration. The procedure for constructing these prostheses is essentially the same up to the point of the patient-approved wax-up. The metal substructure will be designed to provide

**FIGURE 13-43** The teeth are waxed to contour in positions dictated by the record base procedure and are sent to the clinic for try-in and patient approval.

**FIGURE 13-44** Final approval for esthetic display, occlusion, and vertical dimension are all obtained at this clinical visit.

**FIGURE 13-45** The cast framework design is based on available space and tooth position as dictated by the wax set-up from the trial denture base. These dimensions are captured using a buccal index that keys to the master cast.

**FIGURE 13-46** For greatest accuracy, the casting technique for these long-span restorations usually requires a runner bar and multiple sprue attachments to minimize distortion.

**FIGURE 13-47** Using the buccal index, teeth are waxed to the gold casting for try-in. It is usually desirable to have a second try-in appointment to verify the casting accuracy intraorally and to obtain final approval for esthetics.

**FIGURE 13-48** The completed restoration has been processed and is delivered using the manufacturer’s recommended torque at each of the screw sites. The screw access holes can be covered with provisional materials for an interim period but will eventually be filled with cotton over the screws and a composite cover at the surface.

**FIGURE 13-49** Radiographically, the definitive restoration appears to fit passively with all implants functioning successfully after 4 years.

**FIGURE 13-50** An alternative to the gold-casting technique is available using Procera technology that allows the creation of a metal substructure out of a single piece of machined titanium.

**FIGURE 13-51** By entering scanning information into a computer bank, computerized lathes with precisely controlled cutting heads attack the titanium blank to create the milled bar structure.
support for the veneering material and will therefore have a completely different architecture from the hybrid denture tooth design. It may be especially advantageous to use the milled titanium technology for these restorations, since they do not tend to distort through the thermocycling phases of veneering to the same degree as the precious metal alloy cast substructures (Figures 13-55 and 13-56).

Summary

The placement of implants and restoration of the extremely atrophic maxilla is a challenge to both the surgeon and prosthodontist. If conventional implants are to be used exclusively in this setting, extensive bone grafting is usually needed before implant insertion and usually includes sinus lifts and onlay grafts with large amounts of donor bone required. The inconvenience, prolonged treatment, costs, potential complications, lower implant success rates, and donor site morbidity are important considerations. This is further compounded by the patient’s inability to wear a prosthesis for extended periods of time—a factor that keeps many patients from pursuing treatment. With the zygoma implant, bone grafts often may be avoided, treatment time is shortened, donor sites are unnecessary, and the patient may continue to wear a transitional prosthesis. This results in greater patient acceptance while providing the patient with a well-tolerated, stable, and esthetic fixed or removable prosthesis at completion of treatment.

The advantages of considering the zygoma implant include the following:

1. Donor site morbidity is reduced or eliminated entirely.
2. Treatment time is markedly reduced or eliminated entirely.
3. Bone graft survival and consolidation are not considerations.
4. The total number of implants to support a prosthesis is reduced.
5. The treatment is more affordable and less invasive than alternative treatments.

The disadvantages of the zygoma implant include the following:

1. Technically demanding surgery—should only be performed by well-trained surgeons capable of dealing with any surgical situation or complications that might arise
2. Risk of injury to adjacent structures—that is, orbit, orbital contents, facial nerve, lacrimal apparatus, infraorbital nerve
3. Risk of postoperative sinusitis, although less than with sinus lift procedures
4. Fixture failure—although rare, more difficult to retreat
5. Surgical access difficult—deep sedation or general anesthetic required

As with all properly planned and executed implant prosthetic procedures, extensive coordination between the surgeon and the prosthodontist is necessary before initiating treatment. Ideally, the prosthodontist should be available at surgery. Similarly, the surgeon should become familiar with the prosthetic needs and techniques involved with fixture positioning and restoration. Finally, patient education, preparation, evaluation, and informed consent are major parts of the procedure and its ultimate success. Patient understanding, before treatment is initiated, should include the need for meticulous hygiene and maintenance.

The zygoma implant, when understood and appropriately used, provides a treatment alternative for many patients with atrophic edentulous maxillae.

References

Implant Prosthodontics

Thomas J. Salinas, DDS

Biomechanical Considerations

Periimplant 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 is maintained in part by the teeth. Original studies by Waerhaug and Gargiulo and colleagues showed the width of the dentogingival complex surrounding natural teeth approaching 3 mm.1,2 Comparably, a similar study by Cochran and colleagues assimilated the periimplant 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 14-1).3 This distance provides room for biologic width, proper emergence of restoration, and esthetics 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 colleagues have shown that there is a relationship of the underlying bone to soft tissue in the interdental spaces between natural teeth.7 Also a relationship from both implant to natural tooth and implant to implant as well 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 showed the critical distance between implant surface and implant surface approached about 3 mm before the mutually destructive process of lateral bone resorption accelerated each other’s processes (Figure 14-2). Typically, each implant loses peri-

implant bone within the first year and then stabilizes—one criterion of success as outlined by Albrektsson and colleagues.9

Patient Factors

Soft tissue evaluation prior to 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
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Presurgical planning for placement of dental implants and 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 14-1.

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 esthetic 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 implant placement. 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 esthetic 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. It is also well documented that smoking decreases bone density. 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%. 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.

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. However, there are several studies that refute the benefit of hyperbaric oxygen to the long-term survival of dental implants. Standard protocol suggested by Marx and Ames is 20 preoperative dives and 10 postoperative dives.

Systemic factors such as diabetes, connective tissue diseases, autoimmune diseases, and 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.

### Radiographic Evaluation

Periapical radiographs are an excellent way to evaluate single missing teeth since 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 14-3).

Occlusal radiographs for mandibular arch assessment also can give an appreciation of the size of the inner and outer cortices as well as the position of the mental foramina (Figure 14-4). 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 taped to the underside of the patient’s denture or a stainless steel wire attached with sticky tape.

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**Table 14-1 Potential of Creating/Preserving Papilla**

<table>
<thead>
<tr>
<th>Distance from Bone to</th>
<th>Chance of Creating Papilla (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contact Area (mm)</td>
<td></td>
</tr>
<tr>
<td>4.0</td>
<td>100</td>
</tr>
<tr>
<td>5.0</td>
<td>100</td>
</tr>
<tr>
<td>6.0</td>
<td>56</td>
</tr>
<tr>
<td>7.0</td>
<td>27</td>
</tr>
</tbody>
</table>

Adapted from Tarnow DP et al.
wax 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 sinus, 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 work-up 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 14-5). This varies from patient to patient, by location, 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 since 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 cephalograms 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 14-6).

Linear tomography is a useful adjunct when considering a single-tooth implant or definitive positioning of the inferior alveolar canal, concavity of the nasal fossa, and the maxillary sinus. This feature is an extension of most modern panoramic radiographic units. It gives a three-dimensional perspective of the primary radiograph, which can help one anticipate grafting procedures or select an implant length and configuration (Figure 14-7).

Computed tomography (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 prior to augmentive procedures (Figure 14-8).

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.

**Surgical Stents**

Fabrication of surgical stents for implant placement should be part of every case since the placement is permanent and
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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 work-up.35

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 14-9). This information may also be translated from radiographic findings to a surgical stent in the position of the mental foramina (previously described). 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 esthetic 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 since it will be supported with a splinted structure in which cantilevering may be used.

Implant hybrid dentures mandate the use of a surgical stent since 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.

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 esthetic 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 14-10). 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 old 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. Since 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.

Crown-to-Implant Ratio

Ideally, a crown-to-implant ratio of 1:1 or less is desired (Figure 14-11). For this reason, the minimum length needed approaches 10 to 12 mm since the clinical crown...
length frequently approaches this measurement. Standard implant diameters with shorter lengths have been shown to have a high failure rate.\(^36,37\) 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**

There are several axioms in implant dentistry relating to occlusion:

- Avoid lateral component forces whenever possible.\(^38\)
- Establish occlusal forces along the long axis of the implant.
- For added stability, splint implants when 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.)

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.\(^39-41\) In partially dentate cases, the implant restoration should have equal or slightly less occlusal loading than the natural tooth (Figure 14-12). 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**

Full-arch reconstructions of the maxilla should be based on placement of 8 to 10 implants splinted for cross-arch stability.\(^42,43\) Reasonable length implants (> 12 mm) should be considered especially in the posterior maxilla as shorter implants into this relatively soft bone have been shown to do poorly in the long term.\(^44\) 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 14-13).

Full-arch reconstruction of the mandible can involve different considerations as the mandible is a dynamic bone that flexes and rebounds as it opens and closes. Traditionally, mandibular full-arch reconstruction has involved placement of four to six implants between the mental foramina with a minimal cantilever to the posterior.\(^45\) The greater the anterior posterior spread, the greater the amount of cantilever possible. On average, a 16 mm distal cantilever is permitted (Figure 14-14). 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.\(^46\) This allows some flexure of the mandible without transferring stress to the prosthesis and/or implants. Prosthetic screw or implant failure may result if a solid prosthetic connection spans the splinted first molar regions.

**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 14-15). The original applications were
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Cantilevering is about 16 mm.

FIGURE 14-14  Cantilevering is about 16 mm.

FIGURE 14-15  Standard externally hexed implant.

FIGURE 14-16  Press-fit cylinder-type implant.

FIGURE 14-17  Tapered-wall screw implant.

surgical stability in trabecular bone became more apparent. Significant mechanical improvement in abutment and screw-retained components occurred in the early 1990s and markedly decreased complications. Current trends are toward the use of tapered macrotensive 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 14-17. With these trends it is apparent that internal connections are preferable for fixed tooth replacement since abutment screw loosening appears significantly less with internal connections than with butt-joint implants. 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 14-18). 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. Morse tapers are anywhere from 0 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 applications. The development of an extensive armamentarium of abutment connections and restorative components currently exists for restoration with esthetic fixed prostheses. Many well-known systems have this versatility available, which is especially important when considering implant restorations in the esthetic zone. It is advisable for the surgeon to become familiar with the restorative components available when treatment planning for implants cases. Consideration of the components makes it easier to select the appropriate system for both surgical installation and restoration.
Implant Components

There is a wide array of dental implant components for impression procedures, laboratory fabrication, and direct restorative dentistry. The various types of osseointegrated implants are discussed above.

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 14-19. 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 esthetic areas. Cast yellow 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, 55 yellow gold creates a warm appearance in esthetically critical areas. In esthetic areas ceramic abutments have also been used in cemented designs for single and multiple-unit crowns (Figure 14-20). Similar to titanium, these abutments manifest a biologic attachment.

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 < 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 14-21). The open-tray technique is considerably more accurate and is indicated for multiple splinted units. At this point an abutment analog 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 analog 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 14-22). 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.

**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 prior to 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 missing. Horizontal deficits of this type can be restored by using intracoronal anchorage methods (ie, 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, 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. The longevity of an FPD has been examined by a number of studies and is favorable over extended periods of time. Much of the literature indicates standard FPD survival to be in the high eightieth percentile at 10 years and seventieth percentile at 15 years. However, typical complications occurring are related to endodontics, recurrent caries, periodontal factors, and failures in retention. Single-tooth implant studies reveal complications as well. The incidence of complications for single-tooth implant restorations appears to be significant in comparison with other types of implant prostheses; 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.⁵⁹,⁷⁰

The success of removable prostheses relies on the combination of retention, support, and stability, which can be deficient. Implant dentistry today is rooted historically from treatment of mandibular edentulism,⁷¹,⁷² which is currently the most predictable form of dental implant therapy.⁷³–⁷⁶ This success is primarily owing to the high degree of success of osseointegration in the anterior mandible.⁵³ 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. 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 esthetic 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 prior to implant treatment (ie, 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 Esthetic Zone**

Esthetic considerations encompass additional complex concerns such as gingival display, proportion of teeth in the esthetic zone, and bone density support. The esthetic zone is generally considered to be the maxillary anterior area. When considering replacement of a single tooth in the esthetic 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 esthetically pleasing central incisors should be about 66 to 80%.⁷⁷ 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 esthetics for the functional goal of eliminating lateral forces on the restoration/implant. Esthetic 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.

Esthetic considerations for removable prosthetics 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 esthetic integrity so critical to this area. A functional lingual maxillary alveolar seal is essential for correct labiodental consonant 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 periimplant tissues. The axis of implant placement should be aimed through the incisal edge for standard-diameter implants (Figure 14-23). This results in predictable esthetics and manageable soft tissues. If a comparably wider implant is placed (4.3, 5.0, or 6.0 mm) in an esthetic 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 14-24). 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 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 recreate the emergent path as the restoration exits the gingival sulcus.

**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 14-2). Screw-retained prostheses are simplistic to retrieve, 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. Screw retention for single units in the esthetic area may be problematic with respect to hygiene as these sites frequently have a full complement of bone and soft tissue on adjacent teeth (Figure 14-25). This can create an almost unavoidable situation of ridge lapping to provide the palatal access channel needed. Screw-retained prostheses are especially useful in the posterior dentition as 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

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<td><strong>Advantages</strong></td>
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<tr>
<td><strong>Screw Retention</strong></td>
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<td>Retrievalability</td>
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preference but may be tailored to what can be serviced and maintained most easily.

**FPDs in the Esthetic Zone**

Placement of multiunit restorations in the anterior maxilla should bring to mind several anatomic considerations for surgical planning:

- Length of the residual alveolar ridge to the nasal floor
- Buccolingual width of the bony ridge to provide for implant placement
- Available bone for angulation of implants to provide for either screw retention or cement retention
- Participation of the restoration in anterior guidance

Anterior FPDs or any restoration in the esthetic zone should first begin with a diagnostic wax-up or template (Figure 14-26). This will give an idea as to the incisal edge position as well as the available restorative dimension, and should be 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 esthetically 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 augmentative 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 bridgetwork that replaces teeth, soft tissues, and alveolar bone may be more predictable in these circumstances. If the surgical work-up 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 14-27). Superior/inferior positioning of implants is virtually the same as for single units, described above. 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 14-2). 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 becomes occasionally necessary to perform angle correction as there is frequent disparity between the long axes of tooth to the long axis of bone available in the anterior maxilla. An intimate fit of FPDs is far easier to achieve with a 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 esthetic success of the implant restoration. After placement and uncovering of the implants, it is prudent to create provisional restorations to develop soft tissues. Only in this way can an acceptable esthetic outcome become predictable in the esthetic 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 14-28). Tarnow and colleagues 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 (about 2 mm) when the implant abuts a natural tooth. Since 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 14-29). Implant proximity should also be assessed prior to placement for hygiene procedures as 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. In general, 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 14-30). Progressive enlargement of the maxillary sinus is often seen after tooth loss as well as residual ridge resorption. Diagnosis of either of these problems helps one determine the appropriate treatment. If pneumatization has taken place, sinus augmentation procedures can be indicated either with concomitant or delayed implant placement. Residual ridge resorption or traumatic destruction of alveolar bone by trauma or periodontal disease may also have taken place. In these cases, onlay bone grafting may be a more appropriate treatment (Figure 14-31). 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 14-32). In general, horizontal forces acting on implants are considered destructive. It is desirable to use these implants as a vertical stop in the chewing cycle. If lateral components of the chewing cycle are unavoidably placed on the implant restorations, they should be splinted together. 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 14-33).88

**Cantilevered FPDs**

Cantilevered fixed prostheses 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 14-34). Cantilevering requires that a framework be connected at a maximum clamp force; such stability is best achieved with screw-retained frameworks. Occlusal contact created on the pontic should be very light to coincident.

**Restorations for the Edentulous Patient**

**Implant-Retained Overdentures**

Those over 65 years of age are said to represent a significant proportion of the US population, and the average life expectancy has risen by 30 years since 1900.89 This is due mostly to the increase in medical advances and critical care. A sizable portion of this group is edentulous or partially dentate in at least one arch.90 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.91 Increased support and anchorage can be improved with the use of at least two osseointegrated implants in the anterior mandible. The use of stud attachments connected to the implants can be a cost-effective measure to improve retention, stability, and support (Figure 14-35). If a stud-retained denture is planned, the implants should be as parallel
as possible to avoid premature wear of the attachment mechanism. The vertical height of the attachment should be considered as some edentulous mandibular arches do not provide > 4 mm of restorative dimension for the mandibular denture. Preoperative planning calls for the evaluation of the patient’s present difficulty. Reasonable esthetics, 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 prior to exiting the mental foramen; thus, consideration should be given to proper site selection. A radiographic marker such as a piece of foil taken from a film packet or a standardized stainless steel shot can be secured to the patient’s denture and placed in the mouth prior to 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 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 < 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 (Figure 14-36). There are several reasons to plan the implant-retained denture for a bar attachment. First, short (10 mm or less) 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, non-parallel 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 prior to 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 to 4 mm), 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 14-37).

Implant-retained overdentures for the maxilla should always incorporate the use of bar attachments. The literature cites poor long-term success for lone-standing implants supporting overdentures in the maxilla. 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.

**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 intraoraminally. More contemporary literature suggests the use of four widely spaced implants in this region opposing an edentulous arch with equally successful rates. The strategy for using implants in the anterior mandibular
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area allows segments to be cantilevered posteriorly in accordance with the anteroposterior spread of the implants.\textsuperscript{97} On average, this equates to 10 to 20 mm or to the area of the lower first molar.\textsuperscript{98,99} 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.\textsuperscript{100} 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 to the anterior fixtures, which are placed under tension. Also, the mandible may be viewed as a dynamic bony structure undergoing flexure.\textsuperscript{101} 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.\textsuperscript{102,103} Implants planned for support of a prosthesis in the edentulous maxilla should involve at least eight fixtures. This may require the use of sinus augmentation or extended-length implants into the zygomatic process. The use of cantilever extensions in the maxilla should be limited to 10 mm.\textsuperscript{104}

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° taper, allowing a precise path of placement (Figure 14-38). The underside of this overdenture has a cast metallic housing that acts as a guide over the milled-bar attachment (Figure 14-39). 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.

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 bar. This technology, which results in an essentially detachable fixed bridgework, may be prohibitive in costs. This three-level treatment in an edentulous patient has predictable results.

Fixed Detachable Prostheses

One alternative treatment method for an edentulous mandible is the use of a hybrid denture also known as a fixed removable restoration. This restoration contains a screw-retained metal framework with a veneer of acrylic resin and denture teeth, thus earning 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 14-40). 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 must incorporate the use of a surgical stent as the exit sites for the access channels are critical. The surgeon may be cautioned against using a hybrid denture in those patients with a skeletal Class III or severe Class II relationship as revealed by cephalometric radiography. Access to channel location and cantilevering and maintenance of hygiene would be the resultant problems if used in these patients. Recently, application of this immediate-load and immediate-restoration technique has become popular. Prefabricated versions of the technique have also enjoyed widespread success. Chapter 13, “The Zygoma Implant,” elaborates on this topic.

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 would 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 14-41). Implant-supported maxillary overdentures are frequently used in cases of moderate to severe resorption as they replace not only missing mastication and esthetics but also phonetic physiology as well. Speech production may rely heavily on adaptation of the prosthesis to the palatal gingiva. 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 14-42).

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.\textsuperscript{105,106} 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 bony walls. A word of caution is advised for those teeth that have drifted or are not in an ideal location as tooth position influences implant position. Indications for placement into a recent extraction socket are freedom from infection and reasonable orientation of the existing tooth. Ways of facilitating this technique may incorporate orthodontic extrusion to create a smaller socket in the bone, facilitating extraction, and overcorrecting bone apposition to recreate missing architecture (Figure 14-43).\textsuperscript{107} The extrusion should take place slowly, usually over 3 to 6 months.

Surgical Installation Stability

Installation of implants into bone usually is characterized by minimizing the inherent gap between the implant and bone surface. Although this 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.\textsuperscript{108–110} 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 75µm has been shown to inhibit osseointegration to a fibrous tissue deposition instead of bone apposition;\textsuperscript{111} therefore, occlusion placed on a provisional restoration during the critical period of osseointegration must be carefully controlled to eliminate this scenario. Interproximal contact with adjacent teeth should also be eliminated. If this modality is desired, a more controlled technique of protecting the occlusion with a centric relation splint 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 cautiously recommended as long as there are careful control of occlusion and passive splinting frameworks.\textsuperscript{112}

Immediate Restoration

Immediate restoration of a single-tooth implant may be incorporated in the esthetic zone (Figure 14-44). 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
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The advantages of immediate restoration are the establishment and preservation of the periimplant tissues. It is easier to preserve this tissue than to recreate it by using a staged approach. Usually provisional restorations are placed upon single or multiple units during osseointegration.

Immediate Load

Single-Tooth Prostheses Studies of immediately loaded single-tooth implants are not widespread. However, data taken from a selected number of studies indicate an 85% success rate on single-tooth prostheses in the anterior maxilla and other areas. More data are needed before this can be recommended as a standard treatment. Protection of the implant from overloading is critical as osseointegration is interrupted at 50 to 150 µm of repeated movement. Therefore immediately loaded implants should be kept free from interproximal contacts as deflection mesiodistally can also promote micromovement.

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 US general population. 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. Since 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 14-47). 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 since 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, the use of fibular grafts has also shown a promising degree of success in reconstruction of these complex mandibular defects.\textsuperscript{123,124} 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 be also well obtained at surgical installation, and long-term success has been observed (Figure 14-48).\textsuperscript{125} The 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, peri-oral scarring, and adjacent/opposing occlusion. Frequently, the crown-to-implant ratio is seen to be > 1:1 (Figure 14-49). 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. 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 esthetics. 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.\textsuperscript{126} 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.\textsuperscript{127,128} 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 14-50). Recently the use of zygomatic implants has been suggested as an alternative to sinus lifting.\textsuperscript{129,130} The implant protocol for zygomatic implants mandates bilateral placement, and preservation of the defect side of the infraorbital rim may improve surgical stability.\textsuperscript{131} Both of the techniques require a screw-retained bar attachment to be made with the obturator (Figure 14-51).

Craniofacial Defects

Resection of portions of the craniofacial skeleton for disease control can result in...
both functional and esthetic 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 probably the best predictable site for the placement of implants in comparison to frontal nasal areas. 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. Work-up should include computed tomographic images with 2 mm axial cuts while a radiographic stent is worn (Figure 14-52). 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 14-53).

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 following 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. Hyperbaric oxygen therapy has been objectively shown to reduce the risk of osteoradionecrotic complications in both the craniofacial skeleton and intraoral regions. As with any hypoxic wound, increasing oxygen tension above 40 PO2 in comparison to a nonradiated control site increases the likelihood of healing. With this increase of O2 concentration comes angioneogenesis and the subsequent effect of pluripotential 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 esthetic area. Finally, soft tissue depths 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 prior to 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. 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 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 is sizable in cases restored with standard external hex platforms and gold screws. A 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. In these circumstances screw loosening is minimized. Repeated loosening of screws should bring to mind occlusal overload, heavy contact in lateral excursions, or implant mobility.

**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. Pre-machined abutments used for screw-retained restorations can usually be replaced if they fracture.

**Porcelain Fracture**

Porcelain fracture is sometimes seen with implant prostheses owing to dynamic fatigue or contact overload. Proprioceptive feedback is not present with implant restorations and impacts during the chewing cycle should be slightly less than those of natural teeth. This can be verified using 0.001-inch stainless steel shimstock.

**Resin Base Fracture**

Resin base fractures are fairly common occurrences because of unfavorable stress distribution, occlusal overload, and a lack of proprioception. The incidence can range from 1 to 16% over 5 years. Ways to combat this problem are to reinforce the base with a cast metallic housing.

**Maintenance**

Patients restored with osseointegrated implants should receive regular and frequent follow-ups in the first year following implant placement. Factors to evaluate include:

- **Radiographic Bone Loss**
- **Screw Loosening**
- **Abutment Fracture**
- **Porcelain Fracture**
- **Resin Base Fracture**

**FIGURE 14-52** Stent (A) and computed tomography scan showing site selection (B) for implant placement into temporal bone.

**FIGURE 14-53** Facial and intraoral prosthesis anchored with two zygoma and three endosseous implants.
include bone loss, mobility, and pain. Clinical examination should include light percussion and gentle evaluation of soft tissue, which may include a standardized peri-implant probing using nonmetallic standardized force probes. 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 14-54). 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. Since a perimucosal seal exists between the implant and abutment and tissue, it is not suggested that cemented restorations be removed routinely as this may jeopardize the integrity of the restoration and surrounding tissues. However, if the restoration is retrievable, the prosthesis and/or attachment should be removed every 18 to 24 months for débridement, inspection, and polishing. 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 colleagues and remain one of the standards in long-term evaluation of dental implants. Recently additional criteria have been added for the assessment of hard and soft tissue responses. Marginal bone loss of < 4 mm or probing depth of < 4 mm and a crevicular fluid flow rate of < 2.5 mm are considered indicators of success. Mobility, if present, should be tested on an individual basis to best assess a true measure. Therefore, removing the prosthesis (especially if it is splinted with other implants) and gently percussioning 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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Part 3

**Maxillofacial Infections**
Principles of Management of Odontogenic Infections

Thomas R. Flynn, DMD

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 31 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 above, 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 one’s knowledge of the relevant anatomy and surgery fresh, 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 as follows:

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 will examine each of these principles in order and discuss and relate 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 above. 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, contents, and relations of the various anatomic deep spaces that are likely to be invaded by odontogenic infections are described in Tables 15-1 and 15-2.

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 cranial contents. 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 due to swelling or trismus can be classified as having moderate severity. Such anatomic spaces include the masticatory 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. In 1999 Flynn and colleagues devised a severity score (SS) that assigned a numerical value of 1 to 4 for involvement of each of the low, moderate, severe, or extreme severity anatomic spaces, respectively. Table 15-3 lists the severity score for each of the various deep fascial spaces. Thus, a patient with cellulitis or abscess of the right buccal (SS = 1), right pterygomandibular (SS = 2), and right lateral pharyngeal (SS = 3) spaces would have a total severity score of 6, which is the sum of the values assigned to each of the three anatomic spaces. Flynn and colleagues were able to explain by correlation analysis 66% of the length of hospital stay with a model that used the initial SS and the white blood cell count on admission.

### Table 15-1 Borders of the Deep Spaces of the Head and Neck

<table>
<thead>
<tr>
<th>Space</th>
<th>Anterior</th>
<th>Posterior</th>
<th>Superior</th>
<th>Inferior</th>
<th>Superficial or Medial*</th>
<th>Deep or Lateral†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Buccal</td>
<td>Corner of mouth</td>
<td>Masseter m., pterygomandibular space</td>
<td>Maxilla, infraorbital space</td>
<td>Mandible tissue and skin</td>
<td>Subcutaneous</td>
<td>Buccinator m.</td>
</tr>
<tr>
<td>Infraorbital</td>
<td>Nasal cartilages</td>
<td>Buccal space</td>
<td>Quadratus labii superioris m.</td>
<td>Oral mucosa</td>
<td>Quadratus labii superioris m.</td>
<td>Levator anguli oris m., maxilla</td>
</tr>
<tr>
<td>Submandibular</td>
<td>Ant. belly digastric m.</td>
<td>Post. belly digastric, stylohyoid, stylopharyngeus mm.</td>
<td>Inf. and med. surfaces of mandible</td>
<td>Digastric tendon</td>
<td>Platsysma m., investing fascia</td>
<td>Mylohyoid, hyoglossus sup. constrictor mm.</td>
</tr>
<tr>
<td>Submental</td>
<td>Inf. border of mandible</td>
<td>Hyoid bone</td>
<td>Mylohyoid m.</td>
<td>Investing fascia</td>
<td>Investing fascia</td>
<td>Ant. bellies digastric m.†</td>
</tr>
<tr>
<td>Sublingual</td>
<td>Lingual surface of mandible</td>
<td>Submandibular space</td>
<td>Oral mucosa</td>
<td>Mylohyoid m.</td>
<td>Muscles of tongue*</td>
<td>Lingual surface of mandible†</td>
</tr>
<tr>
<td>Pterygomandibular</td>
<td>Buccal space</td>
<td>Parotid gland</td>
<td>Lateral pterygoid m.</td>
<td>Inf. border of mandible</td>
<td>Med. pterygoid muscle*</td>
<td>Ascending ramus of mandible†</td>
</tr>
<tr>
<td>Submasseteric</td>
<td>Buccal space</td>
<td>Parotid gland</td>
<td>Zygomatic arch</td>
<td>Inf. border of mandible</td>
<td>Ascending ramus of mandible*</td>
<td></td>
</tr>
<tr>
<td>Lateral pharyngeal</td>
<td>Sup. and mid. pharyngeal constrictor mm.</td>
<td>Carotid sheath and scalene fascia</td>
<td>Skull base</td>
<td>Hyoid bone</td>
<td>Pharyngeal constrictors and retropharyngeal space*</td>
<td></td>
</tr>
<tr>
<td>Retropharyngeal</td>
<td>Sup. and mid. pharyngeal constrictor mm.</td>
<td>Alar fascia</td>
<td>Skull base</td>
<td>Fusion of alar and prevertebral fasciae at C6-T4</td>
<td>—</td>
<td>Carotid sheath and lateral pharyngeal space*</td>
</tr>
<tr>
<td>Pretracheal</td>
<td>Sternothyroid-thyrohyoid fascia</td>
<td>Retropharyngeal space</td>
<td>Thyroid cartilage</td>
<td>Superior mediastinum</td>
<td>Sternothyroid-thyrohyoid fascia</td>
<td>Visceral fascia over trachea and thyroid gland</td>
</tr>
</tbody>
</table>

Adapted from Flynn TR.®

ant. = anterior; inf. = inferior; lat. = lateral; m. = muscle; mm. = muscles; med. = medial; mid. = middle; post. = posterior; sup. = superior.

*Medial border; †lateral border.
### Table 15-2  Relations of Deep Spaces in Infections

<table>
<thead>
<tr>
<th>Space</th>
<th>Likely Causes</th>
<th>Contents</th>
<th>Neighboring Spaces</th>
<th>Approach for Incision and Drainage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Buccal</td>
<td>Upper bicuspsids</td>
<td>Parotid duct</td>
<td>Infraorbital</td>
<td>Intraoral (small)</td>
</tr>
<tr>
<td></td>
<td>Upper molars</td>
<td>Ant. facial a. and v.</td>
<td>Pterygomandibular</td>
<td>Extraoral (large)</td>
</tr>
<tr>
<td></td>
<td>Lower bicuspsids</td>
<td>Transverse facial a. and v.</td>
<td>Infratemporal</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Buccal fat pad</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infraorbital</td>
<td>Upper cuspid</td>
<td>Angular a. and v.</td>
<td>Buccal</td>
<td>Intraoral</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Infraorbital n.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Submandibular</td>
<td>Lower molars</td>
<td>Submandibular gland</td>
<td>Sublingual</td>
<td>Intraoral</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Facial a. and v.</td>
<td>Submental</td>
<td>Extraoral</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lymph nodes</td>
<td>Lateral pharyngeal</td>
<td>Intraoral-extraoral</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Submental</td>
<td>Lower anteriors</td>
<td>Ant. jugular v.</td>
<td>Submandibular</td>
<td>Extraoral</td>
</tr>
<tr>
<td></td>
<td>Fracture of symphysis</td>
<td>Lymph nodes</td>
<td>(on either side)</td>
<td></td>
</tr>
<tr>
<td>Sublingual</td>
<td>Lower bicuspsids</td>
<td>Sublingual glands</td>
<td>Lateral pharyngeal</td>
<td>Intraoral-extraoral</td>
</tr>
<tr>
<td></td>
<td>Lower molars</td>
<td>Whaton's ducts</td>
<td>Visceral (trachea and esophagus)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Direct trauma</td>
<td>Lingual n.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sublingual a. and v.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pterygomandibular</td>
<td>Lower third molars</td>
<td>Mandibular div. of trigeminal n.</td>
<td>Buccal</td>
<td>Intraoral</td>
</tr>
<tr>
<td></td>
<td>Fracture of angle of mandible</td>
<td>Inf. alveolar a. and v.</td>
<td>Lateral pharyngeal</td>
<td>Intraoral-extraoral</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Submasseteric</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Deep temporal</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Parotid</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Peritonsillar</td>
<td></td>
</tr>
<tr>
<td>Submasseteric</td>
<td>Lower third molars</td>
<td>Masseteric a. and v.</td>
<td>Buccal</td>
<td>Intraoral</td>
</tr>
<tr>
<td></td>
<td>Fracture of angle of mandible</td>
<td></td>
<td>Pterygomandibular</td>
<td>Intraoral-extraoral</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Superf. temporal</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Submasseteric</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Deep temporal</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Parotid</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Peritonsillar</td>
<td></td>
</tr>
<tr>
<td>Infratemporal and deep temporal</td>
<td>Upper molars</td>
<td>Pterygoid plexus</td>
<td>Buccal</td>
<td>Intraoral</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Internal maxillary a. and v.</td>
<td>Superf. temporal</td>
<td>Extraoral</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mandibular div. of trigeminal n.</td>
<td>Inf. petrosal sinus</td>
<td>Intraoral-extraoral</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Skull base foramina</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Superfical temporal</td>
<td>Upper molars</td>
<td>Temporal fat pad</td>
<td>Buccal</td>
<td>Intraoral</td>
</tr>
<tr>
<td></td>
<td>Lower molars</td>
<td>Temporal branch of facial n.</td>
<td>Deep temporal</td>
<td>Extraoral</td>
</tr>
<tr>
<td>Lateral pharyngeal</td>
<td>Lower third molars</td>
<td>Carotid a.</td>
<td>Pterygomandibular</td>
<td>Intraoral-extraoral</td>
</tr>
<tr>
<td></td>
<td>Tonsillar infection in neighboring spaces</td>
<td>Internal jugular v.</td>
<td>Submandibular</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vagus n.</td>
<td>Sublingual</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cervical sympathetic chain</td>
<td>Peritonsillar</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Retropharyngeal</td>
<td></td>
</tr>
</tbody>
</table>

Adapted from Flynn TR.4

*a = artery; div. = division; inf. = inferior; n = nerve; superf. = superficial; v = vein.*
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 to the current signs and symptoms of swelling, pain, trismus, and airway compromise. In their study of hospitalized odontogenic infections, Flynn and colleagues found that the number of days of swelling prior to admission correlated negatively with the initial severity score. This is probably because patients with more severe and rapidly progressive infections were frightened enough to seek hospital care early on.

Odontogenic infections generally pass through three stages before they resolve, the characteristics of which are listed in Table 15-4. 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 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.

<table>
<thead>
<tr>
<th>Table 15-4 Stages of Infection</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Characteristic</strong></td>
</tr>
<tr>
<td>Duration</td>
</tr>
<tr>
<td>Pain</td>
</tr>
<tr>
<td>Size</td>
</tr>
<tr>
<td>Localization</td>
</tr>
<tr>
<td>Palpation</td>
</tr>
<tr>
<td>Appearance</td>
</tr>
<tr>
<td>Skin quality</td>
</tr>
<tr>
<td>Surface temperature</td>
</tr>
<tr>
<td>Loss of function</td>
</tr>
<tr>
<td>Tissue fluid</td>
</tr>
<tr>
<td>Level of malaise</td>
</tr>
<tr>
<td>Degree of seriousness</td>
</tr>
<tr>
<td>Predominant bacteria</td>
</tr>
</tbody>
</table>

The severity score for a given patient is the sum of the severity scores for all of the spaces involved by cellulitis or abscess, based on clinical and radiographic examination.
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 are associated with increased mortality. It can rapidly result in necrosis of large amounts of muscle, subcutaneous tissue, and skin, resulting in severe reconstructive defects (Figure 15-1). 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 15-2). Soon thereafter the skin may become anesthetic. Thereafter frank necrosis occurs.

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. Repeated surgical débridement is the rule, not the exception. Hyperbaric oxygen therapy may also be of benefit.

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, which also straightens the airway and may allow 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 15-3).

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 15-4). If the suspected site of infection is touched with the mirror or tongue blade, acute pain may be elicited, especially as compared to the opposite side. The patient’s report of pain should be distinguished from the gagging that is likely to occur.

Various clinical tests have been proposed with the aim of predicting difficult intubation. The Mallampati test has been correlated with difficult intubation by its
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initial proponent, as have trismus of less than 20 mm and decreased thyromental distance.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.

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. On the other hand, 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 15-5). In a prospective study Miller and colleagues found 89\% accuracy, 95\% sensitivity, and 80\% specificity in identifying “drainable pus” by the combined use of contrast enhanced CT and clinical examination.\textsuperscript{13} 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.\textsuperscript{13}

Step 2: Evaluate Host Defenses

Immune System Compromise

Table 15-5 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 bloodstream, 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.

<table>
<thead>
<tr>
<th>Table 15-5 Factors Associated with Immune System Compromise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes</td>
</tr>
<tr>
<td>Steroid therapy</td>
</tr>
<tr>
<td>Organ transplants</td>
</tr>
<tr>
<td>Malignancy</td>
</tr>
<tr>
<td>Chemotherapy</td>
</tr>
<tr>
<td>Chronic renal disease</td>
</tr>
<tr>
<td>Malnutrition</td>
</tr>
<tr>
<td>Alcoholism</td>
</tr>
<tr>
<td>End-stage AIDS</td>
</tr>
</tbody>
</table>
This disease also appears to decrease host resistance to more severe odontogenic infections such as necrotizing faciitis and deep fascial space infections.

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 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. On the other hand, 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 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

Systemic Reserve

The host response to severe infection can place a severe 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, due to the demands placed on the elderly patient’s physiology.15

In several studies, the white blood cell count at admission has been a significant predictor of the length of hospital stay.6,16 Therefore, evaluation of leukocytosis is 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 15-6 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 turor, and dry mucous membranes, dehydration can be assessed in the presence of normal serum creatinine by an elevated urine specific gravity (over 1.030) or an elevated blood urea nitrogen (BUN), which indicates prerenal azotemia.

Infections in deep spaces that have a severity score of 2 or greater (see Table 15-3) can hinder access to the airway for intubation by causing trismus, directly compress the airway by swelling, or threaten vital structures directly. Thus, an odontogenic infection involving the masticator space, the perimandibular spaces, or deeper spaces indicates hospital admission.

Occasionally general anesthesia is required for patient management due 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.

Table 15-6 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 |
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 importantly, 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, was made possible by their changed surgical policy of immediate establishment of airway security by early 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%. 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 15-7 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 more complicated 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 mentally handicapped individual.

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.

### Table 15-7 When to Go to the Operating Room

<table>
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<th>Indication</th>
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<tr>
<td>To establish airway security</td>
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<tr>
<td>Moderate to high anatomic severity</td>
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<tr>
<td>Multiple space involvement</td>
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<tr>
<td>Rapidly progressing infection</td>
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<tr>
<td>Need for general anesthesia</td>
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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 15-6 illustrates the appropriate locations for extraoral incision placement for drainage of the various anatomic deep spaces. In addition a vertical incision over the pterygomandibular raphe can be used to drain the lateral pharyngeal space as well as the anterior compartment of the submandibular space, as illustrated in Figure 15-7. 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. 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 15-6, 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. The resulting pathway for a through-and-through drain in the submandibular space is illustrated in Figure 15-8.

The advantages of through-and-through drainage are the provision of two pathways for the egression 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 15-9.

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 15-10, 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. 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, and after several days they may cause exudation due to foreign body reaction alone.
Part 3: Maxillofacial Infections

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.\textsuperscript{17–19} The expectant approach to management of severe odontogenic infections has not been supported by empiric investigation.

The alternative strategy, successfully demonstrated by Williams and Guralnick, is the immediate establishment of airway security as necessary, and aggressive early surgical intervention.\textsuperscript{2} Identification of an abscess is not required before surgical intervention. The approach by Williams and Guralnick 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 34 patients hospitalized with severe odontogenic infections, Flynn and colleagues performed surgical drainage on all patients as soon as possible after admission.\textsuperscript{6} In none of their cases did incision and drainage seem to hasten the spread of infection. The need for reoperation was not significantly different between those patients in whom abscess and those in whom cellulitis was found.\textsuperscript{6}

Culture and Sensitivity Testing

Infections that present in the low severity anatomic spaces (see Table 15-3) 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

\textbf{FIGURE 15-8}  \textit{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.}\textsuperscript{31}

\textbf{FIGURE 15-9}  \textit{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.}\textsuperscript{31}
incision and drainage, and empiric 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, since 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 \( \alpha \)-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 \textit{Klebsiella pneumoniae} in diabetes, methicillin-resistant \textit{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 immune compromised, 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.

**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.

Initial temperature has been shown to be a significant predictor of the length of hospital stay with severe odontogenic infections.\(^6,20\) 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 3,100 mL. This would translate to a required intravenous infusion rate of approximately 130 mL per hour, assuming no oral intake and no other extraordinary fluid losses.\(^21\)

The next approach to controlling fever is usually taken by 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. 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.

Fever also increases metabolic demand by 5 to 8% per degree of fever per day. Therefore, it may be necessary to supplement the infected patient’s oral intake, with 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.

**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. The empiric antibiotics of choice for odontogenic infections are, however, listed in Table 15-8.

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. In one pediatric study pain and swelling were significantly better at 7 days with amoxicillin. In all of the above referenced studies the involved tooth or teeth were treated with extraction or root canal therapy. Incision and drainage was performed as necessary. Therefore, penicillin continues to be a highly effective antibiotic for uncomplicated odontogenic infections, owing to its low cost and low incidence of unwanted side effects.

For severe infections warranting hospital admission the antibiotics of choice for odontogenic infections do not include penicillin. In 1999 Flynn and colleagues found a 26% failure rate of penicillin when used empirically in a series of 34 hospitalized cases of odontogenic infection. Of the 31 patients who were placed on penicillin (3 were allergic), 8 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. Therefore, clindamycin has become the empiric antibiotic of choice for 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 *S. 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

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<th>Table 15-8 Empiric Antibiotics* of Choice for Odontogenic Infections</th>
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<td><strong>Severity of Infection</strong></td>
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<td>Outpatient</td>
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<td>Penicillin allergy:</td>
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<td>Inpatient</td>
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*Empiric antibiotic therapy is used before culture and sensitivity reports are available. Cultures should be taken in severe infections that threaten vital structures. IV = intravenous.
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, on the other hand, 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.

Few cephalosporins are able to cross the blood-brain barrier. Some third-generation cephalosporins, such as ceftadizime, can do so. In addition, ceftadizime is effective against the oral streptococci and most oral anaerobes. Among the cephalosporins, therefore, ceftadizime is the alternative antibiotic of choice.

A new fluoroquinolone antibiotic, moxifloxacin has great promise in the treatment of head and neck infections. Its spectrum against oral streptococci and anaerobes is excellent. Its absorption is virtually complete via either the oral or intravenous routes, and it penetrates bone readily. Therefore, this new antibiotic may become 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. 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, and should be used with caution in pregnancy.

**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 chemical qualities of ingested foods, and the basic intestinal tract. 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.

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.

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. Since the serum elimination half-life of penicillin G is 0.5 hours, after 3 hours (6 half-lives) the serum concentration will be approximately 0.3 µg/mL. Since 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. Since 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.

Another practical matter that must always be considered in administering antibiotics is their cost, especially their cost to the patient. When a patient does not have prescription drug insurance coverage, such as in 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 2003 the retail cost of 1 week’s supply of penicillin V 500 mg taken 4 times per day was US$12.09 at a large pharmacy chain in the northeastern United States. The retail cost of 1 week’s supply of clindamycin 300 mg taken 4 times per day was US$58.59. 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 he or she may be forced to either take reduced amounts of the antibiotic, to extend the dosage interval, or to forgo 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 as compared 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 required. 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’s stains and/or culture reports should be available, which may provide some guidance as to the appropriateness of the empiric antibiotic therapy.

If the above 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 15-9. 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 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 15-11 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 15-11A, 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 15-11B, there is continued oropharyngeal swelling surrounding the endotracheal tube at 5 postoperative days. On the other hand 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 15-12). 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.  
5. After waiting for any coughing to subside, the oxygen supply is disconnected.

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**Table 15-9 Causes of Treatment Failure**

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<th>Cause of Treatment Failure</th>
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<td>Inadequate surgery</td>
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<td>Depressed host defenses</td>
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<td>Foreign body</td>
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<td>Antibiotic problems</td>
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<td>Patient noncompliance</td>
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<td>Drug not reaching site</td>
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<td>Drug dosage too low</td>
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<td>Wrong bacterial diagnosis</td>
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<td>Wrong antibiotic</td>
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Adapted from Peterson LJ.32
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 then 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, then extubation can be completed.

8. If the patient is not able to breathe around the tube changer, then 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 it may be due 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 where 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 foreign body, epithelium, tumor, infection, and distal 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 15-13. 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, then 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.

If a thorough search for previously undetected pathogens turns up negative or if another cause for treatment failure cannot be found, then 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 15-10. Because of the necessary time delay in obtaining culture and sensitivity reports, it is occasionally necessary to change from one empiric antibiotic to another. Ideally the surgeon should consider another of the empiric antibiotics of choice listed in Table 15-8. The input of an infectious disease consultant may also be valuable in this situation.

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.

Acknowledgment

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References


Sinus Infections

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Chronic sinusitis is a disease with high prevalence in the American population, affecting up to 13.4% of the population and accounting for almost 2% of all ambulatory diagnoses rendered. This condition is important not only because of its frequency but because complications of sinusitis may carry severe neurologic, ophthalmologic, and systemic consequences. Therefore it is incumbent on all practitioners, particularly those who manage structures of the maxillofacial complex, to be familiar with the features of sinonasal disease. Technologic advances in diagnostic imaging, endoscopy, and surgical instrumentation have revolutionized the diagnosis and treatment of sinusitis. Furthermore, both clinical experience and basic science knowledge have modified our perspective of sinusitis such that we now understand it as an inflammatory disorder, rather than a purely infectious process. This chapter attempts to synthesize a framework for understanding the etiology, clinical presentation, diagnosis, medical treatment, and surgery for sinonasal inflammatory disease. These elements are discussed in the context of our current knowledge base and the latest technologic innovations.

The diagnosis and management of sinusitis has traditionally been based on patient symptomatology and plain film imaging. The advent of sinonasal endoscopy and the wide availability of computed tomography (CT) have enhanced diagnostic accuracy, treatment planning, and surgical capabilities. Prior to these developments, management primarily consisted of antibiotic therapy, with surgery (often performed via facial incisions) reserved for complications. Endoscopy and CT have permitted elective management of sinusitis for symptomatic improvement and the prevention of complications. Advances in our understanding of microbiology, allergy, and pharmacology have complemented these modalities.

The first fiber-optic nasal examination was performed by Hirshman using a modified cystoscope. Instrumentation was then refined after World War II, permitting the development of smaller scopes with improved illumination. Hopkins designed a series of rigid endoscopes in the early 1950s. They were relatively small in diameter and had wide field high-contrast optics and bright illumination. This technology was used by Professor W. Messerklinger of Graz, Austria, for systematic nasal airway evaluation. Importantly, Messerklinger observed that primary inflammatory processes of the lateral nasal wall, particularly the middle meatus, resulted in secondary disease of the maxillary and frontal sinuses. This led to the definition of the osteomeatal complex (OMC; Figure 16-1) as the site of common drainage for the maxillary, frontal, and anterior ethmoid sinuses. Messerklinger demonstrated that even small anatomic variations or inflammatory processes in this location may impair ventilation and drainage of the adjacent sinuses, with subsequent development of significant inflammatory disease in these regions. This observation led him to employ endoscopes for the surgical management of sinusitis such that disease processes affecting the natural sinus drainage pathways could be addressed. Particularly, he showed that even limited surgical procedures directed toward the OMC and anterior ethmoid sinuses can result in improvement of ventilation and drainage of the frontal and maxillary sinuses.

During the 1980s Stammberger, also of Graz, and Kennedy, in the United States, further refined and popularized these techniques. Since that time nasal endoscopy has been employed in the surgical management of sinonasal neoplasms as well as a multitude of both skull base and orbital pathologies. Although indications do exist for external approaches to the paranasal sinuses, endoscopic approaches are typically first line in the surgical management algorithm. Recent advances in surgical instrumentation have included the development of angled forceps, drills, and telescopes. Additionally, the availability of stereotactic navigation imaging has permitted more comprehensive surgery to be performed safely.

The practices of optimal medical therapy,
both pre- and postoperatively, and meticulous postoperative care have further improved our treatment success. The remainder of this chapter highlights the state of the art in the diagnosis and management of sinusitis.

Clinical Presentation

Sinusitis is a clinical diagnosis that is confirmed by physical examination, including nasal endoscopy, and radiographic imaging. The Task Force on Rhinosinusitis sponsored by the American Academy of Otolaryngology—Head and Neck Surgery has established criteria to define a history consistent with sinusitis. These are based on patient signs and symptoms and are grouped into major and minor criteria, as outlined in Table 16-1. The presence of two or more major factors, or one major plus at least two minor factors, is considered a “strong history for sinusitis.” Of note, purulent nasal drainage alone is considered diagnostic for sinusitis. This finding is clearly visible on nasal endoscopy and may manifest as purulence in the middle meatus or within a sinus cavity itself. This is described in greater detail below under “Diagnosis.” It also deserves clarification that fever is only considered a major factor in the setting of acute sinusitis but is otherwise a minor factor. Although the term sinusitis is commonly in use, the process may more accurately be described by the term rhinosinusitis because the nasal and sinus mucosal surfaces are contiguous and it would be impossible to have sinusitis without a coexisting rhinitis. The terms are used interchangeably in the present chapter.

Rhinosinusitis is classified as either acute, subacute, recurrent acute, or chronic. The distinctions are based solely upon the time course or temporal pattern in which the patient has symptoms. Patients may also have episodes of recurrent acute sinusitis superimposed on a baseline state of chronic sinusitis. A diagnosis of acute sinusitis requires that criteria satisfying a strong history for sinusitis are present for 1 to 4 weeks. Patients should exhibit signs and symptoms for at least 1 week before sinusitis is diagnosed because sinusitis typically involves a bacterial process, and the vast majority of patients with symptoms for < 1 week have simple viral upper respiratory infections. Strictly speaking, however, a viral upper respiratory infection is synonymous with an acute viral rhinosinusitis. Subacute sinusitis requires that these

<table>
<thead>
<tr>
<th>Table 16-1 Factors Associated with a History of Rhinosinusitis*</th>
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<tbody>
<tr>
<td><strong>Major Factors</strong></td>
</tr>
<tr>
<td>Facial pain/pressure</td>
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<tr>
<td>Facial congestion/fullness</td>
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<tr>
<td>Nasal drainage/discharge</td>
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<tr>
<td>Postnasal drip</td>
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<tr>
<td>Nasal obstruction/blockage</td>
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<tr>
<td>Hyposmia/anosmia (decreased or absent sense of smell)</td>
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<tr>
<td>Fever (acute sinusitis only)</td>
</tr>
<tr>
<td>Purulence on nasal endoscopy (diagnostic by itself)</td>
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</tbody>
</table>

*Either two major factors, or one major and two minor, are required for a diagnosis of rhinosinusitis. Purulence on nasal endoscopy is diagnostic. Fever is a major factor only in the acute stage.
criteria have existed for 4 to 12 weeks, and in chronic sinusitis the criteria are present for at least 12 weeks. In recurrent acute sinusitis, episodes last < 4 weeks, but the patient is asymptomatic between episodes. Rhinosinusitis may also have significant fungal components and may be influenced by environmental, general host, and local host factors (see below).

Etiology

Anatomy and Physiology of the Nose and Paranasal Sinuses

The pathophysiology of sinusitis must be understood in the context of the normal anatomy and physiology of the nose and paranasal sinuses. The paranasal sinuses are formed early in development as evaginations of respiratory mucosa from the nose into the facial bones. Cavity formation begins in utero, and pneumatization continues into early adolescent life. The ethmoid sinus develops into a bony labyrinth of 3 to 15 small air cells on each side. In contrast, the other sinus cavities develop as a single bony cavity on each side of the facial skeleton, although variations may exist. The ostium of each sinus represents the point at which outpouching initiated.

The lateral nasal wall on each side is lined by three turbinate bones designated as inferior, middle, and superior (Figure 16-2). The space under each is known as either the inferior, middle, or superior meatus, respectively. The OMC is a space within the middle meatus into which the maxillary, anterior ethmoid, and frontal sinuses drain (see Figure 16-1). It is this region where pathology such as anatomic variation or inflammatory disease is most likely to impair sinus ventilation and drainage, resulting in the development of sinusitis. The posterior ethmoid sinuses drain into the superior meatus. The sphenoid sinus drains into an area known as the sphenethmoidal recess, which lies at the junction of the sphenoid and ethmoid bones in the superior portion of the posterior nasal cavity (see Figure 16-2).

The remaining discussion details the anatomy of the middle meatus and the OMC, for this is the critical region in the development of sinusitis. These structures are mainly derived from the ethmoid bone, a T-shaped structure, of which the vertical part contributes to the nasal septum, middle (and superior) turbinate, ethmoid air cell system, and the lateral nasal wall (see Figure 16-1). The horizontal portion forms the cribriform plate of the skull base. The uncinate is a sickle-shaped process of ethmoid bone that lies along the lateral nasal wall. The cleft-like space lateral to this structure is known as the infundibulum, and this is the region into which the maxillary sinus drains. The medial opening of the infundibulum, where it opens into the middle meatus, is known as the hiatus semilunaris. The ethmoid bulla is a prominence of anterior ethmoid air cell(s) along the lateral nasal wall that hangs just superior to the infundibulum. The drainage tract from the frontal sinus courses inferiorly from the sinus medial to the medial orbital wall, lateral to the middle turbinate, and anterior to the ethmoid bulla. This tract, known as the frontal recess, is highly variable and is often lined with variant anterior ethmoid air cells. It is apparent that even minimal inflammatory disease in the OMC can impair sinus ventilation and drainage of the adjacent ethmoid, maxillary, and frontal sinuses.

The paranasal sinuses and the majority of the nasal cavity itself are lined with pseudostratified columnar ciliated epithelium (respiratory type). The cilia suspend a mucous blanket, which is secreted by goblet cells in the mucous membrane (Figure 16-3). The cilia propel this blanket in a predetermined direction (Figure 16-4), in a manner similar to the "mucociliary escalator" of the tracheobronchial tree. This phenomenon is important because in the
paranasal sinuses cilia propel mucus toward the natural ostium. This means that in the maxillary sinus cilia must propel mucus against gravitational forces. Any surgical procedures intended to promote sinus drainage must, however, be addressed to the natural ostium.

One or more of the following local factors may create a predisposition for sinusitis: (1) mechanical obstruction of mucociliary flow, particularly in the OMC region, (2) defects in ciliary capability to propel the mucus blanket, and (3) abnormal quantity or quality of secretions. A combination of these factors results in the development of sinusitis by allowing stasis of secretions, resulting in bacterial colonization and infection with associated inflammation. In turn, this results in further ostial obstruction, stasis, and exacerbation of the inflammatory process. Furthermore, impairment of sinus ventilation creates acidic anaerobic conditions that cause ciliary damage and ineffective mucus clearance. A variety of local and systemic disease processes may promote sinusitis by influencing mucociliary clearance at the anatomic, histologic, immunologic, and biochemical levels (Figure 16-5).

**Anatomic Factors**

Post-traumatic, congenital, or iatrogenic conditions involving the craniofacial skeleton may physically obstruct sinus ostia, contributing to the development of sinusitis. These may include abnormalities of the nasal septum, such as spurs and deviations, or variants of the middle turbinate including turbinate pneumatization (concha bullosa) or hypertrophy. These entities may narrow the middle meatal cleft, thus impairing mucus out-

The presence of accessory ostia, either congenital or iatrogenic, may promote the development of chronic sinusitis by the mucus recirculation phenomenon. This is most apparent in the maxillary sinus. Mucus is physiologically propelled around accessory ostia and toward the natural ostium (see Figure 16-4). However, the presence of an accessory ostium allows mucus reentry into the sinus lumen. Earlier surgical techniques attempting to augment sinus ventilation and drainage included the creation of a “nasoantral window” in the inferior meatus, with the rationalization that this would permit drainage in a gravity-dependent manner. This approach, however, is suboptimal because cilia attempt to direct mucus around the iatrogenic ostium to the natural one.

In children adenoid hypertrophy is a frequent underlying cause of sinus infections. This impairs the outflow of secretions from the posterior nasal cavity into the nasopharynx. The diagnosis is suspected in children presenting with nasal obstruction, mouth breathing, and rhinorrhea. A nasal foreign body may also be observed in children with these findings and may either mimic or be the cause of rhinosinusitis. The
Classic finding in these patients is unilateral foul-smelling rhinorrhea.

Miscellaneous anatomically related conditions that may increase the risk for developing sinusitis include the presence of nasotracheal or nasogastric tubes and barotrauma. Nasal intubation may impair sinunasal drainage, but other mechanisms may be involved as studies have observed sinusitis on the side opposite tube placement.6 Barosinusitis results from tissue edema induced by rapidly changing air pressures during diving, air travel, or hyperbaric oxygen therapy. Any preexisting anatomic narrowing of the OMC predisposes to barosinusitis as air pressure within the sinus cannot effectively equilibrate with the ambient pressure during ascent or descent.

Inflammatory Conditions

The most common inflammatory condition that predisposes to sinusitis is a viral upper respiratory infection, or the common cold, during which approximately 80% of patients have decreased patency of the maxillary sinus ostium secondary to tissue edema.7 Mucociliary clearance is also impaired secondary to destruction and shedding of ciliated epithelial cells. Influenza virus appears to be the most destructive in this regard.8 Rhinovirus is the most common cause, with over 100 serotypes identified, and respiratory syncytial virus, parainfluenza virus, and coronavirus may also be implicated. Regardless of the offending virus, conditions of ostial obstruction and impaired mucociliary flow permit bacterial overgrowth.

Dental conditions may cause maxillary sinusitis secondary to direct extension of infectious or inflammatory processes through the apices of maxillary teeth into the sinus. Infection following a sinus lift procedure appears to be more likely when there is preexisting osteomeatal inflammation. Dental implant and root canal materials may also extrude into the sinus, initiating inflammation via a foreign body reaction or by acting as a nidus for bacterial colonization. Specifically, paraformaldehyde-containing pastes have been implicated.9

Chronic inflammatory disorders affecting the respiratory mucosa appear to correlate with sinusitis. Patients with allergic rhinitis frequently exhibit sinus mucosal disease, and, conversely, a large proportion of patients with chronic sinusitis have positive responses to allergy skin testing. This is thought to be an immunoglobulin E (IgE)-mediated (type I) immediate hypersensitivity, with cell-mediated late-phase responses. Our understanding of the mechanistic relationship between allergy and sinusitis is far from complete, however, and the exact concordance between the disorders is unknown.10 Nonetheless, it appears that atopic patients have an underlying predisposition for mucosal inflammation. Ostial obstruction and impaired mucociliary flow from allergen exposure may result in bacterial overgrowth and exacerbation of the inflammatory process. The effect of allergic disease persists even after surgical procedures that enlarge the natural sinus ostia. In fact, surgery may increase mucosal inflammation by enhancing allergen exposure to susceptible mucosa within the sinus, despite anatomic improvements in the drainage pathway.

Patients with asthma are also predisposed to sinusitis secondary to a generalized reactivity of the respiratory mucosa. Again, the exact relationship between these entities is unclear. However, there is evidence that asthma symptoms may even improve after surgical management of comorbid chronic sinusitis.11,12 One atopic syndrome that deserves discussion is the Aspirin-sensitivity triad (Samter’s triad). These patients develop asthma in association with sinusitis and nasal polyposis, and Aspirin precipitates acute bronchospasm. Overall, it is estimated that up to 25% of patients with nasal polyposis develop bronchoconstriction in response to Aspirin administration.13 Aspirin-sensitivity triad is a defect of arachidonic acid metabolism and may have a genetic basis.14

**FIGURE 16-5** Sinusitis is a multifactorial process, of which bacterial infection is a component.
Over 100 chemicals have been found to cause nasal irritation, many of which are found in cigarette smoke. Pollutants may contribute to sinusitis through several mechanisms. Deposition of irritant particles in the mucous blanket during respiration can increase the relative concentration to which the mucous membrane is exposed, resulting in direct chemical and physical irritation, which subsequently promotes the inflammatory process. The irritant effects of these chemicals may also induce neurogenic inflammation through vasodilation, tissue edema, and leukocyte influx. Specifically, neuropeptides such as substance P from unmyelinated sensory fibers have been implicated. Pollutants may also impair mucociliary clearance through alterations in mucus viscosity, inhibition of ciliary function, and increases in epithelial permeability. The typical chemical components of outdoor pollution have been shown to increase neutrophil counts in nasal lavage specimens. A study in Finland also correlated the increase in nasal polyposis and frontal sinusitis with air pollution. These studies provide circumstantial but objective evidence that pollutants play a significant role in the increasing prevalence of chronic sinusitis.

Recently there has been investigation into a possible role for gastroesophageal reflux disease (GERD) in sinonasal inflammation, particularly in the pediatric population. In fact, GERD has been associated with a multitude of inflammatory processes of the upper aerodigestive tract including esophagitis, pharyngitis, and laryngitis. Evidence for its role in sinusitis, however, is circumstantial, and many feel that it is not a significant predisposing factor. Nonetheless, GERD should be suspected in children whose inflammation appears refractory to medical and surgical management.

Bacteriology of Sinusitis

The type of bacteria involved in a sinus infection depends on multiple factors, including the immune or metabolic status of the host, the duration of the disease process, whether the infection is community or hospital acquired, and antibiotic resistance patterns. In uncomplicated acute sinusitis, Streptococcus pneumoniae and Haemophilus influenzae are the most commonly isolated pathogens; Moraxella catarrhalis may also be a significant organism, particularly in the pediatric population. Staphylococcus aureus, Streptococcus pyogenes, coagulase-negative staphylococci, anaerobes, and gram-negative organisms are found in varying proportions. The pathogenic roles of staphylococcal species in acute sinusitis are unclear as these are found near the maxillary ostium in 60% of healthy asymptomatic adults. Anaerobes, when isolated, are typically a component of a mixed bacterial infection and may be the result of an extension of a dental abscess. It should also be noted that up to 50% of patients diagnosed clinically with acute sinusitis have sterile sinus aspirates. The reason for this is unclear, but it may reflect viral or allergic processes diagnosed as bacterial sinusitis. Nosocomial acute sinusitis may be caused by nasal intubation, nasal packing, patient immobility, chronic debilitation, and/or immunosuppression. The most common species isolated in these cases is Pseudomonas, although S. aureus is also frequently isolated, and the bacteriology may be unpredictable.

Patients with chronic sinusitis typically represent a population with several months to years of symptoms who have received multiple antibiotic courses. Thus the bacterial profile in these patients differs from that of acute sinusitis. Polymicrobial infections and antibiotic-resistant organisms are often found. In general, a higher proportion of S. aureus, coagulase-negative staphylococci, gram-negative bacilli, and streptococci are isolated in addition to the typical pathogens of acute sinusitis. The roles of S. aureus and coagulase-negative staphylococci are controversial as these organisms are known to colonize the anterior nose and are less frequently isolated when the anterior nose is disinfected. Most authors agree, however, that S. aureus is a significant pathogen and should be treated when identified. Gram-negative organisms that may be isolated include Pseudomonas, Klebsiella, and Proteus. Viridans streptococci, organisms commonly found among oral flora, are observed in up to one-third of cases. Interestingly, one study identified anaerobes in 93% of specimens in children with chronic sinusitis. However, because the upper aerodigestive tract is highly colonized with anaerobes, their role in the infectious process is unclear. Postoperatively, the sinonasal mucosa is frequently colonized or infected with Pseudomonas and/or S. aureus, and patients may still be susceptible to acute exacerbations by the pathogens involved in acute sinusitis.

Role of Fungi

Much has evolved in our understanding of the role of fungi in sinusitis, and different patterns of fungal sinusitis exist. Fungal disease can be classified as noninvasive or invasive. Both fungal balls and allergic fungal sinusitis are part of the noninvasive group, although recently it has been suggested that fungus has a wider role as an active factor in the pathogenesis of eosinophilic chronic rhinosinusitis. Invasive fungal disease is typically a fulminant disease in immunocompromised individuals but can also occur occasionally as an indolent disease in patients who are immunocompetent. Fungal balls are typically seen in immunocompetent individuals with chronic (or recurrent acute) symptomatology that is often subtle and restricted to a single sinus. Patients may complain about the perception of a foul odor and occasionally report expelling fungal debris with nose blowing. Most commonly, a fungal ball consisting of Aspergillus fumigatus is found in the maxillary sinus with scant inflammatory cell
infiltration in the surrounding mucosa.\textsuperscript{30} The condition is indolent, and cure is often achieved after surgical removal of the fungus ball and assurance of patency of the natural sinus ostium.

Allergic fungal sinusitis (AFS) is a form of noninvasive fungal sinusitis seen in immunocompetent patients, who exhibit a hypersensitivity reaction to fungal organisms in the nose and sinuses. The disease typically presents with unilateral nasal polyposis and thick tenacious secretions.\textsuperscript{31} The most commonly implicated fungi are those of the Dematiaceae family,\textsuperscript{32} but \textit{Aspergillus} species are also seen. The exact pathophysiology is controversial but is thought to involve IgE-mediated (type I) responses. IgE-sensitized mast cells are activated by exposure to fungal antigens resulting in degranulation, influx of eosinophils, and exacerbation of inflammation via the release of major basic protein. Immune complex (type III) reactions involving IgG have also been identified. Patients have a severe inflammatory reaction with nasal polyposis and inspissated “allergic mucin” consisting of eosinophil breakdown products (Charcot-Leyden crystals) and fungal forms. AFS-like conditions have also been described in which mucin is observed, but fungal forms are not identified microscopically or by culture.\textsuperscript{33} Recent studies by Ponikau and colleagues and Taylor and colleagues, however, revealed that fungi can be demonstrated with increased sensitivity using novel culture and staining techniques.\textsuperscript{34,35} In fact, this group showed that fungi are present in 93\% of 101 patients with chronic sinusitis.\textsuperscript{34} This has led to the hypothesis that the fungi, themselves, may induce an eosinophilic response, and that fungi may play a prominent role in chronic sinusitis, even in the absence of frank AFS. This area of research is progressing rapidly.

Patients with AFS may present with the typical signs and symptoms of chronic sinusitis. Underlying AFS must be suspected in a chronic sinusitis patient whose course is unusually refractory to medical therapy. Additionally, advanced nasal polyposis with inspissated mucin and fungal debris may cause thinning of bone of the adjacent orbit and skull base. The goals for treatment of AFS are to eliminate the fungal antigenic load and to reestablish sinus ventilation, drainage, and mucociliary clearance. Surgery has a prominent role in these regards but must be complemented with medical therapies to both reduce inflammation and eliminate the fungal load.

Immunocompromised patients are at risk for developing fulminant invasive fungal sinusitis. This patient population is composed of diabetics, transplant patients, those receiving cancer chemotherapy, burn victims, the elderly, and patients with congenital or acquired immunodeficiency. In addition to the typical symptoms of sinusitis, patients with invasive fungal disease may present with severe pain, fever, proptosis, visual impairment, cranial neuropathy, other focal neurologic findings, seizures, and altered mental status. Invasive fungal sinusitis may begin as a noninvasive form with subsequent tissue invasion in a susceptible patient. \textit{Aspergillus} and fungi of the Mucoraceae family are often implicated, with the latter being more common in diabetics. Black necrotic eschars of the nasal mucosa are noted during nasal endoscopy, with bone destruction on CT scans. Biopsy of the border of the eschar is essential to confirm the diagnosis. Biopsy is also necessary when pale insensate mucosa is discovered in a patient with a strong history and risk factors for invasive fungal sinusitis. Treatment requires aggressive surgical débridement of infected and devitalized tissues, topical and systemic antifungal medications, and management of predisposing conditions.

The chronic indolent form of invasive fungal sinusitis is more commonly observed in immunocompetent patients and is endemic in Sudan, but it has also been observed in type II diabetics. \textit{Aspergillus flavus} is the most common organism encountered. Symptoms of chronic sinusitis are initially present, but these progress to cause visual and neurologic signs. Nasal endoscopy may reveal granulomatous inflammation.\textsuperscript{31} Bone destruction ultimately occurs. Treatment includes surgical removal of fungal debris and affected tissues, as well as systemic and local antifungal therapy.

**Genetic Disorders**

Little is known regarding genetic influences on the risk of developing sinusitis, and the exact contribution of hereditary variables is difficult to quantify given the multifactorial nature of the disease. However, recently the \textit{ADAM33} gene has been identified as being associated with the closely related disease asthma. Many of the predisposing inflammatory conditions discussed previously, particularly those involving an atopic response, also tend to cluster in families, suggesting a genetic component. Additionally, several defined congenital syndromes are associated with sinusitis. These include defects of metabolism, ciliary structure/function, and the immune system. Some of the more common pathologies with a primary genetic basis are outlined below.

Cystic fibrosis (CF) is an autosomal recessive disorder affecting epithelial transport of chloride and water via mutations in the \textit{CFTR} gene. This results in abnormally viscous secretions, which become inspissated in the lung, pancreas, and sinonasal tract, ultimately leading to chronic inflammation and fibrosis. In the sinonasal tract, patients exhibit florid polyposis and colonization with \textit{Pseudomonas}. A sweat test to detect elevated chloride levels is diagnostic and should be performed on any child presenting with nasal polyposis. Recent data also suggest that heterozygous carriers may be at increased risk for developing chronic sinusitis.\textsuperscript{36} Aggressive medical management against \textit{Pseudomonas} is necessary; treatment also includes surgery to remove...
polyps and chronically infected tissue and to provide sinus ventilation. Pulmonary disease is typically the life-limiting manifestation of CF, but in the era of lung transplantation, patients may live well into the fourth or fifth decade.

Inherited disorders of ciliary structure or function also are associated with chronic sinus disease. Kartagener’s triad is a syndrome involving sinusitis, bronchiectasis, and situs inversus. Sinus, middle ear, and pulmonary diseases are observed in nearly all cases, and male patients are usually infertile secondary to sperm immobility. These manifestations are a consequence of structural defects in the dynein arms of cilia. Light microscopy reveals a reduction in ciliary beat frequency, and structural abnormalities can be observed under electron microscopy. Primary ciliary dyskinesia (or immotile cilia syndrome) is twice as common as Kartagener’s syndrome and has similar sinopulmonary manifestations without situs inversus. These patients often live a normal lifespan with timely management of sinopulmonary infections and prophylactic measures such as avoidance of environmental pollutants.

Young’s syndrome is also associated with chronic sinusitis, lung disease, and male infertility. The etiology of male infertility, however, is secondary to obstruction of the epididymis, and sperm motility is normal. There is no association with situs inversus. Sinus and lung disease usually do not progress beyond childhood, and few require sinus surgery.

Multiple inherited immunodeficiency disorders may be associated with sinusitis. These typically involve defects of antibody-mediated immunity, particularly IgG subclass deficiency, for which the inheritance pattern is unknown. Common variable immunodeficiency (dominant or recessive), IgA deficiency (dominant), X-linked agammaglobulinemia, and complement deficiencies are among the disorders identified. The particular type of immunodeficiency involved may dictate the nature of the superinfecting organism. For example, complement defects are associated with gram-negative infections. Difficult-to-manage sinus disease should inspire an investigation into this area, including the quantitative measurement of immunoglobulins and possibly complement levels.

Diagnosis

Roles of Endoscopy and CT

Sinus infections are typically diagnosed based on clinical criteria described previously (see Table 16-1). Symptom severity and effect on quality of life can be scored on multiple different scales. Acute sinusitis is frequently diagnosed and managed by the primary care practitioner largely based on history, but recurrent acute sinusitis, chronic sinusitis, or that which has failed medical management requires endoscopic evaluation and radiographic imaging. This is important because over two-thirds of patients who meet the criteria for rhinosinusitis have negative results on endoscopy, and over 50% have negative results on CT scans.

Sinusitis can be diagnosed regardless of symptomatic criteria if pus is noted in the middle meatus during nasal endoscopy (Figure 16-6). In patients who have had surgical antrostomy, pus may be seen within the maxillary sinus. This can be cultured during the examination, with the results being useful in antibiotic selection. In addition to purulence, nasal endoscopy can detect mucosal inflammation, edema, polyposis (Figure 16-7), and anatomic variations such as a deviated septum. A recent study demonstrated that the findings of purulence, polyps, or mucosal edema correlate with sinusitis by CT, but anatomic variation was not a significant predictor. Also, negative endoscopy was a good predictor for CT scan results that were normal or indicated...
minimal disease. Overall, these results underscore the need for endoscopy in the diagnostic evaluation of cases other than isolated episodes of uncomplicated acute sinusitis.

Approximately one-third of randomly selected asymptomatic people have some mucosal changes on CT scans, but patients with symptoms and some endoscopic findings do not necessarily have positive findings on CT scans. Thus, although CT is a good predictor of moderate mucosal thickening, it probably should not be considered a gold standard for diagnosis. The decision to treat medically may be based rationally on endoscopic findings because such normal findings are associated with normal or near-normal CT results in over 75% of cases. CT is necessary, however, when surgery is anticipated, complications are suspected, or when there is a significant discrepancy between history and endoscopic examination. In these situations CT not only helps to confirm the diagnosis but also aids in surgical planning. The coronal plane provides the best view of the OMC (Figure 16-8) and can be used to detect opacification, mucosal thickening, and neo-osteogenesis, all of which are indicative of chronic inflammation. Anatomic variations such as a concha bullosa (pneumatized middle turbinate) can be also detected. Scans can additionally be obtained in the axial plane, and images may be reconstructed in three planes: coronal, axial, and sagittal. This technology allows for precise anatomic localization of disease processes and intra-operative stereotactic navigational imaging (see “Surgery,” below). It should be noted that although plain films are widely available and inexpensive, much more precise data is obtained with a coronal CT, whose use has comparable costs and radiation exposure. Although plain films may detect complete sinus opacification or air-fluid levels, chronic inflammatory disease correlates with as little as 2 mm of mucosal thickening, which cannot be identified on plain films.

In an effort to reduce both costs and radiation exposure, protocols have been designed involving lowered radiation doses. These allow adequate bony detail and do not appear to cause diagnostic errors, although soft tissue contrast is slightly reduced. For diagnostic purposes and for routine elective sinus surgery, images in the coronal plane alone are sufficient. These should be obtained at 3 mm cuts, although some centers attempt to further reduce costs by using thicker sections.

Special Considerations

Fungal Sinusitis  Fungal sinusitis, as outlined previously, may manifest in a spectrum of both invasive and noninvasive forms. Endoscopically, with noninvasive or chronically invasive disease, fungal forms may be evident (Figure 16-9), along with mucosal edema and/or polypsis. In allergic fungal sinusitis the allergic mucin that is inspissated among the nasal polyps and fungal debris has a peanut butter–like quality. Histologically this contains fungal forms, eosinophils, and Charcot-Leyden crystals (breakdown products of eosinophil granules; Figure 16-10). The mucous membranes of invasive fungal sinusitis typically contain black necrotic eschars but may be pale or gray in earlier phases. These findings are secondary to ischemic necrosis induced by fungal invasion of the mucosal vasculature and may extend to the gingivae and palate. Suspicion of invasive fungal sinusitis requires biopsy confirmation (Figure 16-11), followed by aggressive débridement of infected and devitalized tissues.

Typically, noninvasive fungal disease appears on CT scans as areas of increased density within the sinuses (Figure 16-12).
mucus retention. Bone thinning or destruction may be observed from the expansile nature of the inflammatory process or owing to tissue invasion.

Complications of Sinusitis Because of the proximity of the paranasal sinuses to the eyes and brain, complications of sinusitis are divided into two broad categories: orbital and intracranial. Infection extending into the orbit and associated soft tissues usually originates from the ethmoids and occurs through one of two mechanisms: (1) direct extension through the orbital wall or (2) retrograde spread through veins between the sinuses and the orbit. Lymphatic spread is not a significant factor because lymphatics are absent in the orbit. The spectrum of orbital complications of sinus infections has been classified in five categories (Figure 16-13).\textsuperscript{53}

Preseptal cellulitis, or periorbital cellulitis, is edema and inflammation of the skin and muscle anterior to the orbital septum secondary to impairment of venous drainage from these tissues.\textsuperscript{54} There are no visual symptoms, restrictions of extraocular movement, or signs of chemosis as the infection has not invaded the intraconal soft tissues. In contrast, orbital cellulitis indicates edema and inflammation of the intraconal contents resulting in ophthalmoplegia, proptosis, and chemosis secondary to obstruction of venous outflow via the ophthalmic veins.

Subperiosteal abscess (Figure 16-14) is a collection of purulent material between the bony orbital wall and the orbital periosteum, usually from direct spread of acute infection in the ethmoid sinuses through the lamina papyracea. Depending on the size of the abscess and the associated mass effect, and the degree of inflammation, ocular muscles and visual acuity are variably affected. Progression of this subperiosteal process may subsequently result in an abscess of the orbital tissues. An orbital abscess may also occur with progression of orbital cellulitis. At this stage, restriction of extraocular mobility, proptosis, chemosis, and visual loss are often observed. When orbital cellulitis or subperiosteal or orbital abscesses are suspected, contrast-enhanced CT examination is necessary.\textsuperscript{55}

Cavernous sinus thrombosis is a grave complication that occurs from direct extension or retrograde thrombophlebitis (via the ophthalmic vein) of ethmoid or sphenoid infections.\textsuperscript{56,57} In addition to restriction of extraocular mobility, proptosis, chemosis, and visual loss, cranial neuropathies and signs of meningitis may be observed. Given the frequency of ocular findings, this entity is often categorized with the orbital complications of sinusitis, but if this or another intracranial complication is suspected, magnetic resonance imaging must be performed. Lumbar puncture may also be indicated.

Intracranial complications occur less frequently than do orbital complications and are most commonly related to the frontal or sphenoid sinuses (Figure 16-15).\textsuperscript{58,59} These complications may occur via either direct spread or retrograde thrombophlebitis. Pott’s puffy tumor is a collection of pus under the forehead periosteum with inflammatory changes of the overlying skin and soft tissues.
This develops secondary to the spread of infection through emissary veins into the cranial bone marrow, and thus essentially represents osteomyelitis of the frontal bone.

An epidural abscess develops from osteitis of the posterior table of the frontal sinus extending into the space between the frontal bone and the dura. Patients present with low-grade fever and worsening headache from elevated intracranial pressure. This complication may be surprisingly indolent because there are no focal neurologic signs and examination of the cerebrospinal fluid (CSF) is often normal. In a manner analogous to the orbital abscess, subdural and brain abscesses can occur from the direct spread of an epidural abscess or from retrograde thrombophlebitis. Increased intracranial pressure is significant in these cases and may lead to herniation and death. Subdural abscess may cause septic venous thrombosis and venous infarction. Brain abscess is associated with brain necrosis.

In contrast to the above intracranial conditions, which usually arise from the frontal sinus, meningitis typically arises from infection of the ethmoid or sphenoid sinus. The typical presenting symptoms and signs are high fever, headaches, seizures, and delirium. Lumbar puncture is necessary to establish the diagnosis and obtain culture results.

**Treatment**

**Medical Management**

The principle of therapy for sinusitis is to break the cycle of impaired mucociliary clearance, stasis, infection, and
inflammation. Treatment for uncomplicated acute sinusitis is primarily medical, with antibiotics representing the mainstay of therapy. In most primary care settings, it is acceptable to initiate antibiotic therapy when the criteria for acute sinusitis are met. First-line drugs for acute rhinosinusitis recommended by the Agency for Health Care Policy and Research Institute include amoxicillin (500 mg PO tid) and trimethoprim/sulfamethoxazole (double-strength tablets, one PO bid). It has been further recommended that cephalosporins, macrolides, penicillinase-resistant penicillins, and fluoroquinolones should be reserved for failures of first-line therapy or for complications. However, some have questioned whether, given the high incidence of pneumococcal and *H. influenzae* resistance in many areas, this graduated antibiotic response is really appropriate. Treatment duration should be at least 10 to 14 days, and antibiotic doses must be adjusted for patient weight (in children) and for hepatorenal function, where appropriate. Recent trends have included the use of culture-directed therapy, which, at least theoretically, allows long-term cost-effective management. This can be performed safely and accurately using a middle meatal swab under endoscopic guidance.

Oral decongestants such as pseudoephedrine and topical decongestants such as phenylephrine and oxymetazoline may be useful by decreasing tissue edema by α-adrenergic vasoconstriction. This allows sinus ventilation and symptomatic relief. Topical decongestants must be used judiciously, however, as continuance of these medications beyond 3 to 5 days is associated with reduced duration of action and rebound vasodilation, a condition known as *rhinitis medicamentosa*. The roles for antihistamines and topical nasal steroids in the management of acute infections are controversial. If allergy is thought to be a significant predisposing or coexisting factor, antihistamines may be indicated. Topical steroids, although useful in chronic rhinosinusitis, have no proven efficacy in the treatment of acute sinusitis but may have a prophylactic effect in preventing recurrent acute episodes. Oral steroids (eg, prednisone or methylprednisolone) are not typically prescribed for acute sinusitis when a significant bacterial component is expected because the immunosuppressive effects may promote the development of complications. However, oral steroids are useful in the management of acute exacerbations of chronic sinusitis to control the baseline inflammatory tendencies of the sinonasal mucosa. Nasal saline irrigations and mucolytics (eg, guaifenesin 600 mg PO bid–qid) may have a role in the treatment of both acute and chronic sinusitis by assisting the mobilization of secretions.

Antibiotic therapy is also a major component in the treatment of chronic (and subacute) sinusitis. The principles of treatment, however, differ from those for acute sinusitis. First, the appropriate duration of therapy may be as long as 3 to 6 weeks. Additionally, empiric therapy requires regimens with coverage of *Staphylococcus* and anaerobes in addition to the common pathogens of acute sinusitis (*S. pneumoniae, H. influenzae*, and *M. catarrhalis*). Culture-directed therapy is essential as antibiotic resistance is a significant problem in this patient population. Virtually all strains of *M. catarrhalis* and over 50% of those of *H. influenzae* are penicillin resistant. Commonly employed regimens include clindamycin (150 mg PO qid) plus either trimethoprim/sulfamethoxazole or a fluoroquinolone. Amoxicillin-clavulanate and selected oral second- and third-generation cephalosporins may be useful as single-agent therapy. New-generation macrolides (clarithromycin, azithromycin) and other cephalosporins may be effective, depending on culture and sensitivity results. Each antibiotic has a unique profile of toxicities and side effects that must be considered. Recent trends have included the use of antibiotic-containing irrigations and nebulized aerosols, particularly in conjunction with endoscopic sinus surgery.

Steroids are also a mainstay in the treatment of chronic sinusitis. Steroids decrease inflammation nonspecifically via a variety of mechanisms. Primarily they inhibit cell-mediated immunity by blocking lymphocyte migration and proliferation. Eosinophil and basophil counts are reduced, and the release of histamine and leukotrienes from basophils is inhibited. Also, steroids decrease both vascular permeability and the secretory activity of submucosal glands.

Topical nasal steroids are effective in reducing mucosal inflammatory changes and are considered safe for long-term use. With initiation of the medication, symptomatic improvement is not realized until >1 week of use. Patients must be counseled in this regard because most patients expect the immediate relief provided by topical decongestants, which cannot be used long-term without rebound vasocongestion. Potential risks associated with nasal steroids include epistaxis and septal perforation. The complications of systemic steroid use, although possible, are rare with topical nasal steroids. Studies have demonstrated increased risk of acute open-angle glaucoma and ocular hypertension with inhaled but not intranasal steroid use. Suppression of the adrenocortical axis has been observed with higher-than-recommended dosages, but other studies have shown that routine daily use is not associated with axis suppression.

Oral steroid therapy can be used intermittently in patients with chronic sinusitis to manage acute exacerbations. Several different steroid compounds are available, and each has its own relative potencies and side effects. Most often either prednisone or methylprednisolone is used. Doses usually begin at 30 mg daily (or equivalent) and are tapered over 2 to 3 weeks. Tapering doses are required after 5 to 7 days of ther-
apy secondary to suppression of the adrenocortical axis. Severe acute exacerbations may require higher dosages, and some patients with recalcitrant chronic rhinosinusitis may necessitate long-term steroid regimens. Often, protracted steroid courses are necessary for management of coexisting asthma in this patient population. Systemic steroid therapy is potentially associated with serious side effects. Long-term use may result in osteopenia or osteoporosis, which may be reversible in early phases. Patients on long-term oral steroids should therefore undergo bone-mineral density studies regularly. Steroid use is also associated with cataracts, hyperglycemia, glaucoma, sodium retention, fat accumulation, and psychosocial changes.

Patients with chronic sinusitis with significant atopic components may be difficult to manage. The most important strategy in this population is avoidance. Antihistamine use should be limited to those with documented allergy by testing or clear allergic stigmata such as frequent sneezing or itchy watery eyes. Antihistamines may cause drying and thickening of nasal secretions resulting in impaired mucociliary flow; therefore, they must be used judiciously. A full discussion of allergy management is beyond the scope of this chapter, but it may include topical and oral steroids, antihistamines, and mast cell stabilizers. There is also mounting evidence supporting the use of immunotherapy, particularly in cases with an allergic fungal component.

Antifungal agents may also have a role in the treatment of sinusitis. Invasive forms often require intravenous therapy with amphotericin B. Use of this medication is limited by renal toxicity. Chronic sinusitis with an allergic fungal component may also be treated with antifungal agents including itraconazole (200 mg PO bid). Topical nasal irrigation with solutions containing amphotericin B or nystatin has also been employed in the treatment of fungal sinusitis. The efficacy of these treatments is an area of active research.

**Surgery**

Indications for surgery include (1) acute sinusitis with a pending or evolving complication, (2) chronic sinusitis that has failed maximum medical management including at least 3 weeks of broad-spectrum antibiotics, and (3) most forms of fungal sinusitis. In cases of complicated acute sinusitis and invasive fungal disease, surgery should be performed on an urgent or emergent basis.

In uncomplicated chronic sinusitis the goals of surgery are to eliminate mechanical obstruction of mucociliary flow, remove chronically inflamed mucocilia, bone, manage/prevent complications, and rule out other disorders such as neoplasia. The determination that “maximal medical management” has failed must be individualized. It should be noted that the indications for surgery are more stringent in the pediatric population, for whom some advocate 3 weeks of intravenous antibiotic therapy prior to consideration of surgery.

Children with severe chronic sinusitis should first have thorough work-up and appropriate treatment for conditions such as allergy, GERD, CF, and immunodeficiency. Simple measures such as avoidance of pollutants (eg, secondhand cigarette smoke) and environmental allergens may avert the need for surgery. One study demonstrated allergies in 80% of children with sinusitis. Children in day-care centers may be prone to upper respiratory infections and consequently chronic sinusitis. Other series have shown that medical treatment of GERD may eliminate the need for sinus surgery in 90% of children otherwise considered surgical candidates.

Prior to surgery it is important to evaluate the CT scan to assess the extent of inflammatory disease and the patient's anatomy. A mental checklist is developed to assess the depth of the ethmoid skull base and the position and integrity of the medial orbital walls. The presence of accessory ethmoid air cells, such as the infraorbital cell or concha bullosa, and anatomic anomalies such as maxillary sinus hypoplasia are noted. Triplanar reconstructions of thinly cut CT scans are used as part of a stereotactic imaging protocol (Figure 16-16). This is useful to assess anatomy and pathology in the axial, coronal, and sagittal planes both preoperatively and intraoperatively, where the surgeon can correlate endoscopic and CT findings during dissection. Use of this technology is indicated when normal anatomic landmarks have been altered, as in patients who have had previous surgery and in cases of massive polyposis. Patients with advanced chronic inflammatory disease, particularly those with nasal polyposis, are treated with oral steroids for up to 2 weeks before surgery. Courses of oral and occasionally intravenous antibiotics are required in selected cases preoperatively.

Surgery is performed under the visualization of endoscopes (Figure 16-17), often with angled lenses, and with a variety of forceps and punches (Figure 16-18). Powered tissue shavers similar to those used in arthroscopic surgery are also used (Figure 16-19). The goals of surgery are to remove chronically inflamed tissue and to restore sinus ventilation, drainage, and mucociliary clearance. Evidence exists that in chronic sinusitis the inflammatory process involves the underlying bone. Thus, it is especially important to resect the bony ethmoid partitions underlying chronically inflamed mucosa. Diseased mucosa is resected, whereas normal mucosa is preserved. It is critical to avoid stripping of normal mucosa because denuded bone results in delayed healing and the regenerated mucosa does not regain normal ciliary density.

In performing maxillary antrostomy, the uncinate process is completely resected and the natural ostium (see Figure 16-19) is identified and subsequently enlarged. The opening must communicate with the natural ostium in a manner that permits
Part 3: Maxillofacial Infections

physiologic mucociliary clearance patterns. The bone of this structure frequently exhibits osteitis. To avoid intracranial complications, special care is necessary during the removal of diseased tissue along the skull base as well as during sphenoid and frontal sinus surgery. Intraoperative stereotactic navigational imaging is useful in performing more comprehensive surgery in these regions (see Figure 16-16).

Prior to the widespread use of endoscopes, ethmoidectomy was performed with a headlight, surgical loupes, or a microscope. Endoscopic technology has greatly improved our ability to perform ethmoidectomy safely and comprehensively. In addition, external approaches including the Caldwell-Luc operation, external ethmoidectomy, and frontal sinus trephination were performed more commonly. The Caldwell-Luc operation, originally described in the late 1800s, is an approach to the maxillary sinus through the labiogingival sulcus and canine fossa (Figure 16-20). In the classically described operation to treat chronic maxillary sinusitis, mucosa of the maxillary sinus was curettaged, and an inferior meatal antrostomy was created. Our knowledge of the mucociliary clearance patterns and our ability to now address the natural ostium have made the classic Caldwell-Luc procedure obsolete in the primary surgical management of chronic maxillary sinusitis. Occasionally a sublabial approach is still required to the maxillary sinus in unusual circumstances; however, given our current understanding of the ability of the mucosa to respond to medical therapy and the long-term problems associated with mucosal stripping, only a very limited mucosal resection is performed when this is required. Overall, external approaches may have a limited role in the management of complicated sinusitis, but endoscopic surgery is preferred when technically possible to address the implicated pathology.

Major complications specific to sinus surgery occur in 0 to 5% and include bleeding, CSF leak and visual problems. Intraoperative blood loss may range from 20 to 500 cc, depending on the extent of disease and surgery. Hemostasis is usually achieved in surgery with local vasoconstrictors.
and/or cautery. Although a small amount of bleeding is typical in the first few days following surgery, excess bleeding is rare and, if it does occur, seldom reaches transfusable quantities. The incidence and severity of postoperative hemorrhage may be increased in patients with acquired immunodeficiency syndrome, diffuse polyp disease, and revision cases.

CSF leak is a risk of surgery performed on the ethmoid bone. This occurs in 0.01 to 1.4% of cases. If recognized intraoperatively, a CSF leak should be repaired in the same operative setting. Patients diagnosed with an iatrogenic CSF leak postoperatively may present with meningitis, which requires medical treatment and surgical repair. The risk of orbital penetration during endoscopic sinus surgery is 2 to 4%, and in one-third of these cases, orbital emphysema is also observed. Fortunately the risk of blindness is low, approaching zero in several series.

This devastating complication is usually secondary to an expanding intraorbital hematoma, although optic nerve injury is possible during surgery of the sphenoid and poste-

rior ethmoid. If blindness is encountered postoperatively, initial management is to remove any nasal packing and perform orbital massage to evacuate any bleeding. Emergent ophthalmologic consultation should be obtained, and lateral canthotomy or endoscopic orbital decompression may be required. Another complication of sinus surgery affecting the eye is nasolacrimal duct injury. Postoperatively, the patient presents with epiphora, or tearing. The nasolacrimal duct courses anterior to the natural ostium of the maxillary sinus and can be injured when the antrostomy is enlarged anteriorly.

The most common complication after endoscopic sinus surgery is the formation of synechiae, observed in approximately 8%. Although these may be asymptomatic, they may also contribute to ostial stenosis and obstruction and, ultimately, the need for revision surgery. Postoperatively, the surgically opened sinus cavities are débrided under endoscopic visualization in the office setting. Patients are asked to use nasal saline sprays and/or irrigations to reduce crusting and facilitate the débridement process. Recalcitrant cases may benefit from the addition of antibiotics to these irrigation solutions.

Postoperative medical management and long-term follow-up care is critically important. Patients are usually put on a course of oral antibiotics to prevent bacterial proliferation in the blood and mucus that may collect in the sinus cavities postoperatively. Antibiotic selection and the duration of treatment are individualized
according to culture results and the degree of inflammation observed. Antibiotics can be discontinued once the mucosa has recovered and ciliary activity can offset the stagnation of secretions. Topical and oral steroids are often prescribed postoperatively to decrease inflammation and reduce scar formation during the healing process. Although some patients require long-term oral steroid therapy, it is preferably avoided, when possible, given the side effects. In contrast, patients almost universally require long-term treatment with topical nasal steroids. This is usually well tolerated and is considered safe.

Overall endoscopic sinus surgery is considered successful in 80 to 90% of patients after at least 2 years follow-up. The natural history for patients with nasal polyps undergoing surgery alone is recurrence since polyposis is multifactorial and is associated with a tendency toward mucosal inflammatory reactivity. One study demonstrated recurrent polyp disease in 55% of patients after a mean follow-up of 3 years and 5 months. Nonetheless, surgery has a clear role in these patients as is evidenced by the observation that over half were asymptomatic or significantly improved, and none were worse. Diligent postoperative care including débridement, medical management, and possibly allergy therapy is essential to reduce or eliminate the tendency toward recurrence, and long-term endoscopic follow-up is required to evaluate for and treat even asymptomatic disease. Studies have also demonstrated that sinus surgery in patients with both asthma and nasal polyposis may decrease both pulmonary and nasal symptoms and reduce the dependency on oral steroids.

Conclusions
Ultimately, additional advancements in our management of sinus disease will require advancements in our understanding of the pathophysiology. At this time, a “common pathway,” through which patients with various risk factors develop sinusitis has not been defined. Sinusitis can be managed effectively, however, with medical therapy in most cases. There are clear roles for surgical intervention in acute sinusitis with complications (or pending complications), chronic sinusitis that has failed medical management, and the various forms of fungal disease. Combined with appropriate medical management, surgical outcomes can be maximized in these cases.

References
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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. The medications linked to osteomyelitis are steroids, chemotherapeutic agents, and bisphosphonates. 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 17-1).

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. More than 500 bacterial taxa have been identified in the mouth. 4–6 The mouth is a 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 17-2)
   b. Progressive
   c. Hematogenous

2. **Chronic osteomyelitis**
   a. Recurrent multifocal (Figure 17-3)
   b. Garre’s (Figure 17-4)
   c. Suppurative or nonsuppurative (Figure 17-5)
   d. Sclerosing (Figure 17-6)

**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.

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 orthopanoramic view. 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.\textsuperscript{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.\textsuperscript{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.
Part 3: Maxillofacial Infections

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 17-7). 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 to the best choice of antibiotics based on the patient’s specific causative organisms. 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 17-6).

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. 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 débridesments 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...
Part 3: Maxillofacial Infections

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 17-8).

**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. 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 17-9 and 17-10). ORN is more common in the mandible than in the maxilla for reasons described earlier in this chapter. A dosage of

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**Figure 17-5** 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 debrided 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.
radiation above 5,000 to 6,000 rads is generally felt to make the mandible susceptible to ORN. 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.

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
Part 3: Maxillofacial Infections

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 debrided 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. 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. There is an excellent skin paddle to provide soft tissue coverage (see Figure 17-7).

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

FIGURE 17-8  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 17-9  A, Panoramic view of the mandible post-radiation in a patient with oral squamous cell carcinoma. Note the large bony sequestrum. B and C, Introra oral views of the right and left mandible showing exposed bone. (CONTINUED ON NEXT PAGE)
proven successful in the dental reconstruction of these patients.\textsuperscript{22}

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.

References

Part 4

Maxillofacial Trauma
Initial Management of the Trauma Patient

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Michael S. Scherer, DDS, 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 exist 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, a few of these deaths have been avoided. The second death peak occurs within the first few hours after injury. The period following 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. Recent analysis of trauma system efficacy suggests that trauma deaths could be reduced by at least 10% through organized trauma systems. 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 based on patients’ injuries and the stability of their 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 observations 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 treat those patients appropriately, while at the same time avoiding unnecessary transport of less severely injured patients (Figure 18-1).\(^6-8\) Over the past three decades many scales and scoring systems have been developed as tools to predict outcomes based on several criteria.
Glasgow Coma Scale

The Glasgow Coma Scale (GCS) was developed in 1974 by Teasdale and Jennet. 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 18-1). 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; therefore, this system 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 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 colleagues to become the Revised Trauma Score in 1989. 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.
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.

In 1989 Champion and colleagues 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.12

### Injury Severity Score

The Injury Severity Score was developed to deal with multiple traumatic injuries. It compares death rates from blunt 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 injury severity score possible is 108 (6² + 6² + 6²). Mortality rates have been found to increase with greater severity of injury and age (Table 18-4).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. Those such factors that have a high correlation with life–threatening injuries include the following16:

- 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 (eg, a burning building or icy water)
- An automobile accident in which it takes > 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 < 5 years or > 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 18-1).

**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, the Trauma and Injury Severity Score, and A Severity Characteristic of Trauma score; recently scales using the ninth edition of International Classification of Diseases nomenclature have been implemented including an International Classification of Disease-Based Injury Severity Score.

**Primary Survey: ABCs**

An algorithm for the initial systemic evaluation and stabilization of the multiply injured patient is presented in Figure 18-2. 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: airway maintenance with cervical spine control, breathing and adequate ventilation, and circulation with control of hemorrhage. Letters D and E have also been added: a brief neurologic examination to establish degree of consciousness, and exposure of the patient via complete undressing to avoid injuries being missed because they are camouflaged by clothing.

**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, or regurgitation of stomach contents. Commonly, the upper airway is obstructed by the position of the tongue, especially in the unconscious patient (Figure 18-3). 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 or the mandible. The chin is then lifted gently anteriorly and the mouth opened, if possible. This method should not hyperextend the neck. The other hand can be used to assist with access to the oral cavity, using the fingers in a sweeping motion to remove such things as debris, vomitus, blood, and dentures that may be responsible for the obstruction. A tonsillographic suction tip is helpful to remove accumulations from the pharynx. Patients with facial injuries who may have basilar skull fractures or fractures of the cribiform plate may, with the routine use of a soft suction catheter or nasogastric tube, be compromised as 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 lock 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, 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. 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.

### Table 18-4 Mortality Rates for Various Injury Severity Scores by Age Groups

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>n</th>
<th>15</th>
<th>25</th>
<th>35</th>
<th>45</th>
<th>55</th>
</tr>
</thead>
<tbody>
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<td>0–49</td>
<td>1,540</td>
<td>3</td>
<td>8</td>
<td>32</td>
<td>61</td>
<td>89</td>
</tr>
<tr>
<td>50–69</td>
<td>316</td>
<td>5</td>
<td>21</td>
<td>56</td>
<td>68</td>
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<td>70+</td>
<td>109</td>
<td>16</td>
<td>45</td>
<td>82</td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>

Adapted from Powers M.
Initial Management of the Trauma Patient

Multiple trauma

- Intubate or secure oral airway.*

Airway patent and secure?
- Yes
  - Administer oxygen.
  - Maintain cervical spine immobilization.
- No
  - 1. Intubate.
  - 2. Assist ventilation.

Check ventilation:
- Yes
  - 1. Hypoventilation?
  - 2. Flail chest?
  - 3. Respiratory distress?
- No

Unilaterally diminished breath sounds (after endotracheal tube repositioning)?
- Yes
  - Insert chest tube.
  - 1. Confirm diagnosis with echocardiogram or needle aspiration, if time permits.
  - 2. Perform thoracotomy.
- No

Pneumothorax or hemothorax?
- Yes
  - Insert chest tube.
- No

Cardiac tamponade (distended neck veins, high central venous pressure, penetrating trauma near heart)?
- Yes
  - 1. Check vital signs and insert intravenous line(s); draw blood for cbc and blood gas determinations.
  - 3. Remove clothing; perform head-to-toe examination.
  - 4. Insert Foley catheter; obtain urine for analysis.
- No

Abdominal trauma (abdominal tenderness, penetrating abdominal trauma, or multiple blunt trauma with altered consciousness)?
- Yes
  - Obtain CT scan or perform peritoneal lavage.
- No

Aortic injury (widened mediastinum, apical cap, first rib fracture, aortic nob obscuration)?
- Yes
  - Obtain aortic arch arteriogram.
- No

Head injury?
- Yes
  - Obtain head CT scan.
- No

Shock present (hypotension, delayed capillary refill, cool pale moist skin)?
- Yes
  - 1. Stop gross external hemorrhage.
  - 2. Insert two or more large-bore intravenous lines.
  - 3. Draw blood for crossmatching cbc.
  - 4. Rapidly infuse crystalloid solution.
  - 5. Maintain cardiac monitoring.
- No

Pulse present?
- Yes
- No

Head injury with unconsciousness or pupil asymmetry?
- Yes
  - 1. Intubate.
  - 3. Administer mannitol 1 g/kg intravenously.
  - 4. Insert chest tube.
- No

Cardiac tamponade (distended neck veins, high central venous pressure, penetrating trauma near heart)?
- Yes
  - 1. Check vital signs and insert intravenous line(s); draw blood for cbc and blood gas determinations.
  - 3. Remove clothing; perform head-to-toe examination.
  - 4. Insert Foley catheter; obtain urine for analysis.
- No

Abdominal trauma (abdominal tenderness, penetrating abdominal trauma, or multiple blunt trauma with altered consciousness)?
- Yes
  - Obtain CT scan or perform peritoneal lavage.
- No

Aortic injury (widened mediastinum, apical cap, first rib fracture, aortic nob obscuration)?
- Yes
  - Obtain aortic arch arteriogram.
- No

Obtain head CT scan.

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 (ie, high-riding prostate, meatal blood, scrotal hematoma). §If not contraindicated (ie, midface or cribiform plate fracture). Adapted from Trunkey DD In: Ho M, Saunders CE, editors. Current emergency diagnosis and treatment. 3rd ed. Norwalk (CT): Lange Publishing Co.; 1990.
confirmed by feeling and listening for air movement at the nostrils and mouth—supplemental oxygen may be delivered by face mask. 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 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 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 (PaO2) should be maintained between 70 and 100 mm Hg. 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 the air to be moved in and out of the pleural cavity with each respiration (Figure 18-4). 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 on three sides of the dressing to the chest. 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

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**Breathing**

With establishment of an adequate airway, the pulmonary status must be evaluated. If the patient is breathing spontaneously—
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 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 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. 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. 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 (Figure 18-5).

A moderate-sized chest tube (32–40F in adults or 26–30F in children) is generally placed either anteriorly in the second intercostal space midclavicular line or in the fourth or 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.

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 fourth intercostal space, an incision is made through the skin along the fifth intercostal space. A gloved finger is used to tunnel transversely through the subcutaneous tissue to the inferior margin of the fourth rib. The intercostal muscles are separated with a large Kelly clamp, and the chest tube is inserted 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

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**Figure 18-4** 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. Adapted from Powers M.15
Part 4: Maxillofacial Trauma

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 18-6). 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 18-7).

**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 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 following 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. 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 $> 200$ to $300$ mL can usually be seen on a good upright chest radiograph with a blunting of the costophrenic angle. The supine radiograph is less accurate.

Treatment of a hemothorax consists of restoration of the circulating blood volume with transfusion of fluids, volume expanders, blood, 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 (36–40F) 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 cm of water). If the chest tube becomes clotted and fails to drain, another chest tube should be put in place rather than an attempt made to irrigate the first tube.

With massive bleeding, autotransfusion of the drained blood is possible until bank blood is available.

A persistent hemorrhage requires surgical exploration. Thoracotomy for intrathoracic bleeding is indicated for the following: initial thoracostomy tube drainage $> 20$ mL/kg of blood; persistent bleeding at a rate $> 7$ mL/kg/h; increasing hemothorax seen on chest radiographic studies; or the patient remaining hypotensive despite adequate blood replacement, and other sites of blood loss have been ruled out, or the patient decompensating after an initial response to resuscitation.

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 18-8). 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

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**Initial Management of the Trauma Patient**

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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 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%.

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 asymmetric and uncoordinated. The region of the fractures may be tender to palpation.

Recommended management of flail chest involves three stages. The first stage is initial stabilization of the loose segment with an external splint, such as a sandbag, rolled sheet, or intravenous bag, taped over the location of the paradoxical movement to both stabilize that segment and to reduce the pain associated with its movement. Although this tends to reduce the vital capacity of the lung, it increases the efficiency of ventilation. This form of treatment can produce atelectasis if used for a prolonged period, but it is adequate for the first 30 minutes until more definitive treatment can be obtained. The next step for prolonged relief is intercostal nerve blocks to block the pain from the fractured ribs, thereby allowing the patient to breathe deeply and cough. The final step involves the use of a volume-cycled respirator with endotracheal intubation to provide PEEP and intermittent mandatory ventilation. 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.

Oxygenation After establishment of a patent airway and sustained breathing, the patient should be given supplemental oxygen to assist reversing of decreased tissue oxygenation during the immediate posttraumatic period. The patient will 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 does not provide for proper delivery of oxygen to the cardiovascular system. Oxygen can be delivered through a nasal cannula, face mask, 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.

Administered oxygen can increase the inspired oxygen to 8 L/min and can increase the fraction of inspired oxygen (FiO₂). A higher FiO₂ 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 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 FiO₂ 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 should be intubated if oxygen resuscitation is required.

Endotracheal intubation helps to protect the airway and facilitates adequate lung inflation with high FiO₂ in the injured patient. Oxygen administered through the endotracheal tube should increase the FiO₂ by 100% (especially if the patient is comatose) until arterial blood gas measurements confirm hemoglobin saturation (PaO₂ > 60–70 mm Hg), at which point FiO₂ can be lowered to between 40 and 60%. Pulmonary oxygen toxicity may result if 100% oxygen is administered continuously for 24 hours; therefore, 100% oxygen delivery is acceptable only until PaO₂ levels can be ascertained. Some concern exists about the suppression of the respiratory drive with oxygen therapy, but the hypoxic drive can be reestablished following 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 hypoxogenation of vital organs 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.

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.

**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, urinary output characteristics, and the mental status of the patient. Blood pressure levels are commonly used to measure cardiac output and to define hypovolemia, but in the emergency situation time does not permit blood pressure level measurement and the physical signs of hypovolemia are more sensitive to 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%.

Blood pressure level may be insensitive to the early signs of shock, and a patient’s blood pressure level may quickly drop following the initial assessment as the compensating mechanisms can no longer provide for the 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 mm Hg but is normally hypertensive may have a significant loss, whereas a healthy young athlete may have a normal systolic pressure of 90 mm Hg 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 < 2 seconds in the normovolemic patient. This indicates that the capillary beds are receiving adequate circulation.30

The rate and character of the pulse is 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 > 120 beats/min, hypovolemia should be expected and investigated further. Older patients generally are unable to exceed rates of 140 beats/min in a hypovolemic state, whereas younger patients may present rates of 160 to 180 beats/min 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, hypovolemic status may not be represented by increased pulse rates.

The location of the pulse may give some indication of the cardiac output. Generally, if the radial pulse is palpable, the patient’s systolic blood pressure is > 80 mm Hg; if the femoral pulse is palpable, the patient’s systolic blood pressure is 70 mm Hg or higher; and if the carotid pulse is noted, the systolic blood pressure is > 60 mm Hg. Pulse rhythm and regularity may also provide clues to increasing hypovolemia and cardiac hypoxia. Cardiac dysrhythmias such as premature ventricular contractions or arterial fibrillations produce an irregular rate and rhythm, signaling the 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/h, 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 a urethrogram can be evaluated for urethral injury. If urethral injury is unlikely, the urinary catheter may be placed with minimal concern. Classic signs of urethral injury include blood at the meatus, scrotal hematoma, or a high-ridding 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 mm Hg. 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 or possibly has suffered 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 that is 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. 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 the pulsus paradoxus (lowering of the systolic pressure by > 10 mm Hg 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 18-9). Because radiographs and physical examination are not helpful, 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 diagnosis or improvement of the patient's condition, only emergent thoracotomy can solve the problem.

Pericardial aspiration through the subxiphoid route involves the insertion of a needle, preferably covered by a plastic catheter (angiocatheter), at 90˚ slightly to the left of the xiphoid process. The needle is inserted until it clears the sternal border and is then directed at 45˚ toward the left scapula 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 cc 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 space, wrist, groin, popliteal space, or ankle may assist in control of hemorrhage distal to the site. Pressure points should only be used if direct wound pressure is not effective alone. 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 face 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

Figure 18-9 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 cc syringe is introduced just to the left side of the xiphoid process. The needle should be introduced at a 45˚ angle to the chest wall, 45˚ 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. Adapted from Powers M.15
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 18-10). Most hemorrhages from facial injuries can be controlled with direct pressure or packing (Figure 18-11). 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.

**FIGURE 18-10** The lateral wall of the nasal cavity (A) and the nasal septum (B) receive a rich blood supply from both the internal and 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.15

**FIGURE 18-11** 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 cribiform 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. Adapted from Leigh JM. Primary care. In: Rowe NC, Williams JC, editors. Maxillofacial injuries. Edinburgh: Churchill-Livingston; 1985. p. 54–74.
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 18-12 and 18-13). 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 1,000 to 2,000 mL for pelvic fractures, 500 to 1,000 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. A slow internal hemorrhage may be controlled by secondary fixation of fractures; by the defense mechanisms of vascular occlusion, refraction, and clot formation; or by open exploratory surgery.

**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 18-5).

**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 an 800 to 1,500 mL loss in the 70 kg adult male. Clinical symptoms commonly expected with this level of blood loss are tachycardia, tachypnea, and a decrease in the difference between systolic and diastolic blood pressure levels. 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 increase 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 1,500 to 2,000 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 to 140 beats/min), tachypnea, marked vasoconstriction, a decreased systolic pressure level, diaphoresis, anxiety, restlessness, and decreased urinary output.

**Class IV Hemorrhage: Blood Loss of > 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 < 60 mm Hg, 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. 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. Two large-bore (16 gauge or larger) short angiocatheters are a minimum for beginning fluid therapy. Initial attempts should be made to place percutaneously 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 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 require 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. 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 of a high-riding prostate (Figure 18-14A). 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 hematomata. A rectal examination should be performed in all trauma patients with suspected pelvic trauma before placement of a catheter. With posterior urethral disruption, the prostate may be forced superiority by a hematoma; if the prostate cannot be palpated, a urethral injury should be suspected (Figure 18-14B). 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 mm Hg, 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...
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 be a significant enough disruption of the urethra to prohibit passage of a catheter safely. 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 development of hematomas over crystalloids. However, there is increased mortality with the use of colloids. Extensive meta-analysis shows a trend toward worse outcomes in patients receiving colloid over crystalloid.39

Blood at the urethral meatus may be 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. Adapted from Powers M.35

expenders (dextran, hetastarch) to treat hypovolemia secondary to trauma. The cost of these materials does not appear to be justified by clinical data. Extensive meta-analysis shows a trend toward increased mortality with the use of colloids over crystalloids. However, there is still support for their use, 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 > 20% of their blood volume.

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 possible immediately 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. 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.

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, a unit of FFP is often given after every 5 U of blood, especially if packed red blood cells are administered in an attempt to prevent coagulation abnormalities. Additionally, platelet levels below < 100,000/mm³ may be an indication for a platelet transfusion. 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, a unit of FFP is often given after every 5 U of blood, especially if packed red blood cells are administered in an attempt to prevent coagulation abnormalities. Additionally, platelet levels below < 100,000/mm³ may be an indication for a platelet transfusion.

If the patient initially responds to therapy, blood may not be required immediately, but the patient will require blood as 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. 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 12.5 g/dL (hematocrit of 38% or higher). Although a hematocrit of 30 to 35% has been recommended in the past, higher levels improve the oxygen-carrying capacity, and the increased viscosity seems to cause relatively little reduction in cardiac output until the hematocrit exceeds 45 to 50%.

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. The ultimate hemodynamic criterion in the treatment of hypovolemic shock is the patient’s response. Adequate resuscitation is achieved when adequate circulation and urine output are restored.

A patient being treated for hypovolemic shock is usually placed in a head-down or Trendelenburg’s 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's position. Alternatively, both of the patient's legs can be elevated while the patient's trunk is maintained in a supine position.\textsuperscript{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 pupil reaction to light requires an immediate CT scan of the head and management with mannitol or fluid restrictions. Be aware of any medications that the patient may have received or drugs he or she may have taken that may affect the pupils.

The Committee on Trauma of the American College of Surgeons recommends the use of the mnemonic AVPU.\textsuperscript{7,8} In this system, each letter describes a level of consciousness in relation to the patient's response to external stimuli: alert, responds to vocal stimuli, responds to painful stimuli, and unresponsive.

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.\textsuperscript{42} Depressed levels of consciousness and narrow pinpoint pupils may result after an opiate overdose. After an overdose with meperidine hydrochloride, the pupils may appear normal or dilated. In both cases, 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 and 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.\textsuperscript{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. 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 18-15).

**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 func-

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**FIGURE 18-15** 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.\textsuperscript{15}
Mass lesions commonly associated with head and skull injuries are expected in the first 12 hours. The secondary assessment includes a subjective and objective evaluation of the injured patient.

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.

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; fractures of the cranium and cerebral contusions; and intracranial bleeding to the brain 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 resulting in a herniation of the brain from the cranial vault (Figure 18-16). 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 < 25% of the time, even when the ICP is found to be > 30 mm Hg and a value > 15 mm Hg 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 18-17), contusion, foreign bodies, and skull fractures. In addition, secondary effects of trauma such as edema, ischemia, infarction, brain shift, and 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 following 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, and evidence of a skull fracture found on physical examination. There is still controversy regarding when a head CT is appropriate. It has been suggested that a CT of the head 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.

Extreme care should always be taken when moving a patient with a head trauma to the CT machine because of the high incidence of associated cervical spine fractures in patients with head and facial traumas. 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 18-18); 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 subcerebral 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.

The GCS (discussed above) provides a simple method of grading consciousness and functional capacity of the cerebral cortex (see Table 18-1). 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.

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 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 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 dilatation of one eye may be due to a developing brain herniation on the ipsilateral side, with bilateral pupillary dilatation 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 18-18). 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 18-18).

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

![Figure 18-18](image-url)
A CT scan should be performed to determine whether there is a fracture site. The head of the bed should be elevated to 90°. 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 proven 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 due 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 of < 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 IV 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 obvious 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 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.

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.

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 with a shift toward the midline, thoracic injuries and fractures that alter lung expansion, and the presence of fluid. Figure 18-19 shows a chest radiograph of a patient without chest trauma. In most instances the trauma patient needs to be immobilized on a backboard (Figure 18-20), 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. Six potentially lethal injuries to consider in the secondary assessment are pulmonary contusion, aortic disruption, tracheobronchial disruption, esophageal disruption, traumatic diaphragmatic hernia, and myocardial contusion.

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 adult 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. 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 intermediate mechanical ventilation.
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 aorta 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 leads 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 left, and a downward displacement of the left mainstream bronchus.7 If an aortic rupture is suspected on clinical or radiographic examination, an aortography should be performed. While waiting for the aortogram, it is important not to let the patient become hypertensive or cough or gag excessively (eg, as may occur with the placement of a nasogastric tube).

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 are contraindications to nasal intubation. Care should 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.

The physical examination should begin with an evaluation for soft tissue injuries. Lacerations should be debrided 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 symmetric 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, and zygomatic arches, and nasal bones should be palpated. Any steps 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. 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 > 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 recent 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 (US).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 life span, 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 mm Hg without the tachycardia, cool extremities, poor perfusion, and decreased urinary output noted in the patient with hypovolemic shock. The neurologic shock is due to dilatation 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 prior to 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 18-20). 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.

Bachulis and colleagues evaluated 4,941 trauma victims between February 1981 and July 1985 and found that 1,923 (39%) had radiographs taken of their cervical spines.51 Injuries to the cervical spine were detected in 94 patients (5%). Ninety of these patients had cervical spine fractures; four 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 either complained of neck pain or of 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 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 injuries may result from axial loading, flexion, extension, rotation, lateral bending, and distraction or combinations of these mechanisms of injury (Figure 18-21).

In the study by Bachulis and colleagues, 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 are difficult, and for patients with a suspected cervical injury that is not detected in screening radiographs.46 A recent study by Griffen and colleagues concluded that CT scanning of the cervical spine should replace plain film studies in blunt trauma patients completely.52

Visualization of all seven cervical vertebrae is important (see Figure 18-21). 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 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 < 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 18-20). Obviously, maintaining a stable airway is critical in patients who have suffered significant head and neck trauma. Cervical neck 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, nasotracheal intubation or cricothyroidotomy should be performed, in that order, always ensuring that the cervical spine continues to be stabilized.

CT should be used for further evaluation of detected or suspected fractures, evaluation of questionable plain films, and to complete radiographic examination of areas not well visualized by plain films. Figure 18-21 Normal cervical radiographs: A, lateral; B, anteroposterior. Radiographs should be examined for prevertebral edema, subluxation, widening of the interspinous distance, widening of the atlantodental interval, bony fractures, malalignment, or jumped facets.
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.\(^7\) 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, patients with unexplained hypovolemia following multiple trauma, patients who are candidates for laparotomy but who have questionable findings, and patients who have experienced severe trauma and who 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 and penetrating trauma. A DPL is usually performed with a sterile intravenous catheter inserted percutaneously through a small midline incision about 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 or 500 white blood cells per cubic millimeter after blunt trauma is sufficient to make a laparotomy mandatory (Table 18-6).

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; it is capable of discerning the presence, source, and approximate quantity of intraperitoneal hemorrhage; and it 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 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

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<th>Table 18-6 Parameters for Evaluation of Peritoneal Lavage Fluid</th>
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Adapted from Powers M.\(^{13}\)

RBC = red blood cell; WBC = white blood cell.
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 < 5 minutes.
- Sensitivity in detecting as little as 100 mL to, more typically, 500 mL of intraperitoneal fluid ranges from 60 to 95% in most recent studies, and specificity for hemoperitoneum is excellent.
- 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.
- 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 the following:

- It does not image solid 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) does not detect the presence of solid parenchymal damage if free intraperitoneal blood is absent, as in subcapsular splenic injury.
- Finally, ultrasonography is poor for detecting a bowel injury in which hemorrhage tends to be inconsequential, and failure to diagnose hollow viscus perforation in a timely manner can have catastrophic results.

Table 18-7 presents indications, advantages, and disadvantages of ultrasonography, DPL, and CT in blunt abdominal trauma.

**Genitourinary Tract**

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. Over 95% 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. Anterior urethral ruptures are also commonly associated with blunt trauma. Most of these injuries occur in men.

Blood at the urethral meatus is the single best indicator of urethral trauma. The meatus must be carefully inspected for even the slightest amount of blood before inserting a urethral catheter. As is discussed above, 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. 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.

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.

Urine should be obtained and evaluated for the presence of blood. A urinalysis of 10 or more red blood cells on a high-power field is suggestive of a urinary system injury. 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.

**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. 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.

Physical examinations 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 rates 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, 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. Fat embolism syndrome has been reported to occur with 30 to 50% of major long-bone and pelvis fractures.59 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, an 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 (eg, muscle wasting, stiff joints, loss of limb length) caused by delays in fracture stabilization and subsequent patient mobilization.60 Studies have shown that early fracture stabilization can significantly decrease mortality, musculoskeletal morbidity, and cardiopulmonary and metabolic consequences commonly associated with multiple trauma.58

Long-bone fractures are a common cause of fat embolisms 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 embolisms.56 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.60

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CHAPTER 19

Soft Tissue Injuries

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In the United States over 11 million traumatic wounds are treated in emergency departments each year. Facial lacerations comprise approximately 50% of these wounds. Facial injuries impact both function and esthetics. There is often a psychological aspect associated with the injury secondary to patient’s concern regarding permanent scarring and subsequent facial disfigurement. According to a recent survey, cosmetic outcome is the single most important aspect of care to the patient.

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. 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.

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 prior to the injury, a 0.5 mL tetanus toxoid boost injection should be given. If the patient has not received a booster within 10 years prior, 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 immunoglobulin, followed by a full course of immunization.

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. Occasionally it is better to delay soft tissue repair until the facial fractures have been addressed. In patients with large avulsion of tissue, definitive early reconstruction of the tissue loss with regional or microvascular flaps may be required.

Anatomic Evaluation

Following 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.

A thorough head and neck examination determines the extent of associated facial wounds. Peripheral cranial nerves are commonly involved with lacerations that involve the face. The facial nerve divides the parotid gland into deep and superficial portions (Figure 19-1). Any injury to the gland should raise suspicion for associated facial nerve injury. The facial nerve exits the stylomastoid foramen and divides into...
five branches within the parotid gland (Figure 19-2). 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 19-3).

Injury to the parotid gland can lead to leakage of saliva into the soft tissue. 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.

FIGURE 19-1 The facial nerve divides the parotid into a deep and superficial lobe.

FIGURE 19-2 Anatomy of the facial nerve.

FIGURE 19-3 Zone of arborization of the facial nerve.
A polymeric silicone (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 19-4). 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.

There are several protocols 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.

**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 where there is a concern that deep structures have been damaged, a general anesthetic affords the best opportunity for exploration and repair. The patient may require repair of other traumatic injuries in the operating room, and on many occasions, definitive repair of associated facial soft tissue injuries can be performed at the same time.

Lidocaine is a popular local anesthetic and ranges in strength from 0.5 to 2%. It is usually administered with epinephrine 1:100,000. Lidocaine has a rapid onset of action, a wide margin of safety, and a low incidence of allergic sensitivity. A thorough evaluation of the seventh cranial nerve should be undertaken prior to injection of anesthetic or administration of a general anesthetic. Injecting local anesthetic prior to 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. 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.

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, which inhibits phagocytosis. Persistent infection at a wound site leads to the release of inflammatory cytokines from monocytes and macrophages, which delays wound healing. An anaerobic environment results and limits leukocyte function. 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 to 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, hypertrophic scarring, and granulomas. A scrub brush and detergent soap may be necessary to remove deeply imbedded foreign material. However, soaps may cause cellular damage and necrosis. A surgical blade may be helpful to scrape foreign material that is deeply embedded. Polymyxin B sulfate can be used to remove residual grease or tar in wounds.

Proper cleaning and good surgical technique are imperative in minimizing infection. Infections are rare when the

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**FIGURE 19-4**  
A, This laceration shows the parotid duct severed and cannulated with a polyethylene tube. B, The duct is sutured over the tubing.
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. 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. It is preferable to suture complex facial lacerations secondary to esthetic considerations. 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 impairment of the vascular supply and may cause an increase in scarring.

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. Margins should be undermined to allow slight eversion of the wound margin. 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 help support the wound margins.

At 7 to 10 days following suture removal the collagen has begun to cross-link. The wound is now able to tolerate early controlled motion with little risk of disruption (Figure 19-5). 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 following 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 to standard wound closure techniques for non-crush-induced lacerations treated less than 6 hours after injury. 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 esthetic concerns.

Suture materials and different surgical techniques do not show substantial differences in relation to outcome. General characteristics of the patient (ie, sex and age) and of the wound (ie, length and site) seem to be important predictors of adverse tissue reaction. Suboptimal appearance is associated with wounds that are infected, wide, 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.

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 those with 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 prior to 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 the fracture site.

**Types of Injuries**

**Abrasions**

Shear forces that remove a superficial layer of skin cause abrasions. The wound should be gently cleansed with a mild soap solution 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 in the wound. Failure to remove all foreign material can lead to permanent “tattooing” of the soft tissue. After the wound is cleansed 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 causes edema and hematoma formation in the subcutaneous tissues. The associated soft tissue swelling and ecchymosis can be extensive. Small hematomas usually resolve without treatment; hypopigmentation or hyperpigmentation of the involved tissue 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 19-6). Lacerations 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. Flap-like 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 “trapdoor” injuries.

**Avulsive Injuries**

Avulsive injuries 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 amount of soft tissue is missing, then a skin graft, local flaps, or free-tissue transfer may be necessary (Figure 19-7).

**Animal and Human Bites**

Dog bites are most common in children and the midface is frequently involved.\(^{26,27}\) Canines can generate 200 to 450 psi when biting, and examination for fractures should be performed.\(^{28}\) Management of bite injuries involves liberal amounts of irrigation and meticulous primary closure.\(^{29}\) 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 *Pasteurella multocida*.\(^{30}\) Cat bites are associated with a twofold higher risk of infection than the more common dog bite wounds. Because their 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.\(^{26,31}\) Amoxicillin-clavulanate is the current drug of choice for bite wounds. Antibiotic prophylaxis should be directed at *Pasteurella multocida* 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.\(^{32}\)

Immediate closure of bite injuries is safe, even with old injuries.\(^{33}\) There is approximately a 6% rate of infection when bite wounds are sutured primarily in lacerations where there are cosmetic concerns.\(^{34}\) Extensive animal bite wounds involving the face should be treated according to the criteria of esthetic reconstructive surgery. Rabies prophylaxis should be given for bite wounds that occurred from an unprovoked domestic dog or cat that exhibits bizarre behavior or from an attack by a wild animal such as a raccoon, skunk, bat, fox, or coyote.\(^{35}\)
**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 (Figure 19-8).8

Ballistic facial injuries are grouped by etiology: gunshot, shotgun, and high-energy avulsive injuries.36 Over the past 20 years advances in imaging and the introduction of craniofacial approaches with rigid fixation have led to an evolution of treating facial injuries. The esthetic 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 esthetic results.

**Scalp and Forehead**

Scalp wounds can occasionally cause a large amount of blood loss due 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, subcutaneous tissue, aponeurosis layer, loose subepicranial space, and pericranial layer.

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. Hollander and colleagues found no significant difference in rate of infection in scalp lacerations that were irrigated compared to those that were not.37

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.38 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, as regrowth of the hair is unpredictable. Closure of lacerations should attempt to salvage as much tissue as possible. Care should be taken 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 19-9). Care should be taken to precisely reapproximate the eyelid margins and the tarsus (Figure 19-10). The conjunctiva and tarsus are closed with resorbable sutures with the knot buried to avoid irritating the cornea. The orbicular muscle is then closed followed by closure of the skin. Injuries involving the upper eyelid may include detachment of the levator aponeurosis and Müller’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 19-11). 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. Repair is accomplished by introducing a lacrimal duct probe into the puncta and into the wound (Figure 19-12). The ends of the lacerated duct are identified and approximated over a polymeric silicone 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. If the patient exhibits chronic epiphora postoperatively, then a dacryocystorhinostomy is indicated.

Avulsive injuries to the eyelids are treated with skin grafts and/or local flaps. Defects of up to 25% of the eyelid length can be closed primarily. Skin grafts harvested from the opposite eyelid provide excellent texture and color match.

**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 polymeric silicone 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 in 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. Local flaps may be required to restore missing tissue (Figure 19-13).

**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 prior to 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. 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.

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 amount are used to avoid devitalizing the region of cartilage (Figure 19-14). Avulsive injuries of the ear can involve a portion of the ear or the entire ear (Figure 19-15). If the avulsed segment is 1 cm or less, it can be reattached and allowed to revascularize.
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 colleagues (Figure 19-16). 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 19-17).

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 vermillion 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 esthetic results. Injuries that involve a greater amount of tissue loss can be reconstructed with a variety of flaps such as Abbe-Estlander or Karapandzic (Figure 19-18).

Neck

Successful management of penetrating injuries of the neck depends on a clear understanding of the anatomy of the region. Injuries can involve deep structures...
Soft Tissue Injuries

affecting the vascular, respiratory, digestive, neurologic, endocrine, and skeletal systems.\textsuperscript{46} The neck is divided into three anatomic zones.\textsuperscript{47} 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.\textsuperscript{46-50}

![Diagram](image.png)

**Figure 19-15**  A, Conversion of a defect to a wedge. B, The use of Burow's triangles. C, Conversion of a defect to a star.
Serial physical examinations alone have been shown to be effective. In cases where serial physical examinations are not possible, mandatory exploration of neck wounds may be more 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 hypertrophic 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. A recent study found no difference in outcome of surgical scars treated with pulsed carbon dioxide laser when compared with dermabrasion.51

Keeping a wound clean and scab free allows for more rapid reepithelialization.52 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 antibiotic ointment is standard. Patients should avoid sun exposure for the first 6 months after the injury to prevent 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 patients to their preoperative state of function and to achieve an esthetic result.

References

Figure 19-18  A, Avulsive lip resulting from a dog bite. The lower lip flap is outlined. B, The pedicled Abbe flap is sutured into place and divided 3 weeks later. C, After division of the flap.
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. Since 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.” 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 which 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.” 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 20-1). 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 where 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 20-2). New bone is laid down by the osteoblasts, forming osteons which cross the gap and impart microscopic points of bony union to the fracture. 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 following 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 only provide stability 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 20-3).

However, various forms of nonrigid fixation are recognized, and there is a continuum between rigid fixation and no fixation at all. There are some forms of nonrigid fixation that 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 stability to allow function even though
FIGURE 20-1  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.
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, and from one fracture to the next. Examples of functionally stable fixation include the single miniplate technique of treating mandibular angle or body fractures (Figure 20-4). 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.

In the late 1950s the Swiss Association for the Study of Internal Fixation (AO/ASIF) promulgated four biomechanical principles in fracture management:

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 20-5). Bone cannot
Part 4: Maxillofacial Trauma

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.

Selection of Fixation Schemes: How Much Fixation (Rigidity) is Enough?

With that prelude into definitions of fixation types, the remainder of this chapter will discuss 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 20-6).

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 fix-
for fractures of the angle of the mandible show that two plates perform much more poorly than does one plate in that location. One must therefore be very careful in applying 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 where 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 20-7). 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 mm, 2.4 mm, 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 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 insufficient 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 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 manufacturers. Examples of load-sharing fixation for angle fractures are demonstrated in Figure 20-1A–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 fixa-
tion. Comminuted or defect fractures, or those where 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 20-8A 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 (Figure 20-8C 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 20-4). 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. 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 tendency for torquing of the fragments to occur, causing mediolateral misalignment of the inferior border. While the arch bar may provide sufficient resistance to the tendency for a gap to form between the teeth under function, a plate

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**Figure 20-8**  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.
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 20-9).

The directions of forces that are distributed through the anterior mandible vary with the activity of the mandible. This means that the classical zones of tension on the superior and compression on the inferior surfaces of the mandible are not absolute.\textsuperscript{7,8} Instead, the anterior mandible undergoes shearing and torsional (twisting) forces during functional activities.\textsuperscript{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 20-1A–H).\textsuperscript{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 from one another 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 from one another. For instance, an atrophic mandibular fracture, where 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 20-10). In such instances a single stronger bone plate should be applied below the inferior alveolar canal (Figure 20-11). 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 20-1F). 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.

\textbf{FIGURE 20-9} Example of a simple isolated mandibular body fracture treated by the application of arch bars and a single 2.0 mm miniplate.

\textbf{FIGURE 20-10} 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.
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Compression versus Noncompression Plate Osteosynthesis

There are many types of bone plates that 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 20-12). While 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 where there is minimal obliquity, and where 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, then 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 “lock” to the plate, thus stabilizing the segments without the need to compress the bone to the plate (Figure 20-13). 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

While 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 20-14).

As with using compression bone plates, lag screw fixation is a technique that should only be used to provide absolute rigid fixation. Micromotion across a fracture secured with lag screws will likely result in dissolution of the bone around the screws, with loss of stability. Therefore, lag screws should only be selected 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 to 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 20-15).

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 fractures 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 plates are selected. One of the undesirable properties of titanium is its brittleness (or lack of ductility) when compared to 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

![Figure 20-13](image1.png) 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.

![Figure 20-14](image2.png) 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.
Part 4: Maxillofacial Trauma

miniplates or 2.0 mm adaptation plates applied to the condylar process, or similar plates applied to the atrophic mandible (Figure 20-16). 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 20-17). 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.

Bone plates applied to such areas of the fractured mandible have to be able to not only acutely withstand the deforming forces applied, but must also 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 (mini-dynamic compression plates) (Figure 20-18) or two 2.0 mm miniplates for condylar process fractures, and reconstruction bone plates for atrophic mandibular fractures. This problem with the atrophic mandible is the reason the AO/ASIF has recommended, “The weaker the bone, the stronger the plate must be.”

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. 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 use either 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. 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 20-19). 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 20-20). 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, then the angle fracture could be treated with a single superior border 2.0 mm miniplate (functionally stable but not rigid fixation).

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, like a 2.0 mm miniplate, or even two 2.0 mm miniplates, may not offer sufficient resistance to the tendency to widen (Figure 20-21A). 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 either use a reconstruction plate applied across the symphysis (Figure 20-21B), or if the fracture is linear, two well-placed lag screws (see Figure 20-1B). The application of two thicker 2.0 mm bone plates (thicker than miniplates) would also suffice (see Figure 20-1C). If one chose to open the condylar process fractures, then the symphysis fracture can be treated as an isolated symphysis fracture, with whatever technique the surgeon usually chooses.

**Figure 20-19** 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 (eg, 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 4-hole 2.0 mm miniplate.

**Figure 20-20** 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 4-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.
Summary

While 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 where 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 plate with plates of varying lengths and thicknesses allows one to choose the appropriate bone plate for almost any location.

References

Management of Alveolar and Dental Fractures

Richard D. Leathers, DDS
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 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 21-1). In the same way, to expedite the healing process, he stressed recapturing proper occlusion, a concept that is 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 cauterization 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 21-1).3

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 anatomical 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 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 2:1.5

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

![Figure 21-1: 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. Reproduced with permission from The Archaeological Museum, American University, Beirut, Lebanon.](image-url)
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 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.11,12

Black and colleagues 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.13

Other groups that are at increased risk of dentoalveolar trauma are those with seizure disorders, mental disorders, and congenital maxillofacial abnormalities.

Lockhart and colleagues reported findings, by the Risk Management Foundation, indicated that damage to the teeth was the most frequent anesthesia-related claim, often resulting in litigation.14 Poor laryngoscopy technique and the unmonitored biting force of the comatose patient also potentially caused dentoalveolar injury.15,16

With direct trauma, maxillary incisors are the most frequently traumatized teeth, especially if they are associated with a Class II division I malocclusion. Trauma to the primary dentition usually results in various luxations (~ 75%), whereas in permanent dentition, crown or crown-root fractures are the normal (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 21-2).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 prior to managing dental injuries. The initial examination should be systematic, methodic, and comprehensive (see Figure 21-2). 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 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.20 Signs of confusion followed by “lucid intervals” may require further radiographic and/or computed tomography (CT) 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 (GI) obstruction until the foreign body is cleared.

Maxillofacial Examination

For medicolegal purposes, consider preoperative photographs prior to invasive treatment.

Include the following in the patient examination23:

- 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 21-1).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 alerts 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.

Prior to any intraoral manipulations, obtain initial radiographic studies (eg, 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.

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 hematomata 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 prior to further treatment.

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 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. Nullness 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.” 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 fibers, which regulate vascular changes and respond to pain stimuli, respectively. As the tooth develops, the pain fibers (ie, myelinated) increase, while simultaneously lowering the electrometric pulp stimulation. 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 development stage of the involved

| Table 21-1  Summary of Tetanus Prophylaxis |
|-----------------|-----------------|-----------------|-----------------|-----------------|
| **History of Adsorbed Tetanus** | **Non-Prone Wounds** | **Tetanus-Prone Wounds** | **Non-Prone Wounds** | **Tetanus-Prone Wounds** |
| Unknown or ≤ 3 doses | Td* | TIG | Yes | Yes |
| ≥ 3 doses† | No§ | No | Yes | Yes |

Td = tetanus and diphtheria toxoids adsorbed—for adult use; TIG = tetanus immune globulin—human.
*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.)
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:

- 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, which is 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 wherein 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.

To ensure completeness, generate a standardized treatment record during the evaluation process, which systematically culminates in a diagnosis, treatment plan, and prognosis. Figure 21-3 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. Following 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.

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**Dentoalveolar Trauma Record**

<table>
<thead>
<tr>
<th>Name: ____________________________</th>
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<tbody>
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</tr>
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<td>Location: __________________</td>
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<tr>
<td>Time: __________________________</td>
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<td>Headache: __________________</td>
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<td>— Nausea, vomiting: _____________</td>
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<tr>
<td>Tooth vitality findings (pulp testing): __________________________</td>
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<tr>
<td>Tooth mobility (+1, +2, +3): __________________________</td>
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</tr>
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<td>Ellis classification (I, II, III, IV): __________________________</td>
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<tr>
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<td>Prognosis: ________________________</td>
<td>Good _____</td>
</tr>
<tr>
<td>Examined by: ________________________</td>
<td></td>
</tr>
</tbody>
</table>

**FIGURE 21-3** Dentoalveolar trauma record.
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 21-4) and Andreasen (Figures 21-5–21-7). 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 (ie, a craze line or crack in the tooth without loss of tooth substance)
  - Crown fracture that is 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)
  - 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 (ie, 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

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 that are 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.
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 following 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, and the immunologic defense systems in the pulp will combat bacterial invasion (Figure 21-8).

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

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
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
This type of fracture is limited to fractures involving the roots only (Ellis IV). Most root fractures occur in the apical and middle one-third 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 21-9). Treatment of mobile root fractures consists of apposition of the fractured segments with rigid splinting for 12 weeks.

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 is 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.

Root 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 periodontal ligament 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


FIGURE 21-8 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.

FIGURE 21-9 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.)
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 that is seen in root fractures and, to a lesser extent, in luxation injuries.

**Internal 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 ingestion 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 21-10).

The potential devastating effects of the resorptive process require immediate and proper treatment of periodontal injuries.

**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 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, permanent teeth will have an increased risk of tooth fracture. The specific luxation classification depends on the force and direction of traumatic impact. Fifteen to 61% of luxation injuries occur in the permanent dentition and 62 to 73% in the primary dentition. Multiple teeth are usually involved in luxation injuries.

**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 by bleeding at the gingival margin crevice (Figure 21-11).

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 pulp necrosis, and endodontic treatment is indicated. Studies show that external resorption will
occur in 4% of these injuries. 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 dislodgement of teeth to these sites when intrusive forces are encountered. Intrusive injuries are the 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. The incidence of pulpal necrosis is relatively high (96%). Inflammatory resorption incidence may reach 52% as a result of the necrotic pulp (Figure 21-12).

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 pulpal necrosis occurs, seek endodontic therapy. In cases of complete root development with closed apices, re-position the toothatraumatically, 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. 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 periodontal ligament.

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%.

It is treated by delicately placing the extruded tooth back into the proper position in the socket. Check and re-check 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 is 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 to 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 periodontal ligament during the progressive eruption of these teeth. \(^{46}\)

The treatment of such injuries must be geared toward early reestablishment of periodontal ligament cellular physiology. The fate of the avulsed tooth depends on the cellular viability of the periodontal fibers that remain attached to the root surface prior to reimplantation. Although extraoral time is a factor, newer physiologically compatible solutions are available that can maintain and/or replenish periodontal ligament cell metabolites. Two such solutions are Hank's balanced salt solution and ViaSpan (Figures 21-13 and 21-14). \(^{47-49}\)

Both Hank's solution and ViaSpan are physiologic with compatible pH and osmolality (Table 21-2). ViaSpan is the solution of choice for organ storage during transport for transplantation. The relative availability and cost effectiveness of Hank's solution makes 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.

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 < 20 minutes. Any periodontal ligament extraoral exposure > 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.

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 minutes); periodontal ligament 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 > 1 mm diameter have a prognosis that is much better than that of the more mature or closed-root apex. Treat the tooth with an open root within the 2-hour time frame 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. 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.

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 21-15 and 21-16). This revascularization process is optimized by the topical application of doxycycline. Individuals who have avulsed teeth with mature or closed apices and who present within the 2-hour time frame 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 prior to splint removal. Final gutta-percha obturation is contingent on resolving canal and/or root pathology (6 to 12 months). 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 periodontal ligament 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 debride 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 21-3 and 21-4).

| Table 21-2 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 |

Table 21-2 Solutions to Replenish Periodontal Ligament Cell Metabolites

<table>
<thead>
<tr>
<th>Time (yr)</th>
<th>Closed apex</th>
<th>Open apex</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>100</td>
<td>90</td>
</tr>
<tr>
<td>5</td>
<td>80</td>
<td>70</td>
</tr>
<tr>
<td>10</td>
<td>60</td>
<td>50</td>
</tr>
</tbody>
</table>

FIGURE 21-15 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 and Andreasen FM.
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 periodontal ligament 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 21-5).

The acid-etch technique is the only system that most closely adheres to these recommendations (Figure 21-17). The arch bar, self-curing, Essig, intracoronary, 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 21-6).

### 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

<table>
<thead>
<tr>
<th>Table 21-4 Treatment Summary for Teeth Avulsed &gt; 2 Hours*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Replant immediately, if possible</td>
</tr>
<tr>
<td>2. Transport in Hank’s solution or milk</td>
</tr>
<tr>
<td>3. Present to nearest qualified facility (decrease time call first)</td>
</tr>
<tr>
<td>4. Check ABCs; evaluate for associated injuries (history and physical examination)</td>
</tr>
<tr>
<td>5. Bathe tooth in sodium hypochlorite for ~30 min vs manual débridement of the periodontal ligament</td>
</tr>
<tr>
<td>6. Perform extraoral RCT</td>
</tr>
<tr>
<td>7. Bathe tooth in citric acid (~3 min)</td>
</tr>
<tr>
<td>8. Bathe tooth in 1% stannous fluoride (~5 min)</td>
</tr>
<tr>
<td>9. Transfer to a 1 mg/20 mL doxycycline bath for ~5 min</td>
</tr>
<tr>
<td>10. Perform radiography (posteroanterior, occlusal, panoramic, chest)</td>
</tr>
<tr>
<td>11. Initiate local anesthesia</td>
</tr>
<tr>
<td>12. Perform tetanus prophylaxis as needed</td>
</tr>
<tr>
<td>13. Initiate antibiotic coverage</td>
</tr>
<tr>
<td>14. Replant tooth</td>
</tr>
<tr>
<td>15. Splint for 7–10 d</td>
</tr>
</tbody>
</table>

ABC = airway, breathing, circulation; RCT = root canal therapy
*Open or closed apex.
Part 4: Maxillofacial Trauma

Severity, use either an open or 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 21-18). 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 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 weeks). 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 (eg, 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

<table>
<thead>
<tr>
<th>Table 21-5 Splint Requirements</th>
</tr>
</thead>
<tbody>
<tr>
<td>The splint should</td>
</tr>
<tr>
<td>1. Be able to be applied directly in the mouth without delay owing to laboratory procedures</td>
</tr>
<tr>
<td>2. Stabilize the injured tooth in a normal position</td>
</tr>
<tr>
<td>3. Provide adequate fixation throughout the entire period of immobilization</td>
</tr>
<tr>
<td>4. Neither damage the gingiva nor predispose to caries and should allow for a basic oral hygiene regimen</td>
</tr>
<tr>
<td>5. Not interfere with occlusion or articulation</td>
</tr>
<tr>
<td>6. Not interfere with any required endodontic therapy</td>
</tr>
<tr>
<td>7. Preferably fulfill esthetic demands</td>
</tr>
<tr>
<td>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</td>
</tr>
<tr>
<td>9. Be easily removed without re-injury to tooth</td>
</tr>
</tbody>
</table>

could lead to unsightly accidental tattooing. If present, carry out meticulous removal within 12 hours, with care not to further inoculate the patient. 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.

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### Table 21-6 Sequence of Acid-Etch Splinting Technique*

<table>
<thead>
<tr>
<th>Step</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Perform alveolar bony reduction and/or replantation</td>
</tr>
<tr>
<td>2.</td>
<td>Perform localized cleansing and débridement</td>
</tr>
<tr>
<td>3.</td>
<td>Isolate and dry area</td>
</tr>
<tr>
<td>4.</td>
<td>Custom fabricate wire (~26 Ga), double-stranded monofilament nylon line, or paper clip</td>
</tr>
<tr>
<td></td>
<td>Extend wire to at least 1 or 2 teeth on either side of the involved tooth or teeth</td>
</tr>
<tr>
<td>5.</td>
<td>Etch the incisal half of the labial surface of the involved and adjacent teeth with gelled phosphoric acid for 30–60 s</td>
</tr>
<tr>
<td>6.</td>
<td>Remove etchant with water stream for ~20 s</td>
</tr>
<tr>
<td>7.</td>
<td>Air dry etched surface; surface should appear chalky white</td>
</tr>
<tr>
<td>8.</td>
<td>Passively place prefabricated wire to involved teeth</td>
</tr>
<tr>
<td>9.</td>
<td>Stabilize splint with fast-setting autocure or light-cure composite resin</td>
</tr>
<tr>
<td>10.</td>
<td>After resin is set, smooth rough edges with a fine acrylic or diamond finishing bur (Check occlusion)</td>
</tr>
<tr>
<td>11.</td>
<td>Perform soft tissue and gingival repair as needed</td>
</tr>
<tr>
<td>12.</td>
<td>Remove splint in 7–10 d</td>
</tr>
</tbody>
</table>

*It may be prudent to use a composite shade that differs from the natural color of the involved teeth as this will facilitate ease of removal and prevent trauma to enamel.

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**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 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 21-19). 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 hypoplasia (Figure 21-20).

Andreasen and Raven 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 21-21).21,22,59

Table 21-7 provides a summary of the treatment regimen.

Table 21-7  Treatment of Pediatric Injuries

<table>
<thead>
<tr>
<th>Type of Injury</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crown fractures</td>
<td>Smooth rough edges</td>
</tr>
<tr>
<td>Class I (enamel only)</td>
<td>1. CaOH or glass ionomer liner over dentin</td>
</tr>
<tr>
<td>Class II (enamel and dentin)</td>
<td>2. Composite resin restoration</td>
</tr>
<tr>
<td>Class III (pulpal involvement)</td>
<td>1. Formocresol pulpotomy</td>
</tr>
<tr>
<td>Vital pulp</td>
<td>2. Coronal coverage</td>
</tr>
<tr>
<td>Nonvital pulp</td>
<td>1. ZnOH-eugenol pulpectomy</td>
</tr>
<tr>
<td>Class IV (root fracture)</td>
<td>2. Coronal coverage</td>
</tr>
<tr>
<td>Apical third</td>
<td>No treatment; follow-up</td>
</tr>
<tr>
<td>Cervical third</td>
<td>1. Remove tooth fragments</td>
</tr>
<tr>
<td></td>
<td>2. Allow apical third to resorb if compromise to permanent tooth bud is expected</td>
</tr>
<tr>
<td>Luxations</td>
<td>Monitor/follow-up</td>
</tr>
<tr>
<td>Subluxation</td>
<td>Realign/remove prn</td>
</tr>
<tr>
<td>Lateral luxations</td>
<td>Realign/remove prn</td>
</tr>
<tr>
<td>Extrusion</td>
<td>1. Allow to re-erupt 4–6 wk</td>
</tr>
<tr>
<td>Intrusion</td>
<td>2. Remove if in contact with permanent successor</td>
</tr>
<tr>
<td></td>
<td>3. Remove if infection presents</td>
</tr>
</tbody>
</table>

prn = as needed.
References


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**FIGURE 21-21** Association of the type of luxation injury with respect to the malformation of the permanent dentition. Adapted from Andreasen JO and Ravn JF.59


Principles of Management of Mandibular Fractures

Guillermo E. Chacon, DDS
Peter E. Larsen, DDS

Management of trauma has always been one of the surgical subsets in which oral and maxillofacial surgeons have excelled over the years. More particularly, our experience with dental anatomy, head and neck physiology, and occlusion provides us with unparalleled skills for the management of mandibular fractures.

The mandible is the second most commonly fractured part of the maxillofacial skeleton because of its position and prominence. The location and pattern of the fractures are determined by the mechanism of injury and the direction of the vector of the force. In addition to this, the patient’s age, the presence of teeth, and the physical properties of the causing agent also have a direct effect on the characteristics of the resulting injury.

Bony instability of the involved anatomic areas is usually easily recognized during clinical examination. Dental malocclusion, gingival lacerations, and hematoma formation are some of the most common clinical manifestations.

In the management of any bone fracture, the goals of treatment are to restore proper function by ensuring union of the fractured segments and reestablishing preinjury strength; to restore any contour defect that might arise as a result of the injury; and to prevent infection at the fracture site. Restoration of mandibular function, in particular, as part of the stomatognathic system must include the ability to masticate properly, to speak normally, and to allow for articular movements as ample as before the trauma. In order to achieve these goals, restoration of the normal occlusion of the patient becomes paramount for the treating surgeon.

Basic principles of orthopedic surgery also apply to mandibular fractures including reduction, fixation, immobilization, and supportive therapies. It is well known that union of the fracture segments will only occur in the absence of excessive mobility. Stability of the fracture segments is key for proper hard and soft tissue healing in the injured area. Therefore, the fracture site must be stabilized by mechanical means in order to help guide the physiologic process toward normal bony healing.

Reduction of the fracture can be achieved either with an open or closed technique. In open reduction, as the name implies, the fracture site is exposed, allowing direct visualization and confirmation of the procedure. This is typically accompanied by the direct application of a fixation device at the fracture site (Figure 22-1). A closed reduction takes place when the fracture site is not surgically exposed but the reduction is deemed accurate by palpation of the bony fragments and by restoration of the functioning segments, for example, restoration of the dental occlusion by wiring the teeth together, using splints, or employing external pins (Figure 22-2).

Fixation must be able to resist the displacing forces acting on the mandible. It can take one of two forms: direct or indirect. When direct fixation is used, the fracture site is opened, visualized, and reduced; then stabilization is applied across the fracture site. The rigidity of direct fixation can range from a simple osteosynthesis wire across the fracture (ie, nonrigid fixation) to a miniplate at the area of fracture tension (ie, semirigid fixation) or a compression bone plate (ie, rigid fixation) to compression screws alone (lag screw technique). Indirect fixation is the stabilization of the proximal and distal fragments of the bone at a site distant from the fracture line. The
Most commonly used method for mandibular fractures is the use of intermaxillary fixation (IMF). A further example of indirect fixation is the use of external biphasic pin fixation in combination with an external frame (Figure 22-3).

Over the past three decades many different techniques and approaches have been described in the literature to surgically correct facial fractures. More recently the use of internal fixation utilizing plates has shown the highest success rates with the lowest incidence of nonunions and postoperative infections.4–6 The origin of plating as a treatment option for fractures can be traced to Dannis and colleagues, who reported the successful use of plates and screws for fracture repair in 1947.7 Later refinement of this technique is credited to Allgower and colleagues at the University of Basel, who successfully used the first compression plate for extremity fracture repair in 1969.8 However, it was not until 1973 that Michelet and colleagues reported on the use of this treatment modality for fractures of the facial skeleton.9 In 1976 following Michelet’s success, a group of French surgeons headed by Champy developed the protocol that is now used for the modern treatment of mandibular fractures. But it was not until 1978 that these findings were published in the English literature.10

Basically, there are two categories of plating systems: rigid compression plates such as the AO/ASIF (Arbeitsgemeinschaft für Osteosynthesefragen/Association for the Study of Internal Fixation) and the semirigid miniplates. The advantages and disadvantages of each system have been extensively discussed; however, the question remains: does compression of fractures really offer a clinically significant advantage in terms of better bone healing and fewer complications?

Proponents of the AO system state that primary or direct bone healing is the main advantage offered by this system. When a fracture is compressed, absolute interfragmentary immobilization is achieved with no resorption of the fragment ends, no callus formation, and intracortical remodeling across the fracture site whereby the fractured bone cortex is gradually replaced by new haversian systems.11 However, in other studies it has been shown that absolute rigidity and intimate fracture interdigitation is far from mandatory for adequate bony healing. Compression is not necessary at the fracture site for healing, and it is questionable whether compression stimulates osteogenesis.12,13

**Biomechanical Considerations**

Studies of the relationship between the nature, severity, and direction of traumatic force on the resultant mandibular injury were made by Huelke and colleagues.14–19 Before this, few experimental studies had been done with regard to the mechanism of mandibular fracture. Most literature regarding the mechanism of fracture was based on clinical impressions and opinions.

Early investigators showed that linear fractures in long bones were initiated by bone failure resulting from tensile strain rather than compressive strain.20 Huelke and Harger applied forces of varying magnitudes and direction to dried mandibles and observed the resultant production of tension and compression.17 They found that > 75% of all experimentally produced fractures of the mandible were in primary areas of tensile strain, which supported a similar observation made earlier in long
bones. A notable exception was that comminuted condylar head injury that was produced by a load parallel to the mandibular ramus was primarily the result of compressive force.

In response to loading, the mandible is similar to an arch because it distributes the force of impact throughout its length (Figure 22-4). However, unlike the arch, the mandible is not a smooth curve of uniform bone, but rather it has discontinuities such as foramina, sharp bends, ridges, and regions of reduced cross-sectional dimension like the subcondylar area. As a result, parts of the mandible develop greater force per unit area, and consequently, tensile strain is concentrated in these locations.

When a force is directed along the parasymphysis-body region of the mandible, compressive strain develops along the buccal aspect, whereas tensile strain develops along the lingual aspect. This produces a fracture that begins in the lingual region and spreads toward the buccal aspect. The mobile contralateral condylar process moves in a direction away from the impact point until it is limited by the bony fossa and associated soft tissue. At this point, tension develops along the lateral aspect of the contralateral condylar neck, and a fracture occurs. If greater force is applied to the parasymphysis-body region, not only will tension develop along the contralateral condylar neck leading to fracture in this area, but continued medial movement of the smaller ipsilateral mandibular segment will lead to bending and tension forces along the lateral aspect and subsequent fracture of the condylar process on the ipsilateral side. Force applied directly in the symphysis region along an axial plane is distributed along the arch of the mandible. Because the condylar heads are free to rotate within the glenoid fossa to a certain degree, tension develops along the lateral aspect of the condylar neck and mandibular body regions, as well as along the lingual aspect of the symphysis. This leads to bilateral condylar fractures and a symphysis fracture (Figure 22-5).

Variation from these standard fracture patterns occurs for two general reasons. First, there is a wide range in the possible magnitude and direction of the impact and in the shape of the object delivering the impact. Second, the condition of the dentition, position of the mandible, and influence of associated soft tissues could not be controlled in these studies. Early observers felt that the presence of posterior dentition tended to reduce the incidence of condylar injury. The implication was that, as the mandible was forced posteriorly and superiorly, the dentition would meet and absorb some of the force, thereby diminishing the force received at the condyle. This was supported

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by the clinical observation that the posterior dentition was often fractured on the side of the condylar fracture. However, more recent findings do not support this theory and show that all types of fractures occur, irrespective of the occlusion, and that no correlation exists between the degree of dislocation, level of fracture, or type of fracture with the presence of a distal occlusion.24 Although the presence or absence of a posterior dentition does not correlate with the incidence of fracture, the presence of specific teeth, particularly impacted third molars, has been shown to markedly affect the incidence of mandibular fractures. It was shown that, when impacted third molars are present, this area represented a region of inherent weakness and the incidence of condylar fractures decreases, whereas the incidence of mandibular angle fractures increases.25

Although unable to show that the occlusion played any role in the type of fracture produced, investigators have found that the relative degree of mandibular opening at the time of impact does play an important role in the type of fracture that occurs.23,26 More recent studies have shown that not only is the incidence of fracture higher when the mouth is open, but the level of fracture varies with degree of opening. When the mouth is opened, the fractures tend to be located more in the condylar neck or condylar head region, whereas when it is closed, fractures are in the subcondylar area.25

**Evaluation of Mandibular Fractures**

Traumatic craniofacial and skull base injuries require a multidisciplinary team approach. Trauma physicians must evaluate carefully, triage properly, and maintain a high index of suspicion to improve survival and enhance functional recovery. Frequently, craniofacial and skull base injuries are overlooked while treating more life-threatening injuries.27 Unnoticed complex craniofacial and skull base fractures, cerebrospinal fluid fistulas, and cranial nerve injuries can result in blindness, diplopia, deafness, facial paralysis, or meningitis.

Following the principles of Advanced Trauma Life Support, during the initial assessment in the emergency department, the first and most critical obligation is to make sure that the airway is patent and free of potential obstruction. The tongue, which may have a tendency to fall back, must be controlled, and objects obstructing the airway must be removed. If an obstruction cannot be removed, a new airway must be established by endotracheal intubation (remembering possible cervical spine injuries) or cricothyrotomy. After the airway has been secured and respiration is occurring, vital signs must be assessed, including pulse rate and blood pressure. Any significant blood loss is likely to be coming from injuries apart from those of the face. Other critical injuries must be ruled out, including intracranial hemorrhages, cervical and other spinal injuries, chest injuries, abdominal trauma, and fractures of the long bones.

Local examination of the face and jaws should be conducted in a logical sequence. The first objective is to obtain an accurate history from the patient, or relative if the patient cannot cooperate. Pertinent to a

![Figure 22-5](image-url)
Principles of Management of Mandibular Fractures

Fractured mandible examination is notation of the size, number, and force of any blows to the face.

Patients often complain of the following:

- Pain or tenderness is often present at the site of impact with the possibility of a direct fracture, or at a distant site in the case of an indirect fracture.
- Difficulty chewing. Pain could be limiting mandibular function or there may be a malocclusion or mobility at the fracture site.
- Malocclusion. The patient may be able to tell the clinician of an alteration in the bite from normal; however, patients are not always reliable and may claim that the bite feels normal when it is not and vice versa.
- Numbness in the distribution of the inferior alveolar nerve. This usually indicates a displaced fracture in the region of the body or angle of the mandible on the affected side. A nondisplaced fracture often does not give rise to numbness in the distribution of the inferior alveolar nerve.

**Clinical Examination**

The clinical examination should consist of inspection and palpation. It is best to proceed in an orderly fashion and to perform this evaluation as a component part of the entire head and neck examination of the trauma patient. The skin of the face and, in particular, the area around the mandible should be inspected for swelling, hematomas, and lacerations. A common site for a laceration is under the chin, and this should alert the clinician to the possibility of an associated subcondylar or symphysis fracture. Typically, the patient who has suffered a fracture of the mandibular condyle will present with facial asymmetry (Figure 22-6). This is owing to the loss of the vertical height of the ramus on the side with the fracture, resulting in a shift of the mandible to the ipsilateral side.

The best routine to evaluate facial fractures is to start at the top and work down, assessing the stability of the anatomic structures in a mediolateral fashion. It is best to begin the examination from behind the seated or supine patient (Figure 22-7). The clinician should palpate the movement of the condyle both over the lateral aspect of the joint and through the external acoustic meatus and observe the movement of the mandible itself. If a unilateral condylar fracture is present, a subjective assessment can then be made between the palpable movement of one side compared with the other. Failure to detect the translation of the condyle, especially when associated with pain on palpation, is highly indicative of a fracture in this area. Palpation will frequently confirm tenderness over the lateral pole of the injured condyle with associated crepitation. However, in the case of fracture dislocations, the condyle may not be palpable.

Any significant deviation on opening may be indicative of subcondylar fracture on the side to which the mandible deviates. To better evaluate this area, the fifth finger is placed in each acoustic meatus and the patient is asked to open and close the mouth. On opening, the mandible frequently shifts even more toward the side of the fracture as a result of decreased translation of the condyle on the injured side. As mentioned before, in unilateral fractures, there is deviation of the occlusion toward the fractured side, with premature occlusal contact in the posterior region on that side. This results because the lateral pterygoid muscle on the fractured side pulls on the fractured segment and does not have any protruding influence on the mandible. The lateral pterygoid muscle on the contralateral side is unopposed and thus causes deviation to the fractured side. The midlines no longer coincide, and there is an open bite in the body region on the contralateral side. This is often accompanied by fracture of the posterior denti-
premature contact is present bilaterally on the posterior dentition with an anterior open bite. The posterior dentition may be fractured on both sides in these situations.

Often the patient with a fracture of the condylar process also has a limited range of motion. This limitation, however, is primarily caused by voluntary restriction as a result of pain. One has to keep in mind that any limitation of mandibular movement may also be a result of reflex muscle spasm, temporomandibular effusion, or mechanical obstruction to the coronoid process resulting from depression of the zygomatic arch. Other less common findings include blood within the external auditory canal and, in the case of fracture dislocation, development of a prominent preauricular depression. Careful otoscopic evaluation of the external auditory canal is of particular importance in patients suspected to have suffered an injury at this level. Occasionally a fracture of the condylar process will produce a tear in the epithelial lining of the anterior wall of the canal, which produces bleeding from the acoustic meatus. It is important to determine that this bleeding is not coming from behind a ruptured tympanic membrane, which may signify a basilar skull fracture.

A detailed intraoral examination should be undertaken with good lighting and immediate availability of suction. The most common intraoral findings are malocclusion, fracture of the dentition, and decreased interincisal opening.

Continuing with the systematic evaluation of the patient, it is suggested that examination of the soft tissues be undertaken next. The gingival tissue should be inspected for tears or lacerations. With the aid of a tongue blade, the floor of the mouth is examined; sublingual ecchymosis is almost pathognomonic of a fracture of the mandible. Next the dentition is examined for evidence of broken teeth and for steps or irregularities in the dental arch. The patient is asked to lightly bite the teeth together and to say whether the bite feels different from normal, following which the occlusion is inspected. Premature occlusal contacts are noted. The three causes of an altered occlusion in the trauma patient are a displaced fracture, a dental injury such as a displaced tooth, and a temporomandibular joint effusion or dislocation.

If the patient is edentulous and has intact dentures with him, these can be replaced in the mouth and the occlusion inspected (Figure 22-9). The mandible should then be grasped on each side of any suspected fracture and gently manipulated to assess mobility. If no fracture can be found but clinical suspicion remains high, the mandible may be compressed by applying pressure over both angles (Figure 22-10). This nearly always gives rise to pain at a fracture site. In the case of subcondylar fractures, firm posterior pressure on the chin will cause pain in the preauricular region.

**Radiographic Evaluation**

To adequately screen for the presence of a mandibular fracture, at least two views at right angles to each other are necessary. A panoramic radiograph and a reverse Towne’s view (Figure 22-11) are adequate screening studies for this purpose. If only one view is used, fractures can easily be missed. In the multiple-trauma patient for whom panoramic radiographs are not possible, lateral oblique views may be substituted. Other radiographic views that may be useful depending on the circumstances are posteroanterior mandibular, mandibular occlusal, and periapical. Linear tomographies of the temporomandibular joints can also be useful in the evaluation of fractures at the level of the condylar process. However, intracapsular fractures...
of the condylar head are often difficult to visualize accurately on plain films.

The typical radiographic findings when a condylar fracture is present are the following: a shortened condylar-ramus length; the presence of a radiolucent fracture line or, in the case of overlapped segments, the presence of a radiopaque double density (Figure 22-12); and evidence of premature contact on the side of the fracture if the radiograph was taken with the patient in occlusion. If more accurate information of the involvement of the temporomandibular joint is required, axial and coronal computed tomography (CT) scans offer an excellent opportunity to study the fracture details.

Indications for CT scans are the following:

1. Significant displacement or dislocation, particularly if open reduction is contemplated
2. Limited range of motion with a suspicion of mechanical obstruction caused by the position of the condylar segment
3. Alteration of the surrounding osseous anatomy by other processes, such as previous internal derangement or temporomandibular joint surgery, to the degree that a pretreatment baseline is necessary
4. Inability to position the multiple-trauma patient for conventional radiographs (CT scans may be the only useful radiograph that can be obtained)

Chayra and colleagues reviewed the need for a complete series of films. They concluded that the initial screening of patients could be effectively undertaken with a panoramic radiograph alone. Ninety-two percent of fractures were seen on a panoramic radiograph alone, compared with only 66% on a routine radiographic series without a panoramic view. However, in order to accurately visualize displacement it is recommended that the standard mandibular views consist of a panoramic radiograph, a posteroanterior mandibular view, and reverse Towne’s view (Figure 22-13). The latter view allows for visualization of the degree of medial or lateral displacement of the fracture and unveils injuries in which only subtle deviation is present, such as is seen in greenstick fractures, which are not readily evident on panoramic view.

The panoramic radiograph usually requires the patient to be able to stand upright and also requires accurate patient positioning for good-quality films. In the severely traumatized patient, this may be difficult to achieve with some machines. Further, mesiolateral displacement in the ramus and body and anteroposterior displacement in the symphyseal regions may also be difficult to visualize. The traditional lateral oblique views of the mandible can be used when panoramic films are not possible. They require accurate positioning of the patient and film to obtain useful views, particularly in the condylar area. A transcranial temporomandibular view may be a good addition in these circumstances.

Accurate assessment of symphyseal fractures may be problematic with the standard views. A mandibular occlusal view is particularly useful in this scenario. It also aids in the assessment of the fracture of the lingual plate, particularly in very oblique fractures. Periapical views may also be necessary for evaluation of the teeth on either side of the fracture line to assess root fractures, periapical and periodontal pathology, and the relationship of the fracture line to the periodontal ligament of each tooth.

Classification

The first step in the development of an appropriate treatment plan is to establish a clear understanding of the type of injury the patient has suffered, in order to provide an adequate surgical solution. In the diagnostic work-up phase, the lack of standardized ways to assess and
characterize the nature and severity of the orofacial injury engenders variation in practice patterns. Probably the most basic question one should ask at the initial evaluation is whether the fractures are displaced or nondisplaced. Depending on the amount of energy transmitted to the facial skeleton and the vector in which such force is directed, there will be more or less disruption of the normal anatomic structures. Muscle attachment and their counteracting forces also play a primary role in the pattern and direction of the fractures. It is the displacing forces of the muscles of mastication that influence favorableness (Figures 22-14 and 22-15). The principle of favorableness is based on the direction of a fracture line as viewed on radiographs in the horizontal or vertical plane. 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. In the parasymphyseal region of the mandible, the combined action of the suprathyroid and digastric muscles on a bilateral fracture can pull on the distal fragment inferiorly in unfavorable fractures, putting the patient at risk for acute upper airway obstruction.

The first concern is whether there are indeed fractures present, and if there are, where they are located anatomically. Mandibular fractures may be further classified by the pattern of fracture (Figure 22-16) present and by anatomic location.

Many systems of classification have been applied to fractures involving the mandibular condyle. The recommended classification parallels the comprehensive classification set forth by Lindahl. As mentioned before, it is imperative that radiographs be taken of the suspected injury in two planes at right angles to each other. The following major relations are noted: the level of the fracture; the relation of the condylar fragment to the mandible, termed the degree of displacement; and the relation of the condylar head to the fossa, or the degree of dislocation.

**FIGURE 22-13** Mandible series of a patient with a left subcondylar fracture. The series consists of posteroanterior (A), Towne’s (B), left lateral oblique, (C) and right lateral oblique (D) views. If a panoramic radiographic machine is readily available, the lateral oblique shots can be replaced by a panoramic view.

**FIGURE 22-14** Diagram of horizontally unfavorable (left) and favorable (right) fracture lines. Arrows indicate displacing forces. Adapted from Luyk NH. p. 410.
Principles of Management of Mandibular Fractures

Anatomic Location

The following classification has been modified from Kelly and Harrigan’s epidemiologic study in which they divided mandibular fractures based on their anatomic location:36

- Dentoalveolar fracture: Any fracture that is limited to the tooth-bearing area of the mandible without disruption of continuity of the underlying osseous structure
- Symphyssis fracture: Any fracture in the region of the incisors that runs from the alveolar process through the inferior border of the mandible in a vertical or almost vertical direction
- Parasymphyssis fracture: A fracture that occurs between the mental foramen and the distal aspect of the lateral mandibular incisor extending from the alveolar process through the inferior border
- Body fracture: Any fracture that occurs in the region between the mental foramen and the distal portion of the second molar and extends from the alveolar process through the inferior border
- Angle fracture: Any fracture distal to the second molar, extending from any point on the curve formed by the junction of the body and ramus in the retromolar area to any point on the curve formed by the inferior border of the body and posterior border of the ramus of the mandible

Pattern of Fracture

The following classification is based on pattern of fracture (see Figure 22-16):

- Simple fracture: A simple fracture consists of a single fracture line that does not communicate with the exterior. In mandibular fractures this implies a fracture of the ramus or condyle or a fracture in an edentulous portion with no tears in the periosteum.
- Compound fracture: These fractures have a communication with the external environment, usually by the periodontal ligament of a tooth, and involve all fractures of the tooth-bearing portions of the jaws. In addition, if there is a breach of the mucosa leading to an intraoral communication or a laceration of the

![Figure 22-15 Diagram of vertically favorable (left) and unfavorable (right) fracture lines. Arrow indicates displacing force. Adapted from Luyk NH.](image)

![Figure 22-16 Types of fractures: A, simple fracture; B, compound fracture; C, comminuted fracture; D, impacted fracture in right subcondylar area and pathologic fracture in the left angle area; E, direct and indirect fractures. Adapted from Luyk NH.](image)
skin communicating with the fracture site, edentulous portions of the mandible may be involved.

- Greenstick fracture: This type of fracture frequently occurs in children and involves incomplete loss of continuity of the bone. Usually one cortex is fractured and the other is bent, leading to distortion without complete section. There is no mobility between the proximal and distal fragments.
- Comminuted fractures: These are fractures that exhibit multiple fragmentation of the bone at one fracture site. These are usually the result of greater forces than would normally be encountered in simple fractures.
- Complex or complicated fracture: This type of injury implies damage to structures adjacent to the bone such as major vessels, nerves, or joint structures. This usually implies damage to the inferior alveolar artery, vein, and nerve in mandibular fractures proximal to the mental foramen and distal to the mandibular foramen. On rare occasions a peripheral branch of the facial nerve may be damaged or the inferior alveolar nerve injured in subcondylar fractures.
- Telescoped or impacted fracture: This type of injury is rarely seen in the mandible, but it implies that one bony fragment is forcibly driven into the other. This type of injury must be displaced before clinical movement between the fragments is detectable.
- Indirect fracture: Direct fractures arise immediately adjacent to the point of contact of the trauma, whereas indirect fractures arise at a point distant from the site of the fracturing force. An example of this is a subcondylar fracture occurring in combination with a symphysis fracture.
- Pathologic fracture: A pathologic fracture is said to occur when a fracture results from normal function or minimal trauma in a bone weakened by pathology. The pathology involved may be localized to the fracture site, such as the result of a cyst or metastatic tumor, or as part of a generalized skeletal disorder, such as osteopetrosis.

Fracture dislocations are caused by missiles such as bullets. Other injuries occur as well and must be considered in the differential diagnosis (Table 22-1).

**Anterior dislocation** occurs when the condyle moves anterior to the articular eminence. This is by far the most common situation and represents a pathologic forward extension of the normal translational movement of the condylar head. Unlike subluxation, which is also a forward extension of the condyle, dislocation is not self-reducing. Dislocation may be caused by yawning, oral sex, phenothiazine use, and trauma. Traumatically induced anterior dislocation is most commonly bilateral, but it may occur unilaterally (particularly if associated with a concomitant fracture elsewhere in the mandible). The diagnosis of an anteriorly dislocated mandible is made by the following clinical features: an anterior open bite with the inability to close the mouth; severe pain in the region

<table>
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<th>Table 22-1 Injuries of the Articular Apparatus</th>
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<tr>
<td><strong>Fracture</strong></td>
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<td>Effusion</td>
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<td>Hemorrhagic or serous</td>
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<td>Soft tissue injury</td>
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<td>Disk</td>
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<td>Capsule</td>
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<td>Ligaments</td>
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<td>Dislocation of the condyle from the fossa</td>
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<td>Without fracture</td>
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<td>With fracture other than condyle</td>
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<td>With associated condylar fracture</td>
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<tr>
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<td>Involving adjacent bony structures</td>
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anterior to the ear; absence of the condyle from the glenoid fossa with a visible and palpable preauricular depression; inability to move the mandible except to open the mouth slightly in a purely rotational manner; difficulty in speaking; and a prognathic lower jaw. Finally, if unilateral dislocation is present, the chin will be deviated to the opposite side (Figure 22-17). Patients with anterior dislocation of the mandibular condyles without other mandibular trauma should be approached using the following treatment protocol: 2 cc of local anesthetic solution should be deposited into the joint capsule followed by manual reduction. If this is unsuccessful or the patient is overly apprehensive, diazepam should be carefully titrated intravenously followed by further attempts at manual reduction. If these measures fail, then general anesthesia with the use of a muscle relaxant may be necessary. It is usually possible to reduce an acute dislocation with these maneuvers. In refractory cases or in cases associated with mandibular body and angle fractures in which the dislocated segment is difficult to control by manipulation, surgical intervention may be required. A percutaneous bone hook placed through the sigmoid notch or wires placed through the angle of the mandible allow for additional downward traction. Following successful reduction, the patient should be instructed to refrain from opening his or her mouth widely and to support the jaw with a hand under the chin when yawning for a period of 3 weeks to allow for healing of the injured soft tissue in and around the joint. IMF is not necessary for a first-time acute anterior dislocation of the jaw, unless it persistently dislocates after reduction. In persistent, recurrent dislocation, contributing factors, such as phenothiazine use, should be identified. A soft diet may also be recommended for several days along with a nonsteroidal anti-inflammatory analgesic.

When a blow to the mandible produces primarily a posterior vector of force and does not result in fracture of the condylar neck, the head of the condyle may be forced into a posterior dislocation. This injury is frequently associated with laceration and fracture of the external auditory canal leading to hemorrhage that is visible at the external acoustic meatus. In most cases maintenance of the patient’s occlusion and treatment of the associated ear injuries are the only management procedures necessary.

Lateral dislocation of the condylar head is always associated with a concomitant fracture either of the condyle or elsewhere within the mandible. The diagnosis of this condition is straightforward. The condylar head is palpable as a hard mass either in the preauricular region or in the lower part of the temporal space. This type of injury is associated with a marked crossbite, which is not attributable solely to the mandibular fracture but instead is secondary to the displaced condyle. Treatment requires reduction of the dislocation through manipulation of the dislocated segment by grasping it with a thumb on the dentition and with the fingers extraorally along the body of the mandible. If the proximal segment size is inadequate for this maneuver, a percutaneous towel clip through the angle or a small incision with placement of a wire through the angle (as described for anterior dislocation) may be necessary. After reduction of the dislocation, treatment of the associated fracture is accomplished, preferably with rigid internal fixation.

Superior dislocation into the middle cranial fossa without associated fracture of the mandibular condyle has been described. The patient is predisposed to this type of dislocation when the condylar head is small and rounded. This injury is more common when the mouth is open at the moment of impact. This type of injury usually occurs with concomitant midface fractures that are telescoped, causing shortening of the vertical dimension of the face and allowing superior dislocation of the mandibular condyle. Superior dislocation of the mandibular condyle is associated with cerebral contusion and basilar skull fracture with facial nerve paralysis and deafness. These patients present with severe restriction of interincisal opening, pain in the area of the temporal-mandibular joint, bleeding from the external auditory canal or hemotympanum, and deviation of the jaw to the affected side. A variety of treatment modalities are recommended, including observation, condylotomy, elastic traction, condylectomy, and manual reduction. Neurosurgical consultation is required.

Effusion and hemothrosis of the temporomandibular joint after trauma occur similarly as in other joints. In most cases this leads to a distention of the joint capsule with varying amounts of discomfort. Frequently deviation of the mandible away from the affected side occurs as a result of downward pressure on the condyle from the production of
fluid within the joint. This produces facial asymmetry and malocclusion (Figure 22-18).

The treatment of traumatically induced effusions of the temporomandibular joint is aimed at the restoration of preinjury occlusion with return to function and relief of pain. If the patient presents with the subjective symptoms of a joint effusion but has a stable and reproducible occlusion, the condition may be managed with close daily observation, nonsteroidal anti-inflammatory medications, and a soft diet. Frequently the condition will resolve in a matter of days. If, however, the malocclusion is significant enough that the patient is unable to achieve a stable occlusion without manipulation of the jaw, Ivy loop wiring or arch bars should be placed and guiding elastics used to produce a stable occlusion. Arthrocentesis, arthroscopy, or both are common therapies for hemarthrosis in other joints and may also be considered. Regardless of the therapy chosen, care should be taken to avoid excessive IMF because this may result in a long-term limitation of function. It has been suggested that this limitation in function is a result of organization of the blood within the joint space with development of fibrosis and subsequent ankylosis. Many authors have emphasized the importance of this proposed mechanism in the development of ankylosis. Aspiration or arthroscopic lavage may alleviate this. It is possible, however, that the development of limited function and ankylosis is more dependent on the inability to maintain a full range of motion during the IMF period rather than on the hemarthrosis. This theory is supported by the failure of experimentally induced hemarthroses to produce ankylosis, and by the absence of ankylosis and limited function after iatrogenically induced hemarthroses during joint injections or arthroscopy. Most likely, decreased range of motion after joint effusion is the result of intra-articular fibrosis potentiated by prolonged IMF.

**Treatment of Mandibular Fractures**

Fractures of the mandible have been reported to comprise between 40 and 62% of all facial fractures, although these figures may not represent the true incidence because isolated nasal fractures are seldom included in such surveys. If these injuries are taken into account, the occurrence of mandibular fractures decreases to anywhere between 10 and 25% of all facial fractures depending on the mechanism of injury. The literature is consistent on the fact that about one-half of all patients who suffer mandibular fractures are involved in a motor vehicle accident. Males are overwhelmingly reported to be affected more frequently than females in a ratio ranging from 3:1 to 7:1 depending on the survey and especially the country involved. Predictably, such studies reveal the most susceptible age group for both sexes is between 21 and 30 years of age.

In most cases, mandibular fractures are encountered in isolation from any other facial fractures. But different studies have revealed that almost 20% of these patients have concomitant fractures in other anatomic structures of the facial skeleton, with the most common one being the zygomaticomaxillary complex. Further injury away from the facial region may also be present, including multiple-system trauma. In the study by Ellis and colleagues of 2,137 patients with mandibular fractures, 10.5% of subjects sustained other injuries outside the maxillofacial region. Injury patterns are largely dependent on the mechanism of injury, with patients involved in motor vehicle accidents sustaining a great percentage of other injuries. The distribution of principal fracture sites has been reported as 33% involving the body, 29% in the condylar region, 23% the angle, and 8% in the symphysis region (Figure 22-19). It is not unusual to sustain more than one fracture site in the mandible. Mandibular fractures are multiple in more than 50% of the cases. The left side is more commonly involved, in particular the left angle, probably because most assailants are right-handed and the left side of the jaw would be the side most likely to be struck. Falls show a greater proportion of subcondylar fractures, as high as 36.3% in one study. When multiple fractures of the mandible are considered, the most common combinations are angle and opposite body, bilateral body, bilateral angle, and condyle and opposite body (Figure 22-20).

The site of fracture is also determined by the size, direction, and surface area of the impacting blow. An impact to the chin...
Principles of Management of Mandibular Fractures

with a line of force through the symphysis and temporomandibular joints will produce a single subcondylar fracture at 193 kg (425 lb.) and a bilateral subcondylar fracture at about 250 kg (550 lb.), whereas symphyseal fractures require force between 250 and 408 kg (900 lb.).

An impact to the lateral aspect of the mandibular body using a 2.5 × 10 cm (1 × 4 in.) impact surface will produce a mandibular fracture at 136 to 317 kg (300–700 lb.). When an impact force is delivered to the mandible, the bone bends inward, producing compressive forces on the impacted (lateral) surface and tensile forces on the lingual (medial) surfaces of the bone opposite the impact site. Fracture results when the tensile strain overcomes the resistance of the bone, beginning on the medial side of the mandible and progressing through the bone toward the impact point.

Direct fracture may occur at the site of impact, but additional indirect fractures may result when higher forces are involved. An example would be a blow to the left angle, causing a direct fracture at the left-angle region and an indirect fracture in the right body. Occasionally, only indirect fracture results, usually in the subcondylar area as, for example, when a blow on the chin results in a fracture of either condylar neck. Indirect fractures demonstrate the opposite tensile strain patterns and fracture outcomes from those of the direct fracture; that is, the tensile strain develops on the side opposite to the impact. In the case of greenstick fractures, the fracture occurs on the tension side and bending occurs on the compression side.

General Approach and Goals of Therapy

Deciding on the correct treatment is often more difficult than administering the treatment itself. The dilemma concerning the appropriate management of fractures of the mandibular condyle is most exemplary of this. Technically easy procedures such as closed reduction have experienced long-term successful results, whereas more complicated and technically demanding procedures of open reduction have continually and cyclically been employed in an attempt to improve on the results obtained with closed reduction. Although anatomic reduction with rigid internal stabilization of the fracture segments may be desirable, it is essential that the surgeon clearly define the goals of therapy and choose the simplest and most effective surgical method available to reach them.

The goals to be achieved in treatment of fractures of the mandible are listed in Table 22-2. Maintenance of a stable occlusion is necessary for both functional and esthetic reasons. Complete range of motion also allows normal mastication and prevents the development of contralateral temporomandibular joint dysfunction. A normal range of motion is most dependent on postoperative retraining of the muscles and elimination of pain. Ideally, the disk-condylar relationship should remain intact without evidence of internal derangement. Some clinical signs of internal derangement such as joint noise can be tolerated if not associated with pain or decreased range of motion. Growth disturbance can result from ankylosis or from injury to the cartilaginous head of the condyle. A goal of treatment should include early mobilization to prevent ankylosis and close follow-up to identify growth changes early in their development. Attainment of an anatomic bony union is not a primary goal in treatment of
condylar fractures, particularly if it must be done at the expense of other more important goals. A malunion or fibrous union that functions normally without pain is preferable to a radiographically excellent reduction that does not eliminate pain or limits motion.

**Treatment Options**

**Closed Reduction** If the principle of using the simplest method to achieve optimal results is to be followed, the use of closed reduction for mandibular fractures should be widely used. According to Bernstein, “It is safe to say that the vast majority of fractures of the mandible may be treated satisfactorily by the method of closed reduction.”65 May and colleagues go further66: “Many fractures are probably overtreated by open reduction. It is important to realize that the majority of fractures can be successfully managed by conservative means (closed reduction).” This concept becomes critical when one considers the economic significance of inflated hospital, operating room material, and personnel costs. Even more important, the need for general anesthesia is obviated. A patient with a mandibular fracture managed by closed technique can be successfully treated as an outpatient with either local anesthesia or conscious sedation.

Therefore, the indications for closed reduction may simply be stated as all cases in which an open reduction is either not indicated or is contraindicated. Several conditions deserve specific mention.

Grossly comminuted fractures are, as a general rule, best treated by closed reduction, because using open reduction techniques would jeopardize the blood supply to the small bone fragments and lead to an increased likelihood of infection. This category also includes gunshot wounds, which are particularly prone to infection.

Fractures in the severely atrophic edentulous mandible represent a difficult clinical situation. On the one hand, there is limited osteogenic potential; the majority of the blood supply comes from the periosteum, so an open reduction further disrupts the blood supply. On the other hand, a stable, nonmobile reduction and fixation of these fractures is difficult with closed reduction techniques. Open reduction with limited dissection of the soft tissue and rigid fixation may be the preferred technique. Later in this chapter we review in more detail the management of this group of patients.

In situations where there is a lack of soft tissue overlying the fracture site, soft tissue flaps have to be transposed to cover a fracture site (particularly if a through-and-through communication exists between the skin and oral cavity). The presence of bone plates, screws, and wires may increase the likelihood of infection under these circumstances.

Fractures in children involving the developing dentition are difficult to manage by open reduction because of the possibility of damage to the tooth buds or partially erupted teeth (Figure 22-21). Closed reduction of fractures of the mandible together with indirect fixation can be achieved by either the application of IMF or by applying a technique to the mandible only.

The overwhelming majority of published clinical series over the past 50 years strongly promote closed reduction for the management of fractures of the mandibular condyle in both adults and children.21,22,32,33,34,67–70 These uniformly excellent results were obtained in all ages of patients treated.71 Conclusions drawn by various authors are the following: no correlation exists between the degree of radiographic displacement and the severity of clinical symptoms; no correlation exists between the radiographic alignment of the fracture segments and postoperative function; growth complications and ankylosis are exceedingly rare; open reduction with internal fixation is fraught with complications; and evidence supports the choice of closed reduction as the primary treatment modality for condylar fractures regardless of the degree of displacement.

Although the majority of the large studies reviewed patients in all age groups, some authors specifically studied children and their response to conservative management of condylar fractures.72–78 All obtained
excellent results with minimal complications when fractures of the condyle in children were treated with closed methods.

The superiority of closed reduction of condylar fractures is also supported by numerous animal studies. Experimentally induced fracture dislocation in rhesus monkeys has resulted in “a workable, usable mandibular articulation regardless of whether the condyle was left remaining at right angle to the ramus, pushed medially or anteriorly, or reduced and maintained via transosseous wire. There was little sacrifice of mandibular growth or symmetry.”

Further studies compared three methods of treatment for fracture dislocations in rhesus monkeys. No difference existed between those treated with internal fixation using wire ligature, those treated with maxillo-mandibular fixation, or those who received no treatment. No incidents of nonunion were reported with any closed technique.

**Length of Fixation** Traditionally the length of IMF used for adult mandibular fractures has been 6 to 8 weeks. However, this length of IMF is not without penalty. Often patients continue to lose weight during this period, they may not be able to return to work, and there is some evidence of histologic changes in the temporo-mandibular joint. Juniper and Awty were able to demonstrate that 80% of mandibular fractures treated by open or closed reduction and IMF were clinically united in 4 weeks. They were also able to demonstrate a clear relationship between the age of the patient and the predictability of early fracture union. These results were confirmed by Amaratunga. He found that 75% of mandibular fractures were clinically stable by 4 weeks, that almost all fractures in children healed in 2 weeks, and that a significant number of fractures in older patients took 8 weeks to heal. It appears that each individual case must be judged on its merits but that most uncomplicated fractures in children are united in 2 to 3 weeks, in adults 3 to 4 weeks, and in older patients in 6 to 8 weeks. Several other factors should be taken into account when deciding on the appropriate regime for a particular patient. The following situations generally require longer periods of IMF: comminuted fractures; fractures in alcoholics, particularly those with nutritional problems; fractures in patients with psychosocial handicaps; fractures treated late; and fractures with teeth removed in the line of the fracture.

**Length of Fixation for Condylar Fractures** Ideally, the period of IMF should allow for reestablishment of the preinjury occlusion and should not be longer. Increased length of the time of fixation may result in limitation in function or ankylosis of the joint. In practice, a wide variety of opinions exists over the length of time that constitutes an adequate period of fixation. Differences depend on the age of the patient, the type of fracture, and the presence of other fractures. Most clinicians agree that a shorter period is needed in children, but they are no closer in agreement over what this time should be.

Animal studies have shown excellent occlusion and postoperative function even in fracture dislocations when no IMF is used. Some studies in humans also agree with this. However, the inability to occlude the teeth without pain is frequently present in patients with condylar fractures and does require some period of fixation. Attempts to predetermine which fractures will need longer IMF than others have been made. The length of time has been based on the presence or absence of teeth, the type of fracture, and the age of the patient. However, Walker has suggested that a relatively short period of intermaxillary fixation is required for all patients regardless of age, occlusion, and type of fracture.

**Intermaxillary Techniques** dentate patients Intermaxillary techniques in dentate patients include application of arch bars (Figure 22-22), direct wiring, Ivy loop wiring (interdental eyelet wiring) (Figure 22-23), continuous wire loop technique (Stout’s method, Obwegeser’s method), cast cap splints, and IMF screws (Figure 22-24).

Methods for dentate patients usually include 0.5 mm (25-gauge) soft stainless steel wires around the teeth. In general, the wires should be handled in a similar fashion for all methods, following certain principles:

1. **Tighten the wires with a continuous tension.**
2. **Direct the force apically when tightening the wires.**
3. **Tighten all wires in a clockwise direction.**
4. **At the end of tightening, turn only half a turn at a time.**
5. **Turn the end of the wire into the interproximal embrasure.**

These additional rules apply when arch bars are used:

1. **Adapt the arch bar closely.**
2. **Use a cuspid wrap wire where indicated.**
3. **Avoid placing the wire across the intermaxillary stabilization lugs.**
4. **Use circumferential wires when single teeth stand alone, and intraosseous suspension or circum-mandibular wires in edentulous areas.**
5. **In the area of the fracture, reduction should be accomplished prior to stabilization of the arch bar on both sides of the fracture.**

![Placement of Erich arch bars for noninvasive treatment of a mandibular fracture.](image)
When IMF is used it may be applied with either elastics or wires. Elastics can be used for fracture reduction and for IMF; however, they apply a constant pressure, which can lead to muscle spasm and pain, particularly in the masseter muscle, and are difficult to keep clean. Wires, on the other hand, are easier to keep clean and are passive. However, they do loosen over time and may need to be tightened or replaced over the period of fixation.

LINGUAL OR LABIAL SPLINT To construct a lingual splint, an impression is taken of the lower arch and a stone model is poured (Figure 22-25). If there is displacement of the fracture site, an upper impression will also need to be taken. The lower stone model is then sectioned at the fracture site, and using the upper model as a guide (Figure 22-26), the correct occlusion is reconstructed. Then the sectioned model is waxed together in the correct relationship, and the lingual surface is relieved with a 1 mm thickness of wax. A hard acrylic splint is then made and holes drilled so that it can be wired to the teeth (Figure 22-27). Just before placement a thin coating of soft liner is applied. The fracture is reduced, and the splint is wired into position.

EXTERNAL PIN FIXATION In external pin fixation usually two pins on both the proximal and distal fragments are placed, if possible. The biphasic extraoral technique uses a special transbuccal trocar set. This is used for each hole through individual skin incisions. A 2.2 mm twist drill is used to drill through both cortical plates at slow speed with constant irrigation. Specially designed self-tapping, coarse-threaded screws are then placed with a socket wrench. A series of locking plates and bars are secured to the four or more pins, and then a self-curing acrylic secondary splint is constructed (Figure 22-28). External pin fixation can be used in edentulous fracture sites in which there is bone loss secondary to gunshot injuries, pathologic fractures, or osteomyelitis, or in cases in which a bone-grafting procedure has been performed. It can also be used in fractures of the atrophic edentulous mandible or in mandibular fractures associated with midface fractures when a quick and simple method of fixation is required.

FIGURE 22-23 In patients with a full and stable occlusion, Ivy loops can be applied to achieve intermaxillary fixation after closed reduction. A, Frontal view. Note there is also an Essig wire in the anterior dentition to help maintain the reduction of the right parasymphysis fracture. B, Right buccal view. C, Left buccal view.

FIGURE 22-24 An option to obtain intermaxillary fixation in patients with a reliable occlusion is the use of intermaxillary fixation screws. In most cases two screws placed on each side is sufficient to maintain the reduction. A, Right buccal view. B, Left buccal view.

FIGURE 22-25 For the fabrication of a lingual splint, the cast must be carefully sectioned along the areas where the fractures are located.
**Principles of Management of Mandibular Fractures**

**Edentulous Patients** Closed reduction in edentulous patients is achieved with Gunning’s splints or splints made from the patient’s own dentures (Figure 22-29).

**Open Reduction** Open reduction of mandibular fractures has developed to become a more frequent treatment option for the management of these injuries over the last decade. With the development of improved fixation systems, which directly translates into reduced IMF times or no IMF at all, both surgeons and patients have become more comfortable with this treatment option. Luyk stated that the significance of the rather large number of successfully managed patients using closed reduction was magnified when one considers that at the time there were no large studies on open reduction showing any improvement in the result or any decrease in the rate of complications. Today, we know that this statement has to be dissected carefully and that depending on the time elapsed between the injury and treatment, and whether the patient is taking antibiotics, this will change the outcome tremendously. In contrast, those recommending open reduction of condylar fractures have failed to report complication rates for the proposed technique or, when cited, reported complication rates that are greater than those seen historically with closed reduction; they experienced complications that have not been seen with closed techniques; and they allowed inadequate follow-up before assessing the outcome.

The major indications for open reduction of a fractured mandible are summarized below.

**Unfavorable or Unstable Fracture** Unfavorable or unstable fractures arise in several circumstances. When an angle fracture is displaced at the time of injury and is horizontally or vertically unfavorable, it is unlikely that simple IMF will maintain the proximal segment in the correct position. Under the influence of the medial pterygoid or the powerful mandibular elevator muscles (temporalis and masseter), the proximal segment most likely will be displaced. This could lead to delayed healing and possibly permanent disruption of the inferior alveolar nerve. When the fracture is both horizontally and vertically unfavorable, an extraoral approach is recommended. Also, most fractures in the parasympyseal region cannot be routinely treated satisfactorily by closed reduction because of the pull of the supraphyoid and digastric muscles. Fractures in this region tend to open at the inferior border and along the lingual surface with the superior aspects of the mandibular segments rotating medially at the point of fixation when closed reduction and IMF are used. With the medial rotation of the horizontal ramus the lingual cusps of all premolars and molars move out of occlusal contact. This results in masticatory inefficiency, and untoward periodontal changes will

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**Figure 22-26** Once the fracture segments have been manually realigned, the cast is waxed together and fast-setting stone is added to rebuild the base and allow for mounting and fabrication of the splint.

**Figure 22-27** A, The splint is fabricated, trimmed, and polished to guarantee a passive fit in the modified cast. B, Drill holes are placed in the interdental areas to allow for wires to be passed and secured around the teeth.

**Figure 22-28** A Joe Hall Morris external fixator is used to manage a severe mandibular fracture resulting from a gunshot wound. This system consists of biphasic transcortaneous pins and an acrylic frame fabricated intraoperatively.
Medically Compromised Patients Some patients with special medical conditions are best treated without IMF. They may be better treated with an open reduction. This group of patients includes those with decreased pulmonary function. Williams and Cawood have demonstrated significant decrease in pulmonary function associated with IMF.89 Patients with gastrointestinal disorders who are on a liquid diet, particularly one based on milk products, may have difficulties. Those with severe seizure disorders in which airway difficulties may arise with IMF and patients with psychiatric or neurologic problems may be candidates for open reduction.

Open Reduction of Condylar Fractures A variety of useful techniques for open reduction have been described.73,91–94 The reason for employing open reduction in each case was to avoid the complications found in closed reduction. No data or follow-up of patients was presented to document this. Tanasen and Lamberg, Zide and Kent, and Raveh and colleagues followed patients with open reduction for up to 37 months.95–97 Complication rates of 85, 50, and 10% were seen, respectively, including concomitant disk repair in their study.98 Eight of nine open reduction patients who were studied for an average of 11 months experienced complications (89%). Six of 12 patients receiving closed reduction were found to have malocclusion at the end of treatment (50%). It is possible that the high incidence of malocclusion in the closed reduction group might be a result of prolonged fixation, inadequate follow-up, and lack of supervised postoperative rehabilitation.87

There is a lack of any controlled clinical data to indicate the superiority of open reduction techniques as a primary mode of management of condylar fractures in
children or adults. Although it is apparent that, in some situations, an unacceptable incidence of complications results when closed reduction is employed, it is inappropriate to assume that an open technique can avoid these complications until this is borne out in controlled clinical trials.

Despite the evidence in favor of closed reduction as the treatment of choice for the majority of fractured condyles in both children and adults, there are indications for the performance of open reduction (Table 22-3).

In the past the indication for open reduction of a condylar fracture was primarily a radiographic one. Essentially, it was thought that the condyle behaved like other areas of the mandible or other bones in the body and that it would respond better and heal with more satisfactory function if an ideal anatomic reduction were obtained. It has been shown that there is little if any correlation between the degree of displacement or dislocation of the fracture and the ability to obtain satisfactory function with a closed reduction. A more functional approach in assessing the need for open reduction was taken by Zide and Kent. According to these investigators, indications for open reduction of condylar fractures should rely on the identification of specific clinical entities that, when treated with closed reduction, would result in a high degree of failure. They also take into account an objective evaluation of function at the time of the planned reduction, the presence and condition of the patient’s dentition, the likelihood of successfully performing a closed reduction, and the presence of other modifying factors such as the patient’s medical condition or the existence of other facial fractures.

**Absolute Indications** Absolute indications for open reduction are present in those situations in which limitation in function is highly probable if a closed reduction is performed or in those situations in which a closed reduction is not possible. Limitation of function may be caused by fracture with dislocation of the proximal segment into the middle cranial fossa, by invasion of the joint by a foreign body, by lateral extracapsular dislocation of the condylar head, or by the presence of any fracture dislocation that produces a mechanical stop, preventing mandibular movement. Inability to perform a closed reduction may result when the fracture is displaced so that it is impossible to manipulate the teeth into an appropriate occlusion.

**Possible or Relative Indications** Possible or relative indications for open reduction also exist and should be assessed on the basis of benefit as opposed to risk:

1. Bilateral condylar fractures with comminuted midfacial fractures. The rationale for open condylar reduction in these situations is that it allows for the establishment of a horizontal and vertical dimension of the midface when this cannot be achieved by other means. If rigid internal fixation of the midface is possible, then open reduction of the condyle may no longer be indicated.

2. Situations in which IMF is not feasible. Certain medical conditions, such as poorly controlled seizures, psychiatric disorders, or severe mental retardation, make maxillomandibular fixation difficult and possibly dangerous. Also, patients with multiple trauma, particularly head injury or chest injury, are at increased risk for complications if placed in maxillomandibular fixation unless tracheostomy is planned. In addition, maxillomandibular fixation is extremely difficult in those patients who have other facial fractures.

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**Table 22-3  Indications for Open Reduction of Fractures of the Mandibular Condyle**

1. **Absolute indications**
   - 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 condylar head
     4. Other fracture dislocations in which a mechanical stop is present on opening, which is confirmed radiographically
   - Inability to bring the teeth into occlusion for closed reduction

2. **Relative indications**
   - Bilateral condylar fractures with comminuted midface fractures in which rigid internal fixation of the midface is not possible
   - Situations when 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)
   - Displaced fractures where dentures or splints are not feasible because of severe mandibular atrophy
   - 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
   - In fracture dislocation in adults to restore the position and function of the meniscus (controversial)
with displaced condylar fractures in whom dentures are not present and splints are not feasible because of severe mandibular atrophy.

3. Bilateral fractures in which it is impossible to determine the proper occlusion. Occasionally, a patient with bilateral fractures will have such an ambiguous occlusion that, even with the use of study models and careful clinical examination, it is not possible to determine the appropriate maxillomandibular relation. This may lead to inappropriate placement of the mandible into malocclusion or to placement of a preexisting malocclusion into a normal relation, thereby predisposing the patient to nonunion or long-term functional disability.

4. Fracture dislocation in an adult patient to restore position and function of the disk. Previous emphasis on indications for open reduction have centered around the need for bony reduction and fixation without consideration of disk position. The unstated implication of most of the literature is that the position of the dislocated disk is not critical for optimal functional results after condyle fracture.\textsuperscript{98} However, this is contradictory, given the present emphasis on the importance of correct condylar disk alignment for management of those patients with internal derangement of the temporomandibular joint. The disk is important in the prevention of post-traumatic ankylosis.\textsuperscript{47} An interesting concept has been raised about the possible necessity for disk repositioning, especially in fracture dislocations, to allow for optimal temporomandibular joint function. Some clinicians have suggested that “open reduction and internal fixation of condylar fractures in conjunction with disk repair is a biologically sound approach….”\textsuperscript{98} Based on their experience, it might be recommended that, in fracture dislocations in which open reduction is indicated, an attempt should be made to reposition the disk at the time of the reduction. However, inadequate data exist to suggest that open reduction performed solely for the purpose of disk repositioning is valid.

\textbf{Surgical Approach  Condylar Fractures}

A variety of surgical approaches to the fractured condyle have been suggested, including intraoral, submandibular, retromandibular, preauricular, and, more recently, endoscopic. The most important factor in determining the approach used is the level at which the fracture has occurred. Modifying factors such as the degree of displacement or dislocation and the planned method of fixation may also have a bearing on the approach selected.

Traditionally fractures in the condylar neck and above were best approached through a preauricular or endaural incision.\textsuperscript{101} This approach also has the added advantage of allowing for surgical manipulation of the soft tissues within the joint, if desired. Subcondylar fractures and fractures extending into the upper ramus region are best approached using a retromandibular or Hinds approach.\textsuperscript{102} The incision begins approximately 1 cm below the lobe of the ear and 1 cm posterior to the ramus of the mandible. The dissection is carried down to the parotid gland, which is retracted anteriorly, providing access to the vertical fibers of the masseter muscle overlying the ramus. These fibers are not stripped but instead are separated bluntly along their vertical course, allowing access to the underlying ramus. Access can easily be gained to relatively high subcondylar fractures through this approach, and a variety of fixation techniques are possible without additional percutaneous puncture, as may be needed if a submandibular approach is used. Low subcondylar fractures, especially those without a significant degree of displacement, may be easily approached from an intraoral incision.\textsuperscript{103} In severe anteromedial fracture dislocations in which the condylar head is not retrievable despite the choice of approach, a vertical ramus osteotomy, followed by removal of the osteotomized segment, has been recommended.\textsuperscript{104–106} This allows for access to the proximal condylar head, which is located medially and is also removed. Rigid fixation with plates or screws is carried out between the ramus segment and condylar head. The unit is returned as a free autogenous bone graft, and the osteotomy is plated. This technique is useful for high dislocated fractures and may be accomplished through a retromandibular approach.

\textbf{Other Mandibular Fractures}

Open reduction of mandibular fractures prior to the advent of antibiotics was associated with a high incidence of infection. Following the introduction of antibiotics, most clinicians used the extraoral approach to the fracture site. This technique, however, is time-consuming, results in a visible surgical scar, and can damage adjacent structures, particularly the marginal mandibular branch of the facial nerve. Transoral open reduction has been advocated as an excellent alternative.\textsuperscript{107–110} The technique is claimed to be quicker to perform, results in no extraoral scar, and does not damage the facial nerve. Less postoperative wound care is required, and it is simple to perform the techniques under local anesthesia. Transoral open reduction of mandibular fractures is useful in tooth-bearing portions of the jaw (ie, in symphyseal, body, and angle fractures). Complications rates and infection rates appear to be similar between the two techniques when large numbers of cases are studied.\textsuperscript{111,112}

Occasionally, a combination of approaches is necessary, particularly in fracture dislocations in which a preauricular approach may be necessary to retrieve the proximal segment, while fixation is performed through another approach.\textsuperscript{113}
Throughout the past decade, surgeons have become interested in the concept of minimally invasive surgical approaches to avoid potential patient morbidity from more traditional open surgical techniques. With the development of these techniques, management of these injuries via an endoscopic approach has gained great popularity among surgeons. In 1994 Ma and Fang were the first ones to describe the use of an endoscope to access the mandibular angle region. Later Jacobovicz and colleagues modified this technique for the management of condylar fractures. Recently, more authors have also described their experience with this approach.

The surgical approach, as described by Miloro, requires a 15 to 20 mm modified Risdon incision to gain access to the lateral ramus. A subperiosteal dissection is then performed blindly to create an “optical cavity” on the lateral aspect of the ramus on the fracture side from the sigmoid notch to the inferior border and from the mandibular notch anteriorly to the posterior border of the ascending ramus posteriorly. A modified Storz retractor with a curved end is then placed through the incision and below the periosteum to engage the sigmoid notch. A 4 mm, 30˚ endoscope is used for retraction and visualization of the surgical site. Following irrigation and the use of a suction elevator, the sigmoid notch, inferior border, mandibular notch, posterior border, and the fracture site can be clearly identified endoscopically. The fractured segments are then repositioned and reduced. Inferior traction on the angle of the mandible, although limited by IMF, can be helpful in the mobilization of the segments. Fixation is achieved with a 2.0 mm titanium miniplate and screws through a preauricular stab incision and trocar. Following reduction and stabilization, the IMF is released for evaluation of the occlusion.

**Methods of Fixation** Once access to the fracture has been achieved, any number of fixation devices may be employed (Figure 22-31). In a given situation, any one of these techniques may have certain advantages over the other. With the development of sophisticated rigid internal fixation systems and instrumentation for their placement, miniplate fixation of these fractures will be the technique most readily employed in most cases. Miniature bone plates can be applied using any of the previously discussed approaches. These plates have the advantage of being available in a wide variety of shapes and sizes; they are now readily available in most operating rooms; and they provide a more stable form of fixation than do wires or Kirschner wires. Theoretically, bone plates have another advantage—they can be placed on a relatively small proximal fragment first, allowing for the creation of a handle to more effectively manipulate the proximal segment into an appropriate reduction. Should the incision selected not allow for total access to the fracture, currently available bone-plating systems are equipped with instrumentation for percutaneous placement of screws.

**Wire** Intraosseal wiring (wire osteosynthesis) can be placed either by an intra- or extraoral route using one of three basic techniques:

1. A simple straight wire across the fracture site (Figure 22-32A). This should be placed so that the direction of pull of the wire is perpendicular to the fracture site. This technique can be either through both the buccal and lingual cortical plate or it may be used on the buccal cortical plate only. This is useful in the angle region, where a third molar socket can be quickly and easily used for a simple straight buccal cortex wire.
2. Figure-of-eight wire (Figure 22-32B). This wiring technique has been shown to have increased strength compared with simple techniques at both the inferior and superior borders in angle fractures.
3. Transosseous circum-mandibular wiring (Obwegeser’s technique) (Figure 22-32C). This is a useful wiring technique when the fracture runs obliquely compared with the inferior border of the mandible. If the fracture line is too vertical the wire could become displaced into the fracture line.

The wire used should be a pre-stretched soft stainless steel, and the fracture should be held in a reduced position while the wire is being tightened so that the wire does not reduce the fracture and possibly lead to wire breakage.
FIGURE 22-31  Previously reported techniques for direct stabilization of condyle fractures: A, Silverman (1925); B and C, Thoma (1945); D, Stephenson (1952); E, Robinson (1960); F, Robinson (1962); G, Messer (1972); H, Kobert (1978); I, Petzel (1982). (CONTINUED ON NEXT PAGE)
RIGID FIXATION

Dissatisfaction with the use of IMF as a means of treatment of mandibular fractures has resulted in the development of open reduction and fixation techniques that do not require the teeth to be wired together. Criticism of the disadvantages of prolonged immobilization of the jaws has included patient complaints of panic, insomnia, social inconvenience, phonetic disturbance, loss of effective work time, physical discomfort, weight loss, histologic changes in the condylar head, and difficulty recovering a normal range of jaw movement. This has led some clinicians to seek alternative methods of treatment, including the use of rigid internal fixation.

The principal disadvantages of the compression plating systems for mandibular fractures are the use of an external approach, thus giving rise to facial scarring and the potential for damage to the mandibular branch of the facial nerve, and the use of very rigid plates, giving rise to “stress shielding,” although this has never been shown to be a problem in mandibular fractures. Also, the position of the teeth and inferior alveolar nerve and the use of bicortical screw fixation necessitate that the compression plates be placed in areas of compression rather than tensile forces, and therefore, additional techniques are required to overcome the tension forces. Another disadvantage is that removal of the plates is advocated.

The second major group of plate fixation techniques is the monocortical miniplate osteosynthesis, which was first described by Michelet and colleagues and then modified and popularized by Champy and colleagues. The principal advantages of this technique over compression plating systems are the use of the intraoral approach and the positioning of the plates in the juxta-alveolar area where tensile strain occurs when the mandible is loaded. The healing that results from the use of this system in humans has not been demonstrated clearly. At least one group of authors claims that the system gives rise to rigid fixation and that it results in primary bone healing, although no evidence is provided to support this assertion. In view of the small malleable nature of these plates and the fact that the system is monocortical, it would seem more likely that the technique is only semirigid and would result in callus formation and secondary bone repair. This is not to imply that it is an inferior technique, because callus formation generally gives rise to quicker and stronger early bone repair.

Special Situations

Edentulous Fractures

The edentulous mandible in the trauma patient has several factors modifying its behavior that the dentate mandible does not. The loss of the teeth results in resorption of the alveolar bone, which weakens the mandible. The loss of bone also means that there is less cross-sectional area of bone in contact in fracture patients and less periosteum and endostem to supply the osteogenic cells for fracture healing. Because of the aging process the majority of the blood supply to the edentulous mandible is from the periosteum rather than the inferior alveolar
A larger percentage of fractures in the edentulous patient are not compound because of the lack of teeth. Minor displacement of the bones can be easily accommodated in the construction of new dentures. The edentulous population also tends to have more health problems resulting from conditions such as osteoporosis, diabetes mellitus, and steroid therapy, which may directly affect bone healing. The site distribution of fractures tends to be different in the edentulous patient, with a higher percentage of body fractures (43.5%) and lower percentages of angle (15.2%) and symphysis (4.3%) fractures (Figure 22-33). A 20% incidence of nonunion has been reported in the treatment of edentulous fractures, particularly when nonrigid fixation was applied in open reduction cases. Longer periods of immobilization have also been shown to be necessary to achieve satisfactory healing.

The anatomic site influences treatment. If the location of the fracture is posterior to the denture-bearing area, then either additional fixation (eg, external pin fixation) or open reduction and fixation may be necessary to control the proximal fragment. Muscle pull on the edentulous jaw is considerably weaker than in a dentate mandible and undisplaced fractures are often closed injuries. Therefore, if the fragments are undisplaced or minimally displaced and not mobile, conservative therapy may be all that is necessary. More definitive treatment will be necessary if the fragments are displaced or excessively mobile. The bilateral body fracture deserves special mention because the pull of the suprahyoid muscles tends to displace this fracture inferiorly. These usually occur in the pencil-thin atrophic mandible. A variety of treatment modalities have been suggested to treat these difficult fractures including open reduction with rigid internal fixation, closed reduction with and without bone grafts, and external pin fixation. When the edentulous mandible is comminuted again because of the poor blood supply to the bone fragments, those fragments are best managed by closed reduction. The use of semirigid fixation systems without some form of IMF is not indicated in this patient subset.

External pin fixation by the biphasic technique is often used in edentulous fractures. It obviates the need for IMF, thus allowing early mobilization of the jaw and improving feeding in some patients. It can be used in comminuted fractures without jeopardizing blood supply to the fractures, and it can also bridge a bone loss gap before bone grafting.

Fractures in Children As previously mentioned, fractures in children are less common than in adults. Their management is complicated by the presence of deciduous teeth, which may be mobile during the mixed dentition stage and whose shape has little in the way of undercut areas, which means that they do not retain wire as well as adult teeth. The presence of tooth buds reduces the area available for interosseous fixation, and there exists a greater potential for ankylosis and growth disturbances in the younger population. Also, children do not tend to tolerate IMF as well as adult patients. On the other hand, fractures tend to heal quicker in children and slight malocclusion problems can be compensated for by growth of the patient.

Children make up about 5% of all mandibular fractures. These fractures are rare in children under 5 years of age because of the greater elasticity of the bone and lighter weight of children, which lowers the
forces of impact during falls. Condylar fractures appear to be common, affecting about 46% of patients either alone or in combination with other fractures.126

Mandibular fractures in children can often be successfully managed by acrylic splint therapy of the mandible only or with eyelet wires and IMF.126,127 A shortened period of IMF, 2 to 3 weeks, is all that is required. When an open reduction is required, it has been successfully accomplished by the extraoral route using inferior border wiring in order to avoid the tooth buds.128,129

If adequate bone height is available below the area where the tooth buds are located, the use of resorbable plates offers a great advantage to fixate these fractures (Figure 22-34).

Complications are rare in this group of patients. Malunion, nonunion, and infection tend to have a low incidence.67 Two serious complications that can occur, however, are ankylosis and growth disturbances. Both of these tend to be more common with intracapsular condylar fractures and when the damage is of a crushing nature.130 The incidence and severity of these complications can be reduced by shorter periods of IMF and close follow-up.

Management of Teeth in the Line of Fracture In the past, teeth in the line of the fracture were always removed.23,131,132 Their removal was advocated because fractures of the dentate portion of the jaws are compound via the periodontal ligament and it was believed that this communication fostered infection, osteomyelitis, and nonunion. However, Neal and colleagues, Kahnberg and Ridell, Schneider and Stern, and Amaratunga have all been able to show that the majority of teeth in the fracture line can be saved if appropriate antibiotic therapy and fixation techniques are used.133–136 The impacted mandibular third molar tooth deserves special mention. Most authors have advocated leaving the tooth in situ if the tooth is not in direct communication with the mouth, no periodontitis exists, and reduction of the fracture is achievable without removal. Shetty and Freymiller reviewed the indications for removal of teeth in the line of the fracture as follows137:

1. Teeth grossly loosened, showing evidence of periapical pathology or significant periodontal disease
2. Partially erupted third molars with periodontitis or associated cyst
3. Teeth that prevent reduction of fractures
4. Teeth with fractured roots
5. Teeth with exposed root apices or entire root surface from the apex to the gingival margin
6. An excessive delay from the time of fracture to definite treatment

Use of Antibiotics

Zallen and Curry demonstrated that with compound mandibular fractures, an infection rate of 50% can be expected in those patients who do not receive antibiotic therapy.138 A prospective trial was undertaken in which only dentate compound mandibular fractures were evaluated. One-half of the patients in this study received “prophylactic antibiotics,” usually penicillin. It was not stated for how long the antibiotic therapy was continued or when it started in relation to the injury. One-half the patients who did not receive antibiotics had infections at the fracture site as opposed to only 6% of those who did receive antibiotics. It seemed to make little difference whether the fractures were treated by open or closed reduction.

All fractures in this study were treated within 36 hours. Another study has confirmed these results in facial fractures and has suggested that short-term prophylaxis as is used in elective surgery may be as effective as the more usual 5-day course of antibiotics.139 This group also found little difference in the incidence of infection whether there was a delay in treatment of mandibular fractures or not.

Penicillin should remain the antibiotic of choice for compound mandibular fractures whether closed reduction or open reduction is contemplated. The antibiotic prophylaxis should begin preoperatively and be continued for not more than 24 hours postreduction.

Complications

Delayed Union and Nonunion

Nonunion is distinguished from delayed union by the potential of the bone to heal. Delayed union is a temporary condition in which adequate reduction and immobilization eventually produces bony union. On the other hand, nonunion may persist indefinitely without evidence of bone healing unless surgical treatment is undertaken to repair the fracture. Nonunion is generally characterized by pain and abnormal mobility following treatment. Malocclusion may be present in dentate cases and mobility exists across the fracture line. Radiographs demonstrate no evidence of healing and in later stages show rounding off of the bone ends. Delayed and nonunion occur in about 3% of fractures.140

There are several causes and contributing factors. The most common reason is poor reduction and immobilization.141 This is more likely in edentulous fractures. Infection is often an underlying cause, and any tooth in the line of the fracture must be carefully assessed for root fracture and vitality. A decreased blood supply can lead to delays in healing. Excessive stripping of

FIGURE 22-34 Use of a resorbable plate for fixation of a symphysis fracture in a 4-year-old child.
the periosteum, especially in comminuted and edentulous fractures, can lead to delayed healing. Metabolic deficiencies and alcoholism are also significant contributors to delayed healing. Cannell and Boyd showed a high incidence of delayed union and nonunion in a group of alcoholic patients. These patients were probably also at increased likelihood to sustain a mandibular fracture. Although the exact reasons for delayed healing in this group of patients is not known, they are known to have metabolic and vitamin deficiencies, poor compliance particularly with IMF, poor bony quality, and impaired local blood supply, all of which could be contributing factors. These patients should be treated whenever possible with closed reductions, because this treatment has a lower incidence of complications in this group of patients.

Treatment of delayed union and nonunion is aimed at eliminating the underlying cause of the problem. When infection is present it must be managed with débridement of sequestra, drainage, and antibiotic therapy. Loose fixation such as wires and plates must be removed, and adequate fixation with IMF, extraoral pin fixation, or even rigid plate fixation should be applied across the fracture site. If there is a gap between the bone ends, a bone graft may be necessary.

**Malunion**

Malunions can be defined as a bone union of the fracture in which some displacement of the bones still exists. Not all malunions of fractured mandibles are clinically significant. Often malunions in edentulous patients or those involving the ramus and condylar area of the mandible result in no clinically detectable alteration in appearance or function. When, the dentate portion of the jaw is involved, however, a malocclusion can result. The rates of malocclusion in patients treated with IMF tend to be very low. In one prospective trial between rigid internal fixation and standard techniques the rate of malocclusion with the rigid fixation was three times higher. However, as the authors concede, they were initially inexperienced with the technique and others have reported a low incidence of malocclusion. Malocclusion can be corrected by further or prolonged IMF in the early stages of healing, and selective tooth grinding, orthodontics, or osteotomies after complete bony union.

Malocclusion that does not result from growth alterations but from a malunion of the condyle fracture occurs infrequently if an adequate follow-up regimen is followed. If malocclusion does persist, its management is similar to the management of malocclusion from other causes. Judicious use of equilibration, orthodontics, and orthognathic surgery allows for restoration of a functional occlusion. Before reconstructing the occlusion to this new articulation, it is necessary to allow a period of 6 to 12 months for complete healing and for any remodeling of the articulative apparatus to occur.

**Nerve Injury**

Traumatic injury to the inferior alveolar nerve is common in displaced fractures of the body and angle of the mandible. There

![Figure 22-35](image)
are few studies documenting recovery of the nerve. Larsen and Nielsen reported a permanent disturbance in mental nerve function in 8% of 229 patients studied.\textsuperscript{147} Return of nerve function depends on the degree of initial trauma to the nerve and an accurate reduction and adequate fixation of the mandibular fracture. Rarely other branches of the mandibular division of the trigeminal nerve can be affected. These include the masseteric nerve, auriculotemporal nerve (both with condylar fractures), and the buccal and lingual nerves associated with intraoral lacerations with body or angle fractures. Also rare is damage to the marginal mandibular branch of the facial nerve with fractures of the condyle, ramus, and angle of the mandible. It is more common to see this nerve damage caused by a laceration along its course.

Most fractures of the mandible heal with relatively simple management. All clinicians must be wary of overtreatment of simple cases that can lead to an increase in cost of treatment for both the patient and society and also an increase in complication rates.

**Growth Alteration**

Growth alterations as the result of condylar injury may occur as the result of two mechanisms. Over- or understimulation of normal growth may result from direct injury to the condyle, or a restriction of normal growth may occur secondary to fibrosis or scarring of the surrounding tissue.

It was once thought that fracture of the condyle produced a growth deficit in proportion to the age of the patient at the time of injury: the younger the child, the greater potential growth problem.\textsuperscript{120} However, although it is true that children undergo several periods of rapid growth during their development and that an injury during one of these growth periods may be associated with a higher incidence of growth alteration,\textsuperscript{78} other factors are involved that alter this simplistic theory. Frequently, complete regeneration of the condyle occurs in young patients, with no residual deficit following fracture, and better regeneration occurs in actively growing patients, particularly those under the age of 12 years.\textsuperscript{148,149} This clinical observation is supported by experimental studies,\textsuperscript{104} which found that, following surgically created fracture dislocations in young monkeys, excellent regeneration occurred with no growth disturbance in any of the animals. This ability for restitution of growth in children under the age of 12 years appears to account for the lack of direct correlation between the age of injury and the degree of growth disturbance—a correlation that would be expected if the sole determinant were the amount of growth left at the time of injury.

The concept that the condylar cartilage acts as a growth center has been replaced by the theory that the cartilage acts as a remodeling center.\textsuperscript{150} The restitution of growth seen after condylar injury (which at times may actually lead to overgrowth of the affected condyle) is a direct result of this remodeling center within the condylar cartilage reacting to a traumatic episode. It is not unusual for a new condylar apparatus to develop, with resorption of the displaced or dislocated condylar head. This compensatory growth seems to depend on the potential space created by the displacement of the stump of the condylar process.\textsuperscript{150} For this reason, it is important to maintain the mandible in its original occlusion, not only for a few weeks during healing, but also for the next several months while bony regeneration and compensatory growth occur. Even when occlusion is maintained and the patient is of the ideal age, 25% of subjects experience a growth disturbance.\textsuperscript{148,149,151} Because of this, adequate patient education and long-term follow-up for several years is necessary in children with fractures of the condyle (Figure 22-36).

**Temporomandibular Joint Dysfunction**

A wide range of temporomandibular joint problems may result from injuries to the condylar apparatus. Internal derangement and ankylosis are perhaps the two most common.

**Internal Derangement** A correlation exists between previous condylar fracture and the development of internal derangement of the temporomandibular joint. There is a greater incidence of temporomandibular joint pain, deviation on opening and joint noise in patients with previous condylar fractures.\textsuperscript{71} The resultant internal derangement primarily occurs in adults and is of two broad types. The first is internal derangement that occurs on the side of the fracture and results from soft tissue injury within the joint. Open reduction with direct repair of the injured soft
Ankylosis Ankylosis is a rare complication of mandibular fractures. It is more likely to occur in children and is associated with intracapsular fractures and immobilization of the mandible. The most commonly accepted etiology is of intra-articular hemorrhage, leading to abnormal fibrosis and ultimately ankylosis. In children, if left untreated, it results in disturbed growth and underdevelopment of the affected side. Prevention is easier than cure, and the use of only short periods of IMF in children can help reduce the occurrence of this complication. Management once the condition is established is surgical with a temporomandibular joint arthroplasty, wide resection of the ankylosis portion of bone, coronoidectomy, and reconstruction with a costochondral rib graft, with active early and prolonged mobilization and exercises.

Although development of internal derangement seems to occur solely in adult patients, ankylosis is much more common in children (Figure 22-37). Factors contributing to the development of ankylosis have been outlined. They include the site and type of fracture, the age of the patient at the time of injury, the duration of IMF, and the extent of damage to the disk.

The site and type of fracture may play an important role in whether or not ankylosis occurs. It is widely accepted that intracapsular fractures are more likely to develop ankylosis. The postinjury relation of the condylar stump with the glenoid fossa is also a factor. With fractures of the condylar head, a greater likelihood exists that there will be intimate contact between the proximal portion of the distal segment and the glenoid fossa, predisposing the patient to ankylosis. Failure to produce ankylosis after experimentally induced condylar fractures, coupled with the clinical observation that the incidence of intracapsular fracture is much higher than that of ankylosis, leads one to believe that other factors besides the site of fracture must be operative in the production of ankylosis.

The condyle of a young child is more easily crushed than fractured, possibly because the cortical bone of the child is relatively thin and the condylar neck broad. The immediate subarticular layer is also extensively vascularized. An impact leading to a crush injury is more common in a child because of these anatomic differences, and the resulting fragments of highly vascularized osteogenic material that are dispersed throughout the joint space may be the cause of ankylosis. This theory helps to explain the clinical observation that there is a greater predisposition for post-traumatic ankylosis in patients sustaining such injuries before the age of 10 years.

It is widely accepted that the length of the maxillomandibular fixation may play a role in the development of ankylosis. Markey was unable to produce ankylosis after experimentally induced fracture with prolonged maxillomandibular fixation. In studies performed by Beekler and Walker, ankylosis occurred with prolonged fixation, while no ankylosis could be created in a moving jaw. This confirms the observation that the duration of immobilization is contributory to the development of ankylosis, although it is not the primary determinant. The location and condition of the disk may be another determinant in the occurrence of temporomandibular joint ankylosis because one never finds the disk in the area of temporomandibular joint ankylosis. Experimentally, ankylosis has been created in a baboon by a combination of bilateral fractures of the condylar process, diskectomy, and prolonged immobilization, while the same procedure without diskectomy did not produce ankylosis. Thus far, this discussion has been limited to the development of true ankylosis with the formation of a bony or fibrous union within the joint itself. There is also the potential for the development of pseudo ankylosis if soft tissue trauma surrounding the joint leads to fibrosis and scarring or (in the case of zygomatic arch and coronoid fractures) a bony union develops between other fractured areas and not within the joint itself.

In summary, it is likely that the following groups of patients will be at high risk for development of ankylosis: patients under the age of 10 years at the time of injury; patients with intracapsular fractures and fracture dislocations with gross telescoping;
Principles of Management of Mandibular Fractures

Other Complications Associated with Condylar Fractures

When the condylar head is forced posteriorly in the process of fracture, some force is directed against the posterior and superior walls of the glenoid fossa. Fracture of the tympanic plate may occur. In addition, partial obstruction of the external auditory canal may result, causing a conductive hearing loss because of the close proximity of the middle ear. Patients with a history of a condylar fracture should undergo a careful otoscopic examination to evaluate the condition of the anterior wall of the external auditory canal, as well as to observe for signs of potential middle ear injury. Appropriate consultation must be obtained if injuries of this nature are indicated by clinical examination or history. Basilar skull fracture along the floor of the middle cranial fossa may also occur from a similar mechanism, resulting in cerebral contusion. The fracture may also spread through the petrous portion of the temporal bone, resulting in injury of cranial nerves VII and VIII and a neurosensory hearing deficit (as opposed to a conductive deficit), facial nerve paralysis, and possibly Battle’s sign.

If either of the fracture segments encroaches on the infratemporal fossa, trauma to the nerves or vessels in this area may occur. Damage of a large vessel can result in hematoma formation or development of a false aneurysm. This expanding hematoma or false aneurysm may also cause injury to the seventh cranial nerve. The third division of the cranial nerve V may also be injured by the displaced condylar segments. If aberrant reinnervation occurs from this injury, the late complication of auriculotemporal syndrome may result.

Postoperative Management

Regardless of the technique employed for treatment of the mandibular fractures, the postoperative management of the patient is critical for long-term successful rehabilitation and return to function.

In cases in which open reduction internal fixation is employed without the use of postoperative IMF, follow-up visits should be used as reinforcement sessions to remind the patient about proper diet and progressive increase in function. It has been our experience that in many respects this group of patients should be monitored more closely than those treated with IMF to prevent possible postoperative complications secondary to their injudicious or untimely return to normal diet and function.

The proper length of maxillomandibular fixation (if used), the duration and frequency of evaluation by the surgeon, the early detection of potential complications, the judicious use of physiotherapy, and proper patient education are all necessary. In most cases some form of IMF will have been employed. The length of the fixation period, as previously discussed, varies between 2 to 8 weeks depending on many factors. At the end of this period, a systematic approach for removal of the fixation is desirable. A follow-up regimen similar to that described by Walker must then be instituted. This allows for wound healing monitoring, oral hygiene reinforcement, and observation of adequate dietary intake. It also gives the clinician the opportunity to control the occlusion in those patients who need further stabilization, while encouraging early movement in those patients who have stable occlusions. It is impossible to predict on the basis of the type of fracture which patients will need continued aggressive elastic guidance to maintain their occlusion. Children of less than 12 years of age rarely require more fixation, but patients over the age of 12 years show extreme variability, regardless of fracture type. If the occlusion is stable and reproducible at the time of IMF release, then jaw-opening exercises are begun. If aggressive physiotherapy is initiated after release of IMF for treatment of a condylar process fracture, the patient should be evaluated in 24 hours to confirm the presence of a stable occlusion. The arch bars are left in place and training elastics are used. The purpose of these elastics is to permit function, while maintaining the occlusion. An effective way to accomplish this is to gradually reduce the use of elastics over a period of time. Initially, elastics should be used 24 hours a day. They should be placed lightly during the daytime to assist in guiding the mandible into occlusion, particularly if significant deviation is present, and applied more tightly at night. After 1 week, it may be possible to completely abandon daytime elastic fixation and continue with relatively tight elastic fixation at night. After another 1 to 2 weeks of this therapy, assuming that continued maintenance of a normal occlusion is present, the patient should be allowed to function without any guiding elastic fixation for approximately 1 week. If, at that time, there continues to be a stable occlusion, further evaluation should continue for other problems, such as limited mouth opening or pain, and the arch bars may be removed. If, on the removal of the IMF or at any time during the training period, the occlusion becomes unstable and nonreproducible, an additional period of tight intermaxillary fixation with wires or elastics is indicated for 1 or 2 weeks. Clinical experience seems to indicate that a longer period of controlled elastic traction is often needed in adults with displaced or dislocated fractures, particularly if these are bilateral. Even with judicious use of guiding elastic fixation, patient education, and careful continued evaluation, malocclusion...
persists in some patients. In these cases one must consider equilibration, orthodontics, osteotomies, or a combination of these to correct the malocclusion.

Throughout the post-IMF period, aggressive maintenance of range of motion is necessary. In some patients this may be as simple as instructing them to open their mouths as wide as possible in a symmetrical manner. Other patients may initially require daily evaluations and forced opening by the surgeon. Manually forcing the teeth apart, use of a ratchet, mouth props, progressive wedging of tongue blades between the teeth, or other more sophisticated physiotherapy devices are all effective means of regaining pre-injury interincisal opening.

The success or failure of any proposed treatment for the fractured mandible, whether by open or closed reduction, will necessarily hinge on the careful adherence to sound physiologic and surgical principles and to close post-operative follow-up.

References
The results of epidemiologic surveys on maxillary fractures differ with the politics and population density of the geographic region studied, the era in which the surveys were performed, the socioeconomic status of the population, and the institution whose experience was reviewed.\(^1\)\(^-\)\(^5\) It is difficult to make generalized statements about the findings of these studies, but trends do exist, and these trends make it clear that maxillary fractures are more frequently associated with motor vehicle accidents and motorcycle accidents than with any other cause. Maxillary 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.\(^9\) In 1893 Otto Lanz also described the creation of an iatrogenic maxillary fracture for access to a tumor.

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 23.1-1).

**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.

**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 den-toalveolus, 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 below 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 zygomaticocotemporal suture is also separated. The fracture then extends along the sphenoid bone, separating the pterygoid plates. The septum becomes separated at the cribiform 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. Virtually all combinations of Le Fort I, II, and III fractures are possible on either side of the face.

In Garretson's 1898 treatise the primary method of treating fractures of the maxillae was to construct a bandage or dressing that elevated the mandible into occlusion and secure it 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.”

Blair gave a very good description of the anatomy of maxillary fractures and of the examination for diagnosing such fractures. He noted that mandibular bandages were insufficient to stabilize maxillary fractures and 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.

Similar treatment modalities were presented by Brophy in 1918; he presented illustrations of the splints as well as 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°. 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 origins of the small facial muscles. The lateral surface of the maxillae forms the infratemporal fossae and 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 lateral nasal walls. This includes the nasal concha and
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 alveolus, which contain the teeth. The posterior border abuts the sphenoid bone and the pterygomaxillary suture. Within the maxilla is the maxillary sinus. This 34 × 33 × 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. 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, and the pterygomaxillary suture. When joined to the maxilla the four bones represent one unit (Figure 23.1-2).

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 palatine bones. The superior border is thick and articulates with the ethmoid bone.

The ethmoid bone is cuboidal and extremely pneumatized; thus, it can be easily fractured and comminuted. The cribriform plate of the ethmoid comprises 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.

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 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 palatine bones and anterior to the pterygoid plates of the sphenoid.

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.

**Diagram**

![Disarticulated midfacial skeleton demonstrates the anatomy of the maxilla, the zygoma, the nasal bones, and the nasal septum.](image)
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. The examiner should also observe the maxilla for movement as a unit, while palpating the forehead, the nasal bridge, and the zygomaticofrontal sutures. The nose should be examined grossly for contour irregularity (Figure 23.1-3). A nasal speculum should be used to identify compound fractures of the septum or septal hematoma. Both hands should be used to palpate the orbital rims and in particular the zygomaticomaxillary suture. The intraoral examination should be complete, and the examiner should note accumulation of blood, debris, or avulsed teeth that could compromise the airway, as well as the presence of laceration, abrasion, or ecchymosis. Abnormal occlusion with an anterior open bite and posterior prematurities should be noted and correlated with pretraumatic occlusion if possible (family members, photographs, dental records).

**Imaging**

Fractures are identified clinically and confirmed radiographically. In the past the Waters’ view and lateral facial radiographs were used in identifying maxillary fractures and may still be used today in remote areas without access to a computed tomography (CT) scanner (Figure 23.1-4). Fine details of the fracture sites are difficult to visualize. Axial and coronal CT scans of the midface should be obtained if a scanner is available (Figure 23.1-5). If clinical evidence strongly indicates maxillary fracture (midface mobility and malocclusion with intact mandible), then CT imaging is a confirmatory test for maxillary fractures. 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 23.1-6).

**Treatment**

Patients do not die of maxillary fractures, but they may die of concomitant injury or failure to manage the sequelae of maxillary fractures. As is true for all injuries initial attention should be directed at establishing an airway and controlling hemorrhage. The most frequent cause of hemorrhage in Le Fort level fractures is a fractured septum. This bleeding may be addressed by placing nasal packs of one of a number of materials, including gauze packing, Merocel packing (Medtronic Xomed), Rhinorocket (Shippert Medical Technologies Corp.), and Epistat (Medtronic Xomed). Bleeding from sites of laceration or abrasion may be controlled by tamponade. Exsanguinating hemorrhage is rarely encountered with facial fractures; however, its occasional occurrence has long been noted: “Hemorrhage, which is not readily amenable to successful treatment, as in the case of rupture of the internal maxillary artery or its terminal branches, may be
followed by fatal results. Should uncontrollable bleeding be encountered, the patient should undergo angiographic evaluation with embolization of the injured artery if indicated. At least one group has suggested caution in the use of embolization because of the possible crossover of the embolic material between the external and internal carotid circulation.

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.

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 can be used, tracheotomy is a final option (Figure 23.1-7). After the airway has been secured and general anesthesia has been administered, arch bars should be placed, along with any required splints or stents. 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 the repair of 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 23.1-8).

Although many wiring techniques have been described in the past, rigid internal fixation is the standard of care. 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 it may be necessary to use fixation to the nasal bones, the orbital rims, or the zygomaticofrontal sutures. 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 with the repair of orbital or zygomatic fractures before 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 by bone contact. For this purpose screws with an outer diameter of 1.5 mm are adequate.
In areas such as the orbital rim or nasal bone, 1.3 mm or 1.0 mm systems may be used. In cases in which bone contact is decreased because of comminution, 1.7 mm 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 23.1-9). 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 in an inferior-anterior movement disimpacts the maxilla. Further assistance may be provided with Hayton-Williams forceps used in conjunction with the Rowe disimpaction forceps.

If the maxillary fracture is incomplete (eg, greenstick fracture), the surgeon may have difficulty in mobilizing the maxilla. The fractured hemimaxilla may be impacted or telescoped, 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 is not as novel as it might sound; in 1914, Blair wrote, “...if the impaction cannot be broken up ... resort may be had to a small, sharp chisel.” After down-fracture the maxilla can easily be moved into appropriate occlusion and stabilized without further difficulty (Figure 23.1-10).

Immediate bone grafting has been advocated for the severely comminuted maxillary antrum. This treatment prevents prolapse of the facial soft tissue into the maxillary sinus and the facial deformation that results. Titanium mesh works well 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 (ie, ciliated respiratory epithelium, goblet cells, squamous epithelium) (Figure 23.1-11).

**Surgical Splints**

In cases of gross comminution, periodontal disease, or inadequate partial dentition (less than three occluding teeth per sextant), occlusal wafers or palatal splints are useful. These splints are fabricated after impressions have been taken and model surgery has been completed. When an occlusal wafer is fabricated it should cover the occlusal surfaces and the heights of contour, but it should not encroach 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, as might be done with an orthognathic surgical splint.

The Gunning’s 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 with arch bars or suspension brackets.

Dentures can also be secured to the jaws...
with bone screws before intermaxillary fixation is attempted.

**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 avulsion injuries associated with maxillary fractures. The priority in treating these injuries is to preserve as much of the remaining tissue as possible. Consideration and administration of a narrow-spectrum antibiotic directed at oral and nasal contaminants, as well as tetanus prophylaxis, are a priority in these injuries. As is true for 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.\(^4^9\) 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.\(^2^0\)

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.\(^4^1\)

**Injuries to Geriatric Patients**  Geriatric patients who suffer a Le Fort injury pose a special concern. Additional medical illnesses and disabilities may render general anesthesia quite risky for these patients. 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.

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’s splint may also be fabricated. Such a splint may be fixed to the zygoma, the anterior nasal spine, the piriform rim, or the palate, either with wires or cortical bone screws.

**Pediatric Maxillary Fractures**  Pediatric maxillary fractures occur infrequently. Because the pediatric sinuses are not highly pneumatized, these fractures tend to be less comminuted in children than in adults. No long-term studies have been undertaken with populations large enough to determine what alterations in maxillary growth will occur after pediatric maxillary fractures. When fixation is undertaken, consideration should be given to the contour and the root length of the primary dentition. The use of occlusal splints and skeletal fixation should be entertained.

Resorbable plating systems have been advocated for use in pediatric patients so that potential complications of translocation, extrusion, and growth restriction can be avoided.\(^4^2,^4^3\) Triana and Shockley reported the use of an L-lactic acid and glycolic acid resorbable plating system; advantages of the system include ease of contouring the plates, appropriate rigidity of the systems, resorption within 12 months, no increased risk of postoperative wound infection, and the apparent absence of growth restriction.\(^4^2\)

**Complications**

Complications associated with maxillary fractures and their repair are listed in Table 23.1-1. 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.

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. 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 re-reduction or the use of bone wax. Hemorrhage from a major artery requires emergency tamponade; if the source 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.

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 often seen in fracture patterns involving the orbit, often with a more severe mechanism of injury. 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. 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. Nonunion of the fractured segments can occur as the result of inadequate blood supply, inaccurate position, movement of segments, infection, or nutritional deficiencies. Infections may be caused by contaminated soft tissue lacerations or foreign bodies, hematomas, or odontogenic infections from previously diseased or fractured teeth. 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.

Acknowledgments
The authors thank Flo Witte, MA, ELS, for her expert editorial assistance.

References

Table 23.1-1 Complications Associated with Maxillary Fractures

<table>
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<th>Complication</th>
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<tr>
<td>Infraorbital nerve paresthesia</td>
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<td>Enophthalmos</td>
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<td>Infection</td>
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<td>Malunion or malocclusion</td>
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<td>Epiphora</td>
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<td>Foreign body reactions</td>
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<td>Scarring</td>
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<td>Sinusitis</td>
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Adapted from Haug RH et al.52
Management of Zygomatic Complex Fractures

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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. 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 Simpson at Los Angeles County/University of Southern California Medical Center found that a majority of the 1,200 zygomatic fractures studied were the result of motor vehicle accidents (MVAs). In contrast, Ellis and colleagues 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 the 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 the zygomaticosphenoid sutures (Figure 23.2-1A 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. 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 (Figure 23.2-1C).

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 23.2-1C). 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 the zygoma and arch and is innervated by...
a branch of the mandibular nerve (see Figure 23.2-1A).

The temporalis fascia attaches to the frontal process of the zygoma and zygomatic arch (Figure 23.2-1D). 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

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accompanied by an antimongoloid (downward) cant of the lateral canthal region caused by displacement of the zygoma (see Figure 23.2-1C).

**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.3,5–7 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 23.2-2). Lacerations in the facial region should lead the surgeon to suspect underlying fracture.

Palpation of the zygomaticofrontal suture, the entire 360˚ 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.

In isolated zygomatic arch fractures, a depression is observed and palpated anterior to the tragus (Figure 23.2-3). 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, retro-orbital 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.

**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.8–10 Radiographs are helpful for
confirmation and for medicolegal documentation and to establish the extent of the bony injury.

**Computed Tomography**
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, infraorbital, zygomaticofrontal, zygomaticosphenoid, and zygomaticotemporal buttresses. Coronal views are particularly helpful in the evaluation of orbital floor fractures (Figure 23.2-4A).9 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° 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 (Figure 23.2-4B). 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.

**Caldwell’s View** Caldwell’s view is a posteroanterior projection with the face at a 15° 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 (Figure 23.2-4C).

**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 classified zygomatic fractures by the direction of displacement on a Waters’ view radiograph.11 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 proposed a method of classification based on the pattern of segmentation and displacement.8 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 23.2-5). High-energy injuries were characterized by comminution in the lateral orbit and lateral displacement with segmentation of the zygomatic arch (Figure 23.2-6).
Gruss and colleagues proposed a system that stressed the importance of recognizing and treating zygomatic arch fractures in association with the zygomatic body.\(^1\) Like Manson and colleagues, Gruss stressed the importance of identifying and treating segmentation, comminution, and lateral bowing of the zygomatic arch.

Zingg and colleagues, in a review of 1,025 zygomatic fractures, classified these injuries into three categories.\(^7\) 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.

**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,
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if they are accepted, are helpful to standardize terminology, to plan treatment, and to 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 esthetic and functional deficits. Treatment may therefore range from simple observation of resolving 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. 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.

Goldthwaite in 1924 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 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 arch. An elevator is placed between the coronoid processes and zygomatic arch, and the fracture is reduced.

The standard technique for treatment of zygomatic arch fractures, first described by Gillies, Kilner, and Stone in 1927, 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 23.2-7A). 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 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 (Figure 23.2-7B). The arch should be palpated at all times as a guide to proper reduction. The wound is closed in layers.

An alternative technique uses a J-shaped curved hook elevator. This is inserted just below the zygomatic arch anterior to the articular eminence through a preauricular stab incision. The tip of the hook is directed under the displaced fragments, and reduction is achieved with controlled lateral traction.

In a series of 2,067 zygomatic fractures, Ellis 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 ½-inch gauze, a nasogastric tube, or a urinary catheter. More conveniently, a transcutaneous circumzygomatic arch wire can be passed and tightened over a foam-backed aluminum eye shield to suspend the arch. Although not a support technique, an aluminum foam-rubber-backed finger splint has been used to prevent the patient from placing undue force on the arch. The splint is formed into a U shape, taped to the face, and maintained for 3 to 5 days.20
Management of Zygomatic Complex Fractures

Open reduction with internal fixation is seldom necessary for treatment of isolated zygomatic arch fractures. 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.21

Middle-Energy Zygomatic Complex Fractures

Middle-energy, displaced zygomatic complex fractures require reduction and internal fixation. Over the past 20 years there has been an increase in the use of open reduction and internal fixation. In 1984, Zachariadis and colleagues 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.22

In 1996, Ellis and Kittidumkerng proposed an algorithm of treatment for isolated middle-energy zygomatic complex fractures that did not require orbital reconstruction (Figure 23.2-8).23 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 transcutaneously into the malar eminence (Figure 23.2-9). 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.

Other authors recommend routine exposure of two or more of the three anterior buttresses for middle-energy injuries: the zygomaticomaxillary buttress, zygomaticofrontal buttress, and the infraorbital rim (Figures 23.2-10–23.2-12). In this manner, multiple buttresses are visualized and the three-dimensional accuracy of the reduction can be confirmed.24–27

High-Energy Zygomatic Complex Fractures

A more aggressive surgical approach should be planned to treat high-energy fractures (Figure 23.2-13).12,23,24,28 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. Additionally, 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 is 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.\textsuperscript{12,23,24,28,29}

**Surgical Approach to 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. A transcutaneous or transconjunctival incision is used to explore and reconstruct the internal orbit.

![Figure 23.2-8](image-url)

FIGURE 23.2-8 Zygomatic complex fracture without need for internal orbit reconstruction. Adapted from Ellis E and Kittidumkerng W.\textsuperscript{23}

**Surgical Approach to 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 23.2-14A and B). If present, a preexisting laceration may be used for exposure of this region.

In 1996, Kung and Kaban described the use of a supratarsal fold incision for approach to the lateral orbit (see Figures 23.2-11 and 23.2-14B).\textsuperscript{32} 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 noticeable 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 23.2-14A). Dissection is continued through the orbicularis oris and the periosteum to the fracture site.

**Surgical Approach to the Infraorbital Rim and Orbit**

Access and exposure for open reduction of the infraorbital rim and orbital floor can be achieved through a transcutaneous subciliary or transconjunctival incision. Protection of the globe with a scleral shield or tarsorrhaphy is recommended.

A subciliary incision is made 1 to 2 mm below and parallel to the lower eyelash margin (see Figures 23.2-13 and 23.2-14A). It should extend from lateral to the punctum in a natural skinfold. The fibers of the orbicularis muscle are separated horizontally at the same level as the skin incision, and a composite skin-muscle flap is elevated anterior to the orbital septum. A periosteal incision is made on the anterior surface of the infraorbital rim. Subperiosteal dissection is then completed to expose the orbital rim and floor.\(^{31,33}\) Multiple variations of this technique have been described including a skin-only flap, a stepped skin-muscle flap, and a subtarsal approach. These have been compared to each other and to the transconjunctival incision.\(^{34-36}\) Regardless of technique, transcutaneous approaches are associated with a higher incidence of ectropion, increased scleral show, and cutaneous scarring.\(^{37-40}\)

To avoid the problems associated with cutaneous incisions, many authors recommend the transconjunctival approach.\(^{37-42}\) Tessier described this approach in 1973 (Figures 23.2-14C and 23.2-15).\(^{43}\) 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 23.2-16).\(^{31}\)

A lateral canthotomy can be used to increase exposure. Meticulous repair of the lateral canthotomy is required to prevent asymmetry.\(^{31,38,40,41}\)

Manson and colleagues described a method to expose the entire lateral orbit, infraorbital rim, and orbital floor through a single incision. This may be performed with a subciliary or transconjunctival approach and requires extended subperiosteal dissection with mobilization of the lateral canthal tendon.\(^{44}\)

**Pitfalls in Surgical Approach to the Infraorbital Rim and Orbit**

All approaches to the infraorbital rim may result in complications. The subciliary and transconjunctival incisions may result in ectropion, entropion, and increased scleral show. Advocates of the transconjunctival approach cite increased rates of ectropion and scleral show with transcutaneous incisions (see Figure 23.2-13J).\(^{37-40}\) In 1993, Appling 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.\(^{39}\)

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.\(^{44}\)

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 colleagues 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.33

Lastly, postoperative support for the lower eyelid with a frost stitch has been proposed as a technique to prevent ectropion. This may encourage re-draping of the lower eyelid tissues.23,44

Surgical Approach to 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 23.2-13F).

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 23.2-13F). The dissection is extended inferiorly at this depth to the zygomatic arch and anteriorly to the lateral orbital rim. The facial nerve is protected within the flap.12,31

Internal Fixation 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.22,45–47
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.” 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. More commonly two- or three-point stabilization is required.

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.

Internal Fixation of the Zygomaticomaxillary Buttress The zygomaticomaxillary buttress provides an ideal location for internal fixation for middle- and high-energy fractures. Anatomic reduction of this fracture assists in restoring malar projection, but is difficult if the buttress is comminuted. The overlying soft tissue is thick, and plate palpability is not a concern. Therefore, this fracture should be stabilized with 1.5 or 2.0 plates.

Internal Fixation of the Zygomaticofrontal Buttress The zygomaticofrontal buttress contains excellent bone for fixation and can accommodate a 2.0 plate. The reduction and fixation of this fracture will reestablish the vertical height of the zygomatic complex. However, because of its narrow inter-

face, this buttress may not be as helpful in evaluating reduction of a rotated fracture. 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.

Internal Fixation of the Infraorbital Rim Unlike the zygomaticofrontal buttress, the infraorbital rim has poor quality bone for internal fixation. Additionally, the lower eyelid skin is quite thin, and large plates are easily palpable. Despite these concerns, fixation of this site is required to define the orbital volume and facial width. The infraorbital rim is typically displaced posteriorly and inferiorly. The fracture should be mobilized anteriorly and superiorly and stabilized. Typically a 1.0 or 1.5 microplate is used to stabilize the infraorbital rim. A potential pitfall in reduction of this fracture is an unappreciated heminasoethmoid fracture (see Figure 23.2-13D). If the infraorbital rim is secured to this undiagnosed displaced segment, postoperative facial widening may occur.

Internal Fixation of the Zygomatic Arch Internal fixation of the zygomatic arch is required for high-energy fractures that demonstrate comminution and lateral displacement. 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.

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.

For middle-energy injuries with exposure of all three anterior buttresses, the zygomaticofrontal fracture may be stabilized temporarily with an interosseous wire. This is followed by fixation of the zygomaticomaxillary fracture and the infraorbital rim. The temporary wire at the zygomaticofrontal fracture is replaced with a plate. The orbital floor is reconstructed after the zygoma has been restored to its correct three-dimensional position.

In high-energy fractures, the zygomatic arch should be reconstructed first.

Management of the Orbital Floor

Patients with middle-energy zygomatic complex injuries and no clinical or radiographic evidence of orbital disruption do not require exploration. Middle-energy injuries with displacement of the orbital rim or floor or herniation of soft tissue into the sinus should be explored (see Figure 23.2-4A). Clinical indications for orbital exploration include enophthalmos, limitation of extraocular muscle function with a positive forced duction test, and persistent diplopia. High-energy fractures require a more aggressive approach, and the orbital rim and floor should be explored and reconstructed.

Fujino and Makino classified orbital floor injuries as linear and pure blow-out fractures (Figure 23.2-17). 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 ten times greater than that required for a linear fracture. Fragments are forced inferiorly into the sinus, producing bony discontinuity.

Indications for exploration of isolated orbital floor fractures include CT scan evidence of a fracture and herniation of orbital tissue, enophthalmos, dystopia,
Role of Bone Grafting  Early bone grafting is indicated for severe injuries in which there is loss of bone or extensive comminution. Commination 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.

Postoperative Care  Zygomatic complex fractures violate the maxillary sinus. For this reason, antibiotics and decongestants are recommended. Ampicillin, amoxicillin, clindamycin, or cephalosporin may be used. A decongestant such as pseudoephedrine is also used to clear the airway.

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 radiographs (Waters’ view and submentovertex view) are obtained to document reduction of the fracture. A CT scan may be obtained in comminuted fractures to evaluate the zygomatic complex reduction and orbital reconstruction.

Complications

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 only become manifest later in recovery.

Infraorbital Paresthesia

The incidence of sensory alterations of the infraorbital nerve following zygomatic trauma ranges from 18 to 83%. Studies by Vriens and colleagues and Taicher and colleagues have found improved recovery of infraorbital sensation following open reduction and internal fixation at the zygomaticofrontal suture compared with reduction without fixation. Presumably, anatomic reduction of the fracture may minimize compression of the nerve and allow for recovery. However, in Vriens’s study, the same degree of improvement was not seen in patients requiring orbital floor exploration and reconstruction.

Malunion and Asymmetry

Inadequate reduction or stabilization of zygomatic fractures may result in malunion or asymmetry. 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.

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. Severe late posttraumatic 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. 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 following orbitozygomatic...
fractures. An increase in orbital volume is the most common etiology.\textsuperscript{57–59}

Grant and colleagues described this clinical problem eloquently by comparing the shape of the orbit to that of a cone. The volume of a cone is $\frac{1}{3} \pi r^2 h$. 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.\textsuperscript{58}

Orbital floor blow-out fracture also may result in enophthalmos by increasing the orbital volume (Figure 23.2-18). With improved CT technology, calculation of orbital volume and its implication regarding orbital floor fractures is possible.\textsuperscript{73,77–80} Raskin and colleagues demonstrated that a 13% increase in orbital volume, at 4 weeks, results in significant enophthalmos (> 2 mm).\textsuperscript{79} The critical size of the orbital defect and herniation of orbital tissues have also been studied. In 2002, Ploder and colleagues reported that a mean fracture area of 4.08 cm\textsuperscript{2} or a mean displaced tissue volume of 1.89 mL, was associated with greater than 2 mm of enophthalmos.\textsuperscript{80} In general, approximately 1 cm\textsuperscript{3} of displaced tissue equals 1 mm of enophthalmos.\textsuperscript{81}

Late repair of enophthalmos is technically challenging. Wide access with osteotomy of the zygoma, repositioning, and grafting is usually required. Re-draping of the periorbital soft tissue including a canthopexy may be required.\textsuperscript{57–59}

**Diplopia**

Diplopia is a common sequela of midfacial fractures. The incidence varies between 17 and 83% and depends on the time of presentation following the injury and the pattern and severity of the injury.\textsuperscript{3,68,82–84} In a review of 2,067 zygomatic complex fractures, Ellis and colleagues noted a 5.4 to 74.5% incidence of diplopia.\textsuperscript{3} Nondisplaced zygomatic complex fractures and isolated zygomatic arch fractures had the lowest incidence of diplopia, while 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 nerves III, IV, or VI. Histologic studies by Iliff and colleagues 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.\textsuperscript{45,82} 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 23.2-19).\textsuperscript{45,60,61,63–65,82}

**FIGURE 23.2-18** 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 demonstrating 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, Post-operative coronal CT scan demonstrating titanium mesh reconstruction of the orbital floor.
Persistent bothersome diplopia that does not resolve may require treatment by an ophthalmologist. The condition may respond to exercise or surgery.\textsuperscript{45,61}

**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 23.2-20). 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.\textsuperscript{88,89}

Management of hyphema consists of supportive therapy including elevation of the head of bed and patching of the injured eye. Medical management includes topical cycloplegics, corticosteroids, and β-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 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.\textsuperscript{45,90}

**Superior Orbital Fissure Syndrome**

Superior orbital fissure syndrome is an uncommon complication following 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.\textsuperscript{91}

**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 1,405 orbitozygomatic fractures, Ord reported a 0.03\% incidence of postoperative retrobulbar hemorrhage with visual loss.\textsuperscript{92} An emergent ophthalmologic consultation is necessary; however, decompression with lateral canthotomy and cantholysis should not be delayed (see Figure 23.2-20).

**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 following 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. Coronoidec- tomy 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.61,93

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References


Orbital and Ocular Trauma

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 24-1). 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 following

![Orbital anatomy diagram](image-url)

midfacial trauma. Orbital fractures that involve the frontal sinuses more commonly result in serious eye injuries.\textsuperscript{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. The frontal sinus begins to form around the age of 6 years and is unilateral in 5% of adults and lacking in another 5%.

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 reach the orbital floor and infraorbital canal by age 2 years.\textsuperscript{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 24-1).\textsuperscript{8} The infraorbital nerve provides sensory innervation to the upper lip, lateral nose, and anterior maxillary teeth and mucosa. 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

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<th>Table 24-1 Orbital Fissures/Canals and Their Contents</th>
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<td><strong>Location</strong></td>
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<td>Superior orbital fissure—lesser and greater wings of sphenoid</td>
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<tr>
<td>Inferior orbital fissure—greater wing of sphenoid; palatine, zygomatic, and maxillary bones</td>
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<tr>
<td>Optic canal—lesser wing of sphenoid</td>
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<td>Anterior ethmoid canal—frontal and ethmoid bones</td>
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<td>Posterior ethmoid canal—frontal and ethmoid bones</td>
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<td>Nasolacrimal fossa—lacrimal and maxillary bones</td>
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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° angle to each other. Each lateral orbital wall forms a 45° 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 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). This gentle outcropping of bone functions as the insertion point for the lateral retinacular structures. 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 retnaculum, which is the actual insertion to the tubercle. 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 papryacea 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, and 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, and 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 sphenethmoidal 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. 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. Average distances for locating these critical structures as they relate to identifiable bony landmarks are contained in Table 24-2. Surgeons should avoid disrupting the medial canthal tendon, lacrimal apparatus, pulley of the superior oblique muscle, supraorbital nerves and vessel, attachments to Whitnall’s tubercle, and the origin of the inferior oblique muscle.

<table>
<thead>
<tr>
<th>Table 24-2</th>
<th>Distance of Vital Orbital Structures from Bony Landmarks</th>
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<tbody>
<tr>
<td>Structure</td>
<td>Reference Landmark</td>
</tr>
<tr>
<td>Midpoint of inferior orbital fissure</td>
<td>Infraorbital foramen</td>
</tr>
<tr>
<td>Anterior ethmoidal foramen</td>
<td>Anterior lacrimal crest</td>
</tr>
<tr>
<td>Superior orbital fissure</td>
<td>Zygomaticofrontal suture</td>
</tr>
<tr>
<td>Superior orbital fissure</td>
<td>Supraorbital notch</td>
</tr>
<tr>
<td>Optic canal (medial aspect)</td>
<td>Anterior lacrimal crest</td>
</tr>
<tr>
<td>Optic canal (superior aspect)</td>
<td>Supraorbital notch</td>
</tr>
</tbody>
</table>
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 24-3). 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 since 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. The upper and lower lids should form a 30 to 40° 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 peristeam and the periosteum of the facial bones overlying the rims. One to two millimeters below the inferior rim, where these layers converge on the facial aspect, is a periosteal thickening called the arcus marginalis. 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, then 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 pre-aponeurotic 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. 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 cyst-like 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,
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), then this accounts for 30 to 55% of all facial fractures.\textsuperscript{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.\textsuperscript{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 $\beta_\text{2}$-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 Caucasians 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.\textsuperscript{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 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.

Visual acuity should be independently tested on each eye using a Snellen chart at a standard 6 m (20 ft.) distance or with reading of standard-type print at 40 cm (16 in.). The patient should wear their corrective lenses during this examination. If over 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, ecchymosis, lacerations, ptosis, asymmetric 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 24-3). Of greatest concern is diplopia in the primary (straight-ahead) and downward gazes. These are the two fields that are used most often. Mild or equivocal restriction (< 5°) 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, then 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. Upward, downward, and lateral motions can be evaluated. 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, then 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 the globe, which is usually concealed beneath the lid (Figure 24-4). 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 post-chiasm 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, then 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. Tonometry indirectly measures intraocular pressure by placing the instrument on the surface of the eye. Normal (10–20 mm Hg) or symmetric 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 is indicative of a retrobulbar hematoma, which may
FIGURE 24-3  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. 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°). 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° inferiorly.
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 lamp-light (Figure 24-5).

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.

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. Other imaging modalities, such as plain radiography, reconstructed three-dimensional CT, MRI, ophthalmic ultrasoundography, 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. Waters’ projection allows visualization of the orbital roof and floor and is particularly useful for evaluating orbital floor blow-out fractures (Figure 24-6). With this 23° (preferably posteroanterior) view, the petrous portion of the temporal bones is projected below the maxillary sinuses and indirect signs of fracture can be noted, such as a teardrop formation or air-fluid levels. This is also an excellent view to assess a ZMC fracture.

If plain films reveal an internal orbital fracture that possibly warrants surgical intervention, then 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 mm axial cuts of the cranium and 5 mm cuts through the facial region. If finer detail or three-dimensional reconstructed images are desirable, then 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 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 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 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 position him- or herself 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 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. This allows a better determination and correlation of any afferent visual defect owing to possible bony impingement.

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. 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. 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.

CT imaging has some drawbacks. As previously mentioned, patients may be unable to position themselves comfortably for direct coronal imaging. 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. 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. 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, then an MRI should be ordered. An MRI should also be performed when an apparent orbital emphysema (focal air collection) fails to resorb rapidly (within several days); this may suggest a space-occupying foreign body.

Ophthalmic ultrasonography is seldom used but is a readily available, safe, inexpensive, and noninvasive imaging modality. 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. Color Doppler imaging is an ultrasound technique that provides simultaneous two-dimensional images and visualization of blood flow. 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.

### 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. 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 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 24-7). 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. 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.

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 asymmetric pupil, without a prior history of surgery, then a globe perforation likely exists (Figure 24-8). Blunt trauma 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 since 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 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.

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
This partial hyphema of the right eye resulted from a punch to the face; a computed tomography scan showed a minimally displaced orbital floor fracture. The slit-lamp examination shows early layering. This patient received nonoperative management.

Orbital and Ocular Trauma
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.

**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 following 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 since individuals seldom require these extreme views for everyday function. Also the patient may complain that the phenomenon is transitory and that sudden 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 following 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 contractions versus a neurogenic motility disorder (cranial nerves III, IV, or VI). Ophthalmologists use diploic visual fields (see Figure 24-3F) 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 24-3). 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 h). Prolonged muscle entrapment with ischemia can lead to fibrosis (Volkman’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, then 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 debrided, 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 monofilament absorbable 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 final skin (with 6-0 nylon or 6-0 fast-absorbing gut). Topical ophthalmic ointment should be prescribed since 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 post repair, if significant lid contracture or focal thickening is noted, then selective judicious steroid injections (triamcinolone acetonide, 40 mg/mL) can be administered with accompanying daily massage by the patient.

In my experience, 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 distal cut end of the drainage system canaliculi since this system is dominant in the vast majority of patients.

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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 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 is best treated early (within 7–10 d) following 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.

I have found that direct canthal tendon reattachment with transnasal wire fixation is best performed by passing a doubled-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 24-12).

Nonoperative Management of Orbital Fractures

Indications for nonoperative or, as it has previously been termed, conservative management of orbital fractures has been controversial for many years. Some historic
perspective and review is warranted since it provides insight into the evolution and current thinking regarding nonoperative orbital fracture treatment. In 1957 Smith and Regan coined the term blow-out fracture and advocated early surgical intervention for orbital floor fracture repair.38 Following this, Converse and Smith endorsed surgical exploration and repair of all orbital fractures within the first 3 weeks of injury.39 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 promoted the concept that orbital floor fractures were overdiagnosed on plain films and, thus, were overoperated.40 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 following surgical exploration of asymptomatic floor fractures.41 In 1974 Puttermann and colleagues reported on a series of 57 patients whom they had observed and on whom they had performed no surgical intervention whatsoever.42 Only a few of these individuals had any persistent diplopia, and there were no visual acuity disturbances 4 months following the trauma. This landmark article created a drastic shift in thinking—nonsurgical treatment of all orbital fractures was advocated. Puttermann 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 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 Puttermann 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 reported that only 50% of all patients with orbital floor fractures required surgical intervention.43 All patients underwent a 2-week observational period; an individual would then undergo surgical intervention if one of the following criteria was present: enophthalmos > 3 mm, large herniation of tissue into the antrum, entrapment with limited upward gaze, or significant diplopia. Nevertheless, these criteria were somewhat subjective and were limited by the current imaging techniques. Crumley and colleagues used similar indications for surgery to those of the Puttermann group, but based on these criteria, almost 90% of all their patients with orbital floor fractures underwent surgical repair.44 Converse and Smith 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.45 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 ocular-plastic surgeons were operating within 2 weeks of injury with few serious complications or sequelae. 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, 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 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 that emphasized the need for surgical repair within 2 weeks following injury in patients with persistent diplopia within a 30° 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.

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, Puttermann has revised his indications for surgical intervention. Puttermann and his colleagues 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 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 used tomography and felt that orbital floor fractures involving > 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 > 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 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. In 1991 Puttermann and colleagues 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.\textsuperscript{52} Despite numerous reports, clinical series, and author suggestions, controversy still remains regarding the management of those patients who develop only mild enophthalmos or hypo-ophthalmos (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 24-13). 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.\textsuperscript{56,57} This phenomenon occurs more in children with linear fractures owing to the elasticity of their bones.\textsuperscript{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°), 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° of their primary gaze, especially the downgaze (used during reading), surgical exploration is warranted. Prior to 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 that are competent in performing transnasal endoscopic optic canal decompression. If at all possible, this should 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 long-standing dogma, post-traumatic fat atrophy does not play a significant role in the development of these deformities.\textsuperscript{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 underlying 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
> 2 mm, then 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 a patient requires surgical intervention, a well-thought-out plan and sequential approach should be developed. Of paramount importance is the determination 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 peripheral and facial fractures prior to 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: the infraorbital, subciliary, and transconjunctival (Figure 24-14). 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 esthetics and generous access, and the fact that surgeons are familiar with their use. It is my opinion that the infraorbital or rim incision results in the worst esthetics and offers no
advantages over the two former approaches; therefore, it should not be employed.

The subciliary incision was popularized by Converse in 1944. 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 prior to 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. 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 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 esthetics, 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\textsuperscript{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.\textsuperscript{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 and Converse and colleagues in 1973 for orbital floor fractures.\textsuperscript{64,65} The two basic variations of this approach to the orbital rim are retroseptal or preseptal approaches. Although the retroseptal approach is more direct, 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.\textsuperscript{66} The preseptal approach (see Figure 24-14) as described by Tessier 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.\textsuperscript{64} Next, a preseptal vertical dissection is carried out down several millimeters below the orbital rim, and the periostium 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 periostium to the orbital septum or other layers.\textsuperscript{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 periostium, then 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%).\textsuperscript{35} 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.\textsuperscript{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.\textsuperscript{69} Both of these basic incisions provide good exposure with excellent esthetics 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 esthetics 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 esthetics (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 periostium, 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 incision 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 are 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. 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 prior to 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 peristeum 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. 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. 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 re-attach the trochlea since, when the soft tissues are re-draped, the trochlea re-adheres on its own. Suturing may actually pierce or violate the trochlear tendon and result in ocular motility disturbances. Closure of the coronoal 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 hemi-coronal 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. This approach provides good access to the lateral and infra-orbital skeleton; however, it is less frequently used because it requires a more complex closure and re-anchoring 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°) esthetics.

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 conjunctival 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 one-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, then 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 as 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.

**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 general result in a bony defect of one orbital wall (typically the medial orbital wall) 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 > 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 esthetics.

Linear fractures are generally caused by blunt forces directly to the globe or partially to the rim and most often result in an esthetic 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 entrap 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 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 esthetic 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 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, I prefer to use thin (0.85 mm) porous polyethylene sheeting. 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. However, I still recommend anchoring the porous polyethylene sheet to the anterior lateral orbital floor with a single titanium screw (Figure 24-15). The greatest advantages of this material are its ease of contouring, in situ carving, burring, and that it can be layered posteriorly behind the orbital equator to achieve proper orbital volume and contour.

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, I still prefer 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.

Blow-out fractures typically involve one orbital wall (usually the anterior or medial portion of the orbital floor) and are < 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. Fat atrophy contributes little, if anything, to the development of early or late enophthalmos. 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 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 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. It is the reconstruction of this posterior medial floor to its normal contour that is the key to restoring normal globe position both anteroposteriorly and vertically. It is this scenario that is problematic in delayed reconstructions since attempts to create a normal anteroposterior or position of the globe may result in inappropriate overpositioning of the globe in a superior direction. I 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

![Figure 24-15](image-url)
contents superiorly, then the reconstructive material can be slid underneath them and overlap the intact bony margins slightly at the majority of areas to provide adequate support. I prefer to use porous polyethylene for moderate to large blowout 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 24-15). Care should be taken to not extend the grafts up to the orbital rim or over the edge since 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 forcedduction test performed prior to 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, it is in this group of individuals that primary repair with normal anatomic realignment is critical for acceptable esthetic 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 prior to rigid fixation of any components with plates and screws (Figure 24-16). This helps one avoid misalignment, over-reduction, 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 prior to 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 anterior inferior floor with a single screw. Sometimes layering of this material with an additional sheet posteriorly is required to achieve correct anteroposterior or 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

FIGURE 24-16  A, An elderly female 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 h) 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)
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 hyper-ophthalmic 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, it is the resolution of the edema that 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 23.2, “Management of Zygomatic Complex Fractures” and Chapter 25, “Management of Frontal Sinus and Naso-orbitoethmoid Complex Fractures.” Some discussion is warranted here, as 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 24-16C–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. Additionally, injuries to the nasal airway and lacrimal system can occur.\(^8\)\(^2\) 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 re-adapt 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, then 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 subbiliary 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, then 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 burr 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 24-12). This is a much easier way to accomplish precise canthal positioning than are direct suturing techniques.

**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.

**References**


Fractures of the frontal bone and the naso-orbitoethmoid (NOE) complex are infrequent, occurring among 2 to 15% of patients with facial fractures. When these fractures 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 > 100 years, no consensus has yet been reached on ideal care after traumatic injury.

Most victims are male (66–91%) and young (usually 20–30 yr of age, range 6–72 yr), and most frontal sinus and NOE injuries are sustained in motor vehicle or motorcycle collisions (44–85%). NOE fractures can occur in isolation, but they most often occur in association with other midface fractures. As many as 60% of patients with NOE fractures have associated nonfacial injuries.

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.

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 eighth or ninth week in utero. 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 twelfth week. The metopic suture in the midline closes during the second year of life. The forehead is displaced anteriorly by sutural growth, inner table resorption, and outer table deposition.

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. Initial pneumatization begins during the fourth month in utero. Secondary pneumatization begins at the age 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. Most pneumatization is completed by the time the child is 12 to 16 years old, but it continues until the age of 40 is reached. The configuration of the sinus and the position of the septa are extremely variable.

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. The mucosa consists of pseudostratified ciliated epithelium, mucous-producing goblet cells, a thin basement membrane, and a thin lamina propria that contains seromucous glands. When the mucosa is healthy, a blanket of mucin
overlies the epithelium. The cilia flow at 250 cycles/min. The mucin blanket flows in a spiral fashion in a medial-to-lateral direction; the flow is slowest at the roof and fastest at the nasofrontal duct. The mucin empties at the nasofrontal duct at a rate of 5.0 g/cm². The physiologic characteristics of the sinus and the status of the nasofrontal duct dictate the treatment of the frontal sinus in trauma.

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 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 foramen 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; the trochlea of the superior oblique muscle is attached to this spine (Figure 25-1).

Paired triangular sinuses are found within the frontal bone. These sinuses are asymmetric and are separated by a frontal septum. The average height of the sinuses is 32 mm, and their average width is 26 mm. The surface area is approximately 720 mm². 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.
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. 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. The relationship of the diploë to the anterior cranial fossae is important to understand because these structures can become a conduit for the spread of infection.

**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 pyramidal-shaped sinus measures 3.5 to 5 cm from front to back.

The ethmoid air cells drain into the middle meatus, as does the nasofrontal duct. The nasofrontal duct 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 may vary from a few millimeters to a centimeter or more (Figure 25-2).

**Medial Canthal Tendon**

The orbicularis oculi muscle has three portions: the orbital, the preseptal, and the 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. 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.

**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

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*The internal concave surface of the frontal bone forms the anterior cranial fossa that houses the brain. The floor of the 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 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.*
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 lacrimalis. The lacrimal canaliculi are lined with nonkeratinized and non–mucin-producing stratified squamous epithelium. The epithelium is 75 to 150 µ thick and consists of a few layers of squamous cells, polyhedral cells, and a basal cell layer.

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. 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.

**Patient Evaluation**

**Clinical Findings**

Periorbital ecchymosis and pain are the most common signs and symptoms associated with fractures of the frontal bone. When the bone bleeds and the periosteum is interrupted, leakage of blood into the adjacent facial planes results in periorbital ecchymosis. Through this same mechanism, subconjunctival hemorrhage may occur. If the nose and zygomas are unaffected, a finding of subconjunctival hemorrhage is sufficient for the diagnosis of frontal bone fracture. 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 (Figure 25-3).

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 of the distribution of the supraorbital and supratrochlear nerves. 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 25-4). 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.40–44

Fractures involving the posterior table of the frontal sinus or the cribiform plate may cause CSF leakage.40–43,46 Confirmation of the presence of CSF can be made by collecting this fluid and comparing its concentrations of glucose and chloride with the patient’s serum concentrations. Concentrations of chloride and glucose can be determined in as little as 0.1 mL of fluid. Chloride concentrations in the collected fluid that are greater than concentrations in serum and glucose concentrations less than those in serum indicate the presence of CSF. Collected fluid can also be tested for the presence of β2-transferrin; a positive result confirms the presence of CSF (Table 25-1).47

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.43,44 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

| Table 25-1 Normal Values of Constituents of CSF, Serum, and Nasal Secretions |
|-----------------|---------------|---------------|---------------|
| Constituent     | CSF           | Serum         | Nasal Secretions |
| Osmolarity      | 295 mOsm/L    | 295 mOsm/L    | 277 mOsm/L     |
| Sodium          | 140 mEq/L     | 140 mEq/L     | 150 mEq/L      |
| Potassium       | 2.5–3.5 mEq/L | 3.3–4.8 mEq/L | 12–41 mEq/L    |
| Chloride        | 120–130 mEq/L | 100–106 mEq/L | 119–125 mEq/L  |
| Glucose         | 58–90 mg/100 mL | 80–120 mg/100 mL | 14–32 mg/100 mL |
| Albumin         | 50–75%        | 55%           | 57%            |
| Total protein   | 5–45 mg/dL    | 6.0–8.4 mg/dL | 335–636 mg/dL  |
| (% of total protein) |         |               |               |
| Immunoglobulin G| 3.5 mg/100 mL | 1,140 mg/100 mL | 51 mg/100 mL  |
| β2-Transferrin (%) | 15%          | 0%            | 0%             |

Adapted from Brandt MT et al.47
CSF = cerebrospinal fluid.
of these fragments may vary, but any movement implies instability and requires open reduction and stabilization. A ruler or caliper should be used to measure the intercanthal distance. The normal distance is 28.6 mm to 33.0 mm for adult women; it is 28.9 mm to 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. The Furness test may also be performed by grasping the skin overlying the medial canthus with a small-tissue forceps (Figure 25-5). A lack of creasing or resistance by the underlying bone is indicative of an underlying fracture. The bimanual examination requires placing an instrument (eg, a 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.

**Imaging**

Poor outcomes after the treatment of NOE fractures and frontal sinus fractures typically result from misdiagnosis, inadequate planning, lack of exposure, inadequate reduction or fixation of soft tissue or bone, stripping of the medial canthus, or loss of nasal contour with insufficient primary grafting. In the past, Waters’ projections, reverse Towne’s projections, lateral skull films, and laminar tomograms were used to visualize midface and upper-face fractures. It is clear that appropriate preoperative imaging can help to prevent misdiagnosis and can aid in proper treatment planning. Today computed tomography (CT) scans are the gold standard for imaging these fractures. The plane of choice for frontal sinus imaging is the axial view, preferably with slice thicknesses of 1.0 or 1.5 mm. 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. 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.

**Patency of the Nasofrontal Duct**

Although the newest CT scanners provide exceptional views and can often provide slices through the nasofrontal duct, evidence of their reliability in detecting obstruction of the ducts is scant. Duct 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. In these situations an open or intraoperative evaluation of patency is indicated. This evaluation is important because the condition of the nasofrontal duct has the most influence on the health of the frontal sinus.

**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. 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.

**Classification of Frontal Sinus Fractures**

Traditional fracture classifications can be used with reference to frontal bone fractures.
Management of Frontal Sinus and Naso-orbitoethmoid Complex Fractures

Numerous 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 ducts and to the presence of concomitant intracranial and craniomaxillofacial injuries. The simplest and most helpful classification schemes distinguish possible complications and treatments on the basis of types of fractures.

Isolated anterior table fractures should be treated so that cosmetic deformities can be prevented. 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 nasofrontal duct 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

Surgical Access

The coronal approach to surgery provides the greatest access to the frontal bone and sinus and produces the most desirable cosmetic results. Although lacerations may be considered as an approach to the fracture, their size and shape rarely provide enough access without undue and unsightly extension. Gullwing 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.

Although the coronal approach has been well described, 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 with 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 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 pericranium in an anterior direction. Raney clips are helpful in achieving hemostasis; however, hemorrhage may recur when they are removed, and electrocautery may need to be used carefully at the end of the procedure as the individual clips are removed. Again, care must be taken to avoid hair follicles to preserve 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

![FIGURE 25-6](image1) The detail of fracture anatomy is clearly superior in computed tomographic (CT) scans when compared with traditional radiography. A, Initial appearance of a patient with an NOE fracture. B, Axial CT scan showing the fracture. C and D, Axial and coronal CT scans of another patient illustrating detailed fracture anatomy.

![FIGURE 25-7](image2) Intraoperative view of the floor of the frontal sinus with nasofrontal ducts.
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.

**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 (Figure 25-9). If contaminated, segments of bone may be cleansed with copious irrigation, scrubbing, and even povidone-iodine solution, and then used for reconstruction as free grafts. Once the anterior table has been removed, access should be adequate for sinus exploration, posterior table inspection, and sinus floor (nasofrontal duct) 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. 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 Nasofrontal Duct**

After access has been obtained and osseous exploration and recovery have been performed, the condition of the frontal sinus floor and the nasofrontal ducts can be assessed by direct visualization (see Figure 25-7). The relative patency of the duct can then be evaluated by placing an

**FIGURE 25-8 Naso-orbitoethmoid fracture classification.**

**FIGURE 25-9 Comminuted frontal sinus segments arranged prior to reconstruction.**
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 of the nasofrontal duct can be confirmed by introducing normal saline and observing its emergence from beneath the medial turbinate or its collection in the posterior pharynx (Figure 25-10). 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 25-11). Fluorescein is an excellent alternative because it is clear, colorless, water soluble, and radiolucent.\(^6^8\) However, its visualization sometimes requires using an ultraviolet light source and then dimming 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.\(^6^0\) Moreover, any spilled radiopaque dye must be completely cleared before additional radiographs or computed tomographic images are obtained. Indigo carmine is another acceptable dye, but Congo red 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.\(^6^9\) Displaced anterior table fractures require open 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.\(^6^4,6^6,7^0,7^1\) 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 25-12). 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.\(^1^6\)

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**FIGURE 25-11** 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. Reproduced with permission from Haug RH and Cunningham LL.\(^6^4\)

**FIGURE 25-12** CT scan demonstrating anterior and posterior table fractures of the frontal sinus.
The surgeon should check carefully for displacement of the fracture, CSF leak, entrapment of sinus membranes, and 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. Communciation of the posterior table, penetrating injury, CSF leak with extensive dural damage, or frontal lobe damage requires frontal sinus cranialization: complete removal of the posterior table, thereby effectively increasing the size of the anterior cranial fossa. In one review of cases, as many as 16% of patients undergoing frontal sinus surgery required a cranialization procedure. In such a case the posterior table would be gently removed, either with a diamond bur or with rongeurs. Care should be taken in the area of the sagittal sinus to avoid 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 or pericranial flap.

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. Failure to remove such elements may result in a mucocele or pyocele. The mucosa is then reflected down into the nasofrontal duct, 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 outer table is reassembled and restored as would be done for a simple anterior wall fracture.

Orbital Roof and Supraorbital Bar Reconstruction

Once the posterior wall and the sinus floor have been explored, inspected, and evaluated for damage, the orbital roof and supraorbital bar may be reconstructed. After these procedures have been completed, a galeal flap should be reflected, the sinus obliterated, and the nasofrontal duct 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 entrapped 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. Mesh has an advantage in that it provides support and consolidation of the segments in three planes of space (Figure 25-13). Titanium mesh has been shown to be compatible with soft tissue, undergoing incorporation with indigenous cells. Resorbable technology continues to show promise, even for frontal bone injury; however, the 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. Once this phase of the procedure has been completed, the nasofrontal ducts may be obstructed (if indicated), the sinus obliterated, the brain isolated with a galeal flap (if indicated), and, finally, the anterior table replaced.

Nasofrontal Duct Obstruction

Nasofrontal duct obstruction should not be confused with sinus obliteration. Sinus obliteration is the elimination of dead space by the introduction of another material. Duct obstruction is one of the methods of isolating the sinus (or brain) from nasal contamination, basically by plugging it with another material.

As stated above, the condition of the nasofrontal duct is the most important factor in maintaining the health of the frontal sinus. This duct permits the exit of mucin, seroma, or hematoma after injury. If the duct is injur ed and obstructed, sinusitis, meningitis, or osteomyelitis may develop. The condition of the duct should be considered in the evaluation of fractures of the NOE complex, the supraorbital rim, or the sinus floor. If the duct is not patent, thorough removal of every possible remnant of sinus mucosa is performed by curettage. This procedure is
followed by removal of additional mucosa from every cul-de-sac and crevice with a small (no. 8 or larger) diamond bur under copious amounts of irrigation and with the aid of magnification. Any remaining remnants of the nasofrontal duct mucosa are then inverted into the nose.

A number of materials can be used to obstruct the nasofrontal duct. Temporal fascia, temporal muscle, or both can be harvested from the adjacent temporal region through a bitemporal flap. 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 and recording. 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. 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. Autologous platelet gel and autologous fibrin glue have also been used for similar indications. 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.

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 duct to block passage of nasal contaminants, followed by inner-table 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 above.

**Sinus Obliteration**

Nasofrontal duct 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, thus causing a seroma or a hematoma. Furthermore, after cranialization, sinus obliteration cushions and protects the brain. Historically, sinus obliteration has been accomplished in a number of ways, including inserting no substance or object (theoretically permitting bone fill after curettage) or hydroxylapatite, glass wool, bone, cartilage, muscle, absorbable gelatin sponge, absorbable knitted fabric, acrylic, or fat. 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. With the open approach the skin is first cleansed with an antimicrobial agent from below the umbilicus to above the escutcheon of the genitalia. A transverse semilunar incision is made within the “bikini” line, 5.0 cm superior to the symphysis pubis (Pfannenstiel’s incision); an incision 5.0 to 8.0 cm long is adequate. An alternative to this approach is a vertical incision from below the umbilicus to above the symphysis pubis. The incision is carried through skin and subcuticular 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 (Figures 25-14 and 25-15).

**NOE Reconstruction**

Early surgical management is important in the reduction of NOE fractures. The deformities that result from unrepaired NOE fractures are severe and difficult to correct, requiring 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. These approaches aid in the treatment of displaced infraorbital rims and maxillary antrum or piriform rims.

Type I fractures are less difficult to treat and 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.

Transnasal wiring is recommended for fractures graded as Markowitz type II or higher. Although we are truly in an era of rigid fixation (bone plates and screws),

![Figure 25-14](image-url)
complete reduction of the NOE area and reattachment of the MCT, or replacement of a small bone segment, seem never 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. 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.22 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.22,36 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.36 In cases in which sufficient medial orbital wall remains, placing a microplate and screw for attaching the MCT behind the lacrimal crest has been suggested.38 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 can be fixated rigidly or with wire.

Medical Therapy of the Sinus Postoperatively
Saline solution nasal spray can reduce symptoms of rhinosinusitis.93 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 be made a part of reasonable 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.94–96 In addition, the trauma of surgery causes edema in the sinus tissues. Mucolytics have been advocated for use in patients with rhinosinusitis to thin the mucus secretions and to improve clearance.93 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 (eg, pseudoephedrine or oxymetazoline hydrochloride) act by stimulating α-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.93,97,98 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.99,100 In cases of contamination by a foreign body, this treatment may be continued for 10 days. In the absence of gross contamination of the wound, a limited number of postoperative doses can be considered, or none at all. Antibiotics used to treat acute rhinosinusitis include amoxicillin, amoxicillin-clavulanate, azithromycin, cefpodoxime proxetil, cefprozil, cefuroxime axetil, clarithromycin, levofloxacin, loracarbef, and trimethoprim-sulfamethoxazole.93 Penicillin is still the drug of choice for treating facial fractures.101
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. The most frequent ocular complication is diplopia. Damage to the superior oblique muscles or trochlea may result in limited range of motion of the globe. Severing of the supraorbital nerve by the injury or during reflection of the osteoplastic flap leaves a permanent anesthesia of the distribution of the forehead. Trauma to the floor of the frontal bone can cause orbital damage. The most frequent ocular complication is diplopia. Damage to the superior oblique muscles or trochlea may result in limited range of motion of the globe.

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. If the nasofrontal duct is occluded, blood may accumulate in the sinus, creating an environment that is conducive to the growth of anaerobic bacteria. Frontal sinus abscess is spread by direct extension through small fractures of the frontal bone or through transosseous anastomotic vessels. The result is brain abscess, meningitis, cavernous sinus thrombosis, or (if the abscess is long term) osteomyelitis.

Mucoceles are the most common chronic problems. 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 pycele. Imaging studies should be performed at 1, 2, and 5 years after surgery or whenever symptoms appear. Complications can occur as late as up to 20 years postoperatively, and patients should be encouraged to have routine yearly follow-ups.

Pain and headache may be chronic and may persist without an identifiable cause. Cosmetic deformities such as contour defects 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. In addition, 23% of patients with high midface fractures report a decreased sense of taste (hypogeusia).

Dacryocystorhinostomy

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.

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 (Figure 25-16).

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 periosseum 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 25-17).

The endonasal approach is conceptually the same procedure, except that the dissection is performed from inside of the nose with the aid of endoscopic instruments and a fiber-optic 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 with 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.111

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, methyloxidacrylate, hydroxylapatite granules, silver, a cobalt-chromium alloy, polytet, polyethylene terephthalate fiber, nylon, polyethylene, and aluminum.112,113 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.114–116 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. 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, a cobalt-chromium alloy, or hydroxylapatite-coated metals. During the operative procedure, the prosthesis is inserted as described above.

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References

Gunshot Injuries

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The greater the ignorance, the greater the dogmatism.
—William Osler

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 residents. 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 thirteenth 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 fourteenth century. 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. Unfortunately, the battlefield has moved to the urban areas with increasing numbers of civilian gunshot injuries.

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. Recently the number of firearm-related deaths and injuries in children and adolescents has declined. 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. Because of past difficulties with surveillance, however, most reports likely underestimate unintentional firearm-related deaths and injuries overall. Interestingly, Patton and Woodward 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.

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.

Currently there are an estimated 135,000 GSWs treated annually in the United States. The incidence of firearm-related injury and death in the United States exceeds that of other developed countries. 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. 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. 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. Clark and colleagues 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. Demetriades and colleagues reported on the extensive experience of the University of California at Los Angeles. Of 4,139 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 following admission. All of the deaths were secondary to injuries to the chest, abdomen, or brain. There were no deaths associated with isolated facial injuries.

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 colleagues 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 one-quarter of 1% of the US health care budget of $950 billion, it is significant considering that the group most affected typically involves younger healthier patients that usually require very little medical care.

**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, 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:

\[
P = m(V_{\text{impact}} - V_{\text{exit}})^2
\]

where \( P \) is power, \( m \) is mass of the projectile, and \( V \) is velocity.

Based on these formulas, the velocity of a projectile has traditionally been considered far more important than its mass in wounding power. Indeed, often guns are classified as low velocity (< 350 m/s), medium velocity (350–600 m/s), and high velocity (> 600 m/s). Considering a typically sized projectile, a velocity of approxi-
mately 50 m/s is required to penetrate the skin, and a velocity of around 65 m/s will fracture bone. See Table 26-1 for a comparison of commonly encountered pistol and rifle cartridges.

In general, there is an inverse relationship between a bullet’s diameter (caliber) and velocity. Unfortunately, the realities of wounding are not as clear cut, and the emphasis on velocity and kinetic energy of the weapon as it relates to treatment strategies is excessive. In an excellent review, Fackler 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 importantly, 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 over-penetrates 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 below) 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 known, however, 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 (eg, fat) may impart far less damage than a larger projectile traveling at a lower velocity and striking an area of high density (eg, 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 below) 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.17
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.”

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 < 600 m/s, and usually cause tissue damage along the bullet tract only. 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 below).

Rifles and handguns are classified by caliber. The caliber of a weapon is the diameter of the muzzle bore, 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 in. (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 in. (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 26-1).

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/s. 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 26-2A). There are some exceptions to this classification scheme. For example, a 410 shotgun has a...
barrel whose internal diameter is 0.410 in. (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 (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 2½ in. (6.99 cm) or 3 in. (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.18 It is also important to note the different makeup of a shotgun shell (see Figure 26-2A). 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 (Figure 26-2B).

Most handguns and rifles have barrels with internal grooves referred to as rifling that impart a spin to the bullet. This keeps the projectile stable in flight over longer distances. In early firearms that were loaded from the muzzle (muzzleloaders), the tight fit between the bullet and the barrel that resulted from rifling significantly slowed loading. For this reason, most early military weapons were smoothbore. Sacrifices in long-range accuracy were a trade-off 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 26-3). 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 gravity.
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. 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 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 26-3). 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) 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 26-4). 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 26-4). 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. 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.

Classification Schemes

Classification of traumatic injuries is helpful in guiding treatment and, more importantly, 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.

Trauma systems have attempted to incorporate gunshot injuries into existing classification and trauma scoring systems. Unfortunately, current schemes have not proven 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 above. 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 non-penetrating (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. Gugala and Lindsey suggested a civilian gunshot injury classification scheme. It takes into account energy (high or low), involvement of vital structures (neural and vascular), wound type (non-penetrating, 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 > 12 m (type III), usually only the skin is penetrated and mortality is rare (0–5%). 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 > 25 cm of pellet scatter. Type II injuries had 10 to 25 cm of scatter. Type III injuries had < 10 cm of scatter and would roughly correspond to a type I injury in the classification of Sherman and Parrish. This classification scheme was developed and applied to abdominal shotgun wounds in an attempt to guide therapy. 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 26-5). The face frequently takes the full effect of the blast, whereas lethal intracranial involvement is avoided. 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 nonlife-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 C-spine injuries. Eye injuries are present in approximately 13% (Figure 26-6). 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 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 the midface (18–36%). Excluding patients that require airway control for associated brain injuries, Demetriades and colleagues found that 17.4% of patients required urgent airway control for facial injuries.12

Gunshot injuries to the neck may result in tracheal damage and require an emergent surgical airway (cricothyroidotomy). Intubation with fiber-optic 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 26-7A); this may influence the decision to proceed with tracheostomy. Associated tracheal injuries are another indication for tracheostomy (Figure 26-7B). Lastly, multiple system injuries with anticipated long-term ventilation is 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 colleagues in Los Angeles reported only 7.5% of patients with isolated gunshot wounds to the face to be in shock upon admission (systolic blood pressure < 90 mm Hg). In their report, 70 patients (28.3% of the total) required angiography, and 10 of these required embolization.12 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. 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 26-8). Standard methods for epistaxis control such as Foley catheters or specially designed balloon catheters will control most midface bleed-
Penetrating Neck Injuries

Gunshot wounds 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:

- 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 (approximately 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 cra-
nial nerves X, XI, and XII. It is the largest area and therefore the most commonly involved zone 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 gunshot wounds that involve mandibular fractures are accompanied by injuries to zone III.

Van As and colleagues 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.35 Many patients had more than one injury. Management strategies for penetrating neck injuries are typically based on the zone(s) involved.36,37 Gunshot wounds 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 26-11). 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 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 as 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 26-12). 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 > 400 to 500 cc/d 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. 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 26-10C). 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.

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 colleagues’ report, 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).35 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 colleagues evaluated 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. 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 gunshot wounds affect young healthy males. Nutritional status becomes an issue only in patients whose injuries preclude oral alimentation for an extended period (> 4 or 5 d). Feeding via nasogastric intubation allows bypass of the oral cavity and improved hygiene in the early days following 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 C-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 26-10B). 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 combined with three-dimensional reconstructions allows the surgeon an unparalleled view of

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**FIGURE 26-11**  A, Initial decision tree for penetrating neck trauma. B, Management of the symptomatic patient with a penetrating neck wound. ATLS = advanced trauma life support.

**FIGURE 26-12**  Chyle leak following penetrating injury to zone I of neck oversewn with nonabsorbable suture and covered with a flap from the sternocleidomastoid muscle.
Part 4: Maxillofacial Trauma

the extent of damage to the maxillofacial skeleton, which lies beneath the skin (Figures 26-13 and 26-14). 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 allows the ability to intervene with embolization of active bleeding vessels that are difficult to approach surgically (see “Penetrating Neck Injuries” above). 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.

Operative Procedure

Paralleling the evolution of firearms has been development in the management of gunshot injuries to the head and neck. 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 gunshot and shrapnel wounds to the face necessitated the development of maxillofacial surgery. Kazanjian and Converse described their approach to gunshot wounds as three phases consisting of initial débridement and suturing, immobilization of bony fragments with splints and ligatures, and, finally, reconstruction following 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. Many surgeons still advocate closed reduction and division of care into early (first 10 d), intermediate (10–60 d), and late (> 60 d) phases. The different nature of civilian gunshot wounds 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. 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 gunshot injuries.

FIGURE 26-13  Three-dimensional computed tomography scan demonstrating fragmentation of the mandible resulting from a gunshot wound. (Courtesy of James R. Koehler, MD, Birmingham, AL)

FIGURE 26-14  A, High-velocity entrance wound of the right cheek. B, High-velocity exit wound of left cheek. C, Three-dimensional computed tomography scan demonstrating extensive bony comminution associated with a high-velocity gunshot wound. (Courtesy of James R. Koehler, MD, Birmingham, AL)
wounds, 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 26-15 and 26-16). The surgeon facing a gunshot injury should consider the concept introduced by Manson for evaluation of four components: soft tissue injury, bone injury, soft tissue loss (true avulsion), and bone loss. After evaluation of the wound, a decision is made regarding early definitive repair versus the need for delayed repair. The majority of civilian gunshot wounds 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 26-15). 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. Otherwise, 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 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 has 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. 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 contrac-
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.46 Wounds in which the bullet traverses the aerodigestive tract or paranasal sinuses are at particular risk. Devitalized tissue and vascular congestion leads 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 gunshot wounds. Extensive surgical debridement is rarely indicated in wounds consistent with low-velocity projectiles to prevent infection.

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.47 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.48 Finally, 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.49,50

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 they 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. Since 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.51

**Specialized Structures**

**Facial Nerve**

Damage to the facial nerve is present in only 3 to 6% of civilian GSWs to the face.12,28 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 high-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 may 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 26-15).

**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, anti-sialalagogues 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 26-17).

**Controversies: Delayed versus Early Management and Closed versus Open Fracture Management**

Proponents exist both for closed management of fractures with delayed reconstruction as well as 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, whereas those advocating more aggressive management report improved functional and esthetic outcomes. Since 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 26-18). 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%. 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 War I, and much of what we know about the healing of free bone grafts was learned following 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. Gruss and colleagues 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 mandible. Some surgeons also advocate immediate bone grafting of mandible defects. 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. Clark and colleagues 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. Rigid fixation maintains the mandibular segments. Even if the titanium plate becomes exposed, wound care will allow it to be maintained until definitive reconstruction. In summary, primary bone grafting in the early phase of gunshot wound 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 or free flap reconstruction offers a predictable result, and in most cases primary grafting of the mandible is not indicated.

Late Reconstruction

Delayed bone reconstructions frequently suffer from a scarred hypovascular environment that does not support the graft. 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. As noted previously, free tissue transfer is usually delayed until after the acute setting to decrease the incidence of flap loss secondary to cloting 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. Anthony and colleagues reported on the use of the fibula in patients in whom previous reconstructive attempts for gunshot injuries had failed. Both cases involved secondary reconstructions. Some surgeons have advocated delayed reconstruction in gunshot wounds 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 prior to undertaking an extensive (and expensive) reconstructive effort. However, Cusick and colleagues found an incidence of only 8% confirmed mortality in the follow-up of 91 patients who had attempted suicide. All were patients who had long-standing chronic mental illness. De Leo and colleagues 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 gunshot wounds.

It should be noted, however, that some authors still recommend delayed reconstructive efforts. Siberchicot and colleagues 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 twentieth 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 in urban trauma centers dealing with gunshot injuries to the face. 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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Gunshot Injuries


Historic Perspectives

The management of craniomaxillofacial trauma, and the treatment of facial fractures in children in particular, has evolved gradually. A review of the historic landmarks in its treatment is important for understanding what has yet to be accomplished.

At the turn of the century Rene Le Fort was the first to document a tendency for the occurrence of specific patterns of midface fractures after direct facial trauma. Within a few years thousands of combined soft and hard tissue facial injuries resulted from the trench warfare of World War I and required urgent treatment and secondary reconstruction. Two physicians in particular, V.H. Kazanjian and H. Gillies, stand out for their work during this period. During and after World War I and again during World War II, these men laid the foundation for what we now know as craniomaxillofacial surgery. Rowe and Killey, Dingman and Natvig, and others refined the basic principles laid down by their mentors, set out to educate their peers, and brought these treatment principles to the civilian population after the two world wars. At the same time, the use of antibiotics and improved airway and metabolic management of the trauma patient increased survival rates.

The extensive surgical procedures that were often required to improve the quality of life of the multiply traumatized patient also became a reality.

Knowledge of the successful repair of traumatic facial injuries brought hope to people with congenital facial deformities. Gillies and Harrison pioneered the elective (extracranial) total midface advancement (Le Fort III osteotomy) for Crouzon syndrome. In 1967 Tessier described a cranial base approach to the management of skeletal deformities associated with Crouzon syndrome and Apert syndrome. His landmark presentation and publications were the beginning of modern craniomaxillofacial surgery. In 1968, Hans Luhr, a young maxillofacial surgeon, proposed that miniature (metal) bone plates and screws could be constructed and used effectively to fixate a mandibular fracture together for improved healing. Despite his enthusiasm these concepts of internal fixation for the craniomaxillofacial skeleton were not put into wide practice until the mid-1980s.

The concept of a hospital-based civilian trauma service that functioned 24 hours a day, 7 days a week, coupled with immediate “in-the-field” emergency reconstruction of the trauma patient followed by rapid transport to the trauma center, was pioneered by R.A. Cowley with the development of the University of Maryland’s shock trauma center. This concept of accurate and rapid verification of injuries by the trauma surgeon, combined with well-trained and immediately available surgical subspecialists, hospital support staff, and technology, led to remarkable patient recoveries in otherwise hopeless situations.

The importance of managing the facial injuries of the multiple-trauma patient became evident early in the trauma center’s experience. Following the basic philosophy of total patient rehabilitation, Gruss and colleagues in Canada and Manson and colleagues in the United States developed new concepts for the management of craniomaxillofacial trauma. Their basic approach incorporated the early accurate preoperative diagnosis of all skeletal injuries by clinical examination with verification using computed tomography (CT) scanning techniques, wide (direct) surgical exposure of all fractures for open reduction of displaced and mobile segments, use of stable internal fixation techniques (plates and screws), and primary autogenous bone
grafting to replace missing or irreversibly damaged skeletal units. The rapid dissemination of their concepts and basic clinical approach to everyday surgical practice around the world is a tribute to Gruss and Manson, who remain dedicated to the highest standards of clinical care, research, and education.

Children with facial injuries have not benefited equally from this rapid refinement in the management of facial trauma in adults. In 1943 Waldron and colleagues were the first to bring to the maxillofacial surgeon’s attention the often unique facial injuries in the traumatized child. MacLennan, and then Rowe, wrote about the rarity of facial fractures in children and suggested a basic approach with a philosophy toward conservatism. Other published articles have also tended toward conservatism, with only limited incorporation of the principles described earlier by Gruss and Manson. Only recently have the distinct advantages of accurate primary repair and the stable fixation of facial fractures been applied to the rehabilitation of injuries in children. Also, resorbable materials have been made available as a fixation option for pediatric craniomaxillofacial fracture management.

**Special Considerations in Children**

The general principles for resuscitating multiply injured patients follow the advanced trauma life-support principles created by the American College of Surgeons. This systematic approach to trauma in adult patients has been modified for the management of trauma in the child, taking into account several critical differences:

- Infants are obligate nasal breathers; at the same time their nasal air passages are relatively narrow and easily obstructed.
- The chest wall in children is pliable; major thoracic injuries may exist with fewer than expected signs of external trauma.
- Children frequently swallow air when they are injured or frightened, resulting in gastric dilatation. This may be a source of confusion when evaluating the patient to rule out an acute abdomen.
- Abdominal girth and the volume of the peritoneal cavity in infants and young children are relatively small. Significant intra-abdominal bleeding results in a rapid change in girth.
- Children may maintain a normal or borderline blood pressure level despite significant fluid loss and then compensate rapidly.
- Children have a larger body surface area-to-overall mass ratio than adults and are therefore more prone to hypothermia.

Children are generally injured in low-velocity accidents secondary to falls from low heights, playground equipment, or riding toys. Most commonly they arrive at the emergency room in a state of hemodynamic stability. With regard to the frequency of organs injured, the kidney is the solid organ that is the most frequently injured, followed by the spleen, liver, and pancreas. Hollow viscus perforations are much less common compared with adult injury patterns. In contrast nonaccidental trauma is more insidious and devastating. The pattern of organs injured, especially in the toddler, is the reverse of that seen in accidental trauma. With child abuse the history is often vague and inconsistent. A history of prior injuries and hollow viscus perforation is common.

Airway management in children with facial trauma has undergone significant change. With the widespread use of soft endotracheal tubes in the 1960s, the number of tracheostomies carried out for perioperative airway management decreased. Use of fiberoptic laryngoscopy has further decreased the incidence of tracheostomy for acute airway management in the pediatric trauma patient. Kaban and Posnick and colleagues reported no tracheostomies for airway management in their series consisting of 262 and 137 pediatric facial trauma patients, respectively. Also cervical spine injuries are exceedingly rare.

**Anatomic Considerations**

Maxillofacial injuries are much less common in younger children than in adolescents and adults. This lower incidence of facial trauma in infants and young children is a result of socioenvironmental, general physical, and craniomaxillofacial anatomic factors.

Before the age of 5 years most children live a relatively protected existence, with close adult supervision, strict limitations on their physical environment, and constant safeguards to limit injury. Although falls from limited heights are frequent the momentum gained by the child’s small body is of a low velocity. These low-impact forces can usually be absorbed by their well-padded skin, elastic skeleton, and cartilaginous growth centers.

After the age of 5 to 7 years, rapid progression of neuromotor development results in a general desire for independent activity, more frequent social interactions with other children, and a wider range of activities outside of the house, with less stringent parental and adult supervision. These factors result in increased opportunity for direct facial trauma. Additionally, increasing numbers of automobiles on the road and participation in pedestrian activities in public areas result in competition for space with motorized vehicles.

Ongoing craniomaxillofacial growth results in a changing anatomy (Figure 27-1). For the first several years of life the cranium follows the rapid pace of brain growth and results in a relatively large and prominent forehead. The ocular globes and orbits also develop rapidly early in life and join the forehead in their relative prominence early in life. This early period of life is marked by a lack of paranasal sinus and dental development, resulting in limited vertical height, horizontal projection, and transverse width of...
the maxillomandibular regions early in childhood. These factors result in a high skull-to-face ratio, leaving the frontal and upper orbital regions more exposed to trauma while the lower face remains relatively protected.

The mandible defines the lower border of the facial skeleton. Its evolving anatomy throughout growth and development significantly affects the pattern of injuries that occur in the lower face throughout childhood. During infancy and early childhood, the condylar process of the mandible has a well-vascularized marrow space with a thick and short neck. The condylar injuries seen involve compression, whereas neck fractures are more rare. This is in contrast with the condylar process’s tall and cortical characteristics later in childhood and adolescence, which leave it vulnerable to neck fractures. The mandible and maxilla continue to grow throughout childhood, maintaining a high cancellous-to-cortical-bone ratio and resulting in greater elasticity of the jaws, with more greenstick and nondisplaced fractures than are seen in adulthood. During the first few years of life the developing permanent tooth buds are small, and the tooth-to-bone ratio of the jaws is relatively low. In the mixed dentition phase (6 to 12 years) a higher tooth-to-bone ratio weakens the mandible in specific locations and encourages fracture through the developing tooth crypts when trauma occurs. After 5 years of age the paranasal sinuses develop gradually, resulting in areas of skeletal weakness, which results in ease of separation of the midface from the base of the skull when facial trauma occurs. Another factor in children is the highly osteogenic periosteum, which results in early healing of a fracture with more extensive remodeling after bone union has occurred.

Prevention
The increased use of age- and weight-specific protective restraints, lower speed limits, more strict alcohol abuse laws, and use of air bags have greatly diminished the incidence of motor vehicle–related trauma. For the infant and young child (less than 100 lb) release of the automobile's air bag may in itself cause trauma and even death (suffocation). The recognition that conventional lap belts do not properly restrain or protect infants and young children is a relatively recent finding. Special harness restraints, marketed since 1967, are required for children weighing less than 44 lb to prevent forward movement, to support the head, and to distribute the force of injury over a larger surface area. Current recommendations state that children weighing less than 100 lb or younger than 12 years should not be placed in an air bag–equipped seat. Infants should face the rear of the vehicle until they are at least 1 year of age. Vehicle safety belts are not to be used until the shoulder belt can be positioned across the chest with the lap belt low and snug across the thighs. Larger children may use booster seats, which have been shown to be protective in many motor vehicle crashes. A booster seat is used until the standard shoulder and lap belts fit appropriately.

Public acceptance, with mandatory laws, has progressively increased their use. Adults have a particular obligation to ensure that children riding in their automobiles are properly restrained in devices that are appropriate for their size and age.

Popular multispeed bicycles, dirt bikes, and off-road vehicles placed in the hands of untrained or unprotected children and adolescents have contributed to an increasing number of maxillofacial injuries in these users. Demas and Braun reviewed the injuries of all-terrain vehicle accident victims at a major pediatric trauma center and found that 37% of these patients sustained facial injuries. Participation in everyday sport activities is another source of pediatric facial fractures. Proper helmets, mouth protectors, and face guards are not always mandatory equipment, even in many organized contact sport leagues.

The awareness and recognition of child abuse and parental and family violence as a cause of facial trauma is another consideration that must not be overlooked by the pediatric or general dentist, pediatrician, and emergency room or trauma physician.

Diagnostic Studies
When facial trauma is suspected in the child, either by history or physical examination, radiographic documentation is mandatory. For the isolated mandible fracture the panoramic tomogram provides an excellent image of the entire mandible. However, for many patients with significant mechanisms of trauma, unclear history, or other factors, CT scanning provides the necessary information to make a complete diagnosis of any facial fractures. CT scanning has for the most part
part supplanted standard radiography as the preferred method of imaging pediatric facial trauma.\textsuperscript{62,63} Multiple CT scan planar views (coronal, axial, sagittal) performed with spiral scanning through all of the facial structures of interest, with three-dimensional reformation of the CT scan data, confirm the location and extent of skeletal, soft tissue, and visceral injuries (ie, brain or eye trauma). The patient is placed in the CT gantry and when necessary given sedation or, occasionally, general anesthesia. The radiation doses required for imaging are generally much lower than that for standard tomograms and have more limited scatter. Spiral and multislice techniques have reduced the dose of radiation significantly when compared with older CT methods.\textsuperscript{64} These techniques also allow for reformatted images in other planes (eg, coronal views) that are of excellent quality. This is helpful in patients who have been immobilized in a cervical collar. For isolated mandibular injury the panoramic tomogram still gives the best overall perspective of dentoalveolar and condylar head (of the mandible) anatomy and injuries and can be taken with a cervical collar in place.

Epidemiology and General Treatment Concepts

The patterns of facial injury in the pediatric population are considerably different than those for adults. Understanding these differences in injury presentation helps the surgeon during the evaluation and treatment phases. The objectives of the study previously published by Posnick and colleagues were to record the pattern of facial injuries treated over a 4-year period at a pediatric tertiary trauma unit and to document the treatment provided and any complications that occurred (Tables 27-1–27-4).\textsuperscript{53} The information gained from this study remains pertinent because it illustrates the common injury patterns seen in pediatric facial trauma at a major referral center for acute treatment.

All patients with acute facial fractures evaluated at a single tertiary care pediatric hospital over a 4-year period and treated by the author (J.C.P.) were enrolled in the study.\textsuperscript{53} The mechanism of injury, location and pattern of facial fractures, and extent of associated soft tissue injuries were evaluated. For each fracture the method of reduction, the type of fixation, and the need for primary bone grafts were recorded. Patients were placed into two groups: (1) those requiring acute care who received their primary treatment and evaluation at a single hospital, by Posnick; and (2) those treated for secondary (or residual) deformity, who were referred to Posnick for management at varying times after their injuries. All perioperative complications were catalogued. Follow-up of the patient group ranged from 1 to 5.5 years at the close of the study.

Fracture patterns were classified according to their complexity. Group 1 included all isolated fractures limited to one bone, group 2 included all multiple fractures occurring in a single bone, and group 3 included multiple fractures occurring in multiple anatomic regions within the facial skeleton. Because of the hospital’s entrance restrictions the oldest child in this population was 18 years.

The facial trauma population consisted of 137 patients (318 fractures) seen over a 4-year period.\textsuperscript{53} Most of the patients (42\%) were between 6 and 12 years of age, and the total population averaged 10.2 years of age. Boys (63\%) outnumbered girls (37\%) in the study (see Table 27-2). Of the 137 patients, 81 were treated for acute fractures (171 fractures) and 56 were evaluated for reconstruction of secondary deformities resulting from the initial fractures (147 fractures). Of the 171 acute fractures, 121 were treated surgically.

Fifty percent of the patients were injured in traffic accidents, followed in frequency by falls and injuries related to sports and altercations (see Table 27-1). Causal mechanisms appeared to be distributed similarly between sexes, except for a slightly higher number of males with fractures attributable to an altercation or

<table>
<thead>
<tr>
<th>Table 27-1</th>
<th>Mechanism of Pediatric Facial Fracture by Age Category</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age Group (year)</td>
<td>Traffic Accident</td>
</tr>
<tr>
<td>&lt; 3</td>
<td>1</td>
</tr>
<tr>
<td>3 to 5</td>
<td>12</td>
</tr>
<tr>
<td>6 to 12</td>
<td>32</td>
</tr>
<tr>
<td>13+</td>
<td>23</td>
</tr>
<tr>
<td>Total</td>
<td>68</td>
</tr>
</tbody>
</table>

Adapted from Posnick JC et al.\textsuperscript{53}

<table>
<thead>
<tr>
<th>Table 27-2</th>
<th>Patient Age and Occurrence of Pediatric Fractures by Region</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age Group (year)</td>
<td>Cranium</td>
</tr>
<tr>
<td>&lt; 1</td>
<td>0</td>
</tr>
<tr>
<td>1 to 2</td>
<td>2</td>
</tr>
<tr>
<td>3 to 5</td>
<td>2</td>
</tr>
<tr>
<td>6 to 12</td>
<td>8</td>
</tr>
<tr>
<td>13+</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>16</td>
</tr>
</tbody>
</table>

Adapted from Posnick JC et al.\textsuperscript{53}
to recreational vehicle accidents. The likelihood of high-velocity injuries increased with age (10% in the 1- to 2-year age group, increasing to 55% in the 6- to 12-year age group). Falls as a cause declined with age (55% in the 1- to 2-year age group, dropping to 8% in the 13+ year age group). The number of facial fractures tended to increase in the summer months; 45% of all fractures occurred between the months of May and August.

Of the 137 children with facial fractures, 66 (48%) sustained isolated fractures (group 1), 27 (20%) had multiple fractures in a single bone (group 2), and 44 (32%) had multiple fractures in multiple sites within the craniofacial skeleton (group 3). Children younger than 3 years were more likely to sustain only single fractures (see Tables 27-2 and 27-3). The children experienced one or more fractures in the following craniofacial regions: mandibular (55%), orbital (30%), dentoalveolar (23%), midface (17%), nasal (15%), zygoma (14%), and cranium (12%). Fracture pattern profiles were similar in both the acute care and secondary treatment groups. Midface (20 of 23) and zygoma (18 of 21) fractures were more likely to occur in children older than 6 years of age (see Table 27-2).

The distribution of fractures by anatomic region and degree of complexity is presented in Table 27-3. Similar anatomic patterns were seen in both the acute and secondary cases. Most of the fractures occurred as part of a complex injury pattern, with the exception of mandibular fractures, which occurred as isolated fractures with nearly equal frequency.

Eighty-one patients with acute injuries were seen for evaluation during the period of the study. These patients sustained 175 fractures, requiring 121 operative interventions. Injuries occurring at high velocity, such as traffic-related events (74%), more frequently required interventions than those occurring at low velocity, such as falls (51%). Boys did not require significantly more operations than girls. Necessity for operative intervention increased significantly with the increasing complexity of facial fractures (group 1 to group 3) but not with age.

Open or closed reduction techniques were used with approximately the same frequency. When closed reduction was used, most patients (93%) underwent reduction and stabilization of the fracture with maxillomandibular fixation (eg, Erich arch bars, skeletal suspension wires, Stout wires). An external fixation device was used for only one patient. Only four fractures were reduced and not stabilized. Thirteen fractures (20%) were opened and explored without any form of fixation. Most of these were orbital floor fractures with associated bone-grafting procedures. Of the fractures treated by open reduction, 35 (55%) were managed with only one form of fixation to stabilize the reduction and 14 (21%) with multiple forms. Use of plates (miniplates or microplates) and screws accounted for 82% (40 of 49) of the internal fixation methods used. Although age was not a factor in the choice of plate-and-screw fixation, review of the data indicated that this method was not used on any patient younger than 3 years (only three of the children in our population were younger than 3 years). Plates and screws were used most often in the mandible (40%) and orbits (26%). Bone grafts (21) were used for fractures of the orbit (16), cranial vault (2), mandible (2), and nose (1). The preferred donor sites included cranium (10), anterior maxillary wall (4), and hip (2).

Complications in treating pediatric facial trauma are rare if good principles are adhered to and precise surgical execution is achieved. This is due, at least in part, to the excellent healing capabilities of most children. Nonunion is very rare due to the excellent healing potential of pediatric bone. Malunion may occur but is

<table>
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<tr>
<th>Table 27-3</th>
<th>Pediatric Fracture Pattern by Anatomic Region and Complexity</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anatomic Region</strong></td>
<td><strong>No. of Subjects</strong></td>
</tr>
<tr>
<td>Cranium</td>
<td>25</td>
</tr>
<tr>
<td>Orbit</td>
<td>41</td>
</tr>
<tr>
<td>Zygoma</td>
<td>21</td>
</tr>
<tr>
<td>Midface</td>
<td>23</td>
</tr>
<tr>
<td>Nose</td>
<td>17</td>
</tr>
<tr>
<td>Mandible</td>
<td>75</td>
</tr>
<tr>
<td>Dentoalveolar</td>
<td>32</td>
</tr>
</tbody>
</table>

*Adapted from Posnick JC et al.\(^{31}\)*

Fracture complexity resulting from trauma was represented by three groups: group 1, trauma involving a single fracture in a single anatomic region; group 2, trauma involving multiple fractures in a single anatomic region; and group 3, trauma involving multiple fractures in multiple anatomic regions.

<table>
<thead>
<tr>
<th>Table 27-4</th>
<th>Management of Acute Pediatric Fractures*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No Surgical Treatment</strong></td>
<td><strong>Closed Reduction</strong></td>
</tr>
<tr>
<td></td>
<td>(No. of Fractures)</td>
</tr>
<tr>
<td>50</td>
<td>Reduction only (4)</td>
</tr>
<tr>
<td></td>
<td>Exploration only (13)</td>
</tr>
</tbody>
</table>

*\(N = 171.\)*

Adapted from Posnick JC et al.\(^{33}\)
usually due to inadequate reduction. In Posnick and colleagues study no deaths, tooth loss, or injuries to the eye or brain were directly attributable to any operative procedure. Two patients developed soft tissue infections that responded to treatment by incision, drainage, and administration of antibiotics. Another developed a small area of alopecia after a coronal flap procedure. One patient, in whom a fracture extended through a tooth root, developed a periapical tooth abscess. This condition was treated with extraction and systemic antibiotic therapy. One miniplate was removed 1 year later because it was palpable and visible below thin forehead skin.

Of the 137 patients in this series, 77 (56%) had associated soft tissue injuries. These included lacerations to the scalp (31%), and injuries to the ear (20%), chin (13%), tongue (8%), forehead (6%), and eyelid (6%). Thirty-three percent in the facial fracture group had injuries to other organ systems. Associated head injuries accounted for 42% of this group, followed by damage to the extremities (24%), eyes (22%), thorax (10%), and abdomen (2%). None of our patients sustained injuries to the cervical spine. As expected, the more complex the facial injury, the greater the likelihood of associated injury \( p = .03 \); 19% of group 1, 26% of group 2, and 36% of group 3 patients had an associated injury. Six percent required emergency endotracheal intubation when first evaluated; no emergency tracheostomies were required.

Patterns of Pediatric Facial Fracture Injury and Methods of Management

Anterior Cranial Vault and Supraorbital Ridge Fractures

Fractures of the forehead and upper orbital regions, combined with brain injury and dural tears with cerebrospinal fluid (CSF) leakage, constitute a frequent pattern of injury in infants and in children younger than 5 years when major anterior craniofacial trauma occurs (Figure 27-2). Isolated cranial vault fractures (18 of 318 fractures, 6%) occurred infrequently in this series. When they did occur, the anterior cranial vault was the most common location (13), followed by the posterior vault (4) and frontal sinus (1). Complete evaluation using CT scanning of the brain, eyes, and craniofacial skeleton, combined with neurosurgical, ophthalmologic, and craniofacial assessment, should be performed to evaluate the injuries completely. A combined neurosurgical and craniofacial reconstructive procedure is necessary for repair of the injured brain, dura, and skeleton. A coronal (skin) incision provides the best exposure of the fractured regions and surrounding normal structures. Once the brain and dural injuries have been managed by the neurosurgeon, reduction and stable fixation (microplates and screws) of all fractures are completed by the craniofacial surgeon. When massive comminution exists, bony defects are present, or complete orbital roof reconstruction is required, then autogenous cranial bone is harvested and used. In a normally developing child the skull will mature into three clinically reliable layers (outer table, medullary cavity, inner table) between the ages of 2 and 5 years. In these instances the bone of the cranial vault is suitable for splitting, yielding bone for grafting. These techniques and a team approach to the early diagnosis and management of combined injuries are cost effective and result in a rapid facial rehabilitation for the injured child.

Naso-orbitoethmoid and Frontal Sinus Fractures

The prevalence of naso-orbitoethmoid fractures closely follows the development of the paranasal sinuses. They are rarely seen in children younger than 5 years, but they become progressively more common in adolescents and adults (Figure 27-3). Rowe reviewed his series of pediatric fractures and found that injuries to the middle third of the face made up only 0.5% of all pediatric fractures. Kaban and colleagues reported no midface fractures in 109 pediatric facial fracture patients from 1965 to 1975. During the next 10 years, with another 184 fractures, they reported only 5 midface fractures, all Le Fort III level injuries. Posnick and colleagues reported that midface injuries seen at a major pediatric trauma center during a 4-year period made up 17% of a series of 318 fractures in 137 patients. Kaban associated this increased prevalence of midface injuries with the increase in survival of persons involved in serious motor vehicle accidents, which may result in more extensive facial injuries in the survivors. When displaced naso-orbitoethmoid fractures do occur in children, we have adopted the same open reduction and internal fixation (ORIF) techniques generally accepted for adult-type injuries. Stable internal fixation techniques (micro- and miniplates and screws) and primary autogenous cranial bone grafts when indicated, result in the anatomic healing required to achieve satisfactory rehabilitation of the child with facial injury.

As in the adult, when the medial canthal ligament is displaced, it usually remains attached to a bone fragment. The medial canthal ligament and bone fragment are repositioned and fixed without the need for a direct medial canthopexy. Formal medial canthopexies often contribute to an unnatural appearance and should be avoided if possible. Often the bony fragment(s) can be repositioned with the aid of microplates and screws with or without the use of a transnasal wire.

Frontal sinus injuries in children are approached in a similar way to those in their adult counterparts. Anterior frontal sinus wall fractures are anatomically reconstructed and stabilized to prevent contour deformity. When the fracture components are severely comminuted, autogenous cranial bone grafts can be used to replace the entire unit. Depending on the
extent of frontal sinus development and injury, the mucous membranes may require débridement with maintenance of a patent frontonasal duct or, in cases of fractures of the ducts, sinus obliteration with sealing of the duct. If the posterior frontal sinus wall is injured, neurosurgical consultation helps determine whether cranialization of the sinus through an intracranial approach is required. Since CSF leaks are common with dural tears in these injuries, it is often helpful to place bone, fibrin glue, and a pericranial flap in the defect to prevent CSF leaking. A double-ring sign is seen on filter paper when CSF is present within nasal sinus. 

FIGURE 27-2 A 16-year-old girl sustained frontal and upper orbital trauma when she hit her forehead on the dashboard in a motor vehicle accident. Initially the cerebrospinal fluid (CSF) leak was repaired through a local scalp laceration; minimal attention was given to her frontal and orbital fractures. Ongoing CSF leak with meningitis and loss of the frontal bone flap occurred, after which she was referred to Posnick and colleagues, and a delayed combined neurosurgical/craniofacial approach was carried out. A, Frontal view before the delayed surgery. B, Frontal view 1 year after reconstruction. C, Oblique view before the delayed surgery. D, Oblique view 1 year after reconstruction. E, Three-dimensional computed tomography (CT) scan of frontal bone defect. F, Intraoperative view of dural tear resulting in traumatic encephalocele. Access craniotomy/osteotomies allow exposure for reconstruction of orbital roof/medio-orbital wall defects. G, CT scan of the anterior cranial base and orbital roof/medio-orbital wall defects. H, Intraoperative view of frontal bone defect and displaced orbital rim fractures. (CONTINUED ON NEXT PAGE)
Part 4: Maxillofacial Trauma

fluid. Alternatively, β2-transferrin can be measured within the nasal fluid to determine if nasal leaking is indeed CSF. Endoscopic techniques with imaging guidance can be used to effectively repair persistent leaking that may occur postoperatively.

**Le Fort (Midface) Fractures**

The prevalence of Le Fort midface fractures increases rapidly once aeration of the maxillary and ethmoid sinus cells has occurred. The rapid development of the sinuses takes place between 6 and 12 years of age. Consequently maxillary fractures in children do not follow the patterns seen in adults. Displaced midface fractures should be treated with ORIF techniques similar to those used in adults.13,78 This is necessary to achieve and maintain anatomic restoration. Closed reduction techniques may be preferred in specific clinical situations to avoid injury to the unerupted permanent dentition, but the dental injuries are generally the result of the trauma event rather than of reduction and fixation techniques that have been carried out by an experienced surgeon familiar with the dentition.

In Posnick and colleagues study 23 patients sustained 31 fractures in the midfacial region. These included nasofrontoethmoid fractures (13 of 31, 42%), Le Fort I (8 of 31, 26%), Le Fort II (5 of 31, 16%), and Le Fort III (5 of 31, 16%). Midfacial fractures generally occurred as part of a complex facial fracture pattern; only 2 of 31 (6%) occurred in isolation. Although few acute midfacial fractures occurred, the majority (9 of 12) required surgery (Figure 27-4). Unstable or displaced fractures were treated with open reduction and internal fixation. The surgical goals in such cases are to restore midface projection, facial width, and orbital volume, and to normalize occlusal relationships. Seven of nine midfacial fractures were stabilized with plates and screws.

A circumvestibular intraoral mucosal incision provides ideal exposure of maxillary fractures through the zygomatic buttress, anterior maxillary wall, and piriform nasal aperture regions. When additional access to the zygomatic arch, frontozygomatic suture, supraorbital ridge, and frontonasal junction is required, a coronal (skin) incision is also used. If specific exploration of the infraorbital rims, orbital floors, and lower aspects of the medial orbital walls is required, a subciliary, lower lid, or transconjunctival incision is added. Palatal incisions are to be avoided, and preservation of the gingiva is important to the child’s periodontal health. As in the case of adults the restoration of normal anatomic position of the midfacial skeleton generally requires open reduction, stable fixation (miniplates and microplates and screws) and may rarely require autogenous cranial bone grafts or the placement of alloplastic materials.

**Zygomatic Complex Fractures**

A zygomatic complex fracture describes a fracture through the frontozygomatic suture, zygomatic arch, infraorbital rim, and zygomatic buttress. Fracture through the orbital floor and lateral orbital wall completes the quadripod injury. The extent of displacement of the zygomatic complex fracture is best clarified through CT scanning in the axial and coronal planes and defines the extent of surgery necessary to restore and maintain preinjury anatomy. The child’s presenting physical findings are similar to those seen in the adult. They generally include periorbital ecchymosis; paresthesia over the zygomatic arch, lateral nose, cheek, upper lip, and anterior maxillary teeth; and subconjunctival hemorrhage.79 Ophthalmologic consultation is essential to determine baseline ocular globe and extraocular muscle injury and dysfunction. Since the base of the lateral orbit is made up of the zygomatic bone,
Fractures within the orbital floor frequently require management in conjunction with repositioning of the zygoma. Some injuries require reconstruction of the orbital floor with autogenous bone or synthetic materials. Of the eight acute zygoma fractures observed in Posnick and colleagues’ study, three were minimally displaced and managed without surgery. The five displaced fractures were comminuted injuries that were treated with open reduction and internal fixation. Three of these fractures were stabilized with plates and screws.

Most zygomatic complex fractures can be approached and reduced using multiple approaches such as maxillary vestibular, lower eyelid, and brow incisions. If a badly comminuted zygomatic arch is associated with a displaced zygomatic complex fracture, a coronal (scalp) incision may be used with intraoral and subciliary (or lower lid or transconjunctival) incisions to expose, explore, reduce, graft, and internally fix all fractured regions. With a minimally displaced or incomplete fractured zygoma, more limited treatment is used to achieve adequate fracture reduction. This can be

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**FIGURE 27-3** A 5-year-old girl who was in a motor vehicle accident sustained orbitonasal, ethmoid, and frontal bone fractures with associated brain and dural injury with cerebrospinal fluid leak. She required a combined neurosurgical/craniofacial procedure. A, Frontal view 6 days after surgery. B, Frontal view 2 years after (single-stage) reconstruction. C, Oblique view 6 days after surgery. D, Oblique view 2 years after reconstruction. E, Worm’s-eye view 6 days after surgery. F, Worm’s-eye view 2 years later. G, Intraoperative view of reconstructed orbitonasal and frontal fractures. Stabilization is with titanium plates and screws. H, Intraoperative close-up view of reduced orbitonasal and frontal fractures stabilized with titanium plates and screw fixation. Medial canthopexies were also carried out (note location of wires). Reproduced with permission from Posnick JC.134
done through a Gillies’ approach within the temporal scalp, an eyebrow incision, or a Keene approach from an intraoral vestibular incision.

**Blow-Out and Blow-In Fractures of the Orbit**

Blow-in and blow-out fractures of one or more orbital walls and/or floor may be associated with more complex fractures (eg, anterior cranial vault/upper orbital, naso-orbitoethmoid, Le Fort midface, or zygomatic complex fractures) or may occur as isolated injuries.\(^{80-83}\) The key to thorough evaluation is complete clinical, ophthalmologic, and CT scan assessments.\(^{84}\) A thin-sliced axial and coronal CT scan is completed to visualize all four orbital walls and/or floors to ensure that the presence and extent of all blow-in or blow-out fractures are recognized. The ophthalmologic assessment may require pupillary dilatation and slit-lamp evaluation in the ophthalmologic suite.

Orbital fractures are common in children and were frequent in Posnick and colleagues’ study; 41 patients sustained 73 separate fractures of the orbit. The distribution of fractures within the orbit included the floor (23 of 73, 32%), medial wall

**FIGURE 27-4** A 14-year-old boy sustained combined Le Fort I and II fractures with bilateral orbital blow-out fractures when he was accidentally kicked in the face while playing competitive soccer. A, Frontal view before repair. B, Frontal view 1 year after (single-stage) reconstruction. C, Occlusal view before repair. D, Occlusal view 1 year after reconstruction. E, Illustration before and after reduction and fixation. (CONTINUED ON NEXT PAGE)
Of the acute fracture group, 21% of the fractures were orbital fractures. These were treated both surgically (59%) and nonsurgically (41%) (see Table 27-4). Most of the orbital injuries that were managed operatively were minimally displaced floor fractures. Thirty-two percent of orbital fractures were managed by exploration, reduction, and grafting with autogenous material but without graft fixation (Figure 27-5). Plate-and-screw fixation was used in six orbital rim fractures and three roof fractures. With the collaboration of a neurosurgeon, displaced roof fractures (blow-in fractures) were routinely treated with open reduction via an intracranial approach. The roof was reconstructed with contoured calvarial bone grafts fixed with plates and screws.

Once a clinically and radiographically significant orbital wall and/or floor injury is recognized, early exploration and repositioning of the soft tissues back into the orbit with simultaneous reconstruction of injured orbital walls and/or floor to appropriate dimensions and overall intraorbital volume is carried out. Because the complications of extraocular muscle entrapment, diplopia, and enophthalmos are difficult to treat later, early evaluation of patients at high risk, followed by prompt surgical intervention, is encouraged. Orbital wall and/or floor fractures heal rapidly in children and result in a higher incidence of scar cicatrization of the herniated orbital soft tissues than in adults.

**Nasal Fractures**

Nasal fractures are also common in the pediatric population. Of the few acute nasal fractures that occurred in the author’s series (12 of 171, 7%), 58% were minimally displaced and did not require surgery, and 33% were treated by closed means. Only one fracture required open reduction. Many isolated nasal fractures were treated on an outpatient basis. The nasal fractures seen by Posnick and colleagues in this study were generally associated with other facial fractures and were therefore not representative of nasal fractures seen in general at the hospital (emergency department).

Development of the nasal septum is thought to be a major factor in midface growth. In theory, trauma to the nasal region early in childhood will negatively impact on midface growth. Although the nose is the most frequently fractured part of the face in a child, extensive midface growth retardation after trauma has only rarely been documented.

Nasal injuries are often recognized but then ignored as unimportant. Two serious pitfalls in treating nasal fractures in children are (1) failure to recognize adjacent bony injuries extending outside the nose and (2) septal hematoma after nasal trauma (which may in theory result in septal necrosis and perforation). Diagnosis of nasal and septal fractures is usually based...
on clinical examination. Radiographic confirmation can be made with CT scans or plain films of the nose, but these are usually not necessary for clinically apparent and isolated nasal septal fractures. Displaced nasal bone and nasal septal fractures should be reduced and stabilized with splints in a similar manner as is done in adults. This should be completed within several days of the injury, as children heal more quickly than adults, making repositioning of the small nasal bone fragments more difficult with time.

**Mandibular Fractures**

The lower jaw of a child represents an evolving anatomy that affects the pattern of fractures seen at varied ages (Figures 27-6 and 27-7). Mandibular fracture patterns are affected by the fact that the child’s jaws are filled with teeth at various stages of development at different ages. Injury to the developing bone and tooth buds may result from the trauma of the fracture, the surgical technique, or complications of treatment (e.g., nonunion, malunion, infection).

In Posnick and colleagues’ study mandibular fracture sites included the condyle (59 of 107, 55%), parasympysis (29 of 107, 27%), body (10 of 107, 9%), and angle (9 of 107, 8%). Thirty-nine percent of all fractures in the study were of the mandible. Of those treated, 18 of 28 (64%) were treated with closed reduction, most of which were condylar process fractures with an element of malocclusion. Only two condylar process fractures were opened. Both were low subcondylar mandibular neck fractures associated with other injuries in the mandible. Minimally displaced body and angle fractures with a satisfactory occlusal relationship were frequently treated with maxillomandibular fixation. Displaced or comminuted fractures were treated with open reduction and internal fixation, and this treatment was used most frequently for parasympysis injuries (53%) and angle fractures (24%) (see Table 27-4).

A surgeon familiar with the evolving dentition is able to apply arch stabilization and maxillomandibular fixation, when indicated, in dentulous children of all ages. Obstacles to the usual application of surgical arch bars are overcome with the use of skeletal fixation: circum-mandibular, circumzygomatic, infraorbital, anterior nasal spine, and piriform aperture wires are used for additional support. When internal fixation techniques are required, careful application of microplate or miniplate and screw fixation, generally with unicortical screws strategically placed along the thick cortical inferior border combined with arch bar stabilization, is often the least traumatic and most stable option. Knowledge of the location of developing teeth allows the surgeon to place internal fixation as needed, with minimal trauma.

The general principles of treating mandibular fractures are the same in children and adults: anatomic reduction is combined with stabilization adequate to maintain it until bone union has occurred. With the exception of mandibular condyle fractures, we frequently find that the judicious use of ORIF is preferable to the closed reduction and immobilization techniques with splints when treating fractures in the deciduous and mixed dentition. Some surgeons believe that minor degrees of malunion may be self-correcting in children or at least amenable to orthodontic alignment. This margin of safety should not be used as an excuse for inadequate treatment.

**Mandibular Condyle and Subcondylar Fractures**

Injury to the mandibular condylar process may affect jaw growth and temporomandibular joint (TMJ) function. The mandible is the final facial bone to complete normal growth, and injury to the condylar growth center before skeletal maturity may lead to growth retardation on the
ipsilateral side, resulting in facial asymmetry and malocclusion.

Once a mandibular condylar fracture occurs, a degree of TMJ degenerative changes or growth restriction is a likely scenario despite the treatment option selected. Condylar injuries represent a wide spectrum of fractures, dislocations, and compression injuries. They may be intracapsular or extracapsular, displaced or nondisplaced, comminuted or noncomminuted, open or closed, located low or high in the condylar neck, medial or lateral pole fractures, and isolated injuries or associated with more complex facial fractures.

The treatment of a fracture of the mandibular condyle remains controversial. Most authors and clinicians continue to advocate a nonoperative approach, whereas a few prefer the use of open reduction techniques. The frequency of less than ideal results seen with varied treatments given for similar injuries is a reflection of the irreversible injury that may occur to the highly differentiated and specialized TMJ structure. Despite a great deal of surgeon interest and experience over the years with open reduction techniques, its proponents have not been able to convincingly demonstrate a lower incidence of growth disturbance, TMJ ankylosis, internal derangement of the TMJ, loss of posterior facial height, or malocclusion in their patients. Although endoscopic techniques have been reported, a detailed analysis of outcomes is lacking and the benefits remain to be seen.

Open reduction of a condyle fracture may be warranted in a child in some instances. Indications may include the following:

- Displacement into the middle cranial fossa
- Unacceptable occlusion after a closed technique trial has failed
- Avulsion of the condyle from the capsule
- Bilateral fractures of the condyles with comminuted midface fractures

We continue to advocate a nonoperative approach for most condylar and subcondylar fractures in young children. A short period of partial immobilization with elastics is generally useful for patient comfort, to encourage soft tissue healing, and to limit the conversion of a greenstick or minimally displaced fracture into a complete or fully displaced one. Ten to 14 days of use of firm elastics is generally enough to accomplish these goals and still allow early increased range of motion to limit the likelihood of the development of TMJ fibrosis or ankylosis. Instituting a regimen of physical therapy for several months is important to avoid TMJ fibrosis or ankylosis.

When a condyle fracture occurs and the use of firm elastics needs to be limited to reduce the incidence of TMJ sequelae, the fixation technique selected for additional simultaneous maxillary and mandibular
fractures should be carefully considered. The common occurrence of a combined parasymphyseal and condylar fracture will warrant a more stable form of parasymphyseal fracture fixation (miniplates and screws) so that early active mandibular range of motion with TMJ function can occur. Instituting a liquid diet for a limited time period even after firm elastic use may be helpful in preventing displacement of parasymphyseal or body fractures. When a mandibular angle fracture occurs in the presence of a condyle fracture, the combined forces may be significant enough to cause displacement unless ORIF at the angle fracture is carried out.

The advantages of continuous passive motion (CPM) for the healing of injured joint surfaces have been well documented in experimental animals. Salter and colleagues concluded that chondrogenesis in the healing of full-thickness defects in the rabbit femur occurs through differentiation of the pluripotential cells of the subchondral
bone to chondrocytes as a result of the stimulation provided by CPM of the joint.\textsuperscript{126,127} They documented improved healing of intra-articular fractures with the use of CPM compared with immobilization.\textsuperscript{128} The use of CPM in the treatment of TMJ disorders and for the early management of acute TMJ injuries seems to have promise but has not been used often. Conversely the use of extended periods of immobilization of the acutely injured TMJ appears to be counterproductive. A regimen of physical therapy for the TMJ after an initial phase of immobilization is recommended for optimal rehabilitation. Also, functional appliances have been used in an attempt to reestablish vertical height to foreshortened fracture sites in the early injury phase. Although case series have shown good results, no outcome data are available that show a clear advantage to using this technique.\textsuperscript{104} Since growth disturbance is a concern with these injuries, long-term follow-up is necessary to evaluate the possible development of asymmetry.

**Parasymphysial Fractures** When marginal reduction and fixation techniques are used for parasymphysial or symphysial fractures, a small dentoalveolar gap often occurs between the two teeth adjacent to the fracture site. Using open reduction techniques with stable (miniplate and screw) fixation at the inferior border, combined with reduction and stabilization at the dentition with an arch bar, gives a more reliable bony union of the injury without displacement. Plating at the tension-band zone is not recommended in the mixed dentition.

**Body Fractures** Body fractures of the mandible usually have favorable “muscle pull” vectors on the segments, which encourage reduction rather than displacement. In these situations closed reduction techniques with maxillomandibular fixation generally suffice. Alternatively the skilled surgeon can place inferior border plates and screws with the aid of a transcortaneous trocar and intraoral incision. When extended maxillomandibular fixation must be avoided (eg, associated condyle fracture or severe trauma), more stable forms of internal fixation (plates and screws) are indicated.

**Dentoalveolar Injuries** Anterior maxillary and mandibular teeth and their supporting alveolar structures often bear the brunt of lower face injuries, and as a result dentoalveolar injuries are very common in the pediatric population.\textsuperscript{43,111,129–131} The teeth may be concussed, subluxed,
partially or totally avulsed, or intruded. In Posnick and colleagues’ study dentoalveolar fractures were evenly distributed between the mandible and the maxilla. Thirty-two children sustained 44 fractures, 8 of which were isolated. Teeth that are loosened should be returned to their normal position in the tooth socket and alveolar segments reduced to their preinjury position. The reduced teeth and alveolar segments should be immobilized until healing occurs. Isolated dentoalveolar injuries may be adequately reduced under local anesthesia and then stabilized with the application of acid-etch bonding techniques and a braided wire. Arch bars can be helpful in select cases but often will extrude the teeth. The selected splinting techniques must meet certain criteria, including easy fabrication, maintenance of only passive forces on the teeth, lack of irritation to the soft tissues, maintenance of normal occlusion, allowance of good oral hygiene, access for subsequent endodontic treatment, and easy removal. Longitudinal reassessment with a pediatric or general dentist is important because ankylosis of primary teeth may prevent the normal eruption of permanent teeth.

Resorbable Fixation Materials

Titanium alloy plates and screws are the standard for craniomaxillofacial fixation. The use of plate and screw titanium fixation in the craniomaxillofacial skeleton has consistently resulted in low complication rates and excellent biocompatibility. However, controversy associated with their use in growing bones has led to the development of resorbable fixation materials. Issues of biocompatibility, strength, bulk, inflammatory response, and predictable resorption rates continue to be discussed. Most resorbable plate and screw fixations use isomer configurations of alpha-hydroxy polyactic and polyglycolic acids.

Possible advantages of resorbable fixation include the following:

- Degradation of the material by the citric acid cycle into CO₂ and H₂O
- No interference with imaging (CT, magnetic resonance imaging, standard radiographs)
- No effect on postoperative radiation treatment
- The possibility of integrating substances such as antibiotics within the fixation material

Possible disadvantages of resorbable fixation include the following:

- Less mechanical strength when compared with titanium alloys of similar sizes
- “Memory” of the material, which may distort reduction of fracture
- Increased reactivity during the degradation phase
- Increased operative working time

Summary

The pattern of craniomaxillofacial fractures seen in children and adolescents varies with evolving skeletal anatomy and socioenvironmental factors. Facial fractures in children may go unrecognized as a result of limited communication, incomplete radiographic examination, or the late presentation of the patient by the family. Recognition of the differences between children and their adult counterparts is important in facial rehabilitation. Consideration should be given to open reduction of the fractures, primary autogenous cranial bone grafting, and the use of stable forms of fracture fixation (miniplates and microplates and screws). Late sequelae of pediatric fractures occur even when appropriate and prompt treatment is instituted. The effects of the trauma event as well as the surgical intervention or lack of treatment on growth and development may be contributing factors. Long-term follow-up by appropriate practitioners is mandatory to monitor these events.

References


Management of Panfacial Fractures

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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 since 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, nasoorbitoethmoid region, maxilla, and mandible. Complex facial injuries such as these are generally the result of high-velocity trauma.22 Prior to 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.

There have been several important advances in the management of maxillofacial trauma that 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 Since gunshot wounds are addressed in Chapter 26, 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 both in vertical and horizontal planes.10,32–34 The vertical buttresses include the nasomaxillary, zygomaticomaxillary, and pterygomaxillary buttresses (Figure 28-1). 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 process of the maxilla, and maxillary tuberosities. Usually the nasomaxillary and pterygomaxillary 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. These include the frontal, zygomatic, maxillary, and mandibular buttresses (Figure 28-2). 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 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 28-3). This approach works well if there is a solitary midpalatal fracture without comminution or avulsion. 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 28-4). This is by no means a foolproof method when both the upper and lower arches are fractured. The more severe the injury (ie, multiple segments), the more difficult it is to establish a preinjury occlusion. If the patient has dental models of his preinjury occlusion
Management of Panfacial Fractures

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 since this is generally a robust bone that can undergo anatomic reduction if attention is paid to detail.

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 lingual cortical surfaces prior to fixation yields better results (Figure 28-5).\textsuperscript{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 temporo-mandibular 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 fixation of the zygomaticomaxillary complex.\textsuperscript{42–44} If other aspects of the facial skeleton are ignored, use of this suture alone can 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 along the internal surface of the lateral orbital wall (Figure 28-6).

Once reduced, a small plate is placed across this fracture for fixation. Since 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.

**Intercanalthal Region**

The intercanthal region may also be used to reestablish midfacial width since the intercanthal distance is fairly constant in the adult facial skeleton. Restoration of the proper intercanthal distance via reduction of the naso-orbitoethmoid complex can help to determine facial width (Figure 28-7). 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 cases 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 advent of computed tomography (CT). CT has improved our ability to image the facial skeleton and obtain details not possible with plain films (Figure 28-8). It allows the clinicians to determine not only the location of fractures but also the degree and direction of displaced segments. Since the introduction of CT, it has undergone an evolution both in 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. 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 28-9), if needed, and decreases the number of repeat scans. 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. This allows the surgeon to view necessary
Management of Panfacial Fractures

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 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 28-10):

- Bicoronal 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:

- Bicoronal 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

Figure 28-9  A and B, Three-dimensional computed tomography images of patient with extensive midface injuries. Note the detail and quality of the images.

Figure 28-10  Surgical approaches to the facial skeleton: bicoronal with preauricular extension (a), paranasal (b), superior tarsal crease (c), subciliary (d), transconjunctival with lateral canthotomy (e), maxillary vestibule (f), mandibular vestibule (g), cervical crease (h).
primary bone grafting and resuspension of the soft tissue after extensive exposure of the facial skeleton.\textsuperscript{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.\textsuperscript{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 bicoronal flap that has already been created during the management of the fractures. These grafts have been shown to resist resorption better than endochondral bone.\textsuperscript{8} Rigid fixation of these grafts has been shown to decrease resorption (Figure 28-11).\textsuperscript{8}

Soft tissue resuspension after surgical access to facial fractures is important for long-term facial esthetics.\textsuperscript{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.\textsuperscript{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 stated that there are two steps to placing the soft tissue back into proper position after exposure of the facial skeleton: refixa-
tion of the periosteum or fascia to the skeleton, and closure of the periosteum, muscle, fascia, and skin where incisions have been made.\textsuperscript{42} 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.\textsuperscript{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.\textsuperscript{42}

**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.\textsuperscript{55–57} Likewise, tracheostomy is an appropriate option to facilitate the management of multiple facial fractures.\textsuperscript{10,56,57} In many cases there are extensive injuries to the naso-orbitoethmoid region, making nasal intubation difficult and hazardous.\textsuperscript{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\textsuperscript{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.\textsuperscript{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 my 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 it 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; howev-
er, fixation at this point may lead to inaccur-
cacies in upper midface position. Instead, a break in the sequence is usually preferred here. The zygomaticomaxillary complex is reduced and fixedated 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-orbitoethmoidal fracture is reduced and stabilized (Figure 28-12).\textsuperscript{62}

The opposite approach, top down and outside in, starts at the zygomatic region. The sphenozygomatic suture is reduced...
FIGURE 28-12  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. 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.
FIGURE 28-13 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. 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.
and fixated inside the orbit. 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 zygomatico-maxillary buttress and piriform rim as a guide. Maxillomandibular fixation can then be established (Figure 28-13).\(^{32}\) 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 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. On the other hand, 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 28-1 and 28-2, two common sequences of management of facial fractures are illustrated. Other sequences exist, but they are variations of these two major approaches.

### Complications

There are many complications that 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 that I will discuss here is widening of the facial complex. This occurs when the 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 28-14). In severe cases three-dimensional computed tomographic reconstruction of the entire facial skeleton can be obtained and, if indicated, a three-dimensional stereolithicographic model can be made.\(^{64,65}\) The model allows the surgeon to identify and recreate 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.

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**Table 28-1** 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 28-12.

**Table 28-2** 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 28-13.
A and B, Twenty-one-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 ON NEXT PAGE)
pattern. Once the proper diagnosis is established, the surgeon should be able to institute an appropriate sequence of treatment.

References
Part 4: Maxillofacial Trauma


Part 5

Maxillofacial Pathology
One of the major roles of the oral and maxillofacial surgeon is that of diagnostician. From private practices in small communities to large tertiary care medical centers, these specialists are 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. Indeed, although the ability to correctly diagnose is important to virtually all professions, 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 obviously important and occasionally critical to the patient so that the most appropriate treatment can be initiated as soon as possible. Early determination of the true diagnosis can further benefit the patient by avoiding the need for expensive unnecessary laboratory studies, the use of ineffective or improper medications, and the inconvenience of additional costly consultation(s).

A variety of terms related to the diagnostic process may be used during the evaluation of the patient. Occasionally the diagnosis is relatively straightforward. Usually, however, a variety of conditions with similar clinical features need to be considered, and a differential diagnosis is prepared. The differential diagnosis represents a listing of the more likely diagnostic considerations for a particular pathologic finding or condition, ranked in descending order of probability. Therefore, the number one consideration from the initial differential diagnosis should represent the culmination of the clinician’s evaluation and is termed the clinical diagnosis (ie, working or tentative diagnosis, clinical impression). Although construction of the differential is 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 29-1). As discussed below, the differential listing may vary widely depending upon the experience and knowledge base of the treating clinician. The designation of final diagnosis is used when the clinician believes that the nature of the disease has been identified to a reasonable degree of certainty. This progression from information to possible diagnoses to final diagnosis is known as the diagnostic process or method. A case example is provided below. Although the determination of a final diagnosis often represents the end of the diagnostic phase of patient care, it is worth remembering that the “final” diagnosis is not always correct. As is stressed below, observation of the patient’s response to therapy and careful monitoring of the subsequent disease course are essential aspects of comprehensive patient management. Should a lesion or condition not behave in the expected manner, reevaluation and revision of the final diagnosis may ultimately be required.

### The Diagnostic Process

The clinician begins the diagnostic process by gathering or accumulating information. In some instances this information includes a significant historic component, whereas in other cases (eg, asymptomatic lesions discovered upon routine examination) the data may be limited strictly to the findings of the physical examination, 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 diagnosticians requires additional information from appropriate imaging or laboratory studies.

**Case Study: From Differential Diagnosis to Final Diagnosis**

A 25-year-old male presents with a 3-month history of gradual painless enlargement of the right anterior lower jaw. His medical history is unremarkable, and he denies recent trauma to the area. Clinical examination reveals a 1.5 cm bony firm swelling of the right mandibular alveolus in the area of teeth no. 26 and 27 causing primarily buccal expansion with an unremarkable overlying mucosa. The area is nontender to palpation, and the adjacent teeth are vital.

Initially, the clinical differential diagnosis focuses on the most common conditions that could present in a fashion similar to the lesion in this patient. Likely considerations include central giant cell granuloma, central ossifying fibroma, ameloblastoma, and odontogenic keratocyst.

Important additional information is easily obtained in this case with routine dental radiography. A panoramic film reveals a 2 cm unilocular radiolucent lesion of the right mandible with a well-defined sclerotic border that contains scattered small particles of radiodense material. With this additional information, the differential diagnosis is revised to exclude conditions not usually associated with calcification and possibly to include other less common conditions that have a radiopaque component: central ossifying fibroma, desmoplastic ameloblastoma, and calcifying epithelial odontogenic tumor.

A biopsy of the lesional tissue reveals a well-circumscribed cellular proliferation of benign spindle cells containing scattered trabeculae of osteoid and bone. The final diagnosis is central ossifying fibroma.

**History**

Most attempts at formulating a differential diagnosis begin with data gathering that includes the history of the specific problem being investigated as well as the patient's medical and social history. The patient's perception of the duration of the lesion can be important as long-standing lesions may suggest a developmental or benign process, whereas rapidly evolving problems often represent reactive, infectious, or malignant disease. Exceptions to these generalizations are numerous, however, since mycobacterial infections may develop slowly, as do some neoplasms that are considered malignant (eg, 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 developing a differential diagnosis. Pain and tenderness (pain on palpation) are often signs of an inflammatory or infectious process, although malignancies can also produce such symptoms, particularly late in their course. A notable exception to this is adenoid cystic carcinoma, which is infamous for the early onset of low-grade intractable pain. Other symptoms such as paresthesia or numbness can also be significant and may be related to pressure on nerves caused by a cystic lesion or tumor mass.

Reported changes in the lesion may also provide important insights. If a mass gradually enlarges, the possibility of neoplasia has to be entertained, whereas a mass that fluctuates in size is more suggestive of a reactive process. In addition, changes in symptoms may be significant. Decreasing pain or ten- derness likely represents a resolving inflammatory or infectious process, whereas pain that develops in a long-standing previously asymptomatic mass may be an indication of malignant transformation (eg, carcinoma arising in pleomorphic adenoma).

**Clinical Examination**

Following a review of the patient’s medical history and history of the present lesion or condition, the clinician typically proceeds with gathering objective data through careful clinical examination. A variety of lesional parameters should be evaluated and recorded, including (1) site, (2) size, (3) character (eg, macule, ulcer, mass), (4) color, including an assessment of its homogeneity, (5) surface morphology (eg, smooth, pebbly, granular, verrucous), (6) the border (eg, smooth, irregular, indistinct, sharply defined), (7) consistency on palpation, (8) local symptoms, and (9) the distribution if multiple or confluent lesions are observed.

The precise anatomic site or location of a lesion can provide essential diagnostic information and is discussed later in greater detail.

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 29-2). As mentioned above, relying on the accuracy (or veracity) of the patient history can be problematic and should be weighted accordingly in the differential diagnosis. Confirmation of the clinical history through other health care practitioners can be helpful in this regard.

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 melanin pigment; a red or purple macule usually represents hemoglobin in either its oxygenated or 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 level of the epithelium. Although additional information regarding the margin or border of a lesion is provided below, it should be mentioned that most pigmented lesions in the oral cavity are relatively homogeneous in color and have a smooth well-defined margin. By contrast, a pigmented lesion that exhibits significant border irregularity and color variegation should be considered as suspicious for melanoma (Figure 29-3).

The surface morphology of a lesion can be virtually diagnostic for certain conditions. Examples include the “tapioca pudding” appearance of the surface of a lymphangioma or the papillary epithelial fronds of squamous papilloma. Similarly,
an irregularly papular or granular surface architecture can be seen with malignant tumors as well as granulomatous processes that can range from deep fungal or mycobacterial infections to foreign-body reactions to immune-mediated conditions such as Crohn’s disease or sarcoidosis.

Palpation of the lesion is necessary to assess its consistency, the 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, whereas an even firmer consistency can reflect metastatic disease within a lymph node. A hard or bony consistency naturally indicates a mineralized or calcified component to the lesion.

The border or margin of a primarily submucosal or subcutaneous lesion is usually described as encapsulated, well-demarcated, or 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 (eg, neurofibroma) and some low-grade malignancies (eg, acinic cell carcinoma) may be well-demarcated, but they are generally less mobile compared with encapsulated lesions. The margins of many malignancies are indistinct, as the tumor invades and blends with the surrounding host tissues.

As noted earlier, the finding of a local symptom such as tenderness is usually associated with an inflammatory process, especially acute inflammation. Although malignant neoplasms may also present with tenderness or dyesthesia (eg, adenoid cystic carcinoma), this feature is usually a later-stage development secondary to tumor invasion of local nerves or surface ulceration. Tenderness may also be a prominent clinical feature of certain benign tumors such as traumatic neuroma.

Finally, the presence of multiple identical or similar lesions can suggest a number of conditions, depending upon their particular character (eg, 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. On the other hand, the finding of a focus of several small relatively painless ulcerations in a unilateral distribution on the left hard palate would be more consistent with a recurrent intraoral herpes infection. Similarly, multiple purplish plaques involving the oral mucosa and skin of a 35-year-old male who is positive for the human immunodeficiency virus would be strongly suggestive of Kaposi’s sarcoma.

Developing the Differential Diagnosis

After collecting the historic and clinical information, the final diagnosis may be obvious; however, in many instances the diagnosis is not readily apparent and the formulation of a differential diagnosis is appropriate. Several approaches have evolved over the centuries of medical practice to assist in the categorization or grouping of diseases. These grouping techniques permit the large number of possible diagnostic considerations for a given lesion to be reduced to the more probable conditions. The resultant narrowing of the differential diagnosis, in turn, aids in the selection of additional diagnostic tests that are most useful in securing a final diagnosis. The major diagnostic strategies or approaches that have been used to group or organize the differential are based on (1) the history and clinical presentation, (2) the potential disease histogenesis, and (3) the disease location (more specifically, the frequency of a given condition in a particular location). In actual practice, more experienced clinicians typically employ all of the categories simultaneously. As a consequence, the specialist is able to rapidly produce a much narrower and usually more precise list of initial diagnostic considerations (see the case study below).

Evaluation of the physical characteristics of a given lesion in the context of the history and clinical setting often permits the clinician to arrive at a reasonable list of diagnostic possibilities. For example, a firm fixed painless 2 cm nodule of uncertain duration in the anterior cervical area of the neck is suspicious for possible metastatic disease or lymphoma. By contrast, if the nodule were soft, mobile, and
tender to palpation, an inflammatory process would be more likely.

Another useful approach to developing a differential diagnosis 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 histogenesis. 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 differential. Similarly, if the lesion presented as a chronic ulceration of the lateral tongue in an adult patient, disorders from the neoplastic (especially malignancies), infectious (eg, mycobacterial or deep fungal infections), and immune-mediated (eg, Wegener’s granulomatosis or regional enteritis) categories would have to be considered.

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 very likely represents a mucocele. By contrast, mucocele would not be included in the differential diagnosis of a painless persistent bluish mass of the attached gingiva as salivary gland tissue is not normally present at that site. This latter clinical finding 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 that has no identifiable source of irritation or trauma would be highly suspicious for squamous cell carcinoma. Salivary gland neoplasia would be a strong consideration for a rubbery firm mass of the posterior 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 differential diagnosis 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. Following 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 differential 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 differential diagnosis 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: Additional Diagnostic Methods

If the final diagnosis cannot be determined based on historic findings and physical examination alone, a variety of procedures and tests can be used to assist in the diagnostic process. Generally, diagnostic tests should be ordered so that the most likely diagnosis can be either confirmed or eliminated. The methodic application of this process together with a proper rationale for selecting each test typically leads to the correct diagnosis in the most rapid cost-effective manner. Tests that do not address the most likely diagnostic possibilities should be delayed as 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 is performed to rule out a rare or unusual condition of serious clinical significance. Finally, diagnostic tests should be interpreted by individuals with specialty training in that area whenever possible to ensure the most timely and accurate result or final diagnosis.

Diagnostic studies are not necessarily complex or expensive. For example, a putative vascular lesion can be evaluated easily 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 bone cavity seen with traumatic bone cyst. Finally, follow-up evaluation of a lesion is a straightforward procedure that can provide important diagnostic insight with respect to biologic behavior. Those conditions that persist or progress 2 weeks after initial inspection often require additional tests to establish the diagnosis.

Diagnostic Imaging

Depending on the clinical setting, imaging studies may be both appropriate and necessary to the work-up of an oral lesion. Additional information on this topic is available in an excellent radiology text edited by White and Pharoah.3 Briefly, imaging studies can include plain radiographic films, sialography, ultrasonography, computed tomography (CT), 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, together with CT, are often the most useful. With the increased use of panoramic radiographs as a screening study in many current dental practices, 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 single radiolucent lesion at the apex of a nonvital tooth most likely represents a periapical cyst or granuloma. A similar-appearing radiolucency below the level of the inferior alveolar canal in the posterior mandible more likely represents a Stafne defect. Sharply defined margins indicate a benign process in most instances, whereas poorly defined margins can sometimes signify malignancy. Notable exceptions to this rule include osteomyelitis and fibrous dysplasia, both of which typically have borders that blend with the surrounding bone. Radiolucent lesions are produced by 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 since 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 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 most useful in the evaluation of deeply seated masses and is often helpful in distinguishing a solid mass from one that is cystic. This technique relies on the fact that different tissue densities result in different degrees of reflection or echo production of a beam of high-frequency sound waves. Although ultrasonography does not expose the patient to ionizing radiation, the tissue resolution is typically less than that achieved with either CT or MRI technology.

CT  CT is a cross-sectional radiologic imaging technique that is particularly useful in the evaluation of bone lesions. Not only can the density and margins of the lesion in question be evaluated with this technique but cortical expansion and fine internal details can often be more readily appreciated compared with plain film images. Use of contrast media has extended the utility of this technique in areas of soft tissue pathology. Furthermore, more recent designs such as spiral CT scanners have made data acquisition much more rapid and have reduced radiation dose to the patient while maintaining or improving resolution.

MRI  MRI is a newer form of cross-sectional imaging that does not expose patients to ionizing radiation. Although primarily used in the evaluation of soft tissue lesions, it is also capable of providing diagnostic information regarding bony lesions. Two distinct views are typically generated: T1 and T2. Adipose tissue has the highest signal in the T1-weighted image, and this view is often used for identifying anatomic structures. By comparison, the T2 image highlights tissues with high water content and is especially useful in depicting inflammatory processes and neoplasms.

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 inflammatory conditions such as osteomyelitis, areas of active skeletal lesions of fibrous dysplasia or osteitis deformans, and metastatic disease.

PET Scan  PET scan is the most recently developed cross-sectional imaging technology. This technique relies on the identification of metabolically active cells, such as metastatic deposits of squamous cell carcinoma, that exhibit preferential uptake of radionuclide-labeled glucose. In conjunction with CT/MRI, preoperative PET imaging of patients with head and neck cancer has lead to increased sensitivity and specificity for detection of oral cavity carcinoma, esophageal carcinoma, and clinically occult metastatic disease in the neck.4–8 PET scans have proved particularly useful in the post-treatment follow-up by helping to distinguish altered anatomic landmarks or areas of fibrosis from recurrent tumor as well as the detection of distant metastases from head and neck primaries.6–8

Exfoliative Cytology  Exfoliative cytology is a relatively inexpensive noninvasive technique that may be used to provide additional information related to lesions of surface origin. The utility of this technique in the diagnosis of conditions such as candidiasis, herpesvirus (herpes simplex virus, human herpesviruses 1 and 2) infections, and pemphigus vulgaris is well documented.

More recently a modified form of cytologic sampling that employs an oral brush instrument to collect epithelial cells followed by automated histopathologic evaluation has been introduced to dentistry. Suggested advantages 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. This new technique does not provide a definitive diagnosis, however, and cannot be used as a substitute for scalpel biopsy and routine histopathologic examination (see below). Therefore, in a clinical setting where the index of suspicion for possible precancerous or cancerous change is high, such as the high-risk areas for oral cancer (ie, ventrolateral tongue, floor of mouth, tonsillar pillars, soft palate), or in a patient with significant risk factors (ie, heavy smoking, heavy alcohol use, or both), use of brush cytology would not be recommended due to the inherent delay in definitive diagnosis of the lesional tissue and any subsequent treatment. In cases in which a persistent mucosal lesion is identified but the index of suspicion is low, the brush cytology technique may be useful in excluding the presence of precancerous or malignant
epithelial changes. For such innocuous lesions, a finding of abnormal cells could trigger scalpel biopsy (and definitive diagnosis) before the surgical procedure might otherwise have been deemed necessary.

**Fine-Needle Aspiration** 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 requires 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, usually in conjunction with tertiary care 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 below).

**Excisional Biopsy** Excisional biopsy is typically used to manage clinically benign lesions that are < 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. Excision of a small but potentially malignant lesion (e.g., squamous cell carcinoma with a primary tumor [T], regional nodes [N], and metastasis [M] staging of T1N0M0) may be appropriate in settings in which the surgeon performing the biopsy is also responsible for final treatment. With rare exceptions, an excisional biopsy should not be performed on a suspected malignant lesion unless the performing clinician is 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 is recommended whenever a clinician suspects that a neoplastic process may have recurrent or malignant potential, including conditions such as epithelial dysplasia or pleomorphic adenoma. This can be accomplished by careful identification of the anatomic margins of the biopsy specimen with suture(s), an accompanying sketch of the specimen, and its 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 neoplastic 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 the exploration of a radiolucent lesion of bone. 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 including digital images can be helpful, particularly for specialists who have dental training such as oral and maxillofacial pathologists. Biopsies of bony pathology should be accompanied by radiographs (originals or copies), whenever possible, as correlation may be needed to help distinguish conditions such as fibrous dysplasia, ossifying fibroma, and focal cemento-osseous dysplasia.

A final piece of information that should always be submitted together with the biopsy specimen is the clinical diagnosis. The clinical diagnosis is important at two levels. First, it helps the pathologist by providing an educated “best guess” as to what the lesional tissue was thought to most likely represent by the clinician. Should the initial histopathology of the submitted specimen appear substantially different 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 working diagnosis, it is the clinician who should proceed cautiously. After discussing the case directly with the sign-out pathologist, 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 surgeon, an important function that ultimately benefits the patient.

For the oral and maxillofacial surgeon, this type of discordance may be minimized if the tissue specimen is initially reviewed by an oral and maxillofacial pathologist. The oral and maxillofacial pathologist receives highly specialized training in the 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 give some perspective, individuals trained in oral and maxillofacial pathology programs review tens of thousands of oral biopsy specimens prior to graduation. By contrast, it is unusual for general surgical (anatomic) pathology residents to examine more than a few hundred specimens from the orofacial region during their training. Furthermore, the oral and maxillofacial pathologist has a command of the terminology used by the dental profession to describe oral disease and can more readily correlate the clinical and radiographic features with the microscopic findings. 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, the professionals who are trained specifically to manage problems related to the oral and maxillofacial region are able to accomplish their respective tasks more efficiently and accurately.

The Microscopic Differential Diagnosis
On occasion a final diagnosis cannot be made after examining routine hematoxylin and eosin-stained sections of a lesion. In such a situation, the pathologist is faced with a microscopic or histopathologic differential diagnosis. 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 appear undifferentiated 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 in the lesional cells 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 (eg, 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 a number of 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 “probes” to be used to characterize the neoplastic process and permit a therapy that is optimized for that particular tumor. Although routine formalin-fixed paraffin-embedded tissue sections can generally be used to perform most IHC studies, an important exception involves tumors that require analysis by flow cytometry. Typically used to permit rapid and highly specific subclassification of lymphomas and leukemias, flow cytometry employs IHC probes, but the tissue samples must not be fixed and should be analyzed immediately following collection. Another exception to this rule concerns the definitive diagnosis of immunobulbous disorders such as cicatricial pemphigoid. When such conditions are considered within the differential, perilesional tissue should be obtained and submitted in a special holding medium known as Michel’s solution (Michel’s Media). A holding medium is necessary because the molecular structure of the diagnostic antigens in these conditions (eg, immunoglobulins, complement, and fibrinogen) is usually destroyed by formalin fixation. These specimens are processed as frozen sections and are evaluated by 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 indirect immunofluorescent studies, patient serum is 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 serum-derived antibody binding to epithelial or basement membrane components.

In a few instances even the more sophisticated immunohistochemical techniques cannot provide a definitive diagnosis. In those situations newly developed molecular techniques are being used with
greater frequency. These techniques include sophisticated cyogenetic studies such as fluorescence in situ hybridization (FISH) as well as molecular probes that use complementary deoxyribonucleic acid (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 work-up may be required (see Figure 29-1). Alternatively, the follow-up findings may indicate that further investigation of the differential considerations is warranted such as biopsy and histopathologic review. Finally, careful follow-up should be considered mandatory for patients who have been previously diagnosed with or treated for oral dysplasia or carcinoma.

Although an important part of the practice of dentistry and medicine, formal guidelines for the management of oral lesions that are not clearly premalignant or cancerous have only recently been suggested.9 Such guidelines are helpful to clinicians as they provide systematic protocols for the management of oral pathologic conditions and serve to reduce the medicolegal risk associated with this important aspect of patient care (Table 29-1).

After the initial evaluation and careful documentation of an oral lesion, a follow-up examination should be scheduled for 7 to 14 days later, with or without any treatment. If there is evidence of lesion enlargement or other physical or symptomatic changes that do not suggest normal healing or resolution, then biopsy is indicated. If the lesion remains relatively unchanged and the index of suspicion for malignancy is low, the clinician should help the patient decide the next course of action based upon experience, advanced training, or both. Whenever available, referral to an oral and maxillofacial pathologist may be helpful in this regard. If the patient and clinician decide to defer biopsy, this decision should be documented and re-evaluation of the area should be scheduled at 1, 3, 6, and 12 months following 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 incisional biopsy (to establish a firm diagnosis). The need for these options varies depending on the concerns of the patient or the experience and expertise of the clinician. At any time point, however, evidence of significant lesional change should immediately trigger a recommendation of 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 above, based upon training, experience, and the clinical judgement of the practitioner.

### Table 29-1 Follow-Up Protocol for Oral Pathology

<table>
<thead>
<tr>
<th>Step</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Initial re-evaluation: 7–14 d following lesion detection/examination</td>
</tr>
<tr>
<td>2.</td>
<td>If no evidence of lesional progression or suspicious clinical alterations, reevaluate at 1, 3, 6, and 12 mo intervals; thereafter, re-examine in conjunction with normal recall visits (every 6–12 mo)</td>
</tr>
<tr>
<td>3.</td>
<td>If lesion progression or suspicious clinical changes noted, incisional or excisional biopsy should be performed as soon as possible, and specimen should be reviewed by oral and maxillofacial pathologist</td>
</tr>
<tr>
<td>4.</td>
<td>If no evidence of preneoplastic change (dysplasia) or malignancy (carcinoma or sarcoma) reported, schedule follow-up as in step 2 and document subsequent findings in patient record</td>
</tr>
<tr>
<td>5.</td>
<td>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</td>
</tr>
</tbody>
</table>

Adapted from Alexander RE et al.9

### References

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 30-1). 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 that form dentin. As the odontoblasts deposit dentin, they induce the ameloblasts to begin forming enamel.

Following 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, following completion of enamel formation, the
enamel organ epithelium atrophies to form a 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 30-2). Normally cell division is divided into four phases: G1 (gap 1), S (deoxyribonucleic acid synthesis), G2 (gap 2), and M (mitosis). A key event is the progression from G1 to the S phase. Genetic alterations, if unrepaired in the G1 phase, may be carried into the S phase and perpetuated in subsequent cell divisions. The G1-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 30-2, 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.

It is the purpose of this chapter 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, on the other hand, are the result of inflammation (Table 30-1).

**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 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 30-3). Since 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 trabeculae within the radiolucency. However, dentigerous cysts are grossly and histopathologically unilocular processes.

![Figure 30-2](image_url) *The cell cycle—a concept of proliferation versus apoptosis. PCNA = proliferating cell nuclear antigen.*
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 > 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 30-4). 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 30-5). 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 is diagnosed histopathologically following removal in an office setting. Such diagnoses would include odontogenic keratocyst and unicystic ameloblastoma.

Large dentigerous cysts may be treated with marsupialization (Figure 30-6) 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.

I emphasize 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

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**Table 30-1 Classification of Odontogenic Cysts**

<table>
<thead>
<tr>
<th>Category</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Developmental</td>
<td>Dentigerous cyst, Eruption cyst, Odontogenic keratocyst, Orthokeratinized odontogenic cyst, Gingival (alveolar cyst of the newborn)</td>
</tr>
<tr>
<td>Inflammatory</td>
<td>Periapical (radicular cyst), Residual periapical (radicular cyst), Buccal bifurcation cyst</td>
</tr>
</tbody>
</table>

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.

**Figure 30-3** This unilocular radiolucency of the left mandibular ramus associated with impacted tooth no. 17 was discovered serendipitously when the patient was evaluated for routine dental work.

**Figure 30-4** The biopsy of the radiolucency in Figure 30-3 shows an atrophic stratified squamous epithelium without significant associated inflammation. The diagnosis is dentigerous cyst (hematoxylin and eosin; original magnification ×40).
representative piece 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. 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. Two variants of this cyst are well known; the sporadic cyst and the 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 of enzymatic activity in the fibrous wall.

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; (2) the odontogenic keratocyst has a higher rate of recurrence than other odontogenic cysts, 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.

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. In his series of 312 cases, Brannon 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 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.

Although it is rare for a dentigerous cyst to appear multilocular on radiographs, it is most common for odontogenic keratocysts to appear multilocular (Figure 30-7). Many

![Figure 30-5 A](image1) A, The dentigerous cyst in Figure 30-3 is treated with enucleation and curettage of the cyst and removal of the etiologic tooth. B, The 5-year postoperative radiograph shows an acceptable bony fill.

![Figure 30-6 A](image2) 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. C, The 5-year postmarsupialization radiograph shows an excellent fill of bone.
Odontogenic Cysts and Tumors

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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 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 30-2). Histologically, the odontogenic keratocyst is readily recognized. A uniform layer of stratified squamous epithelium, usually six to eight cells in thickness, is present (Figure 30-8). 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 as they generally impart a more aggressive character to the cyst.

Treatment and Prognosis Like the treatment of most odontogenic cysts, the odontogenic keratocyst may be treated with enucleation and curettage and must be removed in one piece, which requires acceptable access and lighting (Figure 30-9). 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 my experience and that of others that a large majority of sporadic odontogenic keratocysts may be effectively managed with a thorough enucleation and curettage surgery.18 MacIntosh has advocated the resection of odontogenic keratocysts with 5 mm linear margins as the preferred primary method of treatment, and has reported on 37 patients with 43 lesions emphasizing the efficacy and superior results of resection over all other therapeutic undertakings.20 The reported frequency of recurrence of the odontogenic keratocyst ranges from 2.5% to 62.5% in various studies.11 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 colleagues point out that the recurrence rate for solitary odontogenic keratocysts is 10 to 30%.21 They indicate 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.21 Brannon 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 following enucleation.22 Vedtofte and Praetorius reviewed 72 patients with 75 odontogenic keratocysts and observed remnants of dental lamina between the cyst membrane

Table 30-2 Clinical Features of the Basal Cell Nevus Syndrome

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Clinical Feature</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥50% frequency</td>
<td>Multiple basal cell carcinomas</td>
</tr>
<tr>
<td></td>
<td>Odontogenic keratocysts</td>
</tr>
<tr>
<td></td>
<td>Epidermal cysts of the skin</td>
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<tr>
<td></td>
<td>Palmar/plantar pits</td>
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<tr>
<td></td>
<td>Calcified falx cerebri</td>
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<tr>
<td></td>
<td>Enlarged head circumference</td>
</tr>
<tr>
<td></td>
<td>Rib anomalies (splayed, fused, partially missing, bifid)</td>
</tr>
<tr>
<td></td>
<td>Mild ocular hypertelorism</td>
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<tr>
<td></td>
<td>Spina bifida occulta of cervical or thoracic vertebrae</td>
</tr>
<tr>
<td>15–49% frequency</td>
<td>Calcified ovarian fibromas</td>
</tr>
<tr>
<td></td>
<td>Short fourth metacarpals</td>
</tr>
<tr>
<td></td>
<td>Kyphoscoliosis or other vertebral anomalies</td>
</tr>
<tr>
<td></td>
<td>Pectus excavatum or carinatum</td>
</tr>
<tr>
<td></td>
<td>Strabismus (exotropia)</td>
</tr>
<tr>
<td>&lt;15% frequency (but not random)</td>
<td>Medulloblastoma</td>
</tr>
<tr>
<td></td>
<td>Meningioma</td>
</tr>
<tr>
<td></td>
<td>Lymphomesenteric cysts</td>
</tr>
<tr>
<td></td>
<td>Cardiac fibroma</td>
</tr>
<tr>
<td></td>
<td>Fetal rhabdomyoma</td>
</tr>
<tr>
<td></td>
<td>Marfanoid build</td>
</tr>
<tr>
<td></td>
<td>Cleft lip and/or palate</td>
</tr>
<tr>
<td></td>
<td>Hypogonadism in males</td>
</tr>
<tr>
<td></td>
<td>Mental retardation</td>
</tr>
</tbody>
</table>

Adapted from Gorlin FJ.14
Part 5: Maxillofacial Pathology

Williams and Connor recom-mended a primary enucleation and curet-tage 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 consid-ered 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. 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. 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. 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.

The orthokeratinized odontogenic cyst, once thought to be a variant of the odontogenic keratocyst, is now generally well accepted as being a different clinico-pathologic entity from the more common parakeratinized odontogenic keratocyst; it should therefore be placed in a different category. These cysts usually appear as unilocular radiolucencies, but occasional examples have been multilocular. A major-ity 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 orthoker-atinized, 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.

Nevoid Basal Cell Carcinoma Syndrome
The nevoid basal cell carcinoma syndrome (basal cell nevus syndrome, Gorlin’s syn-drome) is an autosomal-dominant inherited condition that exhibits high pene-trance and variable expressivity. It is caused by mutations in the \( PTCH \) tumor suppressor gene, mapped to chromosome 9q22.3-q31. Affected patients (Figure 30-10A) may demonstrate frontal and temporoparietal bossing, hypertelorism, and mandibular prognathism (see Table 30-2). Other frequent skeletal anomalies include bifid ribs and lamellar calcification of the falx cerebri (Figure 30-10B). The

![Figure 30-9](image1.png)

**Figure 30-9** A, A very thin cyst lining was encountered when performing the enucleation and curettage of the odontogenic keratocyst in Figure 30-7. 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.

![Figure 30-10](image2.png)

**Figure 30-10** A, This 18-year-old shows some of the clinical features of the nevoid basal cell carcinoma syndrome including frontal bossing and mandibular prognathism. B, The radiograph from another patient shows a calcified falx cerebri.
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. Pitting defects on the palms and soles can be found in nearly two-thirds of affected patients (Figure 30-11). 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 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. As a matter of point, I choose to refer to these 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. Marsupialization is a more desirable procedure (Figure 30-12) 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. Although all of the eight cases in the series by Pogrel and Jordan 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.

**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 as 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. Eighty percent of cases occur in the mandible, and a strong predilection for the anterior region of the jaws has been reported, with many mandibular lesions crossing the midline (Figure 30-13). 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 30-14). Waldron and Koh 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.28

Treatment and Prognosis Most glandular odontogenic cysts are treated with enucleation and curettage (Figure 30-15). Some authors, however, point to a recurrence rate of approximately 30% and therefore recommend resection.29

**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.30,31 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. The commentary on the second edition by Kramer, Pindborg, and Shear points out that some COCs appear to be non-neoplastic, but others show an infiltrative pattern of growth.32 They further indicate that more experience with the COC may provide reliable criteria for their reclassification. The review by Hong and colleagues designated 79 of 92 cases of COC as cysts with the remaining 13 cases being neoplastic in nature.30

The COC is predominantly an intraosseous lesion, although 13 to 30% of reported cases occur as peripheral lesions.2 Both the peripheral and 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 about 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. 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 30-16). 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 the same as for the associated tumor. Although only a few cases have been reported,31 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.
Odontogenic Tumors

Odontogenic tumors comprise 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 (Table 30-3). 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 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 30-4). Studies from North America seem to indicate that odontogenic tumors represent approximately 1% of all admissions in oral pathology laboratories, whereas African studies have a much higher incidence of odontogenic tumors. Moreover, the ameloblastoma is more commonly encountered in African and other underdeveloped countries than in North America.

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. 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, 3,677 cases of ameloblastoma were reviewed, of which 92% were solid or multicystic, 6% were unicystic, and 2% were peripheral.

Solid or Multicystic Ameloblastoma

This variant of the ameloblastoma is

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**Table 30-3 Classification of Odontogenic Tumors**

<table>
<thead>
<tr>
<th>Category</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tumors of odontogenic epithelium</td>
<td>Ameloblastoma, Malignant ameloblastoma, Ameloblastic carcinoma</td>
</tr>
<tr>
<td></td>
<td>Calcifying epithelial odontogenic tumor</td>
</tr>
<tr>
<td></td>
<td>Squamous odontogenic tumor</td>
</tr>
<tr>
<td></td>
<td>Clear cell odontogenic carcinoma</td>
</tr>
<tr>
<td></td>
<td>Primary intraosseous carcinoma</td>
</tr>
<tr>
<td>Tumors of odontogenic epithelium with odontogenic ectomesenchyme ± dental hard tissue formation</td>
<td>Ameloblastic fibroma, Ameloblastic fibro-odontoma, Ameloblastic fibrosarcoma, Odontoameloblastoma</td>
</tr>
<tr>
<td></td>
<td>Odontoma</td>
</tr>
<tr>
<td></td>
<td>Compound composite, Complex composite, Adenomatoid odontogenic tumor</td>
</tr>
<tr>
<td>Tumors of odontogenic ectomesenchyme ± included odontogenic epithelium</td>
<td>Odontogenic fibroma, Granular cell odontogenic tumor, Odontogenic myxoma, Cementoblastoma</td>
</tr>
</tbody>
</table>
encountered in patients over a wide age range.\textsuperscript{43} It is rare in children in their first decade of life and relatively uncommon in the second decade.\textsuperscript{44} 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.\textsuperscript{45} About 15\% of multicystic ameloblastomas occur in the maxilla, usually in the posterior regions.\textsuperscript{46–49} A painless expansion of the jaws is the most common clinical presentation; neurosensory changes are uncommon, even with large tumors (Figure 30-17). Slow growth is the rule, with untreated tumors leading to tremendous facial disfigurement (Figure 30-18).\textsuperscript{50}

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 30-19); plexiform, in which the stellate reticulum is located outside 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.\textsuperscript{26} The nuclear PCNA positivity of the unicystic ameloblastoma was notably lower than the positivity of the solid multicystic ameloblastoma.\textsuperscript{26} 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.\textsuperscript{51}

**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.\textsuperscript{22} 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 my strong opinion that this neoplasm is both highly aggressive and curable. This notwithstanding, numerous methods of treatment have

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**Table 30-4 Incidence of Odontogenic Tumors**

<table>
<thead>
<tr>
<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>54,534</td>
<td>1,511</td>
<td>40,000</td>
</tr>
<tr>
<td>Total odontogenic tumors</td>
<td>706 (1.3%)\textsuperscript{*}</td>
<td>289 (19.1%)\textsuperscript{*}</td>
<td>445 (1.1%)\textsuperscript{*}</td>
</tr>
<tr>
<td>Ameloblastoma</td>
<td>78 (11.0%)\textsuperscript{†}</td>
<td>169 (58.5%)\textsuperscript{†}</td>
<td>79 (17.8%)\textsuperscript{†}</td>
</tr>
<tr>
<td>Adenomatoid odontogenic tumor</td>
<td>22 (3.1%)\textsuperscript{†}</td>
<td>18 (6.2%)\textsuperscript{†}</td>
<td>14 (3.1%)\textsuperscript{†}</td>
</tr>
<tr>
<td>Odontoma</td>
<td>473 (67.0%)\textsuperscript{†}</td>
<td>12 (4.2%)\textsuperscript{†}</td>
<td>204 (45.8%)\textsuperscript{†}</td>
</tr>
<tr>
<td>Myxoma</td>
<td>20 (2.8%)\textsuperscript{†}</td>
<td>34 (11.8%)\textsuperscript{†}</td>
<td>24 (5.4%)\textsuperscript{†}</td>
</tr>
</tbody>
</table>

*Percentage of total specimens in respective study.

†Percentage of total odontogenic tumors in respective study specimens.

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**Figure 30-17** 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 no. 18 and 19, indicative of the aggressive nature of this tumor. The incisional biopsy showed solid/multicystic ameloblastoma.
been recommended, ranging from simple enucleation and curettage to resection.53–59 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.60 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 as 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, I recommend resection of the tumor with 1.0 cm linear bony margins (Figure 30-20). This linear bony margin should be confirmed by intraoperative specimen radiographs. Soft tissue margins are best managed according to the anatom-ic barrier margin principles whereby one uninvolved surrounding anatomic barrier is sacrificed on the periphery of the specimen.61 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.62 Moreover, although the persistent and occasionally nonresectable ameloblastoma is radiosensitive, once this otherwise benign tumor defies curative surgical therapy, radiation is of questionable use in salvaging these patients.63,64

Unicystic Ameloblastoma In 1970 Vickers and Gorlin published their findings regarding the histologic alterations associated with neoplastic transformation of ameloblastomatous epithelium.65 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 that does not exhibit the histologic criteria for ameloblastoma published by Vickers and Gorlin.66–69 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.70

Unicystic ameloblastomas are most commonly seen in young patients, with about 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.42 More than 90% of these tumors are found in the

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**FIGURE 30-18** Twenty years of undisturbed growth of a solid/multicystic ameloblastoma led to significant facial disfigurement (A), with an impressive radiographic appearance (B). A segmental resection of the right mandible was performed (C).

**FIGURE 30-19** The incisional biopsy of the patient in Figure 30-17 shows follicular variant of the solid/multicystic ameloblastoma (hematoxylin and eosin; original magnification ×60).
mandible, usually in the molar/ramus region. A unilocular radiolucency, mimicking a dentigerous cyst, is the most common radiographic presentation for the unicystic ameloblastoma (Figure 30-21). Most, if not all, unicystic ameloblastomas are unilocular radiolucencies. 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 30-22). 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 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 the solid or multicystic ameloblastoma.

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 30-23). In a collective sense, the “recurrence” rate of all unicystic ameloblastomas
has been reported as 10 to 20% following enucleation and curettage.\textsuperscript{70} 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 following a well-performed enucleation and curettage surgery should probably be approached with the more aggressive resection. The 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 30-24). The final indication for resection of a unicystic ameloblastoma is in the management of very large tumors (see Figure 30-24) with significant expansion such that an enucleation and curettage surgery would effectively result in a resection of the involved jaw.

**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

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**FIGURE 30-23** A, The luminal unicystic ameloblastoma in Figure 30-21 is treated with an enucleation and curettage surgery. B, The 5-year postoperative radiograph shows an acceptable bony fill.

**FIGURE 30-24** This 18-year-old presented with significant right facial expansion (A) associated with the destructive radiolucency of the right mandible noted on the panoramic radiograph (B). The incisional biopsy documented the mural variant of unicystic ameloblastoma (hematoxylin and eosin; original magnification ×20) (C). A disarticulation resection was performed (D).
same features of the intraosseous form of the tumor. Clinically, these tumors present as nonulcerated sessile or pedunculated gingival lesions (Figure 30-25). Most examples are < 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 30-26).

*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.

**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 in the gingiva, and the linings of odontogenic cysts represent the precursor cells for malignant transformation. Odontogenic carcinomas are classified in Table 30-5. In general, all of these tumors exhibit typical microscopic features of malignancy, with the exception of the malignant (metastasizing) ameloblastoma and the clear cell odontogenic carcinoma. Behaviorally, all of these tumors have the potential for either regional nodal or distant metastases.

**Malignant (Metastasizing) Ameloblastoma** Malignant 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. The most common sites of metastatic disease are the lungs (Figure 30-27), followed by the cervical lymph nodes and visceral organs. Lung metastases have sometimes been regarded as aspiration phenomena, yet the peripheral location of many of these deposits supports hematogenous spread. Eversole 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 30-28), in a recurrence, or in any metastatic deposit. Although ameloblastic carcinomas have been reported to metastasize to the lungs and distant organs, many cases do not metastasize. In Corio and colleagues’ series of eight cases of ameloblastic carcinoma, rapid growth and pain were common symptoms. These symptoms are recognized as being uncommon in patients with benign ameloblastomas.

**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%. Squamous cell carcinomas may also arise from the linings of odontogenic cysts. Cystogenic carcinomas are seen in patients > 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. 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.

**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. 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 30-29). Radiographically, either a unilocular or multilocular lesion is observed.

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**FIGURE 30-28** 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 (hematoxylin and eosin; original magnification ×20 [D] and ×100 [E]).
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 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 asymmetric eruption of the dentition in children (Figure 30-31). 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 30-32). Recurrence after this approach is very rare. Malignant transformation of ameloblastic fibro-odontoma has been reported but is exceedingly rare.

Odontoma

Odontomas are the most frequently occurring odontogenic tumors, with prevalence exceeding that of all other odontogenic tumors combined. As stated

FIGURE 30-29 A, A destructive unilocular radiolucency is present in a 15-year-old boy. B, Incisional biopsy confirmed ameloblastic fibroma (hematoxylin and eosin; original magnification ×40).

FIGURE 30-30 An enucleation and curettage surgery is performed in the patient in Figure 30-29. The associated permanent teeth are removed with the tumor.

FIGURE 30-31 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.

FIGURE 30-32 A, Enucleation and curettage is performed of the lesion in Figure 30-31. The permanent tooth is removed with the lesion. B and C, The histopathology shows ameloblastic fibro-odontoma (hematoxylin and eosin; original magnification ×20).
previously, these lesions are generally well accepted as representing hamartomas. Odontomas present centrally within the jaws in one of two 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 30-33), whereas complex odontomas are typically seen in the posterior maxilla or mandible (Figure 30-34).

**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. 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 30-35). 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 30-36). Radiographically, the odontogenic myxoma appears as a unilocular or multilocular radiolucency that may displace or cause root resorption of teeth in the area 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 Figures 30-35B and 30-37). 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.

**Treatment and Prognosis** Odontogenic myxomas should be treated with resection with 1.0 cm bony linear margins as confirmed with a specimen radiograph (Figure 30-37A).

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**FIGURE 30-33** A, An expansile lesion of the right maxilla. B, Multiple small toothlike calcified structures are removed that represent compound odontoma.

**FIGURE 30-34** A complex odontoma of the left posterior mandible.

**FIGURE 30-35** A large soft tissue mass of the left posterior mandibular gingiva (A) associated with an underlying radiolucent lesion of the mandible (B).
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.

**Calcifying Epithelial Odontogenic Tumor**

The calcifying epithelial odontogenic tumor, also known as the Pindborg tumor, is an uncommon lesion that accounts for < 1% of all odontogenic tumors. It is particularly noteworthy that the three studies depicted in Table 30-4 reported only 15 cases of this odontogenic tumor among a collective series of 1,440 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. Approximately two-thirds of these neoplasms occur in the mandible. 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 30-38).

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 rings (Figure 30-39). 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.

**Treatment and Prognosis**

Although slow growing, the Pindborg tumor is highly infiltrative and destructive and is capable of aggressive behavior. 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 30-40). When this treatment was undertaken for Franklin and Pindborg’s series of tumors, 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. Its clinical features and biologic behavior permit distinction from the ameloblastoma (Figure 30-41). These lesions are limited to young patients, and two-thirds of all cases are diagnosed in the second decade. The tumor is extremely uncommon in patients > 30 years. It has a predilection for the anterior region of the jaws and is found twice as often in the maxilla than 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 30-42). 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 material that may stain for amyloid. Tubular or duct-like structures are characteristic for the adenomatoid odontogenic tumor (see Figure 30-42). 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 30-43). Of the 499 cases of adenomatoid odontogenic tumor reported in the literature, only 1 acceptable case of recurrence has been documented.
5 Part 5: Maxillofacial Pathology

A

594

B

FIGURE 30-43 A, An enucleation and curettage surgery is performed for the patient in Figure 30-41, 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.

References


Benign nonodontogenic lesions of the jaws represent a mixed group of tumors, which in many cases are difficult to classify. Additionally, there are some lesions within this group that actually only seem to occur in the jaws, and, therefore, although they do not contain any histologic or immunohistochemical evidence of odontogenic structures, the mere fact that they only occur in the jaws may mean that they are in fact 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. There is a general consensus 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. Somatic mutations in the GNAS α-gene have been proposed to cause monostotic and polyostotic conditions and Albright’s syndrome.

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. Albright’s syndrome in which multiple lesions are associated with hyperpigmentation and endocrine disturbances, predominantly precocious puberty and/or hyperthyroidism
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 31-1). Classically, the radiographic appearance shows a ground-glass opacity without clearly defined borders (Figure 31-2). 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. 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 in that the patient may perceive a throbbing or discomfort, the swelling increases, and the lesions appear hot on a bone scan (Figure 31-3) and can, in fact, mimic osteomyelitis. In a quiescent phase they may be totally asymptomatic. Teeth can be displaced by the lesion (Figure 31-4). Familial cases of fibrous dysplasia have been noted.

The lesions of fibrous dysplasia may be under hormonal control, particularly in Albright’s syndrome, and cases of increased activity and reactivation during pregnancy have been noted. Although not normally recognized as a premalignant lesion, sarcomatous change has been noted in fibrous dysplasia. Early cases appear to have been associated with the use of radiation therapy for treatment, but cases of spontaneous sarcomatous degeneration have been noted. Additionally, some cases have been difficult to diagnose and may have represented a low-grade osteosarcoma from the outset. Classically, fibrous dysplasia appears to be a lesion that “burns itself out” when the patient is in the late teens or early twenties, 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. 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 following 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.

Cemento-osseous Dysplasia

The cemento-osseous dysplasias represent a pathologic process of the tooth-bearing areas and probably represent the commonest manifestation of fibro-osseous disease; however, since 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, 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. There is a predominance of cases occurring in females and also in African Americans. 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.

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 31-5). Studies indicate that they may occur in around 6% of African American females.24

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 following dental extraction. Since 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.25

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 31-6). Lesions may be associated with superimposed infection and osteomyelitis, and have also been associated with idiopathic bone cysts.26 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 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.8,27,28 However, the role of bacteria in chronic diffuse sclerosing osteomyelitis has proven elusive, and, in general, even authorities who strongly support an infectious origin have had difficulty isolating organisms.29,30

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.31 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 31-7). 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.32,33 The ossifying fibroma is felt 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.34–36 There is, again, a female predominance but no racial predominance, and growth rates are variable. Since it is felt to be a neoplasm, the treatment is surgical; in fact, the lesions often shell out easily at surgery, although there is recurrence, the rate of which has variously been reported from 1 to 63%.37–39 For these reasons, some authorities recommend aggressive treatment for more aggressive lesions, including aggressive curettage,
localized surgical resection, and segmental mandibular resection.\textsuperscript{40,41} When present in the craniofacial complex, treatment may have to be more aggressive to protect the vital structures.\textsuperscript{42}

Juvenile Aggressive Ossifying Fibroma

Juvenile aggressive ossifying fibroma was first described in 1952 as a variant of ossifying fibroma.\textsuperscript{43} 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.”\textsuperscript{44} 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 spheric ossicles and a myxoid component with aneurysmal bone cyst–like areas.

Although felt to be more aggressive than the uncommon 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 commoner in younger patients.\textsuperscript{1}

Osteoblastoma and Osteoid Osteoma

Osteoblastoma and osteoid osteoma are generally felt 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 felt to represent a larger version of the osteoid osteoma. Both are benign processes and are felt to represent true neoplasms. The osteoblastoma occurs primarily in the vertebrae and long bones, but it has been described in the jaws.\textsuperscript{45–47} Clinically it often grows rapidly and the predominant clinical feature is pain, which is generally localized to the lesion itself. Although felt 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.\textsuperscript{48} 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 31-8). The designation osteoblastoma is normally reserved for lesions > 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 either with curettage or local excision. Recurrences are rare but have been reported and may necessitate more aggressive treatment such as en bloc resection.\textsuperscript{49} Rare examples of malignant transformation have been reported,\textsuperscript{50,51} but some of these may be related to an incorrect initial diagnosis.\textsuperscript{45}

The osteoid osteoma represents a smaller version of the osteoblastoma and is felt to be a true neoplasm. It is normally < 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; in fact, whether they ever occur in the jaws or whether they are usually described as chondromyxomas or chondromyxoid fibromas has been questioned. In many cases the true diagnosis in those reported cases is actually low-grade chondrosarcoma. Most reports concern the mandibular condyle, suggesting that these lesions may arise from cartilaginous remnants. The chondroma presents as a painless slowly progressive swelling, which may result in mucosal ulceration. The gender distribution is equal, and most tumors occur under the age of 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 kind of insurance policy.

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). 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. The specific gene associated with the condition has now been identified on the long arm of chromosome 5. 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 should be performed.

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 31-9). 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.

Treatment of osteomas is surgical excision (Figure 31-10). This is often necessary to establish the diagnosis. Asymptomatic cases may be followed up clinically and radiographically without treatment. Following 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 with symptoms consisting of pain and swelling but most often with deviation of the mandible toward the unaffected side (Figure 31-11). The etiology is unknown, but trauma has been suggested. When
these lesions become symptomatic, they should be removed via a standard preauricular approach. Since it is felt 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.\textsuperscript{72} Cases have been reported in which up to 200 of these bodies were present within the temporomandibular joint (Figure 31-12).\textsuperscript{12} Following removal, recurrence has not been reported.

The osteochondroma is felt 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 31-13). Cases have been reported in the mandibular condyle.\textsuperscript{73} 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 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 radiopaque than the surrounding mandible (Figure 31-14). Treatment is symptomatic; when symptoms occur, localized excision is recommended via the normal temporomandibular approach. Recurrence has been reported but is unusual.\textsuperscript{74–77}

**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 as desmoplastic fibromas, which is the hard tissue equivalent of fibromatosis in the soft tissues. Any bone can be affected including the jaws.

The etiology and pathogenesis are in doubt since their aggressive behavior suggests a neoplastic process, but genetic, endocrine, and traumatic factors have also been suggested. Most occur in persons under the age of 20 years, and there is no gender predilection. The mandible is affected more frequently than the maxilla.\textsuperscript{78} Radiographically a unilocular or multilocular radiolucency is noted with poorly defined margins, cortical perforation, and root resorption often being present (Figures 31-15 and 31-16). Histologically the lesion consists of interlacing bundles in a whirled 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 looks benign histologically, it often behaves aggressively,\textsuperscript{79} and the appropriate treatment is aggressive surgery, which often involves mandibular or maxillary resection (Figure 31-17). 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 following 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.\textsuperscript{80,81}
Lesions Containing Giant Cells

There are a number of lesions that 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.) Although not normally considered an odontogenic lesion, the fact that it only occurs 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, 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 showed in a study of a number of non-treated 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. 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.

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 31-18 and 31-19).

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, and the spindle cells are probably the cells of origin of this lesion.

Treatment is usually surgical and consists of local curettage, which is usually curative. 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, and 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.

Since 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.\textsuperscript{94}

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.\textsuperscript{95–98} 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 31-20).

Calcitonin given by subcutaneous injection has also been advocated and has met with some success (Figure 31-21).\textsuperscript{99–106} 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. Since some of the giant cells have been shown to have calcitonin receptors on them, this may explain calcitonin’s effectiveness.\textsuperscript{94}

\(\alpha\)-Interferon given by subcutaneous injection has also been advocated in the treatment of the central giant cell granuloma and has again met with some success.\textsuperscript{106,107} The rationale for this therapy is that the antiangiogenic action of the \(\alpha\)-interferon suppresses the angiogenic component of this lesion, causing healing to occur. In most cases surgery is still required after the \(\alpha\)-interferon treatment, but it may be less radical surgery and there may be a smaller chance of recurrence.

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.\textsuperscript{105}

**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 felt 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 some 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.\textsuperscript{45}

**Hyperparathyroidism**

In hyperparathyroidism (primary, secondary, or tertiary), calcium is mobilized from the bones into the blood stream 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.\textsuperscript{108} Clinically and histologically they are identical to the central giant cell granuloma and cannot be distinguished on either clinical or histologic grounds (Figure 31-22). 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 being required.

**Cherubism**

Cherubism is a familial genetically dominant condition first described by Jones in a family in 1933.\textsuperscript{109} Affected family members 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 31-23). The genetic defect in this condition has been identified on chromosome 4p16.3.\textsuperscript{110,111}

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 31-24 and 31-25).
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 success, suggesting that they are, in fact, different lesions. 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 cosmetic remodeling 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 31-26). 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 moderately aggressive curettage, although hemorrhage can be a problem. Recurrences are rare.

Vascular Malformations

Vascular malformations can occur anywhere in the body and are felt 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 endothelium and is normally present at birth, often enlarges, and then frequently involutes. The vascular malformation generally is not present at
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. 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 asymmetric 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 31-27). 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. Since 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 the central giant cell granuloma, the aneurysmal bone cyst, ameloblastoma, odontogenic keratocyst, and odontogenic myxoma. All of these lesions should undergo needle aspiration prior to biopsy or surgical treatment to rule out a high-flow vascular malformation. When a vascular malformation is suspected or diagnosed, selective angiography is normally performed via a femoral approach (Figure 31-28). 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 31-29). 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 small number of days of embolization. Definitive surgery normally takes the form of resection under
hypotensive anesthesia with adequate resuscitative measures available. Following 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 31-30). Other approaches such as injection of a variety of substances into the lesion including glue, fibrin gel, and platinum coils, for example, have been attempted; also, case reports exist of lesions being treated by means of local curettage following 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

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 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. 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. The Histiocyte Society has attempted to define all of the histiocytic diseases in a logical manner, and Letterer-Siwe disease is now felt 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 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 31-31), 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 31-32). 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 radiolu-
cency, 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. Intraleisional steroids have also been employed with some success, and cases of spontaneous regression have been reported.124,126

It is generally felt 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.121 I have seen the disease in a father and son. The father was diagnosed with the chronic disseminated form of the disease at age 53 years (see Figure 31-31), 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,” above.

Globulomaxillary Lesion
Globulomaxillary lesion was initially defined as a globulomaxillary cyst and was felt 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 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 31-33), it is not, in fact, a fissural cyst since the proposed embryonic derivation is now known to be flawed and the supposed fusion line does not exist. It is felt that most lesions previously diagnosed as globulomaxillary cysts can now be reclassified as odontogenic keratocysts, radicular cysts, periapical granulomas, lateral periodontal cysts, central giant cell granulomas, calcifying odontogenic cysts, and odontogenic myxomas.45 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.

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 felt 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 commonest in the fourth to sixth decades of life. Most cases are asymptomatic and are either 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 31-35). 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 > 7 mm in diameter, the presence of a cyst should be suspected.127

Median Mandibular Cyst
Median mandibular cyst is a rare cyst found in the midline of the mandible. It was originally felt to form at the line of fusion of each half of the mandibular arch. Again, the embryologic theory behind this lesion is no longer felt to be applicable, and it is believed that those lesions found in the anterior mandible represent some other type of odontogenic cyst or tumor.
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 following treatment.

**Traumatic Bone Cyst**

Traumatic bone cyst has been called a number of names, including idiopathic bone cyst, simple bone cyst, and latent bone cyst. 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 31-36). It is not quite as well defined as an odontogenic cyst, and the description made by Howe was that it appears as a “pencil sketch for a final pen and ink drawing.”

The etiology of this lesion is in doubt, and suggestions have included that it may result from intramedullary hemorrhage from trauma, which can be quite mild. 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 blood stream.

Although these lesions have been shown to regress spontaneously, a biopsy is almost always performed to determine a diagnosis. The biopsy is normally curative since 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. Recurrences are extremely rare but have been reported, as have bilateral cases.

**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
shows filling of the defects with the radiopaque media. Cases have also been seen that include lymphoid tissue in the cavity. It is felt that these may represent developmental lesions, although they may not present until 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.131

**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 Antoni B (spindle cells with a more haphazard appearance).

**Neurofibroma**

Neurofibromas are felt 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 associated with the \( \text{NF1} \) and \( \text{NF2} \) genes.

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 31-39). 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 following 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

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**FIGURE 31-37** Appearance of a Stafne's bone defect on panoramic radiograph below the inferior alveolar nerve on the right body of the mandible.

**FIGURE 31-36** A, Bilateral poorly defined traumatic bone cysts. (Bilateral cysts are unusual.) B, Same radiograph shown in Figure A with cysts outlined, showing the size and scalloped margins around teeth.

**FIGURE 31-38** Clinical photograph of a Stafne's bone defect on the lingual side of the mandible. This defect contained lymphoid tissue.

**FIGURE 31-39** A neurofibroma on the left inferior alveolar nerve presenting as a large fairly well-defined radiolucency in the mandibular ramus (arrow). The patient also had café-au-lait spots.
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 lateral neuroma can result (Figure 31-40). 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 following mandibular trauma, resection of pathologic lesions, and nerve involvement following dentoalveolar surgery (Figure 31-41).

If the symptoms are severe, appropriate treatment is resection of the neuroma and appropriate nerve reconstruction. Since 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. 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, 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. One unproven theory is that Paget’s disease may be a delayed or slow reaction to a myxovirus stimulus.

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 since 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 31-42), 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 use of salmon calcitonin or diphosphonates to inhibit bone resorption. Calcitonin can be taken either subcutaneously or by nasal spray, and diphosphonates 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 heart failure and osteosarcoma. Heart failure caused by the excessive blood supply to the remodeling bone 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.

**Gorham’s Disease**

Although first described in 1838, this disease was named after Gorham, who reviewed the literature and added three new cases in 1954. 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 31-44 and 31-45). The bone is usually replaced with fibrous tissue. The majority of cases are monostotic, but polyostotic cases have been reported. There is no specific treatment for this disease; however, radiation therapy and surgical resection have been beneficial in selected cases. Serum biochemistry is usually 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.

**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 31-46). It usually occurs in the second or third decade of life, and has a tendency to grow throughout life. It is tempting to feel 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 prostodontic reconstruction. The common surgical approach is via a double Y-shaped incision (Figure 31-47) 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 31-48). 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 31-49). It may be advisable to insert a dressing plate after the procedure to prevent excessive hematoma formation and possible recurrence of the torus (Figure 31-50).
Benign Nonodontogenic Lesions of the Jaws

Torus Mandibularis

Mandibular tori are bony exophytic growths that present on the lingual aspect of the mandible opposite the bicuspids (Figure 31-51). 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 lower 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’s cartilage and the neomandible, but this is almost certainly not correct.

If surgical removal is required, it is carried out via an extensive gingival margin incision with a possible lingual-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-directed 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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Estimates indicate that more than 1.3 million new cancers will be diagnosed in the United States this year, and 27,700 will be located in the mouth and oropharynx. This number represents approximately 3% of all cancers and is the eighth most common cancer affecting males in the United States. Globally, more than 360,000 new cases of oral cancer will be diagnosed this year. Mortality rates remain high despite some advances in locoregional control. There will be approximately 200,000 deaths worldwide, of which 7,200 will occur in the United States. Most patients will present for diagnosis with either regional or distant disease. Data have shown a trend for African Americans to have more advanced disease compared with white Americans (68% vs 52%) at the time of diagnosis. Even more alarming is the fact that, when compared with equal stages at the time of diagnosis, African Americans have a poorer 5-year relative survival rate compared with other races. A review of trends in 5-year relative survival rates over the past three decades has shown a statistical difference between the time periods of 1974 to 1976 and 1992 to 1996 (54% vs 59%); the improvement in survival again fails to hold true for the African American population.

Approximately 85 to 95% of all oral cancer is squamous cell carcinoma (SCC). However, multiple other malignant lesions can be found in the oral cavity such as sarcoma, minor salivary gland tumors, mucosal melanoma, lymphoma, or metastatic disease from nearly any site in the body.

**Risk Factors for SCC of the Oral Cavity**

The etiology of SCC of the oral cavity has been studied extensively. Numerous risk factors have been suggested as etiologic agents for the development of these malignancies. While no single causative agent can be attributed to the development of all oral cancers, several carcinogens have been identified, and of those tobacco and alcohol appear to have the greatest impact on malignancy development. Both extrinsic and intrinsic factors likely play a role in the development of SCC of the oral cavity. The risk of oral cancer associated with tobacco use is noted to be 2 to 12 times higher than in the nonsmoking population, and 90% of individuals with oral cancer will have a smoking history. The 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, expressed in the hetero- or homogeneity of certain tumor suppressor genes or oncogenes ($\text{TP53,c-my}}$), may lead to the development of a cell line capable of unregulated growth.

Alcohol in itself is not a recognized initiator in the development of oral SCC. 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. This may be related to the effects of contaminants in alcohol and its ability to solubilize carcinogens and enhance their penetration into oral mucosa.

A possible viral etiology has been demonstrated in oral cancers, especially by the human papilloma virus (HPV). The HPV subtypes 16 and 18, similar to those causing cervical cancer, have been implicated. Smith and colleagues showed that when individuals in his study had other risk factors adjusted, such as smoking, alcohol, and
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The presence of HPV in the oral cavity was associated with a 3.7 times greater chance of cancer development than in the noninfected individual. Other authors have noted a unique subset of characteristics in individuals that may develop SCC as a result of HPV infection, showing less association with tobacco or alcohol abuse, frequently involving the tonsils, and having an improved prognosis.

The study of the tumor biology of SCC has exploded in the past decade. The accepted molecular theory concerning genetic alterations of SCC is that of a “multihit” tumorigenesis ultimately leading to unregulated cell growth and function. It is thought that multiple exogenous insults (tobacco, alcohol, viral) can lead to activation of oncogenes or inactivation of tumor suppressor genes. Oncogene dysregulation leads to a gain of function alteration, and transforming growth factor alpha (TGF-α) and eukaryotic initiation factor 4E (eIF4E) are two examples of well-studied genes that have proven up-regulation in SCC. Loss of tumor suppressor gene function requires loss of both normal alleles, which leads to the inactivation of the critical function of that gene. The most studied of the tumor suppressor genes are TP53 and P16. No single gene alteration is responsible for carcinogenesis, but rather a host of altered genes contribute. Attempts have been made to use genes and their products to identify oncologically safe margins operatively with minimal success. Gene therapy trials that target these specific genes hold better promise.

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. Leukoplakia cannot be scraped or rubbed off and is therefore primarily a diagnosis of exclusion. Lesions caused by lichen planus, white sponge nevus, nicot ine 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. It may be found as 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. 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.

The only 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 smokeless, is most closely associated with the development of leukoplakia, and more than 70% of patients with leukoplakia are smokers. While 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 32-1 Typical appearance of floor-of-mouth leukoplakia.

FIGURE 32-2 Common presentation of proliferative verrucous variant of leukoplakia on gingiva.

FIGURE 32-3 Actinic cheilitis of the lower lip secondary to chronic unprotected sun exposure.
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 patient's habits. Waldron and Shafer 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%. 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. They note that, while 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 colleagues 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 32-4). Almost all true erythroplakia demonstrates dysplasia, carcinoma in situ, or invasive carcinoma. Shafer and Waldron's review of biopsies submitted 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 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 erythroplakia or "speckled erythroplakia" (Figure 32-5). These lesions also harbor an ominous potential as rates of malignant transformation have been noted of up to 23%.

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. 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.

Cervical Lymph Node Levels
The neck is divided into six "surgical levels" based on anatomic structures (Figure 32-6). Each anatomic area of the oral cavity has a predictable lymphatic drainage pattern to the over 300 lymph nodes in the neck. Levels I to VI are subdivided and described in text.
by grouping defined nodal groups into surgical levels, clinicians are afforded the ability to communicate with each other. It also allows clinicians to 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 digastic muscles laterally.

Level IB, the submandibular group, is bounded by the posterior belly of the digastic inferiorly, the mandibular body superiorly, the anterior belly of the digastic muscle anteriorly, and the stylohyoid muscle posteriorly.34,35

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).34,35

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.34,35

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.34,35

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 border 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.34,35

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.34,35

Clinical Correlation Based on Site

The boundaries of the oral cavity extend from the vermilionocutaneous junction of the lips to the junction of the hard and soft palate posterior-superiorly and to the line created by the circumvallate papilla posterior-inferiorly. Posterior-laterally 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.36 The sites have defined bound-
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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. 43

**Buccal Mucosa**

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. 36 The buccal mucosa is supported by the buccinator muscle posteriorly and the obicularis oris 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 SCC of the oral cavity (Figure 32-8). 4,37,38,44 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 SCC. 45 First-echelon lymphatic drainage from the buccal mucosa is level I followed by level II. 46 Cervical metastases are observed in 10 to 27% of presenting patients. 44,47,48

**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 to 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. 36 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. 36

Alveolar ridge or gingival carcinoma represents 2 to 18% of oral cancers and occurs predominantly on the mandibular alveolus (64 to 76%). 4,37-41,49,50 At diagnosis, approximately one-third of these tumors exhibit some bony involvement. 50,51 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 mandibular lesions and is found in 24 to 28% of patients at diagnosis. 46,49-51 Alveolar ridge carcinomas are frequently insidious tumors masquerading as inflammatory lesions, periodontitis or gingivitis, tooth abscesses, or denture sores (Figure 32-9).

**Retromolar Gingiva (Retromolar Trigone)**

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 32-10). 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. 4,38,39 Lymphatic drainage from this area is predominantly to the submandibular nodes (level IB) and the upper jugulodigastric nodes (level II). 46,52 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. 53-55

**Floor of the 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 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. 36 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 SCC, and several studies have shown a fairly dramatic increase in incidence (Figure 32-11).\textsuperscript{4,38–41} Two distinct lymphatic drainage systems have been identified in the floor of the mouth.\textsuperscript{56} The superficial system drains bilaterally into the submandibular nodes (level I), while the deep system drains into the ipsilateral submandibular, 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.\textsuperscript{57–59} Shaha and colleagues demonstrated that 60% of individuals with metastatic disease will have multiple levels involved.\textsuperscript{57}

**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 soft component.\textsuperscript{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).\textsuperscript{60–62} 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 SCC (Figure 32-12).\textsuperscript{4,37–39} 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.\textsuperscript{61,63} 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.

**Anterior Two-Thirds of the Tongue (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.\textsuperscript{36} 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%).\textsuperscript{64} Tongue carcinoma represents 22 to 49% of all oral cancer diagnosed (Figure 32-13).\textsuperscript{4,37–41} Several epidemiologic reviews have shown the unfortunate trend of an increase in tongue cancer and an alarming increase in the...
incidence of those diagnosed before 45 years of age. Lymphatic drainage of the oral tongue is principally to level II, followed by levels III and I. 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.

**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 32-1). 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 32-2). M grades distant metastasis and is based simply on its presence (M1) or absence (M0). The AJCC staging system (Table 32-3) 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).

**Assessment of Primary Lesion**

Proper lesional assessment is based on a thorough clinical evaluation. Accurate measurement of the primary lesion before biopsy is essential. Often, biopsied SCCs are referred without accurate measurements, leaving the treating surgeon in a difficult situation relative to properly assigning a T group. Additionally, postbiopsy inflammation could lead to over- or underestimates 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, 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

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**Table 32-1 Primary Tumors (T)**

<table>
<thead>
<tr>
<th>Tumor</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>TX</td>
<td>Primary tumor cannot be assessed</td>
</tr>
<tr>
<td>T0</td>
<td>No evidence of primary tumor</td>
</tr>
<tr>
<td>Tis</td>
<td>Carcinoma in situ</td>
</tr>
<tr>
<td>T1</td>
<td>Tumor 2 cm or less in greatest dimension</td>
</tr>
<tr>
<td>T2</td>
<td>Tumor more than 2 cm but not more than 4 cm in greatest dimension</td>
</tr>
<tr>
<td>T3</td>
<td>Tumor more than 4 cm in greatest dimension</td>
</tr>
<tr>
<td>T4a*</td>
<td>Tumor invades adjacent structures (eg, through cortical bone, into deep [extrinsic] muscle of the tongue, maxillary sinus, skin of face) (resectable)</td>
</tr>
<tr>
<td>T4b</td>
<td>Tumor invades masticator space, pterygoid plates, or skull base or encases internal carotid artery (unresectable)</td>
</tr>
</tbody>
</table>

*Superficial erosion alone of bone or tooth socket by an alveolar primary is not sufficient to classify a tumor as T4.

Adapted from Greene FL et al.

**Table 32-2 Regional Lymph Nodes (N)**

<table>
<thead>
<tr>
<th>Node</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>NX</td>
<td>Regional lymph nodes cannot be assessed</td>
</tr>
<tr>
<td>N0</td>
<td>No regional lymph node metastasis</td>
</tr>
<tr>
<td>N1</td>
<td>Metastasis in a single ipsilateral lymph node, 3 cm or less in greatest dimension</td>
</tr>
<tr>
<td>N2</td>
<td>Metastasis in a single ipsilateral lymph node, more than 3 cm but not more than 6 cm in greatest dimension; or in multiple ipsilateral lymph nodes, none more than 6 cm in greatest dimension; or in bilateral or contralateral lymph nodes, none more than 6 cm in greatest dimension</td>
</tr>
<tr>
<td>N2a</td>
<td>Metastasis in a single ipsilateral lymph node more than 3 cm but not more than 6 cm in greatest dimension</td>
</tr>
<tr>
<td>N2b</td>
<td>Metastasis in multiple ipsilateral lymph nodes, none more than 6 cm in greatest dimension</td>
</tr>
<tr>
<td>N2c</td>
<td>Metastasis in bilateral or contralateral lymph nodes, none more than 6 cm in greatest dimension</td>
</tr>
<tr>
<td>N3</td>
<td>Metastasis in a lymph node more than 6 cm in greatest dimension</td>
</tr>
</tbody>
</table>

Adapted from Greene FL et al.
structures. Assessment of the lateral tongue and posterior pharynx is assisted by anterior and lateral traction on the tongue with cotton gauze (Figure 32-14).

The AJCC describes the possible growth patterns of a tumor as endophytic, exophytic, or ulcerated.36 These characteristics play no part in staging the primary tumor. While depth of invasion 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.70–72 The study performed by Spiro and coworkers 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.70 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%. 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. Warren and Gates first described the notion of synchronous and metachronous tumors in 1932.73 A synchronous tumor is described as a second histologically confirmed malignancy. This malignancy must be 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 described the concept of “field cancerization” secondary to the panmucosal effects of smoked tobacco irritants and alcohol.74 This theory explains the relatively high prevalence of second primary malignancies in the upper aerodigestive tract and has been described on a molecular level.75 Panendoscopy became the gold standard for discovering an often asymptomatic synchronous lesion. McGuirt reported a synchronous primary lesion rate of 16% in his prospective study of 100 head and neck cancer patients.26 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%.77–81 Panendoscopy 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. Additionally with the advent of tomographic 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, while others have called for selective endoscopy to investigate only symptom-driven complaints.81–84 Should multiple primary tumors be discovered during patient evaluation, each lesion should be staged separately.

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%.85 A retrospective study by Snow and colleagues showed a surprisingly high rate of extracapsular spread in even small lymph nodes. His 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.86 Other studies have concurred with this high rate of extracapsular spread.87,88 These drastic

<table>
<thead>
<tr>
<th>Stage</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 0</td>
<td>Tis N0 M0</td>
</tr>
<tr>
<td>Stage I</td>
<td>T1 N0 M0</td>
</tr>
<tr>
<td>Stage II</td>
<td>T2 N0 M0</td>
</tr>
<tr>
<td>Stage III</td>
<td>T3 N0 M0</td>
</tr>
<tr>
<td></td>
<td>T1 N1 M0</td>
</tr>
<tr>
<td></td>
<td>T2 N1 M0</td>
</tr>
<tr>
<td></td>
<td>T3 N1 M0</td>
</tr>
<tr>
<td>Stage IVA</td>
<td>T4a N0 M0</td>
</tr>
<tr>
<td></td>
<td>T4a N1 M0</td>
</tr>
<tr>
<td></td>
<td>T1 N2 M0</td>
</tr>
<tr>
<td></td>
<td>T2 N2 M0</td>
</tr>
<tr>
<td></td>
<td>T3 N2 M0</td>
</tr>
<tr>
<td></td>
<td>T4a N2 M0</td>
</tr>
<tr>
<td>Stage IVB</td>
<td>Any T N3 M0</td>
</tr>
<tr>
<td></td>
<td>T4b Any N M0</td>
</tr>
<tr>
<td>Stage IVC</td>
<td>Any T Any N M1</td>
</tr>
</tbody>
</table>

Adapted from Greene FL et al.35,36
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. While there is no correct order in which to evaluate the neck, each clinician should develop a sequence to use 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 submandibular triangle to the posterior triangle. 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 moveable. Bending the patient’s head forward or slightly to the side will ease taut tissues of the neck allowing for better palpation. Other important palpable structures of the neck to be evaluated in the examination include the parotid gland, the thyroid gland, and the postauricular, occipital, and supraclavicular lymph node chains. The parotid gland should be evaluated for the presence of any palpable disease or masses and the thyroid gland for any nodule, masses, or thyromegaly. The trachea should be inspected for any deviation or fixation.

The past decade has seen a relatively high incidence of observer error. Deficiencies have been observed in both the ability to recognize the presence of a clinically palpable node and also in the ability to assess its size. A study by Alderson and colleagues 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, both computed tomography (CT) 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.

Computed Tomography
CT is generally performed preoperatively with intravenous contrast to help delineate vascular from lymph structures. The scan generally involves 3- to 5-mm slices from the skull base to the clavicles. 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. 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 sphere-like 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 32-15). Only an intranodal abscess or fatty hilar metaplasia can simulate central tumor necrosis.

Magnetic Resonance Imaging
MRI is another method of neck imaging that has gained popularity in the past decade. 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 T1-weighted, fat-suppressed contrast-enhanced image is perhaps the optimal sequence to evaluate cervical metastatic disease. 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 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
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
This specificity can be increased with the use of US-guided fine-needle aspiration. 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.

**Positron Emission Tomography**

The use of 2-¹⁸F-fluoro-2-deoxy-D-glucose (FDG) positron emission tomography (PET) relies on the enhanced metabolic activity of tumoral tissue in the body, of which increased glycolysis is usually the 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 32-16). 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 colleagues 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. As with ultrasound, FDG-PET may have a unique role in the evaluation of the clinically N0 neck. FDG-PET has found a place in the evaluation of an unknown primary with success rates reported from 10 to 60% in the identification of the index lesion. However, the infrequency of distant metastasis was recognized early by Crile. 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. As new therapies lead to better locoregional control of disease, we can expect to see a greater incidence of distant metastasis in long-term follow-up.

**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 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. A study by Holmes and colleagues 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.

**Toluidine Blue**

Oral cancer can have various clinical appearances, ranging from subtle mucosal color or texture changes to gross ulceration or a fungating lesion. These mucosal alterations are particularly difficult to assess in early cancers and dysplasia. It was recognized in the 1960s that toluidine blue stained malignant cells in vivo. Tolu-
Toluidine blue is currently marketed as a commercially available kit. Our opinion is that its use should be limited to the screening of high-risk individuals, and assisting in directing biopsies from a large area of abnormal-appearing tissue. In the end, toluidine blue cannot be substituted for a thorough oral examination and biopsies when clinical suspicion is high.

Biopsy

Once a clinically suspicious lesion is identified in the oral cavity, tissue diagnosis must be obtained prior to rendering any treatment. This 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 prior to biopsy in order to properly stage the lesion. 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 has gained acceptance in the dental community as a safe, minimally invasive technique for use in the screening of clinically suspicious lesions. 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 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 Scibba and colleagues found a sensitivity and specificity of 100%.

Conclusions

SCC of the oral cavity continues to be a common disease worldwide including in the United States. Despite research and advances in surgical and adjuvant therapy, long-term survival remains poor. It is a disease all clinicians will be faced with, and early recognition and diagnosis of premalignant and malignant disease is directly related to outcome. Proper staging of the primary lesion and neck with a thorough clinical examination and imaging is paramount to designing a successful treatment plan.

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Oral Cancer Treatment

Jon D. Holmes, DMD, MD  
Eric J. Dierks, DMD, MD

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 90% of malignancies affecting the oral cavity, and will be the focus of this chapter. Although discussion will be limited to the treatment of squamous cell cancers, oncologic principles outlined in this chapter can be applied to other malignancies affecting the oral cavity.1–3

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.4,5 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 is low. A recent editorial referred to oral cancer as “The Forgotten Disease.”6 Incidence and mortality for oral cancer is nearly double that of cancer of the cervix (30,300 vs 13,500 and 8,000 vs 4,400, respectively); yet few adults can remember their last oral cancer examination, whereas most women are aware of their last gynecologic examination and Pap smear.7 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.8 Although the oral cavity is readily accessible for examination, results of a study by Holmes and colleagues questioned whether health care professionals were screening for asymptomatic cancers.9 Additionally, 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.9 This is unfortunate since 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.10,11 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 squamous cell carcinoma is the most common, other variations require alterations in treatment.

Verrucous carcinoma is generally considered an uncommon variant of squamous cell carcinoma, representing only 5% of oral cancers.12 It has a predilection for the buccal mucosa, and typically appears as a thick white cauliflower-like growth (Figure 33-1). 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 following adequate excision.

Figure 33-1  Verrucous carcinoma of buccal mucosa with extension onto the adjacent maxillary alveolus.
Basaloid squamous cell carcinoma represents a rare aggressive form of squamous carcinoma. It affects males predominately, and is associated with a high rate of cervical and distant metastases. Histologically basaloid cells are arranged in nests or cords. 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. 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.

It is helpful to request from the pathologist a depth-of-invasion measurement on more superficial lesions, given its predictive value in regard to occult metastases, and determining the need for elective neck dissection (see discussion on elective neck dissection in this chapter). Depth of invasion will not influence treatment of deep indurated or fixed lesions. 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 predominately white lesion of the oral mucosa that cannot be characterized as any other definable lesion (Figure 33-2). Worldwide estimates of its prevalence range from 1.5 to 2.6%. Lower socioeconomic status seems to be associated with higher prevalence. The potential for malignant transformation of oral leukoplakia to invasive squamous cell carcinoma is well recognized, and leukoplakia can be considered a precancerous lesion (ie, “a morphologically altered tissue in which cancer is more likely to occur than in its apparent normal counterpart”). Estimated rates of transformation, however, vary widely. This most likely relates to the heterogeneity of the lesions included in most studies. While 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 colleagues 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%. 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.

Predicting which lesions will ultimately transform is currently not possible. Given its asymptomatic nature, the sole 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.

The first Cochrane review on therapy for leukoplakia did not find any reliable therapy to prevent the transformation of leukoplakia to oral squamous cell carcinoma. Also, there were no effective preventive measures to halt the development of oral leukoplakia. No surgical procedures were included in this 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. The ultimate goal remains prevention of subsequent malignant transformation, and unfortunately none of the agents demonstrated this reliably. In addition associated side effects were problematic (see section “Chemoprevention” in this chapter).

Surgical excision remains an alternative for dealing with worrisome lesions. CO2 laser excision has been used to treat widespread superficial lesions in an attempt to limit scarring and morbidity associated with large excisions. Laser ablation allows the destruction of large superficial lesions. It does not provide a histologic specimen, however, and biopsies from any areas of ulceration or erythroplasia are probably indicated prior to ablation. Unfortunately recurrence following laser excision or ablation is not uncommon, and it does not necessarily prevent malignant transformation.

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. 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, 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. 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.23-25

PDT has been used with some success in the endoscopic treatment of dysplastic Barrett’s esophagitis to prevent its transformation to adenocarcinoma.26 Similarly, attempts have been made to treat diffuse oral leukoplakia with PDT with some success.27 In addition to its role in the management of leukoplakia, initial trials of PDT applied to invasive squamous cell carcinoma of selected sites in the head and neck are being reported. Copper and colleagues 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.28 In addition to its application to mucosal lesions, interstitial delivery of light may allow treatment of more deeply situated tumors. 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 discussion that was started with the patient when the biopsy was performed is now continued with the knowledge that a malignancy is present, but that continued 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 prior to making treatment recommendations. It should also be remembered that there is a high incidence of depression in 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. This evaluation may be forgone if panendoscopy or “triple endoscopy” is planned to search for synchronous primary cancers.

Following McGuirt’s 1982 study of panendoscopy, examination of the esophagus, larynx, and bronchus was considered mandatory in the work-up of the patient with cancer of the head and neck.29 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 panendoscopy in their evaluation.29 Recently its routine use has been called into question for a variety of reasons, including cost containment, improved imaging modalities, and lower rates of synchronous primary tumors of the head and neck than previously expected. Some clinicians still feel 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 the 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, the majority of patients with cancer of the head and neck receive a flexible esophagoscope along with placement of a percutaneous endoscopic gastrostomy tube. For these reasons panendoscopy should probably be symptom driven.30

Choosing a Treatment

Once the initial evaluation, data collection, and staging are complete, a discussion regarding treatment is undertaken. The clinician and the patient are faced with deciding which treatment modality or combination offers not only the best chance for cure, but also 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 relies 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 will be 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 with one treatment modality at one session. For most stage I and stage II cancers of the oral cavity, surgical resection with frozen section analysis of the margins is advocated by most clinicians. Although primary radiation to
T1 and T2 lesions may offer similar disease control, the side effects of radiation to the oral cavity 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. Reirradiation, 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, 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. 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. A recent study demonstrated that an altered P53 gene existed in 52% of reviewed patients and that recurrence occurred in almost half of these patients. 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 when present at the resection margin. In an interesting study Thomson performed biopsies in patients with unilateral squamous cell carcinomas or premalignant lesions. These 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. This concept of “condemned mucosa” has caused clinicians to question the ability of surgeons to obtain clear margins in some cancers. Studies have clearly shown, however, a decreased local control and survival in patients with positive margins.

Jacobs and colleagues 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. These studies have led some to investigate addition of brachytherapy to external beam postoperative radiation to patients with unsatisfactory margins, and to subsequently demonstrate improved survival in patients with positive margins who received this intensive radiation. It is agreed by most that surgeons should strive for clear margins, given this impact of positive margins on survival. Excision with 1 to 1.5 cm of normal tissue beyond the obvious tumor edge is generally sufficient. 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 33-3). Mucosal margins as well as deep margin specimens are submitted; however, frozen-section analysis is accurate but not infallible. Indeed the role of frozen analysis of margins in oral cavity cancer has been heavily debated and its cost effectiveness 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.

On occasion a surgeon will be faced with a situation where the frozen margins 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. Secondly, 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. 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 section “Radiation” below). 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. Sutton and colleagues 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.

Surgery in patients with head and neck squamous cancer presents 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 will 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 comorbidities are associated with increased complications and lengthy hospital stays. This is especially true regarding complex reconstructive efforts, such as vascularized tissue transfer. 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 prior to the incision and continued for no more than 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.

**Alcohol Withdrawal** Many patients with oral cavity cancer will be dependent on alcohol. Alcohol withdrawal is common if precautions are not taken and 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 to 10% alcohol with 5% dextrose in water) can be used in the postoperative period and slowly tapered as the patient recovers from surgery.

**Deep Venous Thrombosis** Patients who will be immobilized for a significant time during surgery or following surgery should receive prophylaxis for deep venous thrombosis. 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** Opinions regarding the need for transfusion vary. In general a hematocrit less than 25 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 nutrition 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 or 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. Although rare there have been anecdotal reports of seeding squamous cell carcinoma to the abdominal wall if the PEG is placed prior to resection.\(^43\) This complication, although rare, has led some surgeons to recommend placement in the postoperative period. Even if a PEG is placed, the patient should be encouraged to continue some oral intake, as 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 comorbidities, such as ischemic cardiac disease, chronic pulmonary disease, and alcoholism. Medical manifestations of preexisting chronic disease states, such as myocardial infarction, stroke, and pneumonia, can be precipitated by major surgery, a long general anesthetic, and a prolonged intensive care unit stay. 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.\(^44\) 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, or 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. 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 R’s: repair, reoxygenation, redistribution, and regeneration.

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 man-made 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.\(^45\) 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 impact with H2O molecules to create secondary particles that interact with cellular DNA. Absorbed dose is reported as a gray (Gy), which is one joule of absorbed dose per kilogram. Previously dose was reported as a rad, which was defined as 100 ergs absorbed per gram. One gray is equal to 100 rad and one centigray (cGy) equals one rad (1 cGy = 1 rad).

In the early to mid-twentieth century, radiation was given as orthovoltage (125 to 500 KeV). Currently radiation is delivered as megavoltage (> 1 MeV). Megavoltage results in more radiation delivered to deeper tissues with less superficial (skin) damage. In 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. **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 feel 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.\(^46\)\(^47\)

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 (ie, 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
Three-dimensional conformal radiotherapy (226Ra) or iridium (192Ir), directly in the tumor mass. Brachytherapy has developed a reputation for creating chronic wounds and may lead to osteoradionecrosis when used adjacent to the mandible. Its current use is generally limited to treatment of tongue or tongue base primaries, and is usually combined with external beam radiation. Brachytherapy has also been advocated for treatment of close or positive margins following surgical excision.35 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.50,51

Radiation can be administered with curative intent in the preoperative setting or as an attempt to shrink a tumor presurgically (neoadjuvant). 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 its dependence 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 4 weeks of 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 makes 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 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)
- 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.32 Million and colleagues found that 35% of patients with clinically negative necks converted to positive if the primary cancer was treated with surgery alone.33 This dropped to 5% if radiation therapy was added. Even microscopic evidence of extracapsular extension is associated with a higher rate of recurrence and death.34 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 improved 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,
RTOG 73-03, compared 50 Gy preoperative radiotherapy to 60 Gy postoperative radiotherapy. The 10-year follow-up demonstrated no survival advantage to either regimen, but postoperative radiation treatment demonstrated superior locoregional control. How much is enough? Results from an MD Anderson Cancer Center (University of Texas, USA) study showed that 54 Gy was needed in the postoperative setting, and 57.6 Gy was needed if extracapsular extension was present.

Timing of initiation of radiation therapy following surgery is controversial. Vikram 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. 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.

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 recently been applied to head and neck cancer patients to protect salivary gland function during radiation therapy. 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. 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. Its use is associated with side effects, such as hypotension, and some patients do not tolerate it. 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 section “Chemotherapy” below).

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 reported by 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. 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. Although criticized by some for its lack of a radiation-only control group, the results fostered a renewed interest in 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.

Prior to 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. Since chemotherapeutic agents are most effective against cells undergoing replication, smaller and faster growing tumors are more susceptible.

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 formula above
- 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 only last for 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 percent of patients achieving complete and partial responses. An additional problem with chemotherapy trials is patient selection bias. Increasingly the role of comorbidities 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 discussion below). 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).65,66 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 prior to definitive treatment of the primary cancer site.64 This tactic is generally chosen in an attempt to decrease the size of the primary cancer to make definitive treatment possible. For example, a tumor deemed unresectable may be “downstaged” by neoadjuvant chemotherapy to a resectable tumor. 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. Indeed 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 noncell cycle-specific agents, depending on whether the particular agent requires that the target cell be in a certain phase (G0, S, G1, or mitosis) to be effective. Agents can also be categorized based on their principle mode of action. Antimetabolites, such as methotrexate and 5-fluorouracil, block development of certain metabolites critical for cell metabolism. 5-Fluorouracil 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 only used 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. Other agents used less frequently in head and neck squamous cell cancer
include paclitaxel, which stabilizes microtubular formation and arrests cells in G2, 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 underway.\(^{67,68}\)

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. The question of adding chemotherapy in the postoperative setting remains unanswered. Currently no study has shown definitive improvement. Cooper and colleagues reported on the results of the RTOG 95-01/Intergroup phase 3 trial that evaluated concurrent chemoradiotherapy in postoperative treatment of high-risk squamous cell carcinoma of the head and neck, defined as multiple lymph nodes involved, extracapsular disease, and positive margins. The locoregional control and overall 2-year survival were not improved significantly, and the small improvement in disease-free survival was at the expense of a significant increase in toxicity.\(^{69}\) Adding chemotherapy following surgery and radiation has been shown to decrease the incidence of distant metastases, but this has not been associated with improved survival. At this point chemotherapy in the postoperative setting is not indicated except in cases of known metastatic disease, and its use outside of clinical trials should probably be discouraged.\(^{70-72}\)

Currently the role for chemotherapy in oral cavity cancer is limited to use in unresectable disease in which it is combined with radiation treatment, metastatic disease, or recurrence. 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, and Munro have demonstrated that concurrent treatment is better than neoadjuvant therapy. Locoregional control and survival were improved in advanced head and neck cancers.\(^{73,74}\)

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 section in this chapter 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.\(^{75}\) Treatment involves supradios 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%.\(^{75}\) 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.\(^{75}\)

Other novel techniques for minimizing the systemic side effects of chemotherapeutic regimens are under development, including the PDT discussed above 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. The most commonly used chemotherapeutic regimen for metastatic or recurrent oral cavity squamous cell carcinoma involves combinations of cisplatin or carboplatin and 5-fluorouracil. Median survival rates of 5 to 7 months and 1-year survival of 20% demonstrate the need for improved regimens. 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.\(^{76}\)

Current research in chemotherapeutic agents focuses on 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 epidermal growth factor inhibitors.\(^{77}\) Gene therapy that targets known alterations in head and neck squamous cell cancers, such as TP53, is also an area of growing research.\(^{78}\) Restoration of these altered genes, possibly through viral vectors, holds promise in certain populations.\(^{79}\) A recent review by Milas and colleagues at the MD Anderson Cancer Center offers insight into the current state of chemotherapy in head and neck cancer, as well as newer chemotherapeutic agents on the horizon.\(^{80}\)

### Chemoprevention

An additional area of intensive research is development of chemoprevention agents, which are defined as agents that reverse or suppress premalignant carcinogenic progression to invasive malignancy (see section “Management of Premalignant Lesions,” above). 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 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 in the prevention of second primary cancers. It may act through the up-regulation of a distinct retinoic acid receptor, RAR-β, 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 discontinuing the drug. Secondary primary tumors occur in 4 to 7% of patients treated for head and neck squamous cancer and are the major concern-related cause of death in early-stage cancer. The prevention of these tumors is therefore important. Studies of 13-cis-retinoic acid have shown 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. 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.\textsuperscript{81,82} In addition, Wang and colleagues recently reported on a novel tretinoin biofilm that allows sustained topical delivery to the oral cavity.\textsuperscript{83}

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.\textsuperscript{84} Nonsteroidal anti-inflammatory drugs have also been investigated since chemoprevention activity was found in some cyclooxygenase-2 (COX-2) inhibitors. COX-2 influences several steps in the development of malignancies, such as apoptosis, angiogenesis, invasiveness, and immune surveillance.\textsuperscript{85,86} COX-2 expression has been noted in high-risk premalignant lesions.\textsuperscript{87} In addition to their potential role in chemoprevention, COX-2 inhibitors hold promise in the treatment of invasive squamous cell carcinomas.\textsuperscript{88} Although chemoprevention offers hope for patients at high risk for development of second primary cancers and treatment of patients with high-risk lesions (see discussion on premalignant lesions above), 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**

**The 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 33-5). CO\textsubscript{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 along the inferior alveolar nerve, several years after a lip cancer, can be mistaken for primary intraosseous carcinoma (Figure 33-6).

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)\textsuperscript{89,90}

\textbf{FIGURE 33-5 Vermilionectomy combined with wedge resection of lip cancer associated with diffuse actinic changes across the remainder of the lip.}
Buccal Mucosa

Buccal squamous cell carcinomas represent approximately 10% of oral cavity cancers in the United States compared to 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, cancers that penetrate the buccinator muscle can be difficult to eradicate (Figure 33-7). 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 around 10%. 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. Vikram and Farr concluded that combined therapy for large lesions with surgery and radiation offered the best chance for cure.

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). 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. Diaz and colleagues found 5-year survival rates of 69% with nodal involvement, which decreased to 24% in cases with extracapsular extension.

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, like an oropharyngeal primary cancer (Figure 33-8). Smaller lesions are amenable to wide local excision with or without a marginal mandibulectomy depending on the proximity to the bone (see discussion below). Larger lesions may invade the pterygomandibular space and extend cephalad towards 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 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. In an excellent review of the management of RMT cancers, Genden and colleagues also suggested that the addition of preoperative or postoperative radiation confers a survival advantage.
The Tongue

The oral tongue, that portion anterior to the circumvallate papillae, is the most common location for intraoral squamous cell carcinomas. They typically present as painless indurated ulcerations. If pain is present it is usually due to secondary infection. The behavior and treatment of oral tongue cancers is sufficiently different than that of posterior tongue lesions (oropharyngeal tongue or tongue base) to allow clinicians to determine the epicenter of the tumor and classify it correctly. This may be challenging in the case of larger cancers. The oral tongue poses significant challenges to clinicians. Seemingly small lesions can metastasize early and recur after treatment. Control rates for small lesions of the tongue (60 to 80%) are poorer 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 33-9). 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 colleagues 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 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. Consideration should be given to extending the neck dissection to include level IV. Postoperative radiation should be considered in situations where 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 33-10). 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 33-11). 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 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 should be treated with combined therapy, typically surgery plus 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.

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. Some clinicians have reported 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.

**Floor of Mouth**

The floor of mouth (FOM) is the second most common location for oral cavity squamous cell cancers (Figure 33-12). FOM cancers can extend along the ventral tongue and cause fixation. In addition FOM tumors can become fixed to the mandible or extend into level I of the neck. McGuirt and colleagues 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 mandibulectomy may be considered in tumors that abut the mandible without evidence of invasion (see discussion below). 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.

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).

**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, 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 below. 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 33-13). The pterygoid plates require removal only if there is evidence of invasion through the posterior maxilla. Posterior extension may indicate the need for a transfacial (Weber-Fergusson) approach for adequate exposure. Reconstruction of maxillary defects can be accomplished with local flaps, such as the temporoparietal fascial 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. Prosthetic obturation of the defect...
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offers several advantages, including a simpler operation, easier early detection of recurrence, and replacement of teeth.

Most maxillary alveolus and gingival squamous cells that invade the sinus involve the infrastructure lying below 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 supra-structure. Defects arising from cancers resected below this line are easily reconstructed with obturators. Cancers that require true total maxillectomy (resection of one of the paired maxillae, including the orbital floor) tend to require flap reconstruction. If dura mater is exposed 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 lower alveolar ridge. Poor outcome is associated with advanced stage, perineural spread, and positive margins. Adjunctive radiation is recommended if nodal metastases, perineural spread, or positive margins are present.

The Palate

Squamous cell cancers of the palate, hard and soft, are rare. The soft palate is considered an oropharyngeal subsite (Figure 33-14). Cancers arising from the hard palate 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. 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, as the majority will close spontaneously. Occult cervical metastases are rare among hard palate cancers (10 to 25%), and elective treatment of the neck is generally not indicated except in T3 or T4 lesions. Also, metastases may occur to the retropharyngeal nodes, and consideration should be given to irradiation of the neck to include this area if suspected.

Management of the Mandible in Oral Cavity Cancer

Management of the mandible in oral cavity cancer has been the subject of much controversy. In the past the mandible was routinely sacrificed in the treatment of FOM and tongue cancers, as it was felt that the regional lymphatics coursed through the mandibular periosteum, necessitating an in-continuity resection of the tongue, FOM, mandible, and neck dissection (Commando's operation). The morbidity of this approach was felt necessary to eradicate in-transit metastases, a belief that was likely based on mistranslation by McGregor of an article published by Polya and von Navratil in 1902, in which they actually recommended removal of the periosteum or rim of mandible and not a segment.

Marchetta and colleagues 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. O'Brien and colleagues 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. This finding has led to surgeons advocating preservation of mandibular continuity through the use of a marginal mandibulectomy, in cases without obvious bony involvement. These principles apply only to the nonirradiated mandible. Cancer invasion of a previously irradiated mandible occurs through multiple sites. The clinical and radiographic evaluations of mandibular involvement, however, 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 interpret accurately. A high-quality panoramic radiograph is probably the most commonly used tool to decide on mandibular resection versus segmental resection. CT scanning using DentaScan

FIGURE 33-13  Le Fort I island approach to a posterior maxillary tumor, without Weber-Fergusson incision.

FIGURE 33-14  Squamous cell carcinoma of the right soft palate invading posteriorly to involve the anterior pole of the tonsil.
Part 5: Maxillofacial Pathology

software has also been used, although recent studies have shown that an MRI demonstrating enhancement of the marrow signal was a better predictor 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. At this time the most accurate assessment of mandibular involvement occurs at the time of surgery when the surgeon can inspect the bone. In addition, some surgeons send periosteum for frozen-section analysis, whereas others submit cancellous scrapings for frozen analysis. 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. 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 33-15). 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. Edentulous mandibles are generally not candidates for marginal resection although this is not an absolute rule. Wax and colleagues, and Shah 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 possible return to the operating room if final pathologic analysis reveals an unexpected amount of bone involvement necessitating a segmental resection and more elaborate reconstruction.

Splitting the mandible, or mandibulectomy, is often necessary for access to large cancers, especially of the posterior tongue (Figure 33-16). Technical refinement of mandibulectomy helps avoid complications. Mucosal incisions should not be placed directly overlying the proposed osteotomy site. Division of the mandible at the parasympysis 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 nerve to preserve it. Preadaptation of plates will allow reestablishment of the preoperative occlusion and contour.

Management of the Cervical Lymph Nodes in Oral Cavity Squamous Cancer

Management of the regional lymphatics is a consideration in any cancer. The ability of a cancer to metastasize most commonly manifests itself by growth of cancer in lymph nodes. Surgical treatment of the neck is justified for two reasons: the removal of gross disease in patients with...
Computed tomography scan to avoid postbiopsy inflammatory nodal and is usually accomplished before biopsy is regarded as the first step in this process 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 is low, with an overall error of approximately 30% in several studies. 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 33-17). Specific criteria for nodal metastases, including node size greater than 1 cm (except the jugulodigastric node, which must be greater than 1.5 cm), central necrosis, and morphology (round instead of oval) have increased sensitivity to over 90%. 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 colleagues 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. O-charoenrat and colleagues 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 colleagues, 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 elective neck dissection or irradiation) for thicker primary tumors. Other investigators have suggested that depth of invasion be added to the staging of oral squamous cell carcinomas. Aside from depth, clinicians have looked at other characteristics such as DNA aneuploidy and histologic grade. At this time applications of this technology have not been adopted in the routine clinical setting.

Two additional imaging modalities used for evaluating nodal metastases deserve mention. Ultrasonography and PET with fluorodeoxyglucose 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 hypoechochogenic 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. Knappe and colleagues 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.

Recently PET has 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 work-up (Figure 33-18). Presence of distant metastases may influence the choice of initial treatment. The role of PET scans in the evaluation of occult cervical metastases...
Part 5: Maxillofacial Pathology

is limited by the need for at least 5 to 10 mm$^3$ of tumor for detection. Their role in the work-up of patients with cervical metastases and unknown primaries continues to be explored. PET is also used to examine patients who have undergone chemoradiotherapy for recurrent disease. 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 prior to obtaining a PET scan because of the persistent inflammation associated with radiation and the tumoricidal effects that persist after radiation is completed. PET scan is also used to examine patients who have undergone chemoradiotherapy for recurrent disease. 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 prior to obtaining a PET scan because of the persistent inflammation associated with radiation and the tumoricidal effects that persist after radiation is completed.

Neck Dissection in Oral Cavity Squamous Cell Cancer

The goals of neck dissection are to remove gross disease in patients with clinical evidence of nodal involvement (therapeutic neck dissection) or to remove occult metastases in patients whose tumor characteristics make one suspicious of occult cervical metastases (elective neck dissection or END). The importance of treating the cervical lymph nodes was stressed by Crile in his landmark 1906 paper, and was later popularized by Martin and colleagues. Generations of surgeons were trained in the classic radical neck dissection (Figure 33-19). 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. Revisions were proposed in 2002 to improve communication among clinicians. 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 listed below:

- **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 through 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 3 types:
  - **Type I** preserves the spinal accessory nerve.
  - **Type II** preserves the spinal accessory nerve and the sternocleidomastoid muscle.
  - **Type III** preserves all three structures.

![Positron emission tomography scan showing recurrent cancer in the left neck with three mediastinal metastases. Note the heavy physiologic uptake in the brain, heart, kidneys, and bladder.](image)

![Standard radical neck dissection of the right neck. Note the communication with the oral cavity where an oral cancer has also been resected.](image)
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 33-20). 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. For example, a selective neck dissection for oral cavity cancer would encompass those node groups most at risk (levels I to III) and be referred to as a selective neck dissection (levels I to 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 colleagues 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. Extension on the left side does entail an increased risk to the thoracic duct and attendant chyle leak. Modifications of neck dissections have been made in an attempt to prevent the morbidity of radical neck dissection (Figure 33-21). 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 33-22). 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 (1.6%), and is recommended only if bulky disease is present in level IIa.

If there is clinical evidence of lymph node metastases, controversy exists over the proper type of neck dissection (see section “Therapeutic Neck Dissection,” below). The application of supraomohyoid neck dissection to the N positive neck (therapeutic neck dissection) has yielded mixed results. 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. The question remains as to whether this is due to the type of neck dissection or simply the biology of the tumor. 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 found no adverse impact on survival when neck dissection was performed in a discontinuous manner. Bias might have occurred, however, as smaller lesions were in the discontinuity group. A study by Leemans and colleagues 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 elective neck dissection 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. Although several studies have failed to demonstrate a survival advantage in patients who undergo elective neck dissection 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 elective neck dissection) is becoming increasingly limited. The sentinel node technique, first popularized for melanoma, has been investigated for use in head and neck cancer. 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. The patient is then taken to the operating room where the surgeon may 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 33-23). The surgeon will also use a gamma detection probe counterprobe to identify the node with the highest concentration of radioactive colloid. The node is then removed, 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.

The 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. In addition close proximity of the sentinel node to the primary cancer, for example, an FOM primary cancer and 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 colleagues used the sentinel node technique in 18 oral cavity cancers with N0 necks. They compared sentinel node biopsy to CT images and PET images by obtaining a CT and PET followed by sentinel node biopsy and neck dissection. They found 10 true-positives, 6 positive nodes identified on frozen section, 2 positive nodes 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. Pitman and colleagues further demonstrated the use of the sentinel node biopsy technique for the N0 neck. Hyde and colleagues reported on 19 patients whose radiographic and clinical test results on their necks were negative and who underwent sentinel lymph node biopsy 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, above). In the future the sentinel node biopsy may become the operative procedure of choice for dealing with the N0 neck. In an excellent review Pitman and colleagues concluded that sentinel lymph node biopsy remains an experimental technique in head and neck cancers and has not become a standard of care.

**Therapeutic Neck Dissection**

Patients presenting with nodal disease will usually undergo some type 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. There is some evidence that selective neck dissection may be adequate for the N positive neck in certain carefully selected patient populations (see discussion of selective neck dissections, above). Anderson and colleagues reported the results of three academic centers in which patients with previously untreated clinically and pathologically N positive necks underwent neck dissection. They reported a regional control rate of 94.3%. Their results were comparable to patients undergoing more extensive neck dissections. Patients presenting with massive nodal disease who are going to be treated with chemoradiation therapy or combination of brachytherapy and external beam therapy can present a challenge to surgeons who are faced with the option of surgery before or following radiation. Not infrequently surgeons are faced with complete clinical resolution of disease and the prospects of 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 found that clinical factors did not predict patients with residual disease following 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 following chemoradiation therapy. The role of PET scanning in this situation is unclear, but patients with recurrent cancer following multimodality therapy typically have a poor outcome, making salvage surgery an unattractive alternative.

Surgical management of cervical lymph node metastases, 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.” Clearly the role for 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.

**Recurrent and Follow-Up Surveillance**

In 1984 Vikram and colleagues 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, distant sites, as well as development of second malignant neoplasms in patients treated at Memorial Sloan-Kettering Cancer Center, NY, USA. 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 of 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%. Follow-up protocols vary widely and are intended to detect recurrences early. De Visscher and Manni suggested the following:

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 and can lead to chronic wounds.

Appropriate imaging, including a baseline CT or MRI at the completion of multimodality therapy, is invaluable. The role of PET scanning in follow-up continues to evolve.

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 about 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 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 cancers.\textsuperscript{136,157–159}

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 allow for detection of lung metastases, the most common distant site metastasized for oral cavity cancer, and primary lung cancers, which are not uncommon in the population at risk for oral cancer.\textsuperscript{162} Given the current unavailability of an effective treatment regimen, however, some authors have questioned the use of annual or semiannual chest radiographs.\textsuperscript{163} PET scanning may prove to be a more valuable alternative for detection of distant disease. Yearly lab 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, as many will ultimately become hypothyroid with attendant fatigue and decreased wound healing ability.

Collins 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.\textsuperscript{164} 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.\textsuperscript{164} Patients with recurrent disease are restaged, which requires a similar evaluation as the original. 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 prior to deciding on aggressive retreatment. It the patient does have recurrence that is confined to the locoregional area, treatment decisions are limited by previous therapy. Reirradiation protocols exist but are accompanied by significant morbidity.\textsuperscript{165} Intensive reirradiation and chemotherapy protocols are being investigated and show some promise.\textsuperscript{166} 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 reported on the outcome of salvage surgery for recurrent head and neck cancers, and found benefit in stages I and II.\textsuperscript{160} Success was limited in more advanced disease.\textsuperscript{160} 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 as 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. As cure is no longer a realistic option, treatment modalities to prolong life and improve quality of life assume a higher priority. 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 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 significant side effects in most.\textsuperscript{167} 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.\textsuperscript{164}

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 deal with these very real concerns. Hospice provides an excellent resource, and once enrolled most families are 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. High expression of epidermal growth factor receptors 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. Importantly studies are also demonstrating that comorbidities and performance status predict survival independent of stage at diagnosis. Performance status has been shown to be a predictor of survival independent of tumor, regional nodes, and metastasis (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 cancer-specific survival. Ribeiro and colleagues found that daily alcohol consumption, smoking, poor body mass index, and other comorbidities 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 comorbidities. Frequently 5- and 10-year survival curves are impacted more by these comorbidities than the tumor characteristics recorded in the TNM system (see discussion below). 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 following initial therapy. These patients may benefit from more aggressive combination treatments. 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 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.

**References**


130. Crile G. Excision of cancer of the head and neck with special reference to the plan of dissection based upon one-hundred thirty-two operations. JAMA 1906;47:1780–6.


in the neck following multimodality treatment for advanced head and neck cancer.


Lip Cancer

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G. E. Ghali, DDS, MD

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 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

Epidemiology and Etiology

The incidence of lip cancer varies throughout the world, resulting in 30% of all malignant tumors of the oral cavity in certain regions. In the sunbelt region of the United States, lip cancer is the most common cancer of the oral cavity, and its incidence is second only to skin malignancy of the head and neck. Australia, northern Spain, and Newfoundland have a reported annual incidence ranging from 11 to 50 cases per 100,000 population.9

In the United States, the incidence of lip cancer is 1.8 per 100,000 population, with the state of Utah having the highest regional rate of almost 12 cases per 100,000.10 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 to 98%), with only 2 to 7% arising from the upper lip and 4% at the oral commissures.1,4–6,11–13 The most common age at diagnosis is 54 to 65 years.4 Although a condition seen in middle age, lip cancer occasionally occurs in patients under age 30 years.14 Lip cancer has a predilection for men, with men to women ratios ranging from 35:1 to 6:1, depending on the location of the lesion.4

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 proven. 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, compared with 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.11,17 Although it has never been proven, darker-skinned individuals are believed to have a protective pigment in the vermilion of the lips that provides protection from solar injury.10

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.11–13,16,18–23 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.5,11,15,19,24–28 In 1984, Douglass and Gammon 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.10 Additional case-controlled studies concluded that no statistically significant relation exists between tobacco exposure and lip carcinoma.29,30 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. Because alcohol and tobacco exposure—the two factors most strongly associated with developing oral carcinoma—seem to have limited influence on the developing lip carcinoma, the most consistently associated factor with lip cancer appears to be prolonged and cumulative exposure to ultraviolet radiation from sunlight.4,18–20,24,29,31

Anatomic Considerations

Embryologically, the upper lip forms by fusing the two maxillary processes with a
central median nasal process (Figure 34-1). 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 labiomial 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.” 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.

Lymphatic drainage of the lower lip originates as an interconnecting network of lymph vessels beneath the submucosa of the vermilion. 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. 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 in up to 20% in cancer of the upper lip and commissure. In the upper lip, crossover of lymphatic drainage between the right and left halves typically does not occur. The upper lip also possesses five or six collecting trunks on each side of the midline that originate as delicate lymphatic vessels in the submucosa of the vermilion. The trunks ultimately terminate in the submandibular lymph nodes but occasionally also drain to the ipsilateral preauricular or infraauricular parotid lymph nodes. Metastasis that results from cancer of the lip most commonly involves the submandibular and submental lymph nodes (level 1). 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. Metastasis is found in the submandibular lymph nodes in about 80 to 90% of patients with metastasis from cancer of the lower lip. 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. 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.

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. Lymph node metastasis to the upper jugular digastric chain is seen in only about 15% of all patients who have lymph node metastasis and is almost always seen in conjunction with ipsilateral submandibular metastasis.

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 34-2). Well-differentiated squamous cell carcinomas are often associated with hyperkeratosis and leukoplakia of the vermilion border of the lip. Any lip lesion that
the clinician views as a possible malignancy should 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 the histopathologic type and grade of the tumor.

Staging of lip cancers is similar to that employed for tumors of the oral cavity (Table 34-1). 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 34-2).

Radiographic evaluation of tumors detected at an early stage that involve the lip is generally unnecessary. On the other hand, advanced tumors that adhere to or invade the adjacent mandible require further radiologic evaluation. 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. 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 esthetics of the perioral region. Although external beam irradiation or surgical excision can control small primary tumors of the lip equally well, surgery is quicker and leaves little esthetic or functional impairment. Larger 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 stages of the disease.

<table>
<thead>
<tr>
<th>Table 34-1</th>
<th>Tumor Stage Grouping</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 0</td>
<td>Tis N0 M0</td>
</tr>
<tr>
<td>Stage I</td>
<td>T1 N0 M0</td>
</tr>
<tr>
<td>Stage II</td>
<td>T2 N0 M0</td>
</tr>
<tr>
<td>Stage III</td>
<td>T3 N0 M0 T1 N1 M0 T2 N1 M0 T3 N1 M0</td>
</tr>
<tr>
<td>Stage IV</td>
<td>T4a N0 M0 T4a N1 M0 T1 N2 M0 T2 N2 M0 T3 N2 M0 T4a N2 M0</td>
</tr>
<tr>
<td>Stage IVA</td>
<td>Any T N3 M0 T4b Any N M0</td>
</tr>
<tr>
<td>Stage IVB</td>
<td>Any T</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 34-2</th>
<th>Tumor Node Metastasis System for Lip Cancer</th>
</tr>
</thead>
<tbody>
<tr>
<td>T = Tumor Size</td>
<td></td>
</tr>
<tr>
<td>TX</td>
<td>Primary tumor cannot be assessed</td>
</tr>
<tr>
<td>T0</td>
<td>No evidence of primary tumor</td>
</tr>
<tr>
<td>Tis</td>
<td>Carcinoma in situ</td>
</tr>
<tr>
<td>T1</td>
<td>&lt; 2 cm</td>
</tr>
<tr>
<td>T2</td>
<td>&gt; 2 cm and ≤ 4 cm</td>
</tr>
<tr>
<td>T3</td>
<td>&gt; 4 cm</td>
</tr>
<tr>
<td>T4</td>
<td>Tumor invades through cortical bone inferior alveolar nerve, floor of mouth, or skin of face (ie, chin or nose)</td>
</tr>
<tr>
<td>N = Nodal Metastasis (regional)</td>
<td></td>
</tr>
<tr>
<td>NX</td>
<td>Regional lymph nodes cannot be assessed</td>
</tr>
<tr>
<td>N0</td>
<td>No regional nodes palpable</td>
</tr>
<tr>
<td>N1</td>
<td>Single ipsilateral node &lt; 3 cm</td>
</tr>
<tr>
<td>N2</td>
<td>Metastasis in a single ipsilateral lymph node, &gt; 3 cm but ≤ 6 cm; or in multiple ipsilateral lymph nodes, none &gt; 6 cm; or in bilateral or contralateral lymph nodes, none &gt; 6 cm</td>
</tr>
<tr>
<td>N2a</td>
<td>Single ipsilateral node &gt; 3 cm but ≤ 6 cm</td>
</tr>
<tr>
<td>N2b</td>
<td>Multiple ipsilateral nodes ≤ 6 cm</td>
</tr>
<tr>
<td>N2c</td>
<td>Bilateral or contralateral nodes ≤ 6 cm</td>
</tr>
<tr>
<td>N3</td>
<td>Node &gt; 6 cm</td>
</tr>
<tr>
<td>M = Distant Metastasis</td>
<td></td>
</tr>
<tr>
<td>MX</td>
<td>Distant metastasis cannot be assessed</td>
</tr>
<tr>
<td>M0</td>
<td>No distant metastasis</td>
</tr>
<tr>
<td>M1</td>
<td>Distant metastasis</td>
</tr>
</tbody>
</table>
lip cancers, and surgery is the most common treatment selected for managing lip carcinoma of any size, particularly the larger T3 and T4 tumors.

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.2,11,25,42–44 Small lip cancers, less than 1 cm in greatest dimension, can often be managed with slightly smaller margins of 5 mm.44 In our experience, the locally advanced T4 squamous cell carcinomas of the lip are optimally treated with a slightly larger margin of approximately 15 to 20 mm.

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 34-3).45–54 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.

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, W, or a shield (Figure 34-4). The defects resulting from the V and W excisions can easily be closed primarily with no additional mobilization of adjacent tissues (Figure 34-5). The rectangular form of excision, however, requires advancement of laterally based lip flaps to achieve a satisfactory closure. These forms of excision are selected purely on the basis of cosmetic and functional considerations for all T1 and most T2 lip carcinomas.

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 necessitate 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 will be discussed in detail in the following section on lip reconstruction. Include 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 actually demonstrate radiographic invasion of the mandible, include a segmental mandibulectomy in the treatment plan.

**Lip Reconstruction**

Lip reconstruction following 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 following 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.55–57 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 34-6). Other less commonly used flaps for vermilion reconstruction after a lip shave include cross-lip buccal mucosa flaps and tongue flaps.57–59

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 34-7). The
V-shaped excision design 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. A minimum of a three-layered closure comprising mucosa, muscle, and skin is necessary to avoid unesthetic 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 34-8). 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 (see Figure 34-5).

The need to reconstruct lip defects greater than one-third of the lip length led to the development of various circumoral flap advancement techniques. The most popular of these techniques includes the Karapandzic reconstruction flap (Figure 34-9). This flap consists of a transfer of the remaining lip tissue to reconstitute the lips and mouth opening. 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 where 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 flaps are particularly useful in repairing moderate lip defects of one-third the length of one lip. These techniques transfer a full-thickness segment of lip tissue into a defect on the opposite lip. Estlander and Abbe developed the most commonly used cross-lip flap repair techniques. The Abbe 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 34-10). 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 34-11). All cross-lip flaps are generally referred to as Abbe-Estlander type flaps.

The principle of the Abbe-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 34-12). Make a skin inci-
sion at the previously marked outline of
the Abbe-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 ver-
milion border up to the apex of the flap.
With extreme caution, perform an inci-
sion 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 mobi-
lization of the flap toward the vermilion
border proceeds, separate the musculature
of the upper lip bluntly with a hemostat,
and divide a little at a time small segments
of the muscle fibers 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° to fill the surgical
defect in the lower lip.

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 vas-
cular pedicle across the open mouth, and
perform the second-stage release 3 weeks
later (Figure 34-13). 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, then employ a nonbridged Est-
lander flap (Figure 34-14). This flap is cre-
ated as a single-stage procedure without
the need for secondary pedicle division.
The downside of this type of flap is the
development of a somewhat unnatural,
rounded commissure. The Abbe-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 Burrow’s tri-
angle is often useful for repairing lateral
defects of the upper lip (Figure 34-15).

Several techniques designed for lip
defects are too extensive for reconstruc-
tion using the Karapandzic or Abbe-
Estlander techniques. These tech-
niques 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 tis-
sues of the mental region, and the result-
ing defect is closed by lateral cheek flaps to
form a new lower lip (Figure 34-16). To set
back the commissure and to reduce the
incidence of a “fish-mouth” deformity,
excise 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 prima-
ry tumor. The base of this triangular exci-
sion extends from the commissure of the

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.

Typical appearance of the
cross-lip transfer flap at the time of suture
removal, demonstrated in this lower to upper
lip transfer.

At times an extension into the
labiomial crease may aid in closure of the
defect, as seen marked during this cross-lip
transfer flap.

In situations that do not require
reconstruction of the vermilion surface of the lip,
a cutaneous advancement flap may be appro-
priate, as in this case with Burrow’s triangles to aid
in advancement.
mouth, up to the nasolabial crease, depending on the width of the cheek flap to be mobilized (Figure 34-17A). After excising the triangular wedges, incise the mucosae from their inner aspect, except for the base, and shift the triangular flaps of the upper lip mucosa medially, along with the flaps (Figure 34-17B). Make a counter incision 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 (Figure 34-17C and D).

The main advantage of the Bernard flap is its ability to reconstruct almost the whole lower lip in a single-stage procedure. The main disadvantage is reducing 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 recently shown to be particularly useful is the composite radial forearm-palmaris longus free flap.71,72

**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 with 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 34-18). Thus, even if the patient has no palpable adenopathy (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. 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.73

**Treatment Results**

The cure rate for T1 and T2 lip cancers without regional metastasis is greater than 90% with surgery or radiation.4,48 The
5-year determinate survival is approximately 80%. 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 < 80%, and those that invade deep enough to involve the mandible have a cure rate of < 50%. The primary cause of failure is local recurrence, rather than regional node metastasis. Other adverse prognostic factors include poor histologic grade, tumor thickness > 6 mm, desmoplasia, stromal sclerosis, muscular invasion, and perineural invasion. Angiogenesis has not been shown to have prognostic significance. While TP53 mutations are seen in 50% of lip cancers, the clinical significance of this observation is unknown.

Generally, elective lymph node dissection in the N0 neck is reserved for advanced stage disease (stage III and stage IV). About 5 to 10% of patients with lip cancer will develop evidence of nodal involvement. 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. The risk of developing a metachronous lip cancer is estimated at about 20% by 10 years follow-up.

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.

References

Part 5: Maxillofacial Pathology


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Salivary Gland Disease and Tumors

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The salivary glands consist of three major paired glands (the parotid, submandibular, and sublingual) as well as numerous minor salivary glands, situated mostly in the oral cavity but also found in the pharynx, larynx, trachea, and sinuses. In the oral cavity 700 to 900 minor salivary glands are found, the majority of which are located at the junction of the hard and soft palates. These glands produce saliva, which functions as a lubricant for speech and swallowing, assists taste, has antibacterial and immunologic properties, and contains digestive enzymes.

The salivary glands are affected by many different disease processes, some of which are surgical in nature while others have a medical basis. Surgical diseases include tumors, stones, and cysts, whereas medical diseases include viral infections, autoimmune diseases, and sarcoidosis. This chapter will concentrate on the salivary gland diseases that are of most interest to the surgeon.

Tumors

Tumors of the salivary glands show a wide variety of pathologic types varying from benign to highly malignant. Salivary cancers are comparatively rare and comprise 3% of head and neck cancers, which in turn account for 3% of all malignancies.

These neoplasms will be discussed according to their histopathologic diagnosis and the surgical principles dictated by the site of the tumor.

Histopathology

The large variety of tumors that occur in the salivary glands make an exhaustive list of all types impossible in a chapter of this length. The most common epithelial salivary gland tumors will be reviewed in order to illustrate the fundamentals of management of salivary neoplasia.

Benign Tumors

Pleomorphic Adenoma

The pleomorphic adenoma is the most common benign salivary tumor at all sites. Approximately 80% of all pleomorphic adenomas (PSAs) occur in the parotid, and despite their slow growth they can become extremely large if neglected. This tumor is thought to arise from both salivary ducts and myoepithelial cells and is a true “mixed tumor.” Because of its derivation, histologically, many different patterns can occur, from cellular, glandular, and myxoid types to cartilagenous and even ossified forms. These features can be seen in different areas of the same tumor, accounting for its name, pleomorphic (Greek for many forms). The important feature from a surgical standpoint is the presence of a “pseudo capsule,” which contains outgrowths or pseudopodia of the tumor. Attempts at “enucleation” of the tumor from within its “capsule” will inevitably leave viable tumor cell nests and predispose the patient to multifocal recurrence. Some authorities believe that younger patients with pleomorphic adenomas have a higher chance of tumor recurrence and increased growth during pregnancy. Malignant change is rare and usually takes place in long-standing tumors, the most common type being carcinoma ex pleomorphic adenoma. Prognosis will depend on the type of malignancy and involvement of the capsule. Rarely, malignant change in both elements of the pleomorphic adenoma (ductal and myoepithelial) will occur giving rise to the carcinosarcoma or true mixed malignant (biphasic) pleomorphic adenoma. On rare occasions, an apparently histologically benign tumor will metastasize into the so-called benign metastasizing pleomorphic adenoma.

Warthin’s Tumors

This benign tumor is almost exclusively found in the parotid. It occurs mostly in men and is more common in smokers. It is thought to derive from salivary duct cells that are entrapped in lymph nodes during embryonic development. The tumor consists of large cystic spaces with a surrounding columnar epithelium and a
stroma of lymphocytes. Surgically these tumors may be multiple in one parotid gland or bilateral, or involve lymph nodes adjacent to the parotid gland.1,2

Hemangioendothelioma In children the most common cause of parotid mass is a hemangioma or hemangioendothelioma.3 These are benign tumors that may appear soon after birth and grow rapidly. Usually, conservative treatment while waiting for involution is recommended.

Malignant Tumors Mucoepidermoid Carcinoma Mucoepidermoid carcinoma (MEC) is the most common malignant salivary gland neoplasm in both adults and children, and the most common salivary gland cancer of the parotid and minor salivary glands. This tumor can be of low grade or high grade depending on its histology. Low-grade MECs have multiple macrocysts and abundant mucus-producing cells. High-grade varieties have multiple squamous cells and very few mucus-producing cells or cysts, and mucicarmine or periodic acid–Schiff stains may be needed to identify intracellular mucus to characterize this tumor. There are three cell types of MEC: mucus producing, intermediate, and squamous. The respective ratio of mucus-producing cells to squamous cells will determine the clinical aggressiveness of the tumor (see above). Low-grade MECs can be very slow growing and nonmetastasizing, and can generally behave like a benign tumor. High-grade MECs can exhibit aggressive growth and invasion resulting in widespread metastasis and death. High-grade tumors usually show increased pleomorphism and meiotic figures. High-grade lesions may metastasize to cervical lymph nodes or spread hematogenously to the lung, liver, and bone.

Adenoid Cystic Carcinoma Although this tumor is very slow growing, its relentless course, with repeated recurrence and metastasis via the blood stream, gives low 20-year survival rates.4 Adenoid cystic carcinoma is the most common malignancy of the submandibular gland and is the second most common salivary gland cancer overall. Three histologic types are seen: tubular, cribriform (the classic “Swiss cheese” pattern), and solid. The solid type has the worst prognosis, especially when areas of necrosis are present. The infiltrative nature of this lesion and the frequency of perineural involvement with spread along the nerve mandate wide resection margins. Perineural spread is a bad prognostic sign for both local recurrence and distant metastasis. Clinical and radiologic examination of this tumor frequently underestimate its true extent, and follow-up of 15 to 20 years is required as late recurrences occur.

Low-Grade Polymorphous Adenocarcinoma Low-grade polymorphous adenocarcinoma occurs almost exclusively in the minor salivary glands and is second only to mucoepidermoid carcinoma at these sites. It arises from terminal duct cells and is characterized by cytologically bland monotonous cells that can assume many different patterns (glandular, cribriform, and lobular) within the same tumor. Characteristically “Indian file” cells and perineural involvement are seen. Although this tumor behaves in a very low-grade manner, local recurrence will occur with inadequate excision.5 The important pathologic features seen from the surgeon’s viewpoint are frequent misdiagnosed on initial biopsy, due to the different patterns that may be sampled. Common misdiagnoses are adenoid cystic carcinoma, pleomorphic adenoma, and malignant pleomorphic adenoma. It is also important to be aware that the frequent presence of perineural involvement does not lead to a worse prognosis, as is the case for adenoid cystic carcinoma.

Site of Tumor

Parotid Gland The surgical principles of treating parotid tumors are dictated by the histopathology of the tumor and the need to preserve the facial nerve. Diagnostic imaging with computed tomography (CT) or magnetic resonance (MR) is desirable for superficial lobe tumors but is essential for suspected deep-lobe neoplasms, especially those with a parapharyngeal component. Since 80% of parotid tumors are benign and 80% of these are pleomorphic adenomas, a solitary mass in the parotid with no features of malignancy is most likely a PSA. Open biopsy of such a mass is therefore contraindicated as this will rupture the “capsule” and “seed” the PSA, increasing the complexity of subsequent surgery and chances of recurrence. Fine-needle aspiration biopsy (FNAB) for cytology is the preferred method of diagnosis.6 Clinically only one-third of malignant tumors will have symptoms or signs of malignancy, such as pain, ulceration of skin, facial nerve palsy, or metastatic cervical nodes.7 Thus virtually all parotid tumors will initially be treated as benign unless FNAB shows definite malignancy or there is clinical evidence of malignancy (Figure 35-1). The majority of tumors occur in the superficial lobe, and superficial lobectomy with preservation of the facial nerve has been the standard operation for many years. Recent minor modifications have included the use of a face-lift incision, the use of the superficial musculoaponeurotic system to prevent Frey’s syndrome, the use of flaps or alloplasts to augment defects, and the suggestion that “capsular dissection” without the need to remove the entire superficial parotid may be sufficient.8-10 Superficial lobectomy is suitable for benign and low-grade malignant tumors, and even in high-grade malignancies only branches of the nerve that are actually infiltrated will be sacrificed. If the nerve or portions of it have to be resected, immediate grafting is recommended. In deep-lobe tumors a total parotidectomy is performed, with the superficial lobe being dissected first to expose the nerve. Good margins with
surrounding normal salivary gland tissue are more difficult to obtain on deep-lobe tumors, which tend to be large as they are often detected late. In high-grade tumors, surrounding tissues such as skin, masseter, and mandible may require sacrifice, as dictated by the need to obtain clear margins. In these instances consideration should be given to neck dissection. Where clinically positive nodes are present, a modified radical neck dissection is usually the operation of choice.11 Where the patient is N0 clinically, but at high risk for occult nodal disease, a selective neck dissection of levels I to IV or levels II to IV is indicated. In high-grade tumors postoperative radiation therapy is usually indicated. Chemotherapy has not been shown to convey a survival benefit for these lesions.

Submandibular Gland In suspected submandibular neoplasms, CT imaging, MR imaging, and FNAB are all useful in the diagnostic work-up. Fifty percent of tumors will be malignant, adenoid cystic carcinoma being the most common. In benign neoplasms (PSAs) removal of the submandibular gland with an extracapsular dissection of the tumor and 2 to 3 mm of surrounding soft tissue is sufficient. For malignant tumors the minimum resection will be an en bloc removal of level I. If indicated the overlying platysma superficially and the mylohyoid muscle deeply will be excised. In most malignant tumors with N0 necks, the cervical incision necessary for removal of level I will dictate extending this to a supraomohyoid neck removing levels I to III. The adenoid cystic carcinoma does not usually metastasize via the lymphatics; instead it spreads hematogenously and neck dissection may not be indicated. The mandibular branches of the facial, lingual, and hypoglossal nerves are all in close relation to the submandibular gland. If these nerves appear to be involved by cancer, they should be traced until the nerve appears normal. After resection, frozen sections should be sent from the cut nerve trunk to confirm clearance, although “skip” lesions do occur. Radiation may be useful postoperatively.

Minor Salivary Glands The Palate The majority of minor salivary gland tumors occur at the junction of the hard and soft palates. In this location 50% are malignant, the most common being low-grade mucoepidermoid carcinoma followed by low-grade polymorphous adenocarcinoma. Coronal and axial CT scans with bony windows are helpful to demonstrate bone destruction and involvement of the sinuses or nasal cavity. Biopsy through the middle of the lesion is indicated as the overlying mucosa will be excised.12 In PSA, excision with a 5 mm margin is adequate. The periosteum is a good deep margin if the bone is uninvolved, as is usually the case with PSA (Figure 35-2). In low-grade lesions a 1 cm margin and similar approach
are used. Local flap reconstruction or the use of a palatal plate with subsequent secondary healing by granulation is used for reconstruction. Where bone invasion has occurred, as in adenoid cystic carcinoma or high-grade tumors, a partial maxillectomy will be required. In the case of adenoid cystic carcinoma, attention must be given to the greater palatine nerve, with frozen section clearance obtained. Cranial extension, orbital involvement, and infiltration posteriorly into the pterygoids will increase the extent of surgery and its morbidity, with a decrease in survival (Figure 35-3). Reconstruction is usually with an obturator, although primary maxillary reconstruction has been revisited with the development of interosseous implants and composite microvascular free flaps.

The Retromolar Fossa Although this is a relatively unusual site for minor salivary gland tumors, virtually 100% are malignant and are low-grade mucoepidermoid carcinomas. The surgeon should be aware that a cystic soft tissue mass distal to the third molar, with or without radiographic mandibular involvement, is unlikely to be a mucocele, and incisional biopsy should be undertaken to confirm the diagnosis.

Intrabony Tumors Although intrabony (central) salivary gland tumors are rare, the vast majority are malignant low-grade mucoepidermoid carcinomas. These are mostly seen in the third molar region of the mandible and are frequently multilocular. The tumors are often diagnosed radiologically as ameloblastomas, or odontogenic keratocysts. Resection with a 1 cm margin and sacrifice of the inferior alveolar nerve and overlying soft tissue in areas of perforation are required. Neck dissection is usually not necessary, but if the neck has been opened widely for mandibular resection a supraomohyoid neck dissection can be undertaken. A reconstruction plate is placed and either primary reconstruction with a fibular or deep circumflex iliac artery microvascular flap or secondary posterior iliac crest corticocancellous reconstruction may be used.

Other Intraoral Sites Interestingly, the proportion of benign to malignant tumors varies according to site, with virtually all upper lip tumors being benign and a higher proportion of lower lip tumors being malignant. Salivary gland neoplasms of the tongue and buccal mucosa tend to be malignant and require wide soft tissue dissection to obtain margins.

The Sublingual Gland Less than 1% of all salivary gland tumors occur in the sublingual gland but almost 100% are malignant. Surgical approach will be dictated by the histology and required access for margins. In most cases we have preferred a lip split and mandibulectomy to allow good visualization of the tumor, direct examination of the mandibular lingual cortical plate, and the ability to trace back the lingual nerve when necessary.

Obstructive Disease

Obstruction to the salivary glands is usually seen in the submandibular and parotid glands. It may be due to calcified stones (most common in the submandibular gland) or mucous plugs (most common in the parotid) or strictures of the duct. Stone formation is classically due to stasis of flow, infection, and alteration of the duct contents. Calcified stones are formed by the precipitation of calcium salts around a nidus of mucous plugs, epithelial cells, or microorganisms. Approximately 80% of sialoliths occur in the submandibular gland. Microliths in the minor salivary glands have been described.

As calcified sialoliths increase in size they may give rise to symptoms, especially when they are present in the duct. Classically the patient reports pain and swelling when eating or drinking or sometimes even from the smell of food (Figure 35-4). Examination of the gland may show a tender swelling with inability to milk saliva from the duct orifice.

Plain radiography is used to demonstrate calcified stones, the lower occlusal film for the submandibular gland, and an occlusal or periapical dental film held in the cheek for the parotid. Lateral oblique mandibular films or panoramic radiographs will show parotid duct stones and calcified stones in the hilum or glandular substance of the submandibular gland. CT
scans and ultrasonography have also been used. When a noncalcified (mucus plug) obstruction is suspected, sialography may demonstrate a filling defect (Figure 35-5). Acute infection should be managed with antibiotics prior to sialography. Treatment of the stone will depend on its location.

**Submandibular Gland**

**Anterior Duct** If the stone is palpable in the anterior floor of the mouth close to the orifice of Wharton’s duct, an intraoral approach may be used. Although the anterior duct is traditionally regarded as a line between the first molars, the floor of the mouth slopes downward following the mylohyoid muscle as the premolars are reached, and technical difficulty is increased as the stone is more distal (Figure 35-6). Initially a suture is passed behind the sialolith around Wharton’s duct to use as a traction suture, tenting the duct upward and preventing posterior displacement of the stone during surgical manipulation. An incision in line with the duct is made through the mucosa and dissection carried down to the duct. This is opened in its long axis allowing removal of the stone. The posterior suture is removed and the gland is milked or explored with a lacrimal probe to find other stones. The duct is sutured open to the edges of the mucosa (fish tailed) to prevent stricture.

**Posterior Duct** Stones in the posterior submandibular duct are much more technically difficult to remove intraorally, requiring general anesthesia, excellent light, and retraction, as well as the help of an assistant to push the gland upward into the mouth from extraorally. Even so, irritating bleeding can occur and the lingual nerve must be visualized and protected (see Figure 35-6).

**Stones in the Hilum or Gland**

When the stone is below the posterior edge of the mylohyoid muscle, removal of the gland is necessary. Although intraoral submandibular gland excision has been described, the potential for bleeding from branches of the facial vein and artery and possible scarring of the anterior pole of the gland to the mylohyoid muscle can make this a technically challenging and hazardous procedure. We believe that the conventional cervical approach gives the best access and is the safest procedure.

Under general anesthesia an approximately 5 cm incision is made over the submandibular gland at ½- to 2-finger breadths below the mandible. This incision should be parallel to the neck skin creases, not to the lower border of the mandible (Figure 35-7). The platysma is sectioned and the inferior pole of the submandibular gland visualized. The gland is exposed by subcapsular dissection at the inferior posterior pole. Blunt finger dissection will release the deep surface of the gland. The authors do not routinely tie the facial artery and vein at this stage as these can usually be dissected off the gland, although clipping...
multiple arterial branches to the gland can be tedious. The anterior pole of the submandibular gland is mobilized off the mylohyoid muscle, and in cases of chronic sialadenitis, sharp dissection may be necessary due to dense fibrosis. The superior pole of the gland is dissected in a subcapsular plane and the gland mobilized posteriorly. The posterior edge of the mylohyoid muscle is retracted to expose the lingual nerve and the branch to the gland is tied and sectioned (see Figure 35-7B). The submandibular duct is dissected superiorly into the floor of the mouth as far as possible, tied, sectioned, and the gland removed.

**Parotid Gland**

Most obstructive symptoms in the parotid gland are associated with noncalcified stones or mucous plugs. Although these can sometimes be removed with tweezers following duct dilatation or “milked” from the duct, they often cause repeated bouts of pain and swelling. Sialography is helpful in evaluating the extent of damage to the ductal architecture. Sialograms may show changes varying from mild sialectasis to gross dilatation of Stensen’s duct with loss of secondary and tertiary ducts (Figure 35-8). Sialograms are frequently helpful symptomatically, with cure or improvement in many patients. In advanced cases with no improvement, parotidectomy may be required.

**Stones in the Terminal Duct** Radiographically opaque stones at Stensen’s papilla can be managed intraorally in a similar manner to those of the anterior portion of Wharton’s duct. Following placement of a posterior traction suture, the duct is opened with an incision running in the long axis of the duct.

**Stones in the Posterior Duct** When the stone involves the extraglandular portion of the duct lateral to the buccinator muscle, both intraoral and extraoral approaches are described.16,17 The intraoral approach involves a Y-shaped mucosal incision, dissection through the buccinator muscle, and the use of a traction suture to pull the duct into the mouth. The extraoral approach requires the duct to be displaced laterally with a finger placed in the mouth, with blunt dissection down to the stone, avoiding the facial nerve.

**Parotid Gland Stones** Stones at the hilum of the gland or intraglandular stones usually require a parotidectomy if they are symptomatic. The facial nerve dissection may be challenging due to extensive fibrosis (Figure 35-9).

**Nonsurgical Approaches**

Miniature endoscopes have been used to visualize sialoliths and remove them with baskets.18 Lithotripsy has also been attempted either via endoscopes (intracorporeal) or extracorporeally. Intracorporeal lithotripsy uses shock waves produced by lasers, electrohydraulic sources, or a pneumoballistic source. In a review of 6 series of extracorporeal lithotripsy ranging from 33 to 104 stones, Escudier reported a stone-free range of 18.2 to 52.9% with residual fragments occurring in 47.1 to 81.8% of cases.19

**Mucoceles and Ranulas**

Mucoceles are mostly due to extravasation of mucus from a salivary gland, although a few are true retention phenomena. The most common site is the lower lip, due to trauma (usually following an accidental bite in a child). Mucoceles are simple to treat and they should not recur if the underlying damaged minor salivary gland has been removed. Following a vertical incision through the mucosa over the mucocele, a number of minor salivary glands are usually identified. As it may be impossible
to identify the damaged gland, all these minor glands should be removed before carefully suturing the mucosal incision.

Ranulas are large retention phenomena that occur in the floor of the mouth in relation to the sublingual gland. They may be large enough to elevate the tongue and interfere with speech and swallowing (Figure 35-10). Where dehiscence in the mylohyoid muscle occurs, the mucus can drain into the submandibular space as a “plunging ranula.” The treatment of ranulas has been reviewed at length in a classic paper by Catone. He concluded that definitive therapy was removal of the sublingual gland. Several large series have been reported comparing sublingual gland excision with so-called marsupialization, demonstrating 100% cure for gland excision and 43 to 63% cure for marsupialization.21,22

Despite this evidence some authorities still plead the case for marsupialization or “marsupialization with packing,” which they claim has a lower recurrence rate of 10 to 12%.23 We subscribe to the view that ranulas should be treated by sublingual gland excision.

An intraoral approach is made with an incision along the axis of the gland lateral to the ductal orifices. The submandibular duct is identified, either by dissection or following cannulation with a lacrimal probe. The gland is dissected in a subcapsular plane with meticulous hemostasis. At its posterior pole the lingual nerve is identified as it crosses the duct and is preserved. The sublingual gland is dissected from anteriorly, and the final excision is the posterior pole after visualizing the lingual nerve.

References
Fungal Disease of the Oral Cavity

Fungal diseases of the oral cavity can be classified as superficial or deep in relation to the primary tissue(s) involved in the infection. Most oral fungal infections are opportunistic in nature. Persons living in geographic areas endemic to one or more of these fungi may show immunologic reactivity to the surface antigens without having historic features of active disease. The deep fungi usually infect the lungs before dissemination to other organ systems, including the oral cavity. Deep fungal diseases, including histoplasmosis, coccidioidomycosis, blastomycosis, and cryptococcosis, present clinically as chronic proliferative ulcerated granulomatous tissue lesions that may be single or multiple and painful or asymptomatic. They may simulate clinical features of a malignant neoplasm.

Candidosis

Although numerous deep and superficial fungal diseases can involve the oral cavity, candidosis is by far the most common. The term candidosis is the correct nomenclature describing an infection with one of several species of Candida organisms. However, many publications use the term candidiasis to describe the same disease, even though the suffix “-iasis” is characteristically used to describe parasitic infections such as schistosomiasis or amebiasis.

One or more species of Candida can be found as a component of the normal oral flora in about 60% of healthy adults. The organism can exist in one of three states: the yeast form consisting of blastospores measuring 1.5 µm to 5 µm in diameter, elongated pseudohyphae, and chlamydospores measuring 7 µm to 17 µm in diameter. In its commensal state, the organism usually exists only as spores or pseudohyphae.

Candidosis is usually an opportunistic infection caused by a localized or systemic suppression of the immune system. Commonly recognized causes of candidosis include the use of broad-spectrum antibiotics, xerostomia, chronic diseases of the immune system, and therapy for malignant disease including chemotherapy or radiation.

Oral infections involving Candida species may appear as one of three clinical forms: acute, chronic, and mucocutaneous. Candidosis characteristically shows erythematous mucosa with or without overlying white plaques, which may be rubbed away with light abrasive pressure (Figure 36-1). The dorsum of the tongue usually shows diffuse patches of papillary atrophy (Figure 36-2). Occasional small or confluent ulcerations may be noted. Angular cheilitis is a prominent clinical feature of oral candidosis. Patients characteristically complain of an oral “burning” sensation. Denture-sore mouth (denture stomatitis) is a clinical term used to describe patients with mucosal erythema or inflammatory papillary hyperplasia, usually related to a localized candidosis under a removable prosthodontic appliance.

Clinical features of oral candidosis usually include foci of mucosal erythema, which is the result of inflammation and mucosal atrophy, areas of ulceration, and sometimes white pseudomembranous plaques, which are seen to consist of candidal pseudohyphae and spores if examined microscopically (Figure 36-3). These pseudomembranous plaques, although usually present in acute-onset cases of candidosis, are frequently absent in cases of chronic candidosis such as those related to prosthetic appliances. The lack of white pseudomembranes should not therefore preclude consideration of candidosis in cases of chronic mucositis. Candidosis has also been noted in lesions characterized by focal increases in keratinization such as lichen planus, focal keratosis with or
without dysplasia (leukoplakia), hairy tongue, hairy leukoplakia, and even squamous cell carcinoma. A recent study showed the presence of candidal hyphae and spores in 31% of biopsy specimens showing oral lichen planus. However, a possible cause-and-effect relationship between candidosis and increased keratinization of the epithelium is difficult to show.

Cytologic Preparations  Clinical diagnoses of oral candidosis are easily and quickly confirmed using exfoliative cytology studies. Cytologic specimens are prepared using a wooden tongue blade to scrape the oral mucosa of the involved areas; the exfoliated material is smeared onto a glass slide. The slide is air dried for 5 minutes and then fixed in ethanol (hair sprays with a high alcohol content can be used as fixatives in a clinical setting). The slides are stained with potassium hydroxide, periodic acid–Schiff modified for fungi, or any one of several other stains that delineate the fungal hyphae and spores. The infection can be further delineated as a species using cultures on Sabouraud dextrose or blood agar.

Oral candidosis is the most common diagnosis made in patients whose chief complaint involves a chronic nonspecific mucositis or burning sensation. Other diseases included in the clinical differential diagnosis include lichen planus, pemphigus, pemphigoid, and medication-related toxic mucositis. As stated above, candidosis may be a secondary component of other chronic oral diseases or localized epithelial thickening lesions.

Management  The initial management of oral candidosis following confirmation of the clinical impression with exfoliative cytology studies is the use of one or more antifungal agents. Of greatest therapeutic value in most patients is ketoconazole, administered in one 200 mg tablet daily for 10 to 14 days. If systemic factors contraindicate the use of ketoconazole, clotrimazole troches, administered in one 10 mg tablet dissolved in the oral cavity up to five times daily, or chlorhexidine in a 0.12% mouthrinse in a 5 to 10 mL dose twice daily NPO for 1 hour, are usually effective. Nystatin powder or cream may be used to line dentures in patients with denture sore mouth. Of vital importance is a review of the patient’s past medical history and current medical status in an attempt to identify the causative factors for this opportunistic infection. In patients who have no identifiable predisposing factors or if the predisposing factors are not correctable, multiple recurrences may be anticipated.

Median Rhomboid Glossitis  Although the early reports of median rhomboid glossitis suggested an origin from the tuberculum impar, many investigators now favor classification of this lesion as a localized candidosis (Figure 36-4). Although the exact cause-and-effect relationship is unclear, Candida spp are found in association with many of these lesions, and recent studies have shown the prevalence of median rhomboid glossitis to be higher in adults than in children, a finding contrary to the developmental theory of origin. The lesion appears clinically as an

FIGURE 36-1  A to C, Oral candidosis. White pseudomembranous plaques that can be removed with light abrasion involving buccal, lateral-glossal, and soft palatal mucosa.
erythematous patch, which may be roughly ovoid or rhomboid in shape, is asymptomatic, and is located on the dorsal midline of the tongue just anterior to the circumvallate papillae. The area is usually smooth and devoid of filiform papillae.

Unless the lesion is symptomatic, no treatment is indicated. For symptomatic cases, management with antifungal regimens is usually beneficial. The lesions are generally regarded as having no malignant potential.

Bacterial Infections of Oral Mucosa

Acute Necrotizing Ulcerative Gingivitis

Acute necrotizing ulcerative gingivitis is a rare and clinically painful ulcerative disease that presents with progressive necrosis of the interdental papillae, usually beginning in the mandibular incisor region. The interdental papillae necrosis may spread or remain localized. The necrotic papillae are usually covered with a pseudomembrane of necrotic epithelial cells, plaque, and microbial organisms. The patient may have systemic signs and symptoms including fever and regional lymphadenopathy. Patients have a characteristic rancid halitosis caused in part by the presence of necrotic material in the oral cavity.²³

The probable cause of this disease involves a symbiotic infection by two bacteria, a fusiform bacillus and a spirochete. However, inoculation of these bacteria into healthy tissues does not produce disease, and because moderate numbers of these organisms can be found in otherwise clinically healthy mouths, other factors such as stress and smoking, both of which can affect the host’s immune system, have been implicated as causative factors.

Local débridement by scaling and curettage, sometimes under local anesthesia, usually brings about a marked relief of symptoms. A therapeutic dose of an antibiotic such as tetracycline may be indicated for patients with extensive disease or evidence of regional lymph node enlargement. The use of topical antiseptics such as chlorhexidine or diluted hydrogen peroxide is of value for initial management of the lesions. The lesions usually heal within 2 to 3 weeks, and the interdental papillae often regenerate, seldom requiring gingival surgery. Improved oral hygiene through use of a soft-bristled toothbrush and floss is the best long-term therapy and is aimed at recurrence prevention.

Syphilis

Syphilis is a venereal infection that has been documented extensively, beginning in about the fourteenth or fifteenth century. Before the introduction of penicillin in the early 1940s, over 500,000 new cases were documented in the United States each year. The Centers for Disease Control and Prevention reported 6,657 cases of primary and secondary syphilis in the United States in 1999.⁴

The disease is caused by the spirochete Treponema pallidum and is acquired by contact with an active lesion. The spirochete can also be transmitted by transfused blood, and it crosses the placental barrier from maternal to fetal circulation.

In cases involving transmission from an active lesion, the site of infection forms a chancre or ulceration, which is usually accompanied by regional lymphadenopathy. The ulcer and lymphadenopathy usually persist for 3 to 10 weeks and then resolve spontaneously. This initial disease manifestation constitutes primary syphilis. Assuming no treatment is rendered, secondary syphilis develops following a latency period of several weeks. In this stage the now widely disseminated disease causes fever, malaise, a maculopapular rash, and multiple ulcerations or mucous patches on mucosal surfaces. Broad-based, proliferative slightly raised ulcerations known as condyloma lata may occur during secondary syphilis. These lesions also persist for 5 to 10 weeks and then resolve without treatment. If the patient is still untreated, several recurrences of the manifestations of secondary syphilis may occur or the disease may enter a prolonged latency period lasting months or years.⁵⁻⁷

Fortunately tertiary syphilis develops in only a few patients. There are many manifestations of tertiary syphilis, owing to the extensive involvement of organ systems. Central nervous system involvement can present as a generalized paralysis or tabes dorsalis. Inflammation of the circulatory system can result in aneurysms, especially in the aorta. Intraoral manifestations include granulomatous proliferations known as gummas, as well as a poorly understood generalized glossitis.
The diagnosis of syphilis is usually made following serologic studies, including Venereal Disease Research Laboratory and fluorescent treponemal antibody absorption tests. The treatment of choice for syphilis remains 2.4 million U of benzathine penicillin. For patients allergic to penicillin, erythromycin or tetracycline may be substituted.

**Gonorrhea**

Gonorrhea is currently the most widespread human bacterial infection in the world and is caused by *Neisseria gonorrhoeae*, a gram-negative diplococcus.

Transmission is usually venereal, involving genital, oral, or pharyngeal mucosa. The incubation period is about 1 week with the initial features ranging from no evidence of disease to mucosal ulcers and regional lymphadenopathy. These features, although reported in the oral cavity, are rare compared with the much more common pharyngeal infection. Therefore, in patients who present with chronic aphthous-like ulcerations and erythema predominantly involving the pharyngeal mucosa rather than the oral mucosa, a gonorrheal infection should be part of the clinical differential diagnosis. The microscopic features are nonspecific, and the clinical features of the disease seldom indicate a biopsy. The diagnosis is based on demonstration of the organism in culture media or through the use of immunofluorescent antibody techniques.8,9

The treatment of choice for gonorrhea continues to be penicillin. Occasional penicillin-resistant strains are noted during sensitivity cultures and require management with alternative antibiotics.

**Pigmented Lesions of Oral Mucosa and Skin**

Pigmented lesions of oral mucosa and skin can be divided into generalized lesions, which are diffuse and multifocal, and localized lesions involving one or several locations.

**Generalized Pigmentations**

Some of the common causes of generalized pigmentations are listed in Table 36-1.

The most common type of generalized pigmentation is hereditary or racial. The pigmentation is diffuse, symmetric, and most commonly located on the gingiva and labial mucosa. Pregnancy and ingestion of oral contraceptives may produce melanin pigmentation called chloasma or melasma. Pigmented macules occur on the labial mucosa, forehead, malar prominences, and around the eyes and lips.10–13

Smokers sometimes have melanin pigmentation of the attached gingiva. Numerous medications may cause pigmentation of skin and/or oral mucosa. Antimalarial drugs such as quinine, chloroquine, and amodiaquine may cause pigmentation in approximately 25% of patients taking them for > 3 to 4 months. Cancer chemotherapeutic agents such as busulfan, cyclophosphamide, and bleomycin have been reported to cause pigmentation, primarily of skin. Hydantoins may produce facial pigmentation resembling chloasma. Minocycline may cause pigmentation of skin, bones, teeth, oral mucosa, and the thyroid. Pigmentation secondary to heavy metals is due to deposition of metals in the skin and oral mucosa. This type of pigmentation is not commonly seen today because of their decreased value as therapeutic agents.

Peutz-Jeghers syndrome is characterized by multiple pigmented macules of the hands and feet; areas surrounding the mouth, eyes, and nose; and intraorally on the buccal mucosa, labial mucosa, gingiva, and palate. Multiple hamartomatous polyps are present in the gastrointestinal tract. Patients with this syndrome have an increased incidence of cancer both within and outside the gastrointestinal tract.

Patients with Addison’s disease have increased pigmentation of the skin, lips, gingiva, buccal mucosa, and tongue. Systemic manifestations are prominent and include malaise, weakness, nausea, vomiting, diarrhea, weight loss, and hypotension.

Neurofibromatosis is a relatively common autosomal dominant inherited syndrome. Virtually all patients have six or more brown cutaneous macules > 1.5 cm in diameter known as café au lait spots. Numerous freckles 2 or 3 mm in diameter are often present in the axilla and other intertriginous regions. Other features of neurofibromatosis include multiple neurofibromas, central nervous system tumors, seizures, intellectual handicap, and speech impediments.

**Table 36-1: Generalized Pigmentations of Skin and Oral Mucosa**

<table>
<thead>
<tr>
<th>Category</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hereditary (racial)</td>
<td>Pregnancy (chloasma, melasma)</td>
</tr>
<tr>
<td>Smoking (smoker’s melanosis)</td>
<td>Medications</td>
</tr>
<tr>
<td>Antimalarials</td>
<td>Oral contraceptives</td>
</tr>
<tr>
<td>Busulfan</td>
<td>Cyclophosphamide</td>
</tr>
<tr>
<td>Bleomycin</td>
<td>Phenytoin</td>
</tr>
<tr>
<td>Phenothiazines</td>
<td>Minocycline</td>
</tr>
<tr>
<td>Heavy metals</td>
<td>Bismuth</td>
</tr>
<tr>
<td>Lead</td>
<td>Silver</td>
</tr>
<tr>
<td>Gold</td>
<td>Arsenic</td>
</tr>
<tr>
<td>Mercury</td>
<td>Syndromes and systemic diseases</td>
</tr>
<tr>
<td>Peutz-Jeghers syndrome</td>
<td>Addison’s disease</td>
</tr>
<tr>
<td>Neurofibromatosis</td>
<td>Albright’s syndrome</td>
</tr>
</tbody>
</table>

Neurofibromatosis is a relatively common autosomal dominant inherited syndrome. Virtually all patients have six or more brown cutaneous macules > 1.5 cm in diameter known as café au lait spots. Numerous freckles 2 or 3 mm in diameter are often present in the axilla and other intertriginous regions. Other features of neurofibromatosis include multiple neurofibromas, central nervous system tumors, seizures, intellectual handicap, and speech impediments.

**Localized Pigmented Lesions**

Localized pigmented lesions can be divided into four classes based on their cause and clinical features: (1) melanocytic,
The ephelis or freckle is a Melanoma is a malignant
oral melanotic macule that occurs on sun-exposed areas of skin. It appears in childhood and darkens in the summer and fades during the winter. Microscopically, the ephelis shows increased melanin in the basal cell layer of the epidermis but no increase in the number of melanocytes. Ephelides are not premalignant and require no treatment once the diagnosis is established.

**Melanocytic Lesions** Melanocytic lesions are due to increased amounts of melanin pigment in the tissue and/or a proliferation of melanocytes or nevus cells. Melanocytic lesions are gray, brown, black, or blue and do not blanch on pressure.

**Ephelides** The ephelis or freckle is a small circumscribed brown or black macule that occurs on sun-exposed areas of skin. It appears in childhood and darkens in the summer and fades during the winter. Microscopically, the ephelis shows increased melanin in the basal cell layer of the epidermis but no increase in the number of melanocytes. Ephelides are not premalignant and require no treatment once the diagnosis is established.

**Oral Melanotic Macules** The oral melanotic macule is an oral mucosal pigmentation with similar microscopic features to ephelis. The lesions are well-circumscribed flat macules that are gray, brown, blue, or black. Most are 1 to 3 mm in diameter. The most common locations are the vermilion border of the lip, gingiva, and buccal mucosa. They are often confused clinically with tattoos and nevi.

Microscopically, oral melanotic macules show increased melanin in the basal cell layer, lamina propria, or both. The cause of oral melanotic macules is unknown, although they may be an atypical manifestation of physiologic pigmentation because the microscopic appearance is identical to racial pigmentation. These macules do not recur or undergo transformation into melanoma, but they may be difficult to distinguish it from nevi or melanoma in situ. They should be excised for microscopic diagnosis or checked frequently.

**Lentigo Simplex** Lentigo simplex is a macular brown-to-black lesion that is not associated with sun exposure and may occur on any skin surface. It is not premalignant and requires excision only for microscopic diagnosis. Microscopically, lentigo simplex demonstrates an increased number of melanocytes in the basal cell layer, an increased amount of melanin in the melanocytes and the basal keratinocytes, and elongation of the rete ridges. Macrophages containing melanin (melanophages) are present in the upper dermis.

**Nevi** A nevus is a proliferation of nevus cells or melanocytes. Nevi are extremely common lesions on skin but are relatively uncommon on oral mucosa. Most nevi of skin are absent at birth and appear in childhood. They progress through a series of stages, and then decline in number with increasing age. Nevi begin as junctional nevi, with nests of nevus cells at the dermal-epidermal junction. Compound nevi demonstrate nevus cell nests in the epidermis and upper dermis. Intradermal nevi have nevus cell nests only in the dermis.

Clinically, junctional nevi are flat pigmented macules. The compound nevus is slightly elevated and sometimes has a papillomatous surface. Intradermal nevi are dome shaped and pedunculated. Compound and intradermal nevi may not be pigmented. Normal nevi are round to oval, have a smooth border, and are sharply demarcated from the surrounding skin. They are most commonly found on sun-exposed skin above the waist.

Dysplastic nevi are precursors to melanoma. They may occur sporadically or in an autosomal dominant inherited syndrome in which they are quite numerous. Dysplastic nevi have irregular borders that are indistinct and fade into the surrounding skin. They may demonstrate a mixture of colors, including tan, dark brown, and pink. Dysplastic nevi are typically larger than normal nevi.

It is not necessary to remove normal cutaneous nevi unless they are irritated by clothing. Since dysplastic nevi have an increased potential for developing into melanoma, they should be removed. In patients with numerous dysplastic nevi, the lesions should be closely monitored and excised if they change.

Nevi of the oral mucosa are usually between 1 and 6 mm in diameter and are most commonly located on the hard palate and buccal mucosa. They are occasionally nonpigmented. The majority of oral nevi are raised and thickened, but a significant number may be flat.

Microscopically, the majority of oral nevi have been reported as intramucosal, but blue, compound, and junctional nevi also occur. Because of the small number of reported cases of oral nevi, their potential for evolving into melanoma is not known. Because of this, lesions in which nevus is part of the clinical differential diagnosis should be completely excised.

**Melanomas** Melanoma is a malignant neoplasm of nevus cells or melanocytes. Microscopically, melanomas begin at the dermal-epidermal junction and then may demonstrate two different patterns of growth. In radial growth, or melanoma in situ, melanoma cells grow laterally along the dermal-epidermal junction but do not invade the underlying dermis. A melanoma may remain in the radial growth phase for years, and during this time it does not metastasize. During vertical growth the melanoma cells grow into the dermis and are capable of invading vascular channels and nerves and metastasizing.

Microscopically, melanoma cells are described as epithelioid or spindle shaped. Epithelioid cells are round to cuboidal and form nests. The spindle cells are elongated and do not form nests. The tumor cells demonstrate nuclear pleomorphism, anaplasia, and mitotic figures. The amount of melanin within tumor cells is variable. The Fontana-Masson stain demonstrates melanin in some of the amelanotic-appearing tumor cells. The dopa reaction is more reliable for
Vascular lesions are

A varix is a dilated vein. It occurs on both exposed and unexposed surfaces of elderly persons. It may remain in the radial growth phase for 10 to 15 years before progressing to invasive lentigo maligna melanoma, indicated by the development of nodularity.

Superficial spreading melanoma constitutes about 70% of cutaneous melanomas. It occurs on both exposed and unexposed surfaces, most commonly on the upper back in men and lower legs in women. This melanoma has irregular borders and great variation in color within one lesion, including tan, brown, black, pink, blue, and white areas (Figure 36-5). It often demonstrates nodularity and ulceration. Superficial spreading melanoma remains in the radial growth phase for a shorter time than does lentigo maligna melanoma and thus has a poorer prognosis.

Acral-lentiginous melanoma is the most common melanoma in black persons. It occurs primarily on the palms, soles, and in association with nails. It can metastasize early.

Nodular melanoma presents as a rapidly growing darkly pigmented nodule that is often ulcerated. It is not associated with sun exposure. Since it grows vertically from the beginning, it is often deeply invasive by the time it is diagnosed.

Treatment of cutaneous melanoma usually consists of wide surgical excision. Chemotherapy is sometimes used, but radiation therapy has not proven effective. Incisional biopsy is not thought to cause metastasis of melanomas.

The most important prognostic factor for cutaneous melanoma is the thickness of the lesion. One study reported a 10-year survival rate of 99.5% for patients with melanomas < 0.76 mm thick, as opposed to 48% survival for those with melanomas 3 mm and greater in thickness.23

Melanoma of oral mucosa is a rare neoplasm most commonly located on the hard palate and maxillary gingiva of patients > 50 years of age. About one-third of patients have preexisting melanosis, or macular hyperpigmentation, which probably represents radial growth of the lesion. This may be present for years before vertical growth occurs. At the time of diagnosis, many oral melanomas are large, ulcerated, and have caused bony erosion. A significant number of tumors are amelanotic.

Treatment of oral melanoma is wide local excision with or without lymph node dissection. The prognosis is quite poor. The 5-year survival in one series was only 13%.23 This may be due to the advanced stage of oral melanomas at the time of diagnosis.

**Vascular Lesions** Vascular lesions are due to increased numbers of blood vessels, or blood vessels of increased diameter. These lesions are compressible, blanch on pressure, and are red, blue, or purple.

**Hemangioma** Hemangioma is a proliferation of blood vessels that is usually congenital and may regress spontaneously. It is commonly found on the skin and in the oral cavity.

Hemangioma of skin may present as a soft tissue enlargement or a flat surface lesion. Nevus flammeus, or port-wine stain, is a red-to-blue macule present at birth. Sturge-Weber syndrome (encephalotrigeminal angiomatosis) includes congenital port-wine stain in the distribution of the trigeminal nerve, hemangiomas of the leptomeninges, and ipsilateral hemangiomas of the face, skull, jaws, and oral cavity. The hemangiomas often contain calcifications and may result in seizure disorders and other neurologic problems.

Hemangiomas of the oral cavity are compressible red or blue soft tissue enlargements that blanch on pressure. They present most commonly in the lips, tongue, and buccal mucosa.

Microscopically, hemangiomas are classified as cavernous or capillary. Cavernous hemangiomas consist of large vessels lined with a single layer of endothelial cells, whereas capillary hemangiomas contain numerous smaller vessels. During their period of growth, capillary hemangiomas demonstrate marked endothelial proliferation and only a few capillary lumina.

Hemangiomas are unencapsulated lesions and can be difficult to remove surgically. Other treatment modalities include sclerosing agents, cryotherapy, and laser surgery. Treatment is not recommended unless lesions are a functional or cosmetic problem.

**Varix** A varix is a dilated vein. It occurs most commonly on the lip, buccal mucosa, and ventral surface of the tongue. It increases in frequency with increasing age.

The typical varix is blue, compressible, and blanches on pressure. A thrombosed varix is firm to palpation, does not blanch, and resembles a nevus.
A hematoma is a blood blister or a circumscribed pool of blood outside of a vessel. It is typically caused by trauma and is most commonly found on the buccal mucosa along the occlusal plane. It appears blue to purple and is compressible to palpation. A hematoma requires no treatment and resolves spontaneously in several weeks.

**Kaposi’s Sarcoma** Kaposi’s sarcoma is a malignancy of endothelial cell origin that occurs in three settings. It was first described as a disease involving the skin of the distal portion of the lower extremities in elderly males of Mediterranean or Jewish origin. It is also endemic in black African children and adults. The African form involves viscera and lymph nodes as well as skin. Recently it has become a common lesion in patients with immunosuppression secondary to organ transplantation or human immunodeficiency virus (HIV) infection.

Kaposi’s sarcoma frequently involves the oral cavity, especially in patients with HIV infection. Oral lesions are most common on the hard palate and gingiva. The lesions may be single or multiple, flat or exophytic, and red, blue, or brown. The exophytic lesions blanch on pressure.

The microscopic appearance of early lesions of Kaposi’s sarcoma resembles granulation tissue. Increased numbers of dilated capillaries and a chronic inflammatory infiltrate are present. Advanced lesions have vascular and spindle cell components. The vascular channels are lined with prominent endothelial cells. Strands of pleomorphic spindle cells line narrow slits containing erythrocytes. Extravasated erythrocytes and hemosiderin in the stroma help distinguish Kaposi’s sarcoma from fibrosarcoma.

Treatment of Kaposi’s sarcoma includes radiation therapy, surgery, and/or chemotherapy. African patients and patients with Kaposi’s sarcoma secondary to immunosuppression have a poor prognosis. The disease causes death in 10 to 20% of elderly males with the disease.

**Lesions Owing to Extravasated Blood** Because these lesions are due to the presence of blood outside of blood vessels, they do not blanch on pressure.

**Hematoma** A hematoma is a blood blister or a circumscribed pool of blood outside of a vessel. It is typically caused by trauma and is most commonly found on the buccal mucosa along the occlusal plane. It appears blue to purple and is compressible to palpation. A hematoma requires no treatment and resolves spontaneously in several weeks.

**Ecchymosis and Petechiae** An ecchymosis, or bruise, is caused by diffuse bleeding into the tissue secondary to trauma. It is not palpable. It is initially blue but evolves through many color changes before resolving.

Petechiae are multiple discrete round hemorrhagic spots < 2 mm in diameter. They are more reddish than are ecchymoses or hematomas. Petechiae are a result of capillary bleeding. They may be associated with a viral disease or a blood dyscrasia.

**Tattoos** Tattoos are the most common oral pigmentation. They are the result of intentional or accidental implantation of foreign material, such as amalgam, graphite, ink, or metal, into the skin or oral mucosa. A tattoo on the hard palate is often a result of a child falling on a pencil held in his or her mouth and pushing graphite into the tissue. Amalgam tattoo is usually seen on the gingiva, alveolar mucosa, buccal mucosa, and floor of the mouth.

The most common presentation of a tattoo is an asymptomatic flat nonthickened blue-to-black pigmentation. Occasionally, however, a tattoo may be thickened owing to fibrosis or may enlarge because of phagocytosis of the foreign material by macrophages or incorporation of the material into collagen fibers. Rarely, the foreign material may incite a foreign body granuloma with multinucleated giant cells and macrophages. Radiographs may be helpful in detecting foreign material in the tissue, but not all foreign material can be visualized radiographically.

**Excision of a tattoo is necessary only when a nevus or melanoma is included in the clinical diagnosis.**

**Vesicular, Ulcerated, and Erythematous Lesions**

Numerous diseases cause vesicles and/or ulcers of the oral cavity. Some diseases such as herpes simplex and aphtous ulcers are important because they are frequently encountered in practice. Other diseases such as epidermolysis bullosa and pemphigus are serious life-threatening diseases.

Because vesicles are so transient in the oral cavity, it is usually impossible to determine if an ulcer was preceded by a vesicle. If a vesicle was present, then aphthous ulcers, ulcers of infectious mononucleosis, traumatic ulcers, and ulcers owing to bacteria can be excluded from the clinical diagnosis.

A thorough history should be obtained from patients with vesicular/ulcerative diseases and should include the following questions:

1. How long have the lesions been present?
2. Are the lesions recurrent?
3. If yes, how often do they recur?
4. Do they recur in the same locations?
5. Have you noticed vesicles?
6. Have you noticed lesions on the skin, eyes, or genitals?
7. Have you been aware of fever, malaise, and lymphadenopathy in association with the lesions?
8. What medications do you take?

Since there are a large number of diseases that can cause vesicles or ulcers, one of the convenient ways to classify the diseases is by their cause. The discussion of vesicular, ulcerated, and erythematous lesions below is arranged by the cause of lesions, for example, hereditary, viral, or autoimmune.

**Hereditary Diseases**

**Epidermolysis Bullosa** The most important hereditary vesicular/ulcerative disease
is epidermolysis bullosa (EB). There are at least 18 types of EB including some that are not inherited.

The current classification of EB is based on where the split that forms the blisters occurs, inheritance, and clinical findings. Intraepidermal forms are non-scarring and have autosomal dominant or X-linked inheritance. The split occurs within the epithelium and is associated with defective tonofilaments of the basal squamous epithelial cells. Junctional forms of EB have autosomal recessive inheritance and demonstrate skin atrophy. The split occurs within the basement membrane and is due to decreased numbers of hemidesmosomes and tonofilaments. Dermal forms have autosomal dominant or recessive inheritance with atrophy and scarring of skin and mucosa. The split occurs in the upper dermis or lamina propria owing to defects in anchoring fibrils associated with the basal lamina. Typing of patients requires the use of electron microscopy, immunofluorescence, and immunohistochemistry.

EB simplex Koebner type is an intraepidermal form. Blisters mainly involve the feet, hands, and neck. They begin in infants and are exacerbated by heat. Abnormal nails are sometimes present. Oral blisters are occasionally seen, but the teeth are normal. The disease improves at puberty and is compatible with a normal life span.

EB atrophicans generalisata gravis Herlitz type is a junctional form with autosomal recessive inheritance. Blisters begin within a few days after birth and involve the hands and feet, followed by the trunk, face, and scalp. The nails are lost or dystrophic. Death within the first few months of life is common. Oral blisters and ulcers are found in almost all patients. Enamel is hypoplastic, pitted, and extensively involved with caries.

EB dystrophica Cockayne-Touraine type is a dermal form with autosomal dominant inheritance characterized by blisters of the ankles, knees, hands, elbows, and feet that produce scars. Milia (epidermal cysts) are common. Nails are thick and dystrophic. Onset is birth to 5 years of age, and the condition improves with age. Some patients have oral bullae.

Another dermal type is EB dystrophica Hallopeau-Siemens. It has autosomal recessive inheritance. Blisters are present shortly after birth and may involve any skin surface. Scars form and cause contraction. Formation of a clawhand and/or mitten-like hand are common. Nails are dystrophic or absent. The larynx, pharynx, and esophagus may be involved. Oral bullae and scarring may result in diminished oral opening, ankyloglossia, tongue atrophy, loss of buccal and vestibular sulci, and perioral stricture. Teeth have hypoplastic enamel, delayed eruption, and retention.

EB bullosa acquisita is a noninherited type that begins in adulthood. Blisters form in areas of trauma. Oral lesions have been reported but are rare.

EB is a disease that cannot be cured. The treatment is supportive and symptomatic and includes corticosteroids and antibiotics to fight secondary infections.

Viral Infections

The majority of viral infections are subclinical and asymptomatic. We know of their existence because of the development of antibodies in the patients. Symptomatic viral vesicular and ulcerative diseases often have systemic manifestations of malaise, fever, tender lymphadenopathy, and lymphocytosis. They generally have an acute onset and a vesicular stage, with the exception of infectious mononucleosis. Multiple lesions are present.

The herpesvirus family consists of herpes simplex virus (HSV) types 1 and 2, varicella-zoster virus, Epstein-Barr virus (EBV), and cytomegalovirus. Herpesviruses can assume a latent state in the patient. Cytomegalovirus is important in neonates and immunocompromised patients; it is not discussed further in this chapter.

HSV The primary infection with HSV may occur in seronegative patients of any age and results in acute herpetic gingivostomatitis. The patient experiences the abrupt onset of malaise, fever, and tender cervical lymphadenopathy. Multiple vesicles and ulcers can involve any oral mucosal surface and are accompanied by gingival swelling and erythema. The fluid-filled vesicles contain numerous virions and are infectious. The mouth can become extremely painful, resulting in difficulty eating and drinking (Figure 36-6).

After primary infection of the oral mucosa, HSVs travel centripetally along peripheral nerves to nerve cell bodies of the trigeminal ganglion. The viruses remain latent in the ganglion. Reactivation of the latent virus causes transport of viral
genomes to the epithelial surface, where replication occurs. Recurrent lesions may result. The most important factors associated with recurrent lesions are ultraviolet radiation, immunosuppression, and local trauma. With regard to immunosuppression, patients with defects in cell-mediated immunity have herpes infections that are more frequent and severe.\textsuperscript{28–31}

The vesicles and ulcers of recurrent (secondary) herpes occur in small clusters on the lip, gingiva, and hard palate, and they tend to recur in the same location. The lesions are often preceded by a prodrome of tingling, pain, or numbness in the area. Systemic manifestations are not present.

Recurrent herpetic lesions are often confused with aphthous ulcers. They occur on the lip and keratinized oral mucosa, whereas aphthae occur on nonkeratinized mucosa. Recurrent herpetic lesions consist of multiple small ulcers in a group; aphthae consist of one to several larger widely distributed ulcers.

Herpes simplex infection of the finger is called herpetic whitlow (Figure 36-7). The primary infection presents abruptly with edema, erythema, vesicles, and pain in the infected finger, often accompanied by fever and axillary and epitrochlear lymphadenopathy. The lesions may recur.

Either HSV-1 or -2 can infect the oral mucosa and skin. HSV-1 has a predilection for oral mucosa and skin outside of the genital area, whereas HSV-2 prefers the genital region. Genital HSV-1 infections and oral HSV-2 infections have a greatly decreased incidence of recurrence.

The diagnosis of mucocutaneous herpes is usually apparent on the basis of clinical features, so biopsy is rarely done. Microscopic examination of a fluid-filled herpetic lesion demonstrates an intraepithelial vesicle with marked acantholysis. The epithelial cells have swollen homogenous eosinophilic cytoplasm, known as ballooning degeneration, and one or multiple nuclei. Inclusion bodies may be seen in the nuclei of balloon cells as eosinophilic structures surrounded by a clear halo. Cytologic preparation of a fluid-filled vesicle can also demonstrate multinucleated epithelial cells, and the diagnosis can be augmented by using immunoperoxidase techniques to show antibodies to HSV (Figure 36-8). The diagnosis can also be confirmed by isolating the virus in tissue culture.

Lesions of primary and recurrent herpes resolve spontaneously in 10 to 14 days, and treatment is often unnecessary. When treatment is required, acyclovir is the current drug of choice. Acyclovir inhibits viral replication but has no effect on normal host cell function. However, it does not prevent or eliminate the latent viral state.

Acyclovir is very useful in the treatment of herpes simplex infections in immunocompromised patients. It has been reported to decrease the duration of viral shedding from lesions, the duration of pain, the time to scabbing, and the time to healing of lesions. It can reduce the number of recurrences, but infection can recur after the medication is discontinued.

Acyclovir can decrease viral shedding, time to healing, new lesion formation, and duration of symptoms in primary genital HSV infections. Primary oral herpes would be expected to respond in a similar manner, but the medication must be administered during the first 3 days.

The use of topical acyclovir in healthy patients with recurrent herpes labialis has given conflicting results. To have any effect, the medication must be used during the prodrome, or within the first few hours after onset of lesions. Topical sun-blocking agents are useful in reducing the frequency of recurrences of herpes labialis.

In summary, acyclovir is most helpful in the treatment of herpes simplex infections in immunocompromised patients and in patients with frequent or severe recurrences. It appears to have little value in healthy patients with infrequent minor recurrences of herpes labialis.

Varicella-Zoster Virus The primary infection with varicella-zoster virus causes varicella, or chickenpox. Varicella typically has mild systemic manifestations accompanied by papules, vesicles, and ulcers on the skin and mucosa. Successive crops of lesions begin on the trunk and move to the face and extremities. Lesions in various stages are present at the same time and are quite pruritic. Vesicles and ulcers resembling primary herpes sometimes occur on oral mucosa.

Therapeutic management for varicella is symptomatic and is aimed at reducing the pruritus. Antihistamines and topical lotions are helpful in this respect. Varicella typically has a mild clinical course, and complications are rare, except in neonates,
the elderly, and immunocompromised patients. Complications include bacterial infections of skin, encephalitis, Reye’s syndrome, and pneumonia.

Infection with the varicella-zoster virus results in a latent state, as in herpes simplex. The recurrent disease is called herpes zoster, or shingles. Reactivation of varicella-zoster virus is not as common as with HSV, except in elderly or immunocompromised patients.

Zoster has a prodrome of pain, burning, or paresthesia, followed by grouped vesicles on an erythematous base. The lesions are unilateral and follow the distribution of a peripheral sensory nerve. They are most common on the trunk and in the distribution of the trigeminal nerve (Figure 36-9). Oral lesions can have a painful prodrome that mimics a toothache in some cases. The lesions in zoster resolve in several weeks, but severe pain in the nerve distribution (postherpetic neuralgia) can persist for weeks to months after the lesions have resolved. The prevalence and duration of pain increases with age. Involvement of the facial nerve can cause Bell’s palsy.

The microscopic features of tissues infected with varicella-zoster are identical to those infected with herpes simplex.

Valacyclovir has been shown to be of some value in the treatment of zoster when the drug is started within the first few days of onset of infection.

**EBV** The EBV causes infectious mononucleosis and is also associated with hairy leukoplakia, Burkitt’s lymphoma, nasopharyngeal carcinoma, and lymphoblastic leukemia. EBV infects B lymphocytes and salivary glands and persists within these tissues for the lifetime of the host. The ability of EBV to reactivate depends on the competency of the cellular immune system.

Infants and children infected with EBV usually have an asymptomatic course, but about one-half of infected adolescents and adults develop acute infectious mononucleosis. The clinical features include malaise, fever, pharyngitis, and lymphadenopathy of cervical, axillary, and inguinal chains. Splenomegaly, hepatomegaly, and hepatitis with abnormal liver function tests may be present. Occasionally an erythematous skin rash is seen.

Ulcers may involve the oral mucosa, but a vesicular stage does not occur. The ulcers are secondary to decreased host resistance and appear after the systemic manifestations. Petechiae occur on the palate in about one-third of patients. The oropharynx is inflamed and may be ulcerated. Laboratory features of acute infection include an increase in relative and absolute numbers of lymphocytes and monocytes exceeding 50%, with >10% atypical lymphocytes in the peripheral blood. The atypical lymphocytes are called Downey cells, and they have indented or horseshoe-shaped nuclei and abundant basophilic foamy cytoplasm. The total leukocyte count is between 10,000 and 20,000 by the second or third week of the illness. Serologic findings include high titers of heterophil antibodies, which clump red blood cells of sheep. The antibodies may not appear until several weeks after the onset of signs and symptoms, and they decline during the ensuing 3 to 6 months.

Involved lymph nodes microscopically show reactive lymphadenitis. Lymphoid nodules in the inner cortex are hyperplastic. The germinal centers are markedly enlarged and contain macrophages with nuclear debris and numerous mitoses. Sometimes very large cells with multilobed nuclei and prominent nucleoli resemble Reed-Sternberg cells of Hodgkin’s disease.

There have been reports of a chronic fatigue syndrome associated with EBV. Patients describe this as a flulike illness with muscle aches, pharyngitis, tender lymphadenopathy, low-grade fever, and persistent severe fatigue. Elevated titers of immunoglobulin G (IgG) antibodies to viral capsid or early antigens of EBV are present.

Treatment of infectious mononucleosis is supportive. The acute disease usually resolves within 2 to 4 weeks. Splenic rupture is one of the few fatal complications of the disease, but it is extremely rare.

**Group A Coxsackievirus** The two most important group A coxsackievirus infections involving the oral cavity are herpangina and hand, foot, and mouth disease. Herpangina begins with fever, pharyngitis, and anorexia. Vesicles and ulcers occur primarily on the soft palate, uvula, and anterior tonsillar pillar. The disease resolves in several days and requires only symptomatic treatment.

Hand, foot, and mouth disease has a prodrome of fever, malaise, and headache, followed by macules and vesicles on the palms and soles. Vesicles and ulcers can be located anywhere in the oral cavity. Treatment is symptomatic, and the disease resolves within several weeks.

**Measles** Although a vaccine for measles exists, outbreaks of the disease still occur, primarily on college campuses. Measles begins with high fever, conjunctivitis, photophobia, cough, and nasal discharge. Leukopenia is common during this prodromal phase. Red vesicles with white centers (Koplik’s spots) appear on the buccal mucosa, followed in several days by an erythematous maculopapular skin rash. The rash first appears on the face and then spreads to the trunk and extremities.
Microscopic examination of the oral mucosal vesicles reveals epithelial necrosis, intercellular edema, cytoplasmic and nuclear inclusions, and multinucleated epithelial giant cells. Lymph nodes and tonsils show lymphoid hyperplasia and giant cells (Warthin-Finkeldey cells).

Therapeutic management for measles is symptomatic. It is usually a self-limited disease but may have a number of serious complications, including croup, bacterial pneumonia, otitis media, and encephalitis.

**Rubella** Rubella (German measles) is a mild infectious disease, but it can cause serious fetal malformations when it occurs in pregnant women. The prodrome consists of malaise, fever, mild conjunctivitis, and lymphadenopathy. Oral vesicles and ulcers may be present, but they are not distinctive. A maculopapular skin rash begins on the face and spreads downward to the trunk and extremities. It usually lasts for about 3 days. Arthralgia may involve wrists, fingers, and knees. Rubella may be completely asymptomatic or consist of lymphadenopathy without the rash.33,34

Congenital rubella syndrome usually results from maternal infection during the first trimester of pregnancy. The classic parts of the syndrome include cardiac malformations of patent ductus arteriosus, interventricular septal defect, or pulmonic stenosis; eye lesions of cataracts, chorioretinitis, and microphthalmia; mental retardation; and deafness.

Rubella is usually a benign disease requiring only symptomatic treatment. A live attenuated vaccine is effective, but it should not be given to pregnant women or to those who may become pregnant within 2 months of vaccination.

**HIV** HIV infects and destroys helper T lymphocytes, resulting in profound immunosuppression that predisposes to opportunistic infections and malignant tumors.

HIV is transmitted by sexual intercourse, through contact with blood or blood products, and perinatally. It is found in saliva, but transmission by saliva is unlikely.

The clinical spectrum of HIV infection includes an acute viral syndrome with malaise, fever, and lymphadenopathy; an asymptomatic carrier state in which there are circulating antibodies to HIV, and a wasting syndrome. Neurologic disorders are common and range from subtle memory loss to dementia. Numerous opportunistic infections, both fatal and nonfatal, and malignant neoplasms are a characteristic part of acquired immunodeficiency syndrome (AIDS). Many of these can be present in the oral cavity.35

**Oral Manifestations Opportunistic Infections** A common oral disease in HIV-infected patients is candidosis. Four clinical types of candidosis can be present in HIV patients. Pseudomembranous candidosis appears as white plaques that rub off, leaving an erythematous and/or bleeding base. Hyperplastic candidosis presents as white rough plaques that do not rub off. Erythematous candidosis is characterized by diffuse or localized patches of red mucosa. Angular cheilitis presents as cracks or fissures of the commissures, sometimes associated with white plaques. Candidosis in HIV infection responds to antifungal medications, but it is chronic and recurrent.36

Hairy leukoplakia consists of unilateral or bilateral white rough plaques that do not rub off, most commonly found on the lateral surface of the tongue. It is seen mainly in homosexual males but is also found in other HIV-risk groups. Deoxyribonucleic acid (DNA) hybridization demonstrates EBV in epithelial cells of the lesion. Hairy leukoplakia is pathognomonic of HIV infection and is highly predictive that the patient will develop AIDS.36,37

Microscopically, hairy leukoplakia is a lesion of squamous epithelium demonstrating hyper-keratosis, acanthosis, and swollen ballooning epithelial cells.

Hairy leukoplakia is usually an asymptomatic infection requiring no treatment. For those patients requiring treatment, acyclovir 200 mg tablets 12 times per day for 3 weeks has been used with some temporary success. In addition, cytology smears for candidosis should be performed and antifungal medication prescribed for patients with candidal organisms.

Herpes simplex and herpes zoster are more frequent and severe in HIV patients as are nonspecific aphthous-like ulcers. Prolonged postzoster neuralgia can be extremely painful. High-dose acyclovir can be useful in the treatment of either disease.38,39

**Periodontal Disease** A unique form of periodontal disease is present in many HIV patients. Clinical features include chronic gingival erythema, severe pain, soft tissue necrosis, and rapid destruction of alveolar bone and the periodontal attachment. Pocket formation is minimal or absent. The cause of HIV periodontitis may be an overgrowth of virulent organisms possessing tissue-damaging capabilities. This is probably a result of compromised immunity owing to HIV infection.

HIV periodontitis does not respond to conventional therapy alone. However, it does reportedly respond to twice-daily rinsing with chlorhexidine combined with conventional methods.40

**Malignant Neoplasms** The most common malignant neoplasms involving the oral cavity in HIV patients are Kaposi’s sarcoma, non-Hodgkin’s lymphoma, and squamous cell carcinoma. Most HIV patients with Kaposi’s sarcoma have oral lesions, and these may be the first sign of the disease. The lesions are red, blue, or purple and may be flat or elevated. They are most common on the hard palate and gingiva. Treatment includes radiation therapy, laser surgery, and/or chemotherapy.41–43

Non-Hodgkin’s lymphoma of the oral cavity in HIV patients is characterized by...
rapid growth, tendency to occur on the palate or alveolar ridge, and poor prognosis. Most of these lymphomas are of B-cell origin, and in situ hybridization techniques often reveal Epstein-Barr virus DNA in the tumor cells.

Other oral manifestations of HIV infection include salivary gland enlargement, xerostomia, and ulcerations similar to aphthous ulcers.36

Autoimmune Diseases

Autoimmune diseases typically have a gradual onset and a chronic progressive course with exacerbations and remissions. Lymphadenopathy is rare.

It is important to perform an incisional biopsy to establish a definitive diagnosis. A gingival biopsy should be avoided, if possible, because nonspecific gingival inflammation makes microscopic diagnosis difficult. Topical or systemic corticosteroids usually control but do not cure autoimmune diseases.

Pemphigus Two types of pemphigus, vulgaris and vegetans, have oral manifestations. Pemphigus vulgaris is the most common and is characterized by flaccid bullae that quickly rupture forming painful ulcers. Large areas of skin and mucosa can be involved, causing serious problems with infection.

Oral mucosal lesions are almost always present, and they are the initial lesions in the majority of cases. Extensive areas of mucosa may be involved, making eating extremely painful and difficult. Rubbing or blowing air on clinically uninvolved mucosa creates a blister, a phenomenon called Nikolsky’s sign. A Nikolsky’s sign is most commonly associated with pemphigus vulgaris and benign mucous membrane pemphigoid, but it may also be present in bullous pemphigoid and lichen planus. In pemphigus vegetans the blisters have a rough warty surface.44

Pemphigus is caused by circulating autoantibodies directed against desmosomes of squamous epithelium. This results in loss of epithelial cell cohesion and formation of an intraepithelial blister in the lower spinous cell layer. The basal epithelial cells remain attached to the underlying connective tissue. Acantholytic epithelial cells floating in the vesicle are termed Tzanck cells. They have rounded cytoplasm and large hyperchromatic nuclei. Cytologic preparation made from an early blister and stained with Papanicolaou’s stain can demonstrate Tzanck cells. A cytologic smear gives only a preliminary diagnosis and does not replace a biopsy.

Direct immunofluorescent studies, using the patient’s own skin or mucosa, reveal in vivo bound IgG antibody in the intercellular spaces of the epithelium in almost all cases (Figure 36-10). IgA, IgM, and C3 are present less often. Indirect immunofluorescence tests for autoantibodies in the patient’s serum. In pemphigus, circulating IgG antibodies can be demonstrated in the serum in most patients at some time during the course of the disease. Indirect immunofluorescence is not as sensitive as the direct technique. However, the titer of antibodies in the serum is often proportional to the severity of the disease.

Early diagnosis of pemphigus is important because it is a serious disease requiring aggressive treatment with corticosteroids. It is often fatal if not treated. Even with treatment, 10 to 15% of patients die owing to the effects of corticosteroids.

Cicatricial Pemphigoid In cicatricial pemphigoid (benign mucous membrane pemphigoid [BMMP]), autoantibodies are formed against components in the epithelial basement membrane. This results in painful vesicles and ulcers that may heal with scarring. BMMP has a marked predilection for females and adults past middle age.46

BMMP initially involves oral mucosa in almost all cases. The lesions consist of erythema, vesicles, and ulcers, most commonly involving the gingiva. Because the epithelium becomes detached from the connective tissue, BMMP is sometimes called chronic desquamative gingivitis, a nonspecific clinical description that can be applied, less commonly, to pemphigus and lichen planus (Figure 36-11). BMMP can also involve conjunctiva; nasal, pharyngeal, esophageal, and vaginal mucosa; and skin. Scarring and adhesion between the bulbar and palpebral conjunctivae (symblepharon) causes visual impairment.

The microscopic features of BMMP include subepithelial vesicle formation and nonspecific inflammatory infiltrate in the connective tissue (Figure 36-12). Direct immunofluorescence reveals linear
Bullous Pemphigoid  Bullous pemphigoid (BP) and BMMP have similar causes and microscopic features but a different distribution of lesions. The skin in all patients with BP demonstrates large thick-walled bullae, but oral mucosal lesions are less common.

Direct immunofluorescent findings are identical in BMMP and BP. Indirect immunofluorescence reveals circulating IgG antibodies against the basement membrane in the vast majority of BP patients but only rarely in patients with BMMP. There appears to be no correlation between antibody titer and disease severity in BP.47,52

Lupus Erythematosus  Lupus erythematosus is an autoimmune disease in which autoantibodies form to a wide variety of tissues including skin and oral mucosa. The autoantibodies can be directed against the cell’s nuclear material (antinuclear antibodies [ANA]) or cytoplasmic antigens.53,54

Discoid lupus erythematosus (DLE) is a skin disease that most commonly involves the face, scalp, and ears. The skin lesions appear as erythematous patches, often scaly and hyperpigmented. Older lesions may have atrophic scarring centrally and hyperkeratosis at the periphery.

Oral lesions of DLE are uncommon in the absence of skin lesions. They characteristically show central erythema with white spots and a border zone of white striae surrounded by telangiectases. Less typical oral lesions can resemble lichen planus or hyperkeratosis.

Systemic lupus erythematosus (SLE) is a chronic multisystem disease most common in young women between the ages of 15 and 40 years. Arthritis is typically present, often at the onset. Central nervous system manifestations include seizures and psychoses. The leading cause of death is renal disease, leading to destruction of glomeruli and hypertension. Other manifestations include vasculitis, Raynaud’s phenomenon, pleurisy, and pericarditis.

Numerous laboratory abnormalities may be present in SLE. The most important include elevated titers of antibody to native DNA, positive LE cell preparation, persistent false-positive serologic test for syphilis, anemia, leukopenia, thrombocytopenia, proteinuria > 0.5 g/d, and cellular casts in the urine.

The classic skin lesion of SLE is an erythematous rash located on sun-exposed surfaces such as the malar eminences. The oral lesions are similar to those of DLE. Oral ulceration is a well-known manifestation of SLE. Oral candidiasis secondary to corticosteroid therapy is common in SLE.

Certain medications have been reported to cause lupus-like reactions. The most common of these include procainamide, hydralazine, phenytoin, penicillamine, methyldopa, trimethadione, primidone, thioracil, and carbamazepine. Systemic involvement is less common with the drug-induced syndrome, and the signs and symptoms usually resolve when the drug is withdrawn.

The microscopic appearance of lupus is variable. The epithelium is hyperkeratotic and shows alternating areas of atrophy and hyperplasia. The lamina propria is edematous and has dense perivascular and deep inflammatory infiltrates. Periodic acid-Schiff stain demonstrates deposits subjacent to the epithelium and thickening of blood vessel walls.

Direct immunofluorescence on oral lesions reveals deposits of IgG and C3 in the basement membrane zone of the epithelium in the majority of cases of DLE and SLE.

Therapeutic management of oral lesions of lupus includes topical and/or systemic corticosteroids and antifungal medications as necessary for candidiasis. DLE has a good prognosis. The prognosis for SLE depends upon the extent of systemic involvement of the disease.

**Idiopathic Vesiculoulcerative Diseases**

Idiopathic diseases have causes that are unknown or poorly understood. They do not have clinical characteristics common to the entire class, and they must be considered individually when formulating a clinical differential diagnosis.

**Aphthous Ulcers**  Aphthous ulcers are common painful lesions that have periodic recurrences. Most patients have only a single ulcer during a given episode, although occasionally two or three ulcers may be present (Figure 36-13). Vesicles do not occur. Unlike recurrent herpes, aphthous ulcers are found on nonkeratinized oral mucosal surfaces. They have an acute onset, and each ulcer heals spontaneously without scarring in 10 to 14 days. There are no systemic manifestations and usually no lymphadenopathy.55–61

Major aphthae, also known as periadenitis mucosa necrotica recurrens, or Sutton’s disease, is characterized by multiple large mucosal ulcers. A patient has at
least one ulcer present all the time. The ulcers may take up to 6 weeks to heal, and healing is accompanied by scarring.

Behçet’s syndrome is a systemic disease that can affect most organ systems. The most common lesions are recurrent oral aphthous ulcers, genital ulcers, skin lesions, and eye lesions. The skin lesions consist of erythema nodosum–like eruptions and thrombophlebitis. The eye may be affected by recurrent iritis, uveitis, and retinitis, which can lead to blindness. Other less common problems include arthritis, ileal and colonic ulcers, aneurysms, arterial and venous occlusion, and a variety of central nervous system diseases.

Aphthous ulcers have been associated with a number of factors, but the cause is unclear. Aphthae do not appear to be caused by deficiencies in serum vitamin B12, red blood cell folate, iron, or total iron-binding capacity, or malabsorption enteropathies. An allergic response to certain foods such as walnuts, strawberries, and tomatoes does not appear to be important.

Genetic factors are significant as the frequency of human leukocyte DR7 antigen is significantly increased in aphthae patients, and aphthae are more common in related persons. Women commonly state that the ulcers appear with the onset of menstruation, supporting the role of endocrine factors.

The microscopic features of aphthous ulcers are those of any nonspecific ulcer and are not diagnostic. The history and clinical findings determine the diagnosis.

The goal of therapeutic management is to decrease the inflammatory response; topical corticosteroids accomplish this for most patients. Patients with major aphthae usually require systemic corticosteroids.

**Lichen Planus**  
Lichen planus is a chronic disease of skin and mucosa. Skin lesions are often extremely pruritic and appear as violet-colored flat-topped papules and plaques with a shiny surface. The lesions are most commonly located on the volar surface of the wrists, anterior surface of the legs, and penis.

Oral lesions of lichen planus are most common in adults > 40 years. The lesions have several forms. The reticular form presents as a network of lacy white lines called Wickham’s striae (Figure 36-14). The plaque form appears as white homogenous plaques. The white lesions in both forms are nonpainful, rough to palpation, and do not rub off. The atrophic form consists of erythematous mucosa plus a reticular keratotic pattern along the periphery. The erosive form combines ulcerations with atrophic features (Figures 36-15 and 36-16). The atrophic and erosive forms are typically symptomatic. Occasionally, vesicles are seen, which quickly rupture to form painful ulcers. Ulcers and erosions can involve the attached gingiva producing a desquamative gingivitis pattern in 25% of patients. Candidosis is another common finding in patients with lichen planus. Oral lesions of lichen planus are multifocal and can involve any mucosal surface. The most common locations are the buccal mucosa, followed by gingiva and the tongue. One-fifth of patients with oral lesions have skin lesions.

Drugs can cause lichenoid reactions of skin and mucosa that are clinically similar...
to lichen planus. The reactions resolve when the drug is discontinued. The most commonly implicated drugs include methyldopa, amiphenazole, chloroquine, hydroxychloroquine, quinacrine, chlorpropamide, tolbutamide, tetracycline, chlorothiazide, practolol, dapsone, furosemide, phenothiazines, quinidine, triprolidine, para-aminosalicylic acid, arsenicals, bismuth, gold salts, and mercury. Lichenoid reactions also occur during the chronic phase of graft-versus-host disease following bone marrow transplantations.

Lichen planus is considered a disease of the cellular immune system involving T lymphocytes, Langerhans’ cells, and macrophages. The Langerhans’ cells and macrophages process antigens and present the antigenic material to T lymphocytes. The lymphocytes proliferate and become cytotoxic for basal cells of the squamous epithelium. A similar immune mechanism has been reported in graft-versus-host disease following bone marrow transplantations.

The microscopic features of lichen planus are variable, and clinical features are important in establishing the diagnosis. The primary microscopic features include hyperkeratosis and a band-like inflammatory infiltrate, consisting primarily of lymphocytes, subjacent to the epithelium (Figure 36-17). The epithelium–connective tissue interface is obscured owing to liquefaction degeneration of the epithelial basal cell layer and/or infiltration with lymphocytes. An eosinophilic band may be seen between the inflammatory infiltrate and the epithelium. The spinous cell layer is often hyperplastic. Colloid or Civatte bodies, representing necrotic epithelial cells, are occasionally seen as eosinophilic bodies in the lower layers of the epithelium.

Direct immunofluorescence reveals fibrinogen deposition in the basement membrane zone in almost all cases, and less commonly in colloid bodies and walls of blood vessels.

Lichen planus is a chronic or recurrent disease that only rarely undergoes spontaneous remission. The goal of treatment is control of symptoms. Asymptomatic lesions require no treatment, whereas symptomatic cases are usually controlled with topical and/or systemic corticosteroids. In one study of 570 patients with oral lichen planus, 63% experienced improvement and 29% experienced complete remission while maintained on corticosteroids. Antifungal medication is necessary if candidosis is present.

It appears that oral carcinoma occurs in lichen planus patients at a slightly higher rate than in the general population. However, the frequency of malignant transformation is unknown, and the classification of lichen planus as a premalignant lesion does not appear justified. Periodic recall examinations are necessary.

**Erythema Multiforme** Erythema multiforme (EM) can involve skin and oral mucosa independently or simultaneously. It has traditionally been described as acute and self-limited, requiring an average of 3 weeks for resolution. Some patients have a variable pattern of recurrence. In other patients EM has a chronic course.

The cause of EM is unknown, although it appears to be some type of immune dysfunction. It may be related to immune complexes deposited in walls of blood vessels in the dermis or submucosa. In about half the cases EM appears to be triggered by infections or drugs. The most common infections reported include herpes simplex viruses, tuberculosis, and histoplasmosis. The most frequently implicated drugs are sulfonamides, barbiturates, phenylbutazone, oxyphenbutazone, phenazone, penicillins, chlorpropamide, phenytoin, and carbamazepine.

The skin lesions of EM include macules, papules, vesicles, and bullae. The most characteristic lesion, known as the iris or target lesion, appears as a central vesicle surrounded by erythematous and skin-colored rings. The lesions are symmetrically distributed, most commonly on the extremities and face.

One-fourth to one-half of patients with skin lesions have oral lesions (Figure 36-18). Ulcers are present, most commonly on the lips, buccal mucosa, and tongue, as well as erythematous mucosa. The oral lesions vary from mild to so severe that patients cannot speak or eat. The lesions may be accompanied by headache, fever, and malaise.

Stevens-Johnson syndrome is a severe form of EM with more serious systemic manifestations. Extensive skin lesions, conjunctivitis, and oral and genital mucosal lesions are present. The oral lesions often begin as vesicles, which rupture forming painful ulcers. Lesions on the labial mucosa may have a bloody crust.

Toxic epidermal necrolysis is an even more serious form of EM characterized by large flaccid bullae and sloughing of the epidermis in large sheets. Oral lesions may be prominent, especially on the buccal

![FIGURE 36-17 Lichen planus. Focal hyperkeratosis, basal cell liquefaction degeneration, and a superficial infiltration of lymphocytes (×60 original magnification; stained with hematoxylin and eosin).](image)
mucosa. Toxic epidermal necrolysis is usually caused by drugs. The patient is acutely ill, and the disease is often fatal.

The microscopic features of EM are not diagnostic. The epithelium demonstrates edema and necrosis of keratinocytes. The connective tissue contains perivascular infiltrates of lymphocytes, plasma cells, and macrophages. Immuno-fluorescence reveals deposits of IgM and C3 in the vascular walls, suggesting immune complex deposition is important in the pathogenesis.

Treatment may not be necessary for mild forms of EM, which have a good prognosis, although they may be recurrent. More serious types respond well to corticosteroids; however, Stevens-Johnson syndrome is occasionally fatal.

References


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 comprises 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 as 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 1,000 in the continental United States, 1 in 100 on the island of Kauai, and > 2 in 100 in Australia.3

Epidemiologic studies demonstrate a positive correlation between ultraviolet (UV) radiation exposure and the incidence of NMSC. NMSC is predominantly a Caucasian disease (≥ 98%) but does occur in Blacks and Hispanics.2 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 > 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 2,000 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 classified skin into six different groupings or types (Table 37-1).13 Each group was categorized based on the 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. 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. Human immunodeficiency virus 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, their

| Table 37-1  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 min of sun exposure at midday in the northern hemisphere. + indicates a relative level of pigment darkening, with +++ being the highest.

From Fitzpatrick TB.  

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. There are several syndromes that 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 37-1) but act just like common nodular BCC. Control with a CO2 laser or curettage and electrodesiccation (C and E) is critical before enlargement destroys anatomic structures.
  - Xeroderma pigmentosan is an autosomal recessive disorder resulting in defects in repair of deoxyribonucleic acid (DNA). UV radiation results in skin DNA damage; therefore, xeroderma pigmentosan 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 37-2)
- 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 37-3); therefore, the American Academy of Dermatology recommends treatment. All suspicious lesions should undergo biopsy. Treatment options include chemical peel, laser, cryotherapy, C and 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

Immunologic Factors  Immunosuppression predisposes a person to several types of cancers including skin cancer. Immunosuppression 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. There are several syndromes that predispose a person to skin cancer:

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tumors appear years earlier than in any control population. 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.  

**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. UVA, originally thought to be harmless, is now known to enhance the effects of UVB as a cocarcinogen. 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 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. 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. 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.
Basal cell tumors originate from pluripotential cells in the epidermis and hair follicles. They are often slow growing and may take years to enlarge significantly. Typically, patients with BCC are categorized as Fitzpatrick types 1 to 3 with a history of sun exposure. Eighty to 93% of the cancers occur on sun-exposed areas of the head and neck, and 26 to 30% occur on the nose. BCCs can be divided into several subtypes: superficial, nodulo-ulcerative (or nodular), pigmented, infiltrative, micronodular, morphea-like, and basosquamous.

Superficial BCC represents approximately 10% of all BCCs (Figure 37-4). They present as slightly elevated plaques or discrete macules that may be scaly. They can resemble eczema or fungal infections. Nodulo-ulcerative BCC is the most common type, accounting for approximately 75% of all BCCs (see Figure 37-4). 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.

Death from BCC is rare, with a rate of metastasis of 0.0028 to 0.1%. Size, depth of invasion, and histologic type are important predictors for metastasis. 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.

Squamous Cell Carcinoma

SCC is the second most common skin cancer and accounts for 20% of all NMSC cases. SCC is a malignant proliferation 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.

Men with a fair complexion who are > 50 years and have had heavy sun exposure in the past several years typically get multiple actinic keratoses and SCC. SCC presents as a painless poorly defined erythematos nodule with raised borders (Figure 37-5). 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 > 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%. 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, and periorbital and preauricular areas have higher rates of recurrence and metastasis (10–14%).

The most common precursor for SCC is AK. The rate of transformation of AK to SCC is 1 in 1,000 per year. Approximately 40% of people > 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.

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 > 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 metastasis rate between 18 and 38%. With SCC, the first shot at cure is crucial as recurrent SCCs have a metastatic rate of 24 to 45%; if they metastasize the 5-year survival rate is around 50%.

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 > 40,000 new cases of melanoma will be diagnosed this year in the United States alone. An estimated 1 in 75 people develop melanoma in their lifetime, up from 1 in 150 persons in 1985. Melanoma accounts for over three times more deaths than the combined fatalities from all other skin malignancies.

Risk Factors

People in Fitzpatrick groups 1 and 2 are the most susceptible to melanoma. The role of UV is not precisely known for
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 > 100 of these moles have a tenfold risk of developing melanoma.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 37-6). 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: **asymmetry**, **border irregularity**, **color changes or variation**, **diameter of lesion (< or > 6 mm)**. The practitioner should not place the patient under
casual observation (ie, not perform a biopsy) just because these common indicators might be absent. Approximately 40% of board-certified dermatologists and 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, 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 asymmetric borders; it can be present for up to 5 years prior to 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. Around 5% of nodular melanomas lack pigmentation and are pinkish “amelanotic” melanomas. Nodular melanomas are thicker and metastasize rapidly.

Lentigo maligna melanoma comprises 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 Caucasians 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 > 2 cm or those located at anatomically restricted areas (eg, eyelids, ears). The biopsy should be at the most raised site or the darkest area of the lesion (Figure 37-7). Full-thickness excisional biopsy with a 2 mm margin is the preferred method for lesions < 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 classification) or by determining the anatomic level of invasion (Clark classification). Melanomas measuring < 0.76 mm have a 5-year survival rate of > 93%, whereas lesions > 4 mm thick have a 5-year survival rate of < 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. The most frequent sites of melanoma metastasis include the skin, lymph nodes, lung, liver, brain, bone, and gastrointestinal tract. On the other hand, 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 < 1 mm or > 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% (ie, from 35 to 45%) (Tables 37-2 and 37-3).\(^52\)

**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.

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 versus an invasive lesion. Shave biopsy is contraindicated in potential melanomas. A “pseudoshave” biopsy might be performed with a curetted specimen prior to 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 as the punch may force the tumor deeper into tissue.

Incisonal and excisional biopsies are well known to surgeons. Deciding whether to use one revolves around whether the diagnosis is obvious (eg, 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

<table>
<thead>
<tr>
<th>Table 37-2 Melanoma: Survival Rates</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Breslow Classification</strong></td>
</tr>
<tr>
<td>No evidence of primary tumor</td>
</tr>
<tr>
<td>Thickness &gt; 0.76 mm</td>
</tr>
<tr>
<td>Thickness 0.76–1.5 mm</td>
</tr>
<tr>
<td>Thickness 1.51–3.0 mm</td>
</tr>
<tr>
<td>Thickness &gt; 3.0 mm</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 37-3 Melanoma: Biopsy Strategies</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Tumor Thickness</strong></td>
</tr>
<tr>
<td>Lentigo maligna, in situ</td>
</tr>
<tr>
<td>Melanoma &lt; 1.00 mm</td>
</tr>
<tr>
<td>Melanoma 1.01–4.00 mm</td>
</tr>
<tr>
<td>Melanoma &gt; 4.01 mm</td>
</tr>
</tbody>
</table>
might include liquid nitrogen cryotherapy, standard excision, Mohs’ micrographic surgery (MMS), radiation, C and E, topical chemotherapy, laser ablation, photodynamic therapy, interferon, and retinoids. We review most of these options below.

**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. Excision may be done under local anesthesia or in the outpatient surgery setting. Tables 37-4 and 37-5 outline acceptable margins for clearing most lesions.

The lesion and indicated margin for clearance is 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 37-8).

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. 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 prior to the surgery exactly where the scars will be located. Delayed reconstruction has been proven beneficial for patients receiving skin grafts as 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 prior to reconstruction, which may be performed on an elective basis often up to a week later. The only surgical difference between immediate (within 24 h) and delayed reconstruction (≥ 48 h) 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 37-9).

### Table 37-4 Margin Control for Squamous Cell Cancers

<table>
<thead>
<tr>
<th>Tumor Description</th>
<th>Margin Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small, well differentiated</td>
<td>5 mm with orientation</td>
</tr>
<tr>
<td>&gt; 1 cm</td>
<td>Increase margin size</td>
</tr>
<tr>
<td>Lesion on upper lips, eyelids, nose, ears, etc.</td>
<td>Consider Mohs’ micrographic surgery</td>
</tr>
</tbody>
</table>

### Table 37-5 Margin Control for Basal Cell Cancers

<table>
<thead>
<tr>
<th>Tumor Description</th>
<th>Margin Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 mm or less</td>
<td>2 mm</td>
</tr>
<tr>
<td>5 mm–1 cm</td>
<td>3–4 mm</td>
</tr>
<tr>
<td>1–2 cm</td>
<td>5–7 mm</td>
</tr>
<tr>
<td>&gt; 2 cm, morphea-like, or unusual pathologic behavior</td>
<td>7–10 mm margin or Mohs’ micrographic surgery or delayed reconstruction following permanent histology</td>
</tr>
<tr>
<td>Unusual pathologic behavior, recurrent tumors, tumors on lips, ear, nose, medial canthus, eyelids</td>
<td>Mohs’ micrographic surgery or delayed reconstruction following permanent histology</td>
</tr>
</tbody>
</table>

**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 re-excised...
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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 the 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 nodulo-ulcerative 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° toward the center of the tumor. The specimen is anatomically 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 < 2 cm treated with MMS approach 99% (vs 90–95% by routine pathologic examination).56,57 Standard vertical breadloaf sections evaluate < 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
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 < 1.5 cm in diameter. I, The excisions have been made. J, During the closure the entire nasal dorsum is undermined submuscularly and supraperichondrally. 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.
rate from 94 to 99% as opposed to 90% for non-Mohs’ techniques. Recurrent SCC cure rates with MMS approach 90% as opposed to 76% for other treatment modalities.

Under these circumstances MMS is indicated for the treatment of recurrent BCC, histologically difficult BCC (ie, micronodular, infiltrative, and morphea-like), and BCCs in which conservation of tissue is critical (eg, on the nose, lip, ear). For SCC, MMS might be indicated for lower lip cancer, some poorly differentiated SCCs, and areas where maximum tissue preservation is essential.

**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.

RT of tumors < 2 mm has a cure rate of 90% and 85 to 95% for BCC and SCC, respectively. However, larger lesions have a much lower success rate. For melanoma, local recurrence rates of up to 50% have been reported. Thus, RT for melanoma is only a viable option 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%. 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.

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 and 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 and 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 and 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 following C and E. BCCs < 5 mm have an 8.5% recurrence rate after C and E by an experienced clinician. Lesions of the nose, ear, and perioral and periorcular areas may recur at a rate of 16%. This rate soars to 26% for lesions > 20 mm. Therapeutic C and E is therefore contraindicated for larger lesions, poorly differentiated SCC, 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-α and messenger ribonucleic acid 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.

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.

**Laser**

The CO₂ 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 CO₂ 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 BCC, and SCC. We have found its greatest benefit in ablation of superficial AK and superficial SCC, both on the skin and 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 O₂ species are generated within the cells producing cell death.68

Cure rates for photodynamic therapy for AKs, superficial SCC, and BCC are reported to be > 90% in some studies, but tumors thicker than 2 mm are photoresistant.69

**Interferons**

Interferons are cytokines that may effect cell growth and differentiation and accent immune responses and antiviral activity. Intranasal injection of interferon-α can attain cure rates of > 80% for superficial and nodulo-ulcerative 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 preventative is a long-term proposition as the effects of the drug plateau at around 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 as follows: 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 pigmented 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 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.79 The principle 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.71,80

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 subcutaneous fat. A septocutaneous vessel travels through the septal fascia and courses parallel to the skin surface with an accompanying vein. Named septocutaneous vessels (eg, 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 “angio-some” 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 d 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.

Esthetic flaps are not mere hole fillers. They are designed to complement natural esthetic units and facial borders. Defects that trespass multiple esthetic 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 esthetic and tumor considerations. Ideally, grafts to the nose are well matched with preauricular skin, but any supraclavicular facial graft (from the blash 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 (eg, bacitracin and/or polymyxin B sulfate). The outer edges of the wound are coated with an adhesive (eg, 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 37-10).

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

![Figure 37-10](image)

**Figure 37-10** 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.
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. Over-stretching the skin collagen effaces the blood vessels under tension; thus, necrosis secondary to decreased perfusion to a distal flap may occur (Figure 37-11).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 [LME]) 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 LME. 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 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. Additionally, there are skin stretchers that are made for this purpose (Figure 37-12).

Finally, biologic creep is a slow methodic stretching of skin, yielding brand new skin.88 Skin expanders do just that (Figure 37-13).

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. On the other hand, 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 subcutaneous planes, thereby reducing shearing forces and allowing the skin to slide and redrape in 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.

Flap Designs Advancement Flaps An advancement flap is advanced linearly over a defect. It consists of a classic elliptic 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. 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:

1. Do nothing; this approach works well on the scalp as bunched up tissue lies down with time
2. Close opposite lines of uneven lengths by spreading out the problem—halving (Figure 37-14A)
3. Remove the excess to a hidden area—an end or middle triangle (Figure 37-14B)
4. Lengthen the incision. This eliminates bunching (Figure 37-14C)
5. Perform an M-plasty (sometimes called a T-plasty), which shortens the problem (Figure 37-14D)
6. Reverse the S loop and hide the excess elsewhere (Figure 37-14E)
7. Advance the dog-ear as a flap (subcutaneous “island”) or use it as a free graft (Figure 37-14 F; also see Figure 37-6)

**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. Rotation flaps rarely fit
perfected geometric schemes of success. Rather, the surgeon often finds himself adjusting to the specific variables of a given situation (Figure 37-15). There are two exceptions to this complexity that have been worked out fairly precisely:

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 adjustments103

Advantages to rotational flaps include broad-based reliable vascularity, flexibility in design, and easy placement of scars into esthetic/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 37-16). 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 impact the geometry of the flap design, with the classic transposition being a rhombus.

The rhombic flap is an equilateral parallelogram with oblique angles. The (Limbberg) rhombic flap, first described in 1963, was an equilateral rhombus with 60˚ and 120˚ 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 LME. These LME are always perpendicular to RSTLs and run in the direction that tissue stretches most efficaciously.

The rhombic flap, whose short diagonal line parallels the LME 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˚ 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˚ or a square defect. As
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. 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 37-6). RSTL = relaxed skin tension lines.
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 parallel to the long diagonal completing the second flap. The Webster 30˚ flap allowed for easier closure by bisecting the 60˚ angle into two 30˚ angles via an M-plasty.

Here, the short diagonal had to be at least 110˚ 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 Abbe (Figure 37-17) and Estlander flaps and the paramedian forehead flap. A full description of these flaps is discussed in Chapter 38, “Local and Regional Flaps.”

**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 prior to skin grafting.

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 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 multiperated 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 the 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 (eg, from suture packaging) (Figure 37-18). Since an FTSG contracts by 10 to 15% after 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 prior to peripheral suturing.98 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.

 FIGURE 37-16  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 37-17  A, This 70-year-old man has a large basal cell cancer removed by Mohs’ micrographic surgery. B, Subsequently a midline axially based Abbe flap is inserted. C, Three weeks later, the Abbe flap is divided and inset as an office procedure. The esthetic result 2 weeks later shows the flap is not exactly in the midline but simulates the philtral area effectively.
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. 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. Contraindications include areas that might compromise functional or esthetic 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.

There are varying types of dermatomes ranging from machine to manual. The Brown dermatome allows for precise modification of graft thickness. The graft dimensions should be at least 25% larger than the wound defect. 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.

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 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 STSG. A good compression dressing and/or bast- ing stitches may suffice to promote adherence between the graft and under- lying tissue and to prevent fluid accumu- lation. A variation to placing interrupted basting sutures is the spiral bast- ing stitch. The suture is started at the edge of the graft with the “tail” left long. 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. Com- posite 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 FTSG and STSG and is attributed to the high metabolic demands of the grafts. Harvesting (donor) adjacent dermis attached to the composite graft and inserting the de-epithelialized dermis into adjacent subcutaneous tunnels (recipient) may improve vascularity substantially. Cooling the composite graft with ice for 24 hours also helps.

Regardless, composite grafts are tech- nique sensitive. Generally, composite grafts should be no larger than 1.5 to 2.0 cm. Avelar and colleagues have shown composite grafts greater than 2.0 cm grafted successfully to nasal and auricular defects. Similarly, Skouge has effective- ly grafted larger defects using a “tongue and groove” technique and turndown hinged flaps. The postoperative appearance of composite grafts is distinc- tive. 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 pro- cedures. 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 > 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, non-steroidal 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 since there have been several documented cases of stroke when anticoagulants were stopped prior to 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 prior to 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 underlying tissue is detrimental to flap circulation because it creates tension, and it acts as a physical barrier preventing cohesion to the underlying tissue base. Additionally, stagnating blood may promote wound infection.

An early hematoma may often be pushed out and washed away, but a reforming 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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